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Mandibular Attachment Device Effects on African American Veterans with Heart Failure

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This is to certify that the doctoral dissertation by

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2020

Abstract

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MSW, Florida Agricultural and Mechanical University, 2009

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Dissertation Submitted in Partial Fulfillment

of the Requirements for the Degree of

Doctor of Philosophy

Public Health, Epidemiology

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Abstract

Heart failure is a growing epidemic that affects people nationwide and is disproportionate to African Americans. The purpose of this quantitative repeated measures study was to determine whether mandibular attachment device (MAD) therapy impacts symptoms of heart failure in African American male veterans diagnosed with obstructive sleep apnea (OSA). The oxidative stress theory was applied in this study to assess whether MAD therapy received from Veterans Affairs (VA) dental clinics impacted heart failure symptoms, after controlling for patient body mass index (BMI) levels and smoking status. Research questions examined whether MAD had a significant effect on symptoms of heart failure in male African American veterans with OSA and whether BMI and smoking caused a moderating effect on MAD therapy treating efficacy on symptoms of heart failure. Secondary data from the VA was captured through the VA informatics and computing infrastructure. Data obtained from 29 records were analyzed using the statistical package for the social sciences. The repeated measures multivariate analysis of variance and the repeated measures multivariate analysis of covariance were used to assess the magnitude of change in heart failure symptoms (ejection fraction, systolic blood pressure, diastolic blood pressure, oxygen saturation, brain natriuretic peptide, n-terminal pro-brain natriuretic peptide, and troponins levels) while controlling for the 2 covariates. Results showed a positive mean change in systolic blood pressure while using the MAD and a negative moderated effect after controlling for BMI at 4-months on oxygen saturation. This study will aid positive social change by providing new data that can be used by the public health field towards an alternative treatment.

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Dedication

This study was dedicated to my late grandfather Willie James Hueston for his constant support, inspiration, the lessons he taught me, and encouragement he always showed.

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I would like to first thank God for all the things he has done in my life, for his guidance, grace, and mercy and for giving me the strength, drive, patience, and knowledge to pursue my Doctorate. I would also like to thank my late grandfather for instilling in me the importance of having a love for people, showing me how to build and do for self, and for providing me structure. To my grandmother for her encouragement, keeping me grounded, instilling in me a passion for the health field, honesty, and ability to think outside the box. To my mother for showing me sacrifice, love, and my father for displaying perseverance. To my aunt for the life lessons, alternative perspectives, and advice. To my brother and sisters, for your love and support. To my wonderful wife and best friend for your sacrifices, which allowed me to pursue my dreams, and for being there through the good and tough times, I love you. To my four children for being my motivation and providing me with the determination to succeed. Lastly, to my extended family, godparents and brothers, mother-in-law, brother and sister-in-law, close friends, co-workers, and mentors who have motivated, guided, reinvigorated, and enlightened me throughout my life's journey. It does take a village; I thank you all.

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Chapter 1: Introduction to the Study

Introduction

Obstructive sleep apnea (OSA) is a common respiratory disorder in the United States that affects over 25 million adults (American Academy of Sleep Medicine [AASM], 2018; Gillespie, Knauret, & Naik, 2015). Adams et al. (2017) indicated that over 82% of adult males in the United States remain undiagnosed for OSA. There are 5.9 million that have been diagnosed with the disease (AASM, 2016). In 2015, those diagnosed with OSA had an economic burden of $12.4 billion in the United States (AASM, 2016). OSA has been identified as a condition that contributes to heart failure as a result of varying amounts of oxygen being received by the body (Chan et al., 2016).

Heart failure is a disease that impacts 900,000 people in the United States annually (American Heart Association [AHA], 2017a). Heart failure symptoms frequently occur in those who have OSA occurring in over approximately 50%-80% of patients (Abraham et al., 2013). Heart failure is present in 5.7 million people in the United States and costs the nation roughly $30.7 billion every year (Centers for Disease Control [CDC], 2016). Heart failure is the result of one in four deaths in the U.S. (CDC, 2017b).

According to Colvin-Adams, Sharma, and Yancy (2014), African Americans appear to be the highest affected ethnic group afflicted with heart failure. Several studies reveal that African Americans are disproportionately affected by heart failure, and few studies involve African Americans and heart failure (Akinboboye, Jean-Louis, Mitchell, Ogedegbe & Olafiranye, 2013; Becker et al., 2014). Continuous positive airway pressure

(CPAP) use on those with OSA and heart failure has been effective, though historically had low compliance issues, which limits its efficiency (Cistulli et al., 2013). Nevertheless, there has been insufficient data to determine whether heart failure symptoms are significantly affected by using a mandibular attachment device (MAD), which historically has high compliance from those who use the device (Booth, Djavadkhani, & Marshall, 2014).

The following study has examined whether treatment with the MAD effectively affects heart failure symptoms in African American male veterans with OSA and whether treatment efficacy differs in terms of body mass index (BMI) and smoking status. This investigation may result in positive social change for African American male veterans diagnosed with OSA and heart failure. In addition, this examination may contribute to the increased efficacy and use of MAD treatment. This chapter includes the following: background, problem statement, the purpose of the study, research questions, theoretical framework, nature of the study, definitions of terms, assumptions, limitations, the scope of the review, and significance of this study.

Background

Sleep-disordered breathing, such as OSA, has been strongly linked as a comorbidity to heart failure (Abraham et al., 2013). Obstructive sleep apnea is the most widespread of all sleep disorders (Al-Shorman & Shydfat, 2015) and was four times more predominant in men than in women (Ohn & Tin, 2016). An unknown portion of the population suffers from sleep apnea without knowing it until confirmed by medical professionals (Booth et al., 2014). Approximately 25 million people in the United States

have been affected by OSA (AASM, 2018). More than 80% of the cases of moderate to severe sleep apnea go undiagnosed until confirmed by medical professionals (American Sleep Apnea Association [ASAA], 2017). OSA has significantly been linked to heart failure (Barbe, Drager, Lorenzo-Filho, McEvoy & Redline, 2017; Colvin-Adams, Sharma, and Yancy, 2014).

Heart failure is a significant public health concern worldwide and within the United States (Lung & Saverese, 2017). In between 2011 to 2014, over 6.5 million of the U.S. population had heart failure (Benjamin et al., 2017). Heart failure in the U.S. is expected to increase by approximately 46% or roughly 8 million by 2030 (Benjamin et al., 2017; Farmakis, Papingiotis & Parissis, 2017; Lung & Saverese, 2017). According to Arynchyn et al. (2009), in people of younger ages, heart failure, and higher BMIs are present at higher rates in African Americans. African Americans diagnosed with heart failure who are less than 50 years old are 20 times more likely to have increased mortality than Whites less than 50 years old diagnosed with heart failure (Colvin-Adams et al., 2014).

Babson, Bonn-Miller, Del Re & Woodward (2013), using the Veteran Healthcare Administration (VHA) database, have demonstrated that OSA has been linked to secondary physical complications, including heart failure. The AHA (2017a) indicated that high BMI levels and smoking present significant concerns for many Americans suffering from sleep apnea and heart failure. Clark, Fonarow, and Horwich (2014) revealed that high levels of BMI and being overweight (25-29.9 kg/ m2) or obese (≥ 30 kg/ m2) are factors that increase the risk of cardiac issues such as heart failure. Smoking

impairs endothelial function, increases oxidative stress and vascular inflammation, which increases the risk of heart failure (Bauer et al., 2012).

Heart failure has been linked to multiple indicators, including smoking, obesity, hypertension, lack of physical activity, and lifestyle (AHA, 2017a). According to the AHA, 2018), an increase in heart failure diagnosis promotes work loss, household productivity loss, and encourages premature mortality. Heart failure diagnosis in the United States is expected to increase the direct national cost from $30 billion to an anticipated $70 billion by 2030 (Greenberg, 2017).

Treatment of sleep apnea has gained attention as treatment methods have expanded to include the use of CPAP, surgery, and oral devices (Gillespie, Knauret, & Naik, 2015). Sleep apnea treatment mechanisms have been created to increase airflow and reduce gaps in breathing during sleep (Booth et al., 2014). Akinnusi, Anandam, El-Sohl, Jaoude, & Patil (2013) revealed that sleep apnea treatments relating to the use of CPAP have led to reductions in cardiovascular symptoms. A combination of therapies that include CPAP coupled with the use of the MAD has been piloted and shown to have a significant positive effect on cardiovascular symptoms, though they also display patient adherence concerns (Akinnusi, Churder, El-Solh, Lafornara & Moitheennazima, 2011).

CPAP usage in some studies has shown to be effective against OSA and reducing symptoms of heart failure by promoting oxygen, increasing endothelial functioning, reducing apnea-hypopnea index (AHI), and lowering blood pressure (Marklund, Randerath, & Verbaecken, 2012; Pinto & Sharma, 2018). According to Murariu, Pang & Rotenburg (2016), there is a consistent implication across studies over a twenty-year time

frame from 1994-2015 of 34.1 % noncompliance with CPAP usage. This high rate of non-compliance accentuates the patient need for an alternative treatment for heart failure that is similarly effective, cost-effective, noninvasive, and promotes high compliance with treatment (Al-Shorman & Shydfat, 2015; Anderson et al., 2016; Bajaj, Kant, & Singh, 2017; Marklund et al., 2012). One potential alternative treatment includes the MAD.

The MAD is an oral insertion device received from a dental clinic that moves the mandible forward and allows for an increase in airflow when sleeping (Abraham et al., 2013; Bratton, Gaisl, Kohler, & Wons, 2015). Many of the studies that have used MAD to address OSA have primarily included individuals from European or White backgrounds as participants. However, African Americans are the racial group most affected by OSA (Colvin-Adams et al., 2014). In current practice, the MAD is an independent or collaborative method used with and without a CPAP for those who are noncompliant with OSA treatment (Abraham et al., 2013). Due to the high patient noncompliance rates for the CPAP to treat symptoms of heart failure treatment, the MAD may provide an alternative first-line noninvasive option for treatment. Proper use of the MAD reduces gaps in breathing, promotes blood flow, and is potentially more cost-efficient over time compared to the CPAP (Bajaj, Kant, & Singh, 2017; Bennett et al., 2014; Marklund et al., 2012). The MAD has not explicitly been tested for its efficacy in reducing symptoms of heart failure (Abraham et al., 2013; Akinnusi, Anandam, El-Sohl, Jaoude, & Patil, 2013; Berge, Gjerde, Lehmann, Johansson, A. & Johansson, A. K., 2015). However, the MAD may potentially be a non-invasive sleep apnea solution for

reducing heart failure symptoms in African Americans with OSA and therefore requires further study to assess how the MAD affects symptoms of heart failure (Abraham et al., 2013; Akinnusi et al., 2013).

Colvin-Adams et al. (2014) have shown that in the United States, African Americans have a higher heart failure prevalence than whites. To further reduce heart failure rates among African American males, the goal of this study was to find an alternative noninvasive OSA treatment that would significantly reduce symptoms of heart failure. This study was necessary to address the gap in the literature, related to CPAP noncompliance, which indicates a further need in the public health and medical field for an observatory study to identify if the MAD is an alternative first-line treatment for those with OSA and heart failure over the age of 20. The need for a further assessment of the MADs effect on heart failure symptoms was present due to the unknown cardiovascular outcomes resulting from its upper airway stimulation.

Problem Statement

Obstructive sleep apnea is commonly undiagnosed and affects veterans nearly two times more frequently than other sampled communities (Babson, Bonn-Miller, Del Re & Woodward, 2013). Capaldi, Guerrero, and Killgore (2011) found that "redeployed combat veterans commonly suffer significant sleep-disordered breathing such as OSA" (p. 879). Approximately 26-36% of veterans served by the VHA have OSA (Casey, Knepler, Panos, & Samson, 2012). Of the diagnosed cases of OSA found in veterans treated by the VHA, 12-17% of veterans had severe OSA (Casey, Knepler, Panos, & Samson, 2012). Additionally, of the 118,105 veterans with OSA served at the Cincinnati

Veterans Affairs Medical Center, approximately 13.5% had heart failure (Casey et al., 2012).

In between 2007-2009, the prevalence of OSA has increased and is now considered a health epidemic that negatively affects African Americans more so than any other ethnic race (Bakker et al., 2015). This condition has been strongly linked to cardiovascular diseases such as heart failure, and mortality rates continue to climb (Colvin-Adams et al., 2014). The use of MAD therapy by patients has shown some improvement in vascular functioning and may have similar potential in terms of reducing heart failure symptoms (Booth et al., 2014). Nevertheless, there are limited studies that have investigated how OSA affects African Americans with heart failure. Currently, CPAP machines are the gold standard therapy method for treating OSA (Hwang, Mereddy, Parthasarthy, Pepin, & Tamisier, 2018). There appears to be a gap in the literature related to the effects of the MAD and cardiovascular symptoms. In a limited study by Akinnusi et al. (2013), it has been suggested there may be a possible benefit from the MAD on heart failure or cardiovascular disease, however, further analysis was needed.

One in five people in the United States has OSA, while one out of 15 has moderate to severe OSA (Bergerhenryent, 2017). African Americans under the age of 25 and over age 65 are the most affected by OSA (Akinbobye, Jean-Louis, Mitchell, Ogedegbe, & Olafiranye, 2013). There remains a debate on the most affected ethnicity from OSA between the ages of 26 through 59 years (Akinbobye et al., 2013).

In my review of the literature, limited information was identified explicitly addressing the use of the MAD to treat OSA and symptoms of heart failure in African American males creating a gap in the literature. Duran, George, & Norris (2014) has indicated that African Americans appear to be underused in research due to several issues including lack of knowledge on behalf of the researcher regarding cultural differences, the sensitivity to research among minorities resulting in ineffective communication and the lack of consent from African Americans to participate in research. Other issues relating to the underuse of African Americans in research include the recruiting of minorities requiring extended time before acceptance and participation, the lack of consideration from researchers for minorities, and minorities mistrust of researchers (Duran, George, & Norris, 2014). Further issues that contribute to the underuse of African Americans include both the researcher and the participant's fear of unintended outcomes and lack of confidence due to social repercussions related to stigmas for participating in research (Duran et al., 2014). Duran et al. (2014) also indicated that barriers involved in the underuse of African Americans in studies may be manifested from overt or subtle racism, prejudice, and discrimination from the researcher, participant, or community. The focus on African Americans in this study was necessary due to the overwhelming number of African Americans who suffer from OSA and heart failure and the limitations they experience with regard to treatments that are conducive and accessible to meet their needs. This study was conceptualized because of a gap in knowledge regarding the isolated use of the MAD as an alternative noninvasive treatment for male African Americans veterans diagnosed with OSA and heart failure.

Purpose of Study

The purpose of this quantitative repeated measures study was to examine whether MAD therapy impacts symptoms of heart failure in African American male veterans diagnosed with OSA. This study has also determined whether treatment efficacy differs after controlling for patient BMI levels, including those who are underweight (less than 18.5 kg/m^2), have normal weight (18.5-24.9 kg/m^2), are overweight (25-29.9 kg/ m^2), obese (≥ 30 kg/ m^2), or smoke (Downs, Langton, Neyra & Niebuhr, 2016). Secondary medical data gathered from the VHA were used to address the research questions. The independent variable in this study was the use of the MAD. The dependent variables in this study were the symptoms of heart failure (ejection fraction, systolic blood pressure [SBP], diastolic blood pressure [DBP], oxygen saturation, brain natriuretic peptide [BNP], n-terminal pro-brain natriuretic peptide [NT-proBNP], and troponins levels). Identified covariates in this study include BMI and smoking status.

Research Questions and Hypotheses

There are three primary research questions that drive this research. The purpose of this study was to assess the changes in the values of dependent variables observationally. This study used secondary data to evaluate whether the independent variable, the use of MAD therapy, impacted the dependent variable, symptoms of heart failure in African American male veterans diagnosed with OSA. This study also determined whether treatment efficacy differs after controlling covariates, such as BMI levels and smoking status. The research questions are:

RQ1-Quantitative: Does use of MAD therapy have a statistically significant effect on the symptoms of heart failure in male African American veterans diagnosed with OSA?

H_01: Use of MAD therapy does not have any statistically significant effect on the symptoms of heart failure (ejection fraction, SBP, DBP, oxygen saturation, BNP, NT-proBNP, and troponins levels) in male African American veterans diagnosed with OSA.

H_a1: Use of MAD therapy does have a statistically significant effect on the symptoms of heart failure (ejection fraction, SBP, DBP, oxygen saturation, BNP, NT-proBNP, and troponins levels) in male African American veterans diagnosed with OSA.

RQ2-Quantitative: What is the moderating effect of BMI on the use of MAD therapy for treating symptoms of heart failure in male African American veterans diagnosed with OSA?

H_02: BMI levels do not have a statistically significant moderating effect on the use of MAD therapy for treating symptoms of heart failure (ejection fraction, SBP, DBP, oxygen saturation, BNP, NT-proBNP, and troponins levels) in male African American veterans diagnosed with OSA.

H_a2: BMI levels do have a statistically significant moderating effect on the use of MAD therapy for treating symptoms of heart failure (ejection fraction, SBP, DBP, oxygen saturation, BNP, NT-proBNP, and troponins levels) in male African American veterans diagnosed with OSA.

RQ3-Quantitative: Does smoking status have a statistically significant moderating effect on the use of the MAD therapy for treating symptoms of heart failure in male African American veterans diagnosed with OSA?

H_03: Smoking status (smoker or nonsmoker) does not have a statistically significant moderating effect on the use of MAD therapy for treating symptoms of heart failure (ejection fraction, SBP, DBP, oxygen saturation, BNP, NT-proBNP, and troponins levels) in male African American veterans diagnosed with OSA.

H_a3: Smoking status (smoker or nonsmoker) does have a statistically moderating effect on the use of MAD therapy for treating symptoms of heart failure (ejection fraction, SBP, DBP, oxygen saturation, BNP, NT-proBNP, and troponins levels) in male African American veterans diagnosed with OSA.

This study used the repeated measures multivariate analysis of variance (rMANOVA) and repeated measures multivariate analysis of covariance (rMANCOVA) to test this correlational study. The principal focus of this research was to use descriptive statistics to examine how MAD use affects heart failure symptoms in African Americans with OSA, who have various levels of BMI and smoke statuses. The independent variable in this study was the use of the MAD. Dependent variables used in this study as symptoms of heart failure were ejection fraction, SBP, DBP, oxygen saturation, BNP, NT-proBNP, and troponins levels. Body mass index and smoking have been used as covariates that may affect the relationship between the use of the MAD and symptoms of heart failure in this study. Identification of diagnosis and measurement baselines of heart failure and OSA have been gauged by the international classification of disease (ICD-10) code diagnosis found in archived records from the medical problem list of the VA electronic medical record. Heart failure was assessed by using a ratio level of measurement for assessing the magnitude of mean change in the seven dependent

variables (ejection fracture, SBP, DBP, oxygen saturation, BNP, NT-proBNP, and troponins levels). Obstructive sleep apnea was assessed by reviewing deidentified VA archival records containing the identified medical diagnosis of OSA. The initial measurement of the use of MAD treatment in archived records was assumed to begin upon receipt of the MAD device from the dental service in the VA electronic record. Study results have been utilized to provide information on appropriate outcome use of the MAD to the broader African American male population.

Theoretical Framework

The theoretical framework used in this study was Denham Harman's 1972 oxidative stress theory, which was initially modified from the free radical theory (Birch-Machin, Bowman & Kandola, 2015; Bisht & Dada et al., 2017; Gladyshev, 2014; Sanz, 2016). Free radicals are reactive oxygen species (ROS) that can contribute to oxidative stress and damage to cellular and DNA structures (Birch-Machin et al., 2015). Oxidative stress theory suggests that free radicals induce stress on the mitochondria resulting in its dysfunction leading to increased oxidative damage to organs in the body (Goswami & Maulik, 2015). The oxidative stress theory suggests that oxidative stress contributes to the mechanism of disease progression and heart failure through oxidative damage to the cells of the heart resulting in loss of function (Bobak et al., 2015; Bokov et al., 2009; Goswami & Maulik, 2015). Oxidative stress theory was applied to this study to explain how the MAD might impact symptoms heart of failure before and after controlling for patient BMI levels and smoking status by increasing oxygen flow and decreasing the stress on the heart. The primary objective of the oxidative stress theory used to explain

the effect the MAD has on heart failure is the amount of oxidative stress build-up resulting in oxidative damage to lipids, DNA, and proteins in the body that ultimately contribute to heart failure (Bokov et al., 2009). This theory was further explored in Chapter 2.

Nature of the Study

This quantitative nonexperimental study used a repeated measures design. Archival data used in this study were collected from the VHA. The targeted population was African American male veterans over the age of 20 who obtained treatment in VA dental clinics. I assessed secondary data through direct observations of quantitative data on mean dependent variable changes. De-identified archived data gathered to measure mean dependent variable changes were collected by VA for medical purposes placed in medical charts, which include both standardized tests and inquiries.

A repeated measure test was employed to empirically identify the statistically significant changes in heart failure symptoms when using the MAD for the target population. As a result, the qualitative design was not appropriate because patient observations or opinions were not warranted (Atlasti, 2018). Additionally, the quasi-experimental design was not appropriate because random assignments, cutoff scores, and time-series design were not required (Trochim, 2006).

Definitions

Body Mass Index (BMI): A medical determination of a person's weight in kilograms divided by the square of height in meters used to assess weight categories, possibly leading to health problems (CDC, 2015a).

Brain Natriuretic Peptide (BNP): A medical term that indicates an active hormone release in the same molecule that releases Nt-proBNP in reply to changes in pressure inside the heart (Cleveland Clinic, 2018b). BNP is a protein produced primarily by the left ventricle of the heart (Mangla, 2014).

Diastolic Blood Pressure: A medical term for the bottom number in a blood pressure reading. This number measures the pressure in a person's blood vessels when the heart beats (CDC, 2017d).

Ejection Fraction: A medical term that describes the percentage of blood leaving the two ventricles of the heart chambers during each contraction cycle (Mayo Clinic, 2018b).

Heart Failure: A medical term for a chronic progressive cardiovascular disease that involves the heart muscle not being able to pump enough blood to supply the body's oxygen and blood needs (AHA, 2017a).

Mandibular Attachment Device (MAD): One of three approved categories for dental sleep devices (Food and Drug Administration [FDA], 2017). MADs are oral devices used to move the mandible forward to open airways (Bajaj et al., 2017).

N-Terminal Pro-Brain Natriuretic Peptide (NT-proBNP): A medical term that indicates a nonactive prohormone released molecule that releases BNP in reply to changes in pressure inside the heart (Cleveland Clinic, 2018b).

Normal weight: A medical determination calculated according to BMI. A BMI ranging from 18.5kg/m^2 to 24.9 kg/m^2 is considered a normal and healthy weight (CDC, 2017a).

Obese: A medical determination calculated according to high BMI. A BMI above 30kg/m^2 is considered obese, resulting in high prevalence of heart failure (Clark, Fonarow, and Horwich, 2014).

Obstructive Sleep Apnea (OSA): A medical diagnosis for a form of sleep-disordered breathing that causes repetitive gaps in upper airway breathing during sleep, resulting in a diminished airflow (hypopnea) or complete cessation of airflow (apnea; Dallas, Holty, Owens, Qaseem, & Shekelle, 2014).

Overweight: A medical determination calculated according to BMI. A BMI ranging from 25 to29.9 kg/m^2 is considered overweight, resulting in high prevalence of heart failure (Clark et al., 2014).

Oxygen Saturation (SpO2): A medical term for peripheral oxygen saturation, which describes how much oxygen the blood is carrying.

Oxidative stress: A term to describe the result of reactive oxygen species damage to proteins, lipids, nucleic acids as shown in degeneration, aging, and other conditions (Huang, Ray, & Tsuji, 2012).

Reactive Oxygen Species: A term that consists of radical and non-radical oxygen species formulated due to an increase or decrease in ROS or an overwhelmed cellular antioxidant defense system. The formation of ROS is the result of a partial reduction of oxygen, as a result of oxidative stress (Huang et al., 2012).

Smoking: A term for the inhaling and exhaling of burning tobacco nicotine fumes from apparatuses such as cigars, cigarettes, or pipes (National Institute of Health [NIH], 2020).

Systolic Blood Pressure: A medical term for the top number in a blood pressure reading. This number measures the pressure in a person's blood vessels when the heart rests between beats (CDC, 2017d).

Troponins (cTnl and cTnT): A medical term for essential proteins found in the blood that produce muscular contractions and provide information on cardiac injuries such as acute cardiac syndromes (Jarolim & Mahajan, 2011).

Underweight: A medical determination calculated according to BMI. A BMI below 18.5kg/m^2 was considered less than a healthy weight (CDC, 2017a).

Assumptions

Assumptions provided were outside of the researcher's control and assumed to be correct. Methods to address these assumptions have been provided and were used to maintain the effectiveness of this study. An assumption made in this study consisted of using secondary data that was collected and recorded efficiently and accurately. This assumption was important to verify there was no missing data that may skew study outcomes. To address this assumption, I have checked for accuracy by reviewing the data for the identifier variable pertinent to the study and the time frame of the study (Cheng and Phillips, 2014).

Other assumptions made consisted of assuming archived VA electronic record data included appropriate information needed to answer the study questions. This assumption was important to verify that the research questions being asked could be explained by the data being used. To address this specific assumption, I provided operational definitions for the independent variables, dependent variables, outcome

variables, and covariates to ensure appropriate information was being obtained (Cheng and Phillips, 2014).

Further assumptions were that the quantitative repeated measures study was the best method to address the research problem accurately. This assumption was important to verify that the study was making the best use of the information provided by the data to answer the research question. To address this assumption, I reviewed the criteria for the repeated measures design, reviewed the variable data to be used, and developed a data plan that included the variables and covariates to be used in the study data set (Cheng and Phillips, 2014). Lastly, another assumption was that smoking consisted of tobacco usage unless specifically identified as another smoking substance. To address this assumption, I reviewed the smoking data received and verified there were no indications in excess of smoking tobacco. Assumptions are important and need to be addressed for the study to remain purposeful (Simon, 2011).

Scope and Delimitations

Heart failure as a byproduct of OSA in the United States was the focus of this study. The purpose of this study was to determine whether treatment using the MAD effectively reduces symptoms of heart failure in male African American veterans over 20 years of age who have been diagnosed with OSA and whether treatment efficacy differs after controlling for patient BMI and smoking status. This study does not include populations outside of self-identified African Americans. African Americans were used in this study due to being underutilized in multiple studies and disproportionately affected by heart failure (Bergerhenryent, 2017; Duran et al., 2014). Study findings could provide

information on alternative non-invasive treatment that will help African American veterans over twenty who have acquired the OSA and heart failure diagnosis.

When considering the study design, I initially considered using the health belief model. Browne et al. (2015) indicated that the health belief model's emphasis was on individual awareness, adaptations, and change in health behavior relating to MAD effectiveness. After consideration, the oxidative stress theory was selected because it suggests oxidative stress to the mitochondria may be due to OSA leading to increased oxidative damage in those with heart failure symptoms. This theory has been used in this study to explain oxidative stress, oxidative damage, and the effect the MAD intervention has on the dependent variables before and after controlling for patient BMI levels and smoking status.

The generalizability of this repeated measures study gathered from secondary data allows for statistical inference to be made with fewer VHA records (Jeyabalasingham, Lanka, & Ragavan, 2011). A delimitation that I have used within this study was to use secondary data to limit my errors when collecting of data error. Other delimitations were the use of only including identified African Americans and no other groups to maintain the focus of the study. The study was further delimited by the data collection time frame between 2016 and 2017. Another delimitation was the use of data only being gathered from the VA dental clinics in the U.S. that provided the MAD. Further delimitation in this study was the use of 29 archived records that contained the necessary inclusion criteria needed for the study. Lastly, another delimitation was the use of the VA

informatics and computing infrastructure (VINCI) program due to it being the only database to house the VA archived records on the dependent variables in this study.

Limitations

Limitations exist within this study involving secondary data from archived records. Cheng & Phillips (2014) have argued that limitations of using secondary data can consist of existing data not specifically addressing the exact research questions or hypothesis's articulated in a new study due to some variables not being available for analysis. Circumstances causing variables not to be available for analysis were due to them not being important in the initial collection of secondary data relating to a population subgroup, or geographic region (Cheng & Phillips, 2014). Circumstances of this type could result in omissions crucial to variable analysis within the study. Therefore, limitations in secondary data could result from the data being used, not specifically addressing the desired focus of this study or population, resulting in skewed or unusable results to answer research questions.

Cheng & Phillips (2014) have further argued that another limitation using secondary data was that confidential data may have been deleted to protect categories such as race, age, ethnicity, or other covariate variables. This limitation could result in data not reflecting the specific population for the study and excess time being spent reviewing the data. According to Cheng & Phillips (2014), researchers collecting data may not always be the same person analyzing the data. The change in personnel, knowledgebase, and awareness of study distinctions from the researcher could result in data inconsistencies. The result of these limitations may be that small specific nuances of

information may be missed or glanced over. Cheng & Phillips (2014) have also revealed methods to mitigate missed details that include utilizing data from an appropriate population, and that covers an accurate geographic location discussed in the study. Other methods discussed by Cheng & Phillips (2014) consist of verifying that the secondary data used has the correct focus needed for the study and that it utilizes appropriate variables that can be resolved through careful examination of relevant documents.

Significance

This study was significant because it has the potential to provide useful information regarding a possible first-line alternative treatment to the CPAP machine for treating people suffering from OSA and heart failure. According to Abraham et al. (2009), the use of the MAD in patients resulted in a 52% average improvement apnea-hypopnea rate <10 for those who use it. Bratton, Gaisl, Kohler, & Wons (2015) indicated that there was no difference between MAD use and CPAP use in terms of blood pressure, which can be used as a marker for cardiovascular diseases. Though very little data was available regarding the impact of the MAD on heart failure among those with OSA, the MAD may have the potential for similar efficacy values for treating symptoms of heart failure symptoms (Akinnusi et al., 2013; Booth et al., 2014).

Social Change Implications

Cistulli et al. (2013) used one-month trials when reviewing the MAD in comparison to CPAP treatments relating to cardiovascular functioning. This research study supports professional knowledge, practice, and policy by providing a longer-study term than has previously been conducted that references the relationships between the use

of MAD and changes in heart failure symptoms (Marklund et al., 2012). This research can be beneficial to patients wanting to objectively assess the MAD's impact on heart failure symptoms across time in terms of its effectiveness on the underused and under-assessed African American male U.S. veteran population. Additionally, this study supports both the public health and healthcare fields' professional practice by serving as a reference for local, state, and government agencies and medical providers regarding whether to request MAD. Casey et al. (2012) indicated that 26-36% of the veteran population served by VHA who have OSA, and the 13.5% that have heart failure. Findings from the study could be used to promote progressive social change by adding new information to the public health and medical field that can be used for medical providers, patients, and policymakers to develop a better understanding of MAD treatments for OSA and symptoms of heart failure. Moreover, this study could encourage patient self-efficacy by providing information that can better equip medical personnel to explain and estimate the effect the MAD may have on heart failure symptoms in African American male veterans with OSA.

Findings from the study could be used to contribute knowledge regarding how patient's use of MAD therapy impacts OSA and symptoms of heart failure in African American male veterans. The results could provide data that can be used to promote the use of a cost-effective alternative treatment as opposed to the cumbersome and often expensive CPAP to treat those with both OSA and symptoms of heart failure. The results of this study may provide both providers, patients, policymakers, and African American male Veterans an awareness of the benefits of the MAD that may trigger a reevaluation

of the types of treatment options dental providers, hospitals, and healthcare organizations offer. Healthcare professionals could use the information to promote and advocate for healthcare policy changes in hospitals, clinics, and agency dental and medical care guidelines for the treatment of OSA and heart failure that could encourage insurance coverage of the MAD to treat OSA and possibly reduce symptoms of heart failure in African American veterans.

Summary

In this chapter, I have distinguished that both OSA and heart failure are significant public health concerns in the United States. African Americans are severely affected by both OSA and heart failure (Ambrosy et al., 2014; Arynchyn et al., 2009; Badr, Pranathiageswaran, Rowley, & Severson, 2013; Becker et al., 2014; Colvin-Adams et al., 2014; Dudley & Patel, 2016). Heart failure is a rising epidemic amongst minorities, though there have been some successful non-pharmacological and limitedly invasive methods such as CPAP in place to provide maintenance and control for its reduction (Kato, Kasai & Suda, 2014). According to Colvin-Adams (2014), African Americans have higher heart failure prevalence, and death rates once admitted to hospitals for heart failure than any other ethnic group. Additionally, the rate of heart failure-associated deaths and morbidity are highest amongst African Americans (Colvin-Adams et al., 2014). Forces behind the growing pervasiveness of heart failure include being overweight, hypertensive, smoking, obesity, and lifestyle.

This study looked to assess the MAD effect on heart failure symptoms and the two main covariate factors smoking and BMI by establishing quantitative research

questions that can be explained within the theoretical framework. A repeated measures study design was used to provide clarification regarding the research problem by reviewing the same archived variables at different monthly intervals throughout the study (Goff, Schwartz &Wilson, 2018). Results from this study possibly could lead to alternative methods of treatment that would encourage compliance and improve air intake needed to reduce heart failure levels among African Americans. As a result, this study may lead to social change, reduce symptoms of heart failure, promote increases in life expectancy, life quality through increased airflow, and reduce healthcare costs. Chapter 2 includes influential and peer-reviewed articles and studies to address the study focus relating to literature search strategies, the theoretical foundation, and a literature review related to key variables.

Chapter 2: Introduction

Introduction

This study evaluated whether the use of the MAD significantly reduces heart failure symptoms in male African American veterans with OSA above the age of 20 and whether treatment efficacy differs in terms of BMI and smoking status. Chapter 2 includes information on literature search strategies, the theoretical foundation, and a literature review related to key variables. This chapter covers the impact that OSA, BMI, heart failure, and smoking have had on African Americans as well as current treatment methods for OSA and the need for an alternative solution. The current literature review covers BMI, smoking, African Americans, BMI and smoking, BMI, smoking and African Americans, OSA, heart failure; biomarkers of heart failure, prevalence of heart failure, prevalence of OSA and heart failure, race, OSA and heart failure on race, BMI and heart failure; smoking and heart failure, oral devices and obstructive sleep apnea, MAD, CPAP, CPAP versus MAD, CPAP versus MAD economic cost, summary, and conclusion.

Literature Search Strategy

Advanced search engines and libraries that were used to access literature and databases included Google Scholar, the Florida Agricultural and Mechanical University Library, the VA Library, and the Walden University Library. Literature databases used to gain access to data, national websites of organizations, peer-reviewed articles, journals, online books, reports, and studies, consist of the American Medical Association, BioMed Central, Centers for Disease Control, Google Scholar, Horizons, Medline, Medline Plus,

NIH Public Access, ProQuest, PubMed, Science Direct, and SpringerLink. Search phrases or combination of phrases used to locate literature were African American heart failure, BMI, cardiovascular mortality, continuous positive airway pressure, heart failure, heart failure risk factors, heart failure mortality, oral devices, MAD, mandibular oral device, OSA, overweight, oxidative stress theory, mandibular respiratory device, mandibular respiratory splint, obesity, respiratory appliance, sleep medicine, sleep apnea, sleep breath, smoking, and veteran. Data and reports used in this study were published between 2010 and 2019. This literature review includes 77 seminal and peer-reviewed articles. While performing research, there appeared to be contradicting information related to CPAP versus MAD efficacy and limited information related to the effect the MAD has on African American veterans with heart failure, which supported the necessity of this study. Research on evaluating whether the use of the MAD significantly reduces heart failure symptoms in male African American veterans with OSA has been limited. Partialized information from various studies was used to describe gaps in the literature and the need for further observatory evaluations of the study topic that include longer study periods.

Theoretical Foundation

The oxidative stress theory serves as the theoretical foundation for this study. The oxidative stress theory was created by Denham Harman and extracted in 1972 from the free radical theory to explain how molecular damage occurs in the body (Birch-Machin et al., 2015; Sanz, 2016). The oxidative stress theory was used to indicate how oxidative stress affects the mitochondria, leading to increased oxidative damage (Goswami &

Maulik, 2015). This theory implies that increased oxygen stress levels caused by OSA contribute to heart failure under normal metabolic processes causing oxidative damage to macromolecules including lipids, DNA, and proteins resulting in loss of function (Lagouge & Larson, 2013).

The purpose of this theory was used to indicate that oxidative stress on cells was the primary cause of the mitochondria being attacked, resulting in oxidative damage to the body. This theory was driven by the theoretical belief that the mitochondria generates significant amounts of cell energy, consuming over 90% of cellular oxygen, and is a target of ROS, ultimately setting the limit on lifespan as a result of a loss of function (Lagouge & Larson, 2013). In sum, when oxidative stress attacks mitochondria, it leads to increased oxidative damage, which causes a decrease in efficacy and a loss of functional integrity (Birch-Machin, Bowman, & Kandola, 2015; Sanz, 2016). Eisele, Markart & Schulz (2015) have used the oxidative stress theory among humans with OSA in relation to cardiovascular diseases, indicated that there was growing evidence to support the pathophysiology of untreated OSA causing oxidative stress resulting in morbidity and mortality. Additionally, this study has indicated that OSA treatments such as CPAP have been used to effectively reverse these abnormalities (Eisele, Markart & Schulz, 2015).

Studies from Cistulli et al. (2014b) and Marklund, Randerath, & Verbaecken (2012) have also displayed that the use of MAD may also advance oxidative stress and endothelial function. Due to past studies identifying the usefulness of CPAP as an OSA treatment method to address oxidative stress, this study may represent the step necessary

to support the additional impact of MAD on heart failure symptoms. The oxidative stress theory concept was used to suggest that those who suffer from OSA and heart failure related to oxidative stress have potentially impacted mitochondria resulting in oxidative damage that possibly affects symptoms of heart failure.

Bokov et al. (2009) argued that the use of oxidative stress theory on secondary data has shown a correlation between increased lifespan, reduced oxidative stress, and oxidative damage. Richardson & Schadt (2014) and Bokov et al. (2009), has also revealed that experimental manipulations have increased rodent lifespans and displayed a reduction in oxidative damage to macromolecules. The oxidative stress theory was used in this study to explain how damaged cells that result in increased heart failure symptoms are impacted by the MAD while controlling for BMI and smoking status. This theory was used to indicate how reducing oxidative stress on heart failure symptoms can be associated with lowering oxidative damage, therefore increasing heart function. If impactful, the oxidative stress theory could yield useful information relating to the effect of the MAD on heart failure symptoms while providing data to suggest the use of alternative heart failure treatments.

Literature Review Related to Key Variables

Sleep apnea awareness began in the mid-1960s after the first recorded polysomnography test, which was used to record apnea episodes during sleep (Bahammam, 2011; Resmedica, 2011). Resmedica (2011) has shown that in 1978 in the je Remmers study indicated that OSA was identified to occur at the point of the airway closure in the oropharynx. Obstructive sleep apnea leads to adverse medical

consequences when left unmanaged (Javaheri, Javaheri, & Javaheri, 2013). Unmanaged OSA causes large intrathoracic pressure swings, which increase both the left and right ventricular pressure, potentially resulting in cardiac arrhythmias, atrial distensions, and pulmonary congestion (Javaheri et al., 2013). Additionally, OSA causes multiple sleep arousals, which may lead to increased voluntary activity, decreased involuntary activity, increased blood pressure, and heart rate during sleep (Javaheri et al., 2013). Obstructive sleep apnea also results in oxygen desaturation during sleep, followed by a period of recovery where reoxygenation and reduced carbon dioxide occur. As a result of increased ventilation causing sympathetic activation resulting in pulmonary arteriolar vasoconstriction, pulmonary artery pressure, right ventricular afterload, and abnormal enlargement of the heart may occur (Ayas, Badran, & Laher, 2014; Javaheri et al., 2013). This recurrent cycle of hypoxia and reoxygenation, known as ROS, results in endothelial cell dysfunction (Chen et al., 2017). Obstructive Sleep Apnea contributes to approximately 12-53% of heart failure cases (Ayas, Badran, & Laher, 2014). Obstructive sleep apnea promotes systematic hypertension and may result in complications involving left-sided heart failure and ejection fraction (Ayas et al., 2014; Javaheri et al., 2013).

Heart failure has been indicated as an epidemic due to its spread across ethnicities and its specific effect in the U.S. (Dunlay & Roger, 2014). Common risk factor for heart failure includes hypertension, obesity, smoking, physical inactivity, excessive alcohol intake, poor diet, hyperlipidemia, atherosclerosis, and diabetes mellitus (Colvin-Adams et al., 2014). Diagnosis of this disease is often identified by either a physician, respiratory therapist, or dentist.

Treatment methodologies for those with OSA resulting in adverse health outcomes such as heart failure have expanded over the years, and now depending on the level of the diagnosis, may include invasive or noninvasive treatment (Colvin-Adams et al., 2014). Economically, untreated OSA potentially translates into healthcare dollars over economic cost and are higher for those undiagnosed with OSA (Gillespie et al., 2015). Non-invasive treatment methods currently involve CPAP use initially (Kasai et al., 2014). There are several clinical practice guidelines from the American Academy of Sleep Medicine and the American Academy of Dental Sleep Medicine [AADSM] recommendations for oral device use (Chervin et al., 2015). Practice guidelines for the receipt of the MAD suggest that it was provided upon patient request, if the there was a sleep physician recommendation or if there was patient intolerance with CPAP, rather than no therapy at all (Chervin et al., 2015). Additionally, if provided an oral device for OSA, most qualified dentists use a custom titratable oral device over a non-custom device, provide oversight, conduct follow-up sleep testing, and request patients return for periodic office visits for monitoring (Chervin et al., 2015).

BMI

The BMI is a frequently used measurement for assessing body composition (Downs et al., 2016). The BMI has been identified by the World Health Organization (WHO) in a scale indicating those who have BMI < 18.5 as underweight, those with a BMI between 18.5-24.9 as normal, those between 25-29.9 as overweight and those with BMI >30 as obese (Downs et al., 2016). In 2015, 159 million United States adults, approximately 69%, were listed as obese or overweight (Arnett et al., 2015).

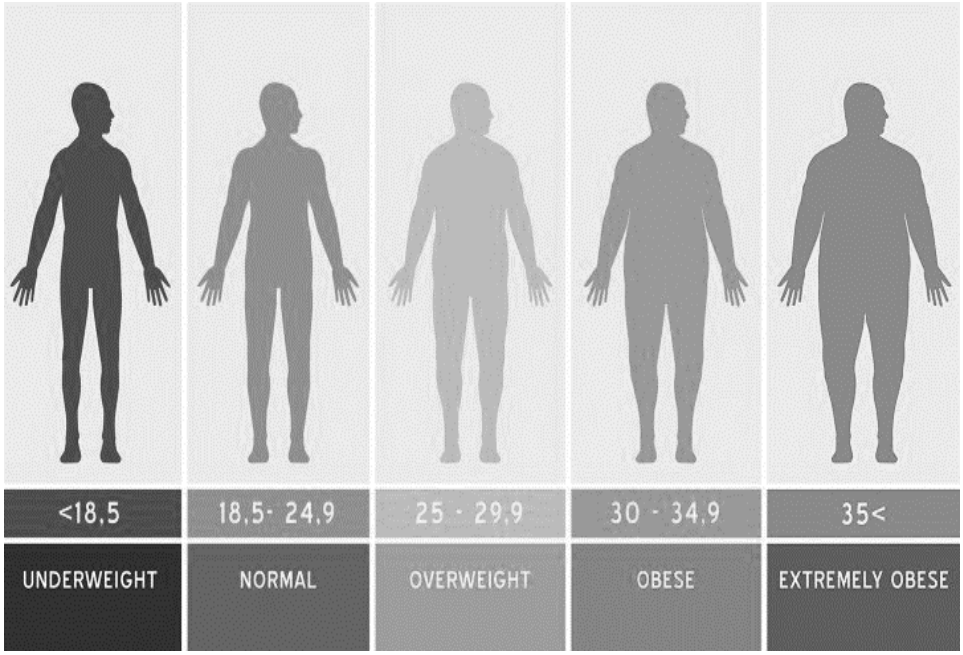


Figure 1. From "Higher BMI Means Higher Risk of Heart Disease and Heart Failure: BMI and heart disease have a cause-and-effect connection, not just a link" by J.J. Brown, 2018 (https://www.everydayhealth.com/heart-failure/living-with/bad-news-if-your-bmi-is-high-fat-causes-heart-failure/).

Downs, Langton, Neyra & Niebuhr (2016) reported data from 2011-2012 that has indicated that the overweight prevalence of those with a BMI ≥ 25 kg/m^2 in the United States, ages 20 to 39 is greater than 60%. Obesity is the fifth leading cause of death in the world (Dare, Mackey & Pell, 2015). The obesity prevalence of those with a BMI of ≥ 30 kg/m^2 in the United States, ages 20 to 39, is higher than 30% (Downs et al., 2016). Dudley & Patel (2016) have shown African Americans compared to all other ethnicities have a 51% greater likelihood of obesity even after controlling for age, sex, and comorbidities. The prevalence of obesity among African American men in the United States in 2012 was 37.1% and 56.6% for African American women (Dudley and Patel,

2016). In multiple studies by Dudley & Patel (2016) and Chaturvedi & Marulanda-Londono (2017), an increased BMI was associated with OSA and suggests an increased risk for heart failure. In sum, BMI has a significant effect on African Americans ages 20 and older in the United States and was a factor for heart failure.

Smoking

Both smoking and high BMI are major public health concerns that are increasing globally (Dare et al., 2015). Smoking is the inhalation of carcinogenic chemicals from tobacco (Institute of Medicine, 2015). There are over 7,000 toxic carcinogens in tobacco (Institute of Medicine, 2015). Smoking is the principal cause of preventable mortality in the United States (Dixon-Williams, Krishnan & Thornton, 2014). There are approximately 5-6 million people who die annually as a result of smoking. Though smoking prevalence has dropped in the United States, rates are still as high as 30% in some states (Dixon-Williams et al., 2014). Currently, approximately 40 million Americans smoke, leaving themselves at risk for premature mortality and morbidity (Dixon et al., 2014). Over 29.8 % of those that smoke in the United States are African Americans (CDC, 2018b). Smoking is a major contributor of illnesses such as cancer, respiratory, heart disease, stroke, and cardiovascular disease (CDC, 2018b; Dare et al., 2015; Dixon et al., 2014; Institute of Medicine, 2015).

Smoking was estimated to cause over 480,000 deaths annually in the United States; 160,600 are linked to cardiovascular disease (Institute of Medicine, 2015). A single puff of smoke from a cigarette exposes the smoker to over 10^{15} free-radical causing a significant amount of oxidative stress and immediate oxidative damage

(Institute of Medicine, 2015). In sum, smoking is a substantial concern for African Americans, causing various life-threatening diseases and conditions. Smoking also causes oxidative stress resulting in oxidative damage to the body contributing to cardiovascular disease.

African Americans

According to the United States Census Bureau (2019), in 2018, there were approximately 47.8 million African Americans in the United States. Over 13.5% of African Americans in the United States are in fair to poor health, while 11.3% of those over 65 have no insurance (CDC, 2017b). There are approximately 22.9 million African American males in the United States (United States Census Bureau, 2019). Of the African Americans living in the United States, there are 21.6% of men that smoke (CDC, 2017b). Over 88% of African Americans are more likely to have OSA than whites (Dudley & Patel, 2016). In an analysis of data from the National Health and Nutrition Examination Survey (NHANES), African Americans appear to be more strongly associated with hypertension related to cardiovascular disease as a result of the diagnosis of OSA than Hispanics or whites (Dudley & Patel, 2016). The leading cause of death in African American males as of 2015 was heart disease at a rate of 23.9% (CDC, 2018a). Heart disease has been the leading cause of death in African Americans by race, ethnicity, and gender since as late as 2013 (Heron, 2017; The National Academic Press, 2017). In sum, the data provided by the CDC (2018a) and Dudley & Patel (2016) indicate the significant impact of heart failure on African American males relative to the

population living in America. Therefore, there is a need to utilize African Americans in this study as a significantly affected population.

BMI and Smoking

Body mass index and smoking are growing health epidemics that have been linked to heart failure. There is a complicated relationship between high BMI and smoking that may not be completely understood and has had a history of conflicting study results (Bush et al., 2011). There also appears to be some reverse causation relating to smoking and BMI, suggesting that those who are overweight are more likely to start smoking than those not overweight (Dare et al., 2015). Additionally, reports have also indicated that fear of weight gain has resulted in former smokers relapsing. Reports from this study further indicated that those who have a high BMI and who smoke are more likely to be among those who are socioeconomically deprived. Additionally, BMI was sometimes derived from self-reported weight and height measurements, which may tend to be overestimated and weight underestimated.

Smoking and BMI have been synonymous through myth and association. Myths include the idea that smoking protects the user from weight gain (Dare et al., 2015). The study by Dare, Mackay & Pell (2015) was developed to explore the association between smoking and BMI > 30 kg/m^2 in adult's age 31 to 69 years. Results showed those who currently smoke were less likely to have a high BMI than those who never smoked (Dare et al., 2015). However, those who were former smokers were more likely to have a higher BMI than those who never smoked (Dare et al., 2015). Nevertheless, obesity increased with the amount smoked and that former heavy smokers had a higher BMI than former

light smokers (Dare et al., 2015). Therefore, showing that the synonymous myth that smoking protects a person from weight gain was overgeneralized (Dare et al., 2015).

Another study by Plurphanswat & Rodu (2014) showed that smokers have a lower BMI and a probability of obesity greater than those who have never smoked. This study's focus was to evaluate the association amongst smokers, selected demographics, and BMI on those 25-64 years of age (Plurphanswat and Rodu, 2014). Results indicated that males who smoked had a 6.9% lower BMI mean than those who had never smoked or who were former smokers (Plurphanswat and Rodu, 2014). Study results have also indicated that African Americans and Hispanics had a higher BMI than any other ethnicity within the study (Plurphanswat and Rodu, 2014). In sum, multiple studies have shown that there may be reverse causation amongst factors of BMI and smoking and that males appear consistently more affected by both factors. Therefore, these two characteristics should be reviewed as covariates in this study due to the effect they may have on African Americans with OSA.

BMI, Smoking, and African Americans

Body mass index and smoking are important risk factors that are strongly linked to a variety of health concerns for African Americans. In a follow-up prospective cohort study by Bethea et al. (2014) on a pooled all-cause analysis of BMI and mortality among African Americans from several studies indicated that obesity was linked to a higher risk of mortality in African Americans than whites or Asians. The study used a multitude of additional studies that included 239,526 participants, from ages 30 to 104 (mean age of 52 years old), all followed for an average of 11.7 years, half of the participants reported

smoking (Bethea et al., 2014). Of the population studied, a third were overweight with a BMI between 25-29 kg/ m^2, and another third were obese with a BMI over 30 kg/m2. Data indicated that being overweight was higher in men than in women (Bethea et al., 2014). In both male and female African Americans, it was noticed that in subjects whose BMI increased, there was an increase in hazard ratios resulting in 11,386 deaths (Bethea et al., 2014). There was a more significant effect in women, the closer they were to BMIs of 27.4 kg/m2 and the higher men were to BMIs greater than 30 kg/m2 (Bethea et al., 2014). However, with increased BMI, there was a continuous reduction in smoking. The prevalence of those who were overweight and obese was shown to increase drastically among African Americans and in recent decades in the United States, are expected to continue growing in African Americans (Bethea et al., 2014).

In a randomized control study by Antoni et al. (2018), it identifies that African Americans between 18-65 years of age gained an excessive amount of weight after attempting to quit smoking. This study's main focus was to study the pattern in weight gain by participants and study the association between smoking, weight gain, and smoking cessation treatment in African Americans (Antoni et al., 2018). This study has shown that African Americans experience the highest rate of tobacco-related deaths than any other ethnicity and have a higher level of obesity (49.5%) and percentage of those who are overweight (76.7%) than whites (Antoni et al., 2018). Additionally, over 80% of smokers gain weight in the first three months after they have attempted to quit, followed by a smoking relapse (Antoni et al., 2018). Results indicated that there was an overall weight increase of 0.62 pounds per month or 4-10 pounds throughout the study for all

participants who did not abstain from smoking (Antoni et al., 2018). Those that did abstain from smoking gained an increased average of 1.2 pounds per month or 7.26 pounds throughout the study (Antoni et al., 2018).

In the study by Gapstur, Hildebrand & Patel (2014), it has indicated that BMI and smoking are highly significant in blacks and that those who smoke and are underweight or in high weight category pertaining to BMI had a higher risk for mortality. This study assessed the risk of mortality according to BMI in both whites and blacks who smoked and had a prevalent disease (Gapstur, Hildebrand & Patel, 2014). Results revealed that BMI was highest in those who had never smoked or who had a prevalent disease and that BMIs in those considered overweight were linked to a significantly high risk of mortality (Gapstur et al., 2014). Additionally, smoking was associated with greater mortality in blacks with both a prevalent disease and those who smoke (Gapstur et al., 2014). Further results have indicated that the absolute mortality rates were higher in blacks than whites in each BMI category and that BMI was strongly associated with mortality prior to age 70 (Gapstur et al., 2014). In sum, the information in the above studies indicates that with an increase in BMI, smoking declines. Additionally, there was an indication that weight gain was less associated with smoking for those who do smoke and was more likely for those that do not smoke contributing to the current public health condition of BMI and smoking. As a result of the significance of African Americans affected by these two epidemics, this study used both BMI and smoking as covariates on African Americans.

OSA

Obstructive sleep apnea occurs involuntarily and repeatedly when the back of the throat collapses on the airway while sleeping, which stops or slows down oxygen from entering the lungs (American Lung Association [ALA], 2018). This study has several risk factors consisting of gender, age, and those who are excessively overweight (ALA, 2018). Symptoms of the OSA condition consist of snoring pauses in breathing, daytime sleepiness, and difficulty in control medical issues (ALA, 2018). OSA affects approximately 24.9 million adults in the United States (Watson, 2016). African Americans are 3.5 times more likely to receive OSA than any other ethnicity (Farinde, 2015). Obstructive sleep apnea appears 2-8 times more in males than females and shows a significant increase with age after age 20 through 65 and higher (Ayo-Yusuf, Motloba, Solomons & Sethusa, 2015).

Obstructive sleep apnea has been shown as a significant health issue over the last 20 years, resulting in various increased health risks, including cardiovascular issues (Gillespie et al., 2015). Gillespie et al. (2015) revealed that within the Wisconsin Sleep Cohort study, approximately 13 million Americans over age 30 had moderate to severe OSA, and 9% were men (Dempsey, Palta, &Young, 1993 as cited in Gillespie et al., 2015). Additionally, 29.5 million Americans over age 30 have mild to severe OSA, and 24% are men (Gillespie et al., 2015). As a result of untreated OSA, patients may be at risk for cardiovascular morbidities and hypertension as well as all-cause mortality or high cardiovascular mortality with a 2.4 relative risk for heart failure (Gillespie et al., 2015). Additionally, due to untreated OSA, heart failure was found likely to occur six times or

more compared to control having a reduced left ventricular ejection fraction (Gillespie et al., 2015).

Obstructive sleep apnea has also been linked to a fall in oxygen saturation levels, vacillating heart rate, and blood pressure (Badr et al., 2013; Booth et al., 2014). However, if left untreated, OSA was linked to cardiovascular diseases such as heart failure, stroke, and a three to four times higher mortality risk rate (Badr et al., 2013; Booth et al., 2014). The link between OSA and heart failure was considered to be due to the continual amount of low levels of oxygen being inhaled and getting to the heart, resulting in changes in blood pressure and carbon dioxide levels (Cleveland Clinic, 2018a). In sum, OSA is an involuntary condition that was likely to occur with increased weight and age. This disease also affects African Americans more than any other ethnicity and contribute to both heart failure and death.

Heart Failure

Heart failure is a condition of OSA and is often called congestive heart failure, which occurs when the heart fails to pump blood through the body as effectively as it should (AHA, 2018; Mayoclinic, 2018a). The first research on heart failure came about in 1971 as a result of the Framingham study, which linked to the failing health and imminent death of President Franklin D. Roosevelt to heart disease (Gordon, Kannel & Schwartz, 1971 as cited in Levy, Mahmood, Vasan & Wang, 2013). The Framingham Study indicates an evolving heart failure diagnosis that began in the 1960's and continues to grow to include elevated lipids (troponins), myocardial infarction (which precede ejection fraction), and high blood pressure (Gordon, Kannel & Schwartz, 1971 as cited in

Levy et al., 2013). This disease is both acute and chronic, resulting in symptoms related to difficulty breathing, weakness, swelling, limited movements, irregular heartbeat, and chest pains (Mayo Clinic, 2018a).

The majority of research related to reducing heart failure has focused on ethnicities affected, epidemic prevalence, risk factors, diagnosis, and standard treatment methodologies. The effect of heart failure is one that has been shown to affect all ethnicities, including 6.6 million people in the United States (Colvin-Adams et al., 2014). Heart failure has resulted in approximately a 50% death rate within 5 years of receiving the disease (CDC, 2016). This disease disproportionately affects African Americans more than whites or any other ethnicity (Akinboboye & Cuyjet, 2014). This disease specifically affects African Americans at a rate of 4.6 per 1,000 person-years (Colvin-Adams et al., 2014). Heart failure incidence and prevalence is attributable to an increased burden of traditional risk factor amongst African Americans (Albert et al., 2017).

Akinboboye & Cuyjet (2014) showed that within 82 African Americans out of 316 white participants, African Americans had a 50% risk of death and mortality versus whites. This affliction has been compacted by the result that the population most affected, African Americans, not having experienced the same benefit from treatment as whites (Colvin-Adams et al., 2014). The literature review provided in this study indicated that the skew in effect on African Americans may be a result of risk factors associated with heart failure being considerably more prevalent in African Americans. Nevertheless, African Americans are underrepresented in heart failure trials, though the reason is not well understood (Albert et al., 2017). The significance of African Americans being

underrepresented in studies makes it difficult to draw accurate conclusions about ethnicities and races (Clark, Ehlen & Paul, 2016). In sum, heart failure is a result of OSA that affects African Americans disproportionately and, in some cases, maybe due to not undergoing similar benefits from treatment as other ethnicities and being underutilized in trials.

Biomarkers of Heart Failure

Kimmenade & Januzzi (2011) argued that heart failure has been indicated as a considerable burden on the medical healthcare field and population. The report focuses on identifying appropriate biomarkers for elective, supplemental diagnostic for heart failure (Kimmenade & Januzzi, 2011). In this study, the researchers identified various perspectives on evaluating heart failure, including alternative methods. The researchers have created standards of which to evaluate biomarkers such as biomarkers should be measurable at a reasonable cost, able to increase knowledge through the use of a clinical workup, and be beneficial in the management of cardiovascular disease (Kimmenade & Januzzi, 2011). Other quantifiable methods are that biomarkers identify the cause, presence, and absence of heart failure while indicating its severity (Kimmenade & Januzzi, 2011). Researchers have identified several promising and useful biomarkers in identifying heart failure. Specific biomarkers that have proven to be historically practical in identifying heart failure symptoms such as BNP and NT-proBNP, which assist in identifying vessel stretching, injury, and hypoxia (Kimmenade & Januzzi, 2011). Another biomarker that was indicated was t ST2, which identified the myocardial strain and was used to monitor heart failure severity (Kimmenade & Januzzi, 2011). Additionally, the

researchers have used troponins as a method to assess myocardial necrosis or cell death in a failing heart (Kimmenade & Januzzi, 2011).

In the study by Anand et al., (2017), the focus is on gathering and summarizing existing literature on guidance, utility, and availability of heart failure biomarkers. A biomarker has been defined as "any substance, structure, or process that can be measured in the body or its products and influences or predicts the incidence of outcome or disease" (Anand et al., 2017, p. e1055). The researchers found that symptoms of heart failure, such as BNP, can accurately diagnose heart failure patients presenting to emergency departs with 90% sensitivity. Researchers have indicated that appropriate biomarkers form the blood accuracy level, while Nt-proBNP has a higher sensitivity ruling out heart failure (Anand et al., 2017). Other heart failure symptoms, such as troponins, strongly predict heart failure risk, can detect heart failure severity, assess risk, guide therapy, and prevention (Anand et al., 2017).

Gaggin & Januzzi (2015) sought to provide a distinction between different biomarkers as it related to heart failure. This article displays the heart failure biomarkers by identifying myocardial stress/injury through markers such as BNP, NT-proBNP, proBNP, and troponins, neurohormonal activation through Mid-regional pro-adrenomedullin (MR-pro ADM), remodeling through the ST2 gene and other comorbidities that contribute to heart failure (Gaggin & Januzzi, 2015). Gaggin & Januzzi (2015) indicated that the AHA has shown that both BNP and NT-proBNP have been recommended for assessing both diagnosis and prognosis. Additionally, troponins have received approvals for aiding in identifying the prognosis and the finding of acute

myocardial infarction as the preliminary of acute heart failure (Gaggin & Januzzi, 2015). Biomarkers displayed have value in informing change from clinical care and also indicate if there was an improvement in prognosis. The information yielded from the researchers in this article have shown identifiers that can be used to monitor, assess, and benchmark heart failure. In sum, identified biomarkers used in this study as dependent variables are both viable and reliable to accurately identify and monitor heart failure.

Prevalence of Heart Failure

Over 5.7 million are diagnosed with heart failure, costing the nation 30.7 billion dollars annually in missed days of work, healthcare, and medications (CDC, 2016). Emory Healthcare (2018) has displayed there are over 500,000 new cases of heart failure a year. Of those identified with heart failure, 1.4 million are under the age of 60, and more than 5% are between the age of 60 and 69 (Emory Healthcare, 2018). Emory Healthcare (2018) has displayed that African Americans are 1.5 times more likely to develop heart failure than whites.

In a compendium review article by Colvin-Adams et al. (2014) on information about heart failure in African Americans related to management and challenges, it has been indicated that heart failure is increasing despite 20 or more years of medical and surgical therapies. Colvin-Adams et al. (2014) has shown that as a result of the AHA data indicates that African Americans receive heart failure at a rate of 9.1 per 1,000 person-years versus whites at 6 per 1,000 person-years. This outcome was accompanied by a similar result from the artherosclerosis risk in community's study, which has shown that new onsets of heart failure are acquired at a 4.6 per 1,000 person-years in African

Americans versus Chinese at 1.0 per 1,000 person-years and whites at 3.5 per 1,000 person-years (Bahrami, Bluemke & Kronmal (2008) as cited in Colvin-Adams et al., 2014).

In a study by Cheriyath, Guduru, Jadhao, Komanduri & Went (2016), it shows that despite population awareness and medical advancements, the same risk factors for heart failure continue. Cheriyath et al. (2016) analyzed NHANES data from 2013-2014, which explained the changes in the prevalence of the heart failure epidemic. The researchers indicated that heart prevalence in Americans is over 23 million, affecting 1.9% of the population (Cheriyath, Guduru, Jadhao, Komanduri & Went, 2016). Of those affected by heart failure, the males appear to be significantly more affected, with a prevalence of 1.8% (Cheriyath et al., 2016). Additionally, researchers from this study have indicated that heart failure continues to grow at a significant rate, potentially attributable to awareness and advancement in diagnosis (Cheriyath et al., 2016). Cheriyath et al. (2016) have shown that the primary risk factor consists of coronary artery disease and hypertension, those over 65 years old, and increasing obesity. In sum, heart failure is an epidemic that considerably affects African Americans more disproportionately than other ethnicities, and is increasing at a significant rate. The Cheriyath et al. (2016) result provides information indicating the need for this study to attempt to find an alternative method to reduce this major health concern.

Prevalence of OSA and Heart Failure

Obstructive sleep apnea has been linked to heart failure and estimated to cost 3.4 billion annually (Akinboboye, Jean-Louis, Mitchell, Ogedegbe & Olafiranye., 2013). In

the study by Lombardi, Losurdo, Montano, Parati & Tobaldini (2017), it has been indicated that there is increasing evidence to show that there is a relationship between OSA and cardiovascular disease, including heart failure and other preliminary measures such as hypertension (Lombardi, Losurdo, Montano, Parati & Tobaldini, 2017). Lombardi et al. (2017) indicated that severe untreated OSA was linked to both fatal and nonfatal cardiovascular events and mortality rates. Heart failure rates in those with obstructive sleep apnea have increased from 10% to 20% (Lombardi et al., 2017). Floras (2017) has indicated that in 80% of heart failure patients with diminished or preserved left ventricular ejection fraction, they experience 5 apnea events per hour and that those likely may increase to an OSA rate of 61% in the future. The researchers found that in observational studies, which include heart failure therapy patients, there was a doubling rate of mortality risk that may occur if OSA is identified and presented (Floras, 2017).

According to Gillespie et al. (2015), the AHA assessed, the cost of treating heart failure in the U.S. in 2015 was approximately 44.6 billion, with other comorbidities identified. A study review by Ambrosy et al. (2014), performed a global review of heart failure characteristics, management, results, predictors, quality enhancement initiatives, regional differences, and limitations regarding available data. The researchers found that 26 million people globally suffer from heart failure, and 5.7 million people in the United States (Ambrosy et al., 2014). Ambrosy et al. (2014) has shown that heart failure was the leading cause of hospitalization, with over 1 million people hospitalized annually in Europe and the United States and over 670,000 initial cases a year. The researchers revealed that those admitted to hospitals for heart failure are between 70 and 75 years old

and have a standard deviation of 15 years. However, admission globally is based on the standard of living, and those in North America are admitted at an older age than patients in other countries (Ambrosy et al., 2014). The researchers indicated that heart failure admission in the United States affects females at 40-50% more than males compared to the rest of the world (Ambrosy et al., 2014). Ambrosy et al. (2014) has shown that African Americans (20%) and Hispanics (7%) appear to be affected more, at a younger age and higher prevalence of medical comorbidities than any other ethnicity. Though there have been recent developments in drug and device treatment for those with ambulatory heart failure, there has been a little progression in its management, which has contributed to low in-hospital mortality rates and high risk for readmission post-discharge (Ambrosy et al., 2014). In sum, the studies used to show the prevalence of both global and domestic OSA and heart failure and how the commonality of these factors has grown. The prevalence of this disease can also be seen across ethnicity, age, and sex. Therefore, the information provided by Ambrosy et al. (2014), Lombardi et al. (2017), and Floras (2017) has displayed a need to explore this growing epidemic in this study as a result of the number of potential lives it affects.

Race

In a study review by Clark, Ehlen & Paul (2016), it has been indicated that race plays a role in sleep-disordered breathing. Clark et al. (2016) haves suggested that African Americans experience a higher rate of sleep-disordered breathing than any other race, though there have been few studies that compare across different ethnic groups. A result from the few studies that have reviewed sleep-disorders across ethnic groups has

yielded under-diagnosis of sleep apnea in African Americans, and African Americans are afflicted with sleep apnea at younger ages than whites (Clark et al., 2016). Additionally, the researchers have indicated that the predictor of sleep apnea in African Americans has been both BMI and socioeconomic status (Clark et al., 2016). Lastly, African Americans develop OSA and cardiovascular diseases at a higher rate than whites (Clark et al., 2016).

Dudley &Patel (2016) have shown that within the disparities and genetic risk factors of OSA, it has been indicated that there was a racial disparity regarding African Americans and OSA. The researchers suggested that African Americans consistently have the highest OSA rate out of all ethnicities (Dudley & Patel, 2016). Within the Dudley & Patel (2016) study, results have indicated that there may be soft tissue and skeletal contributors specific to African Americans with OSA, such as the enlarged tongues. Other risk factors that have been linked to racial differences in African Americans include the level of obesity (Dudley & Patel, 2016).

Albert et al. (2017), have shown that there are racial disparities that exist for African Americans, including health literacy. Researchers show that incidence rates of failure are highest amongst African Americans and that they are the race most likely to be hospitalized and develop heart failure (Albert et al., 2017). The researchers indicated that African Americans are susceptible to an increased window of opportunity for clinical issues that contribute to other diseases, including socioeconomic status (SES), high salt intake, high caloric intake, and genetics (Albert et al., 2017). Lastly, Albert et al. (2017) have shown that there are some racial disparities that relate to African Americans having an increased likelihood to receive emergency room care and utilizing less medical

therapy when needed than whites. In sum, race was important in sleep-disordered breathing, which has in some cases, been underdiagnosed in African Americans causing the belief that African Americans experience an increased rate of OSA and cardiovascular disease more than any other ethnicity due to genetic disposition, health literacy, SES, and medical decisions.

OSA and Heart Failure on Race

Booth, Djavadkhani, & Marshall (2014) OSA has been identified as a form of sleep-disordered breathing. Obstructive sleep apnea is clinically divided into three subsections on the apnoea-hypopnea index, including mild 5-15/h, moderate 15-30/h, and severe >30/h. Untreated OSA may cause job impairment, vehicle accidents, weight gain, and memory problems (Bergerhenryent, 2017). Approximately one in five peoples have OSA. African Americans are the highest ethnic group affected, and of those of middle age, 9% of women and 25% of men are affected, and 38,000 cardiac deaths are attributed to OSA annually, including heart failure (Bergerhenryent, 2017).

Akinboboye et al. (2013), revealed there was a need for further research on the impact amongst OSA and cardiovascular disease, particularly in blacks. The purpose of Akinboboye et al. (2013) was to evaluate data that indicates the association of OSA and cardiovascular disease (including heart failure) and to assess it for the influence of racial differences. The researchers have indicated that previous studies have been conducted on non-black populations (Akinboboye et al., 2013). However, reviews from the researchers have suggested that there has been a significant prevalence of blacks with OSA under age 25 and older than age 65 more than other racial groups (Akinboboye et al., 2013). The

researchers have also indicated that OSA as a precursor to heart failure has been linked to hypertension to blacks and that blacks had a higher baseline level of blood pressure and oxygen desaturation, and AHI than whites. Akinboboye et al. (2013) have shown that while OSA and cardiovascular disease disparities continue, there were limited research studies that have investigated the role of OSA on CVD in minorities or on the interface of race on the association of OSA in those with increased cardiovascular risk.

In the prospective study by Badr, Pranathiageswaran, Rowley, & Severson (2013), the focus was on assessing if the severity and mortality of sleep apnea are higher in African American patients with OSAHS compared to whites. The researchers have performed a prospective study that included data from July 1996 to February 1999 on 512 patients (244 men and 228 women) greater than 18 years old. Badr et al. (2013) explored confounding variables, including gender, age, BMI, and comorbidities. Researchers have shown that out 340 African Americans and 172 whites of the same age, the apnea-hypopnea index was highest among African Americans under 39 years old and those between 50 to 59 years old, therefore indicating OSA was higher in African Americans than whites (Badr, Pranathiageswaran, Rowley, & Severson, 2013). Badr et al. (2013) suggested that race did not predict mortality, and all confounders were found to be significant and useful modifiers. The researcher also has displayed the general population and subcategories affected by the prevalence of obstructive sleep disorder and the potential racial disparities in cardiovascular disease in African Americans (including heart failure) in the United States. Badr et al. (2013) indicated that OSA contributes to mortality and specifies that increased mortality was higher in those with severe OSA.

The article by Abraham et al. (2013) displays the history of sleep-disordered breathing in heart failure. Researchers indicated that widely unrecognized commonality, an increased relationship of both OSA in heart failure relating to mortality and morbidity (Abraham et al., 2013). Abraham et al. (2013) also indicated the increased need for suspicion of heart failure and sleep-disordered breathing correlations, the need for optimal treatment for this population remains unclear, and the need for further study. In sum, OSA and heart failure on race has been indicated to be impactful on the lives of African Americans. However, there appears to be a limited amount of studies to quantify this pressing issue. Abraham et al. (2013) have also indicated a need to begin to fill the gap in the literature regarding the limited amount of studies related to the role of OSA on CVD in African Americans.

BMI and Heart Failure

Those whose BMI was considered overweight or obese have been identified as a factor that increases the risk of cardiac issues including heart failure (Balfour et al., 2015; Clark et al., 2014). Clark et al. (2014) indicated that the Framingham study had 581 white middle-aged to elderly patients that heart failure rose by 7% in women and by 5 % in men for every unit of BMI increased. Albert et al. (2018) looked to evaluate the influence of ethnicity on the relationship between BMI and heart failure mortality with ejection fraction and heart failure. Albert et al. (2018) indicated that heart failure affects 5.7 million people, and high BMI/obesity affects 35% of people in the United States. Albert et al. (2018) proposed that increased BMI levels are linked to lower mortality in heart failure patients. Researchers have indicated that minimal information is known about the

link between BMI and heart failure mortality prevalence among diverse patients (Albert et al., 2018). Lastly, Albert et al. (2018) indicated that minorities such as blacks and Hispanics had a higher obesity rate than any other ethnicities, potentially contributing to cardiovascular disease.

The study by Christensen et al. (2013) proposed to identify whether factors such as inflammation, endothelial dysfunction are associated with BMI and CHF. It has been indicated by researchers this study that cardiac natriuretic peptides including, BNP and NT-proBNP, are significant heart failure factors that increase impaired outcomes (Christensen et al., 2013). These markers have also been determined to be inversely associated with BMI and found to be high in patients with cardiac issues (Christensen et al., 2013). Additionally, Christensen et al. (2013) have indicated that low BMI was linked to a poor outcome relating to heart failure. Researchers have shown that increased NT-proBNP is a direct effect that was linked to the wasting process or loss of function seen in heart failure (Christensen et al., 2013).

Additionally, Aune et al. (2016) have shown that there was an association between those who are overweight, obesity, and heart failure. Researchers have focused on clarifying if the relationship amongst general and abnormal adiposity and the risk of heart failure (Aune et al., 2016). Aune et al. (2016) indicated that there was a strong correlation between BMI, waist circumference, and waist-to-hip ratio, and heart failure. Researchers argued that there was an observed relationship between African Americans, BMI, and heart failure that was observed; however, it requires further study (Aune et al., 2016). In sum, researchers have shown that BMI levels are linked to heart failure

mortality rate and poor heart failure outcomes and indicated a need to be assessed as a covariate BMI on heart failure in this study.

Smoking and Heart Failure

Bauer et al. (2012) have argued that there was an association between smoking and heart failure. Bauer et al. (2012) focused on assessing if there was an association between smoking status and incident heart failure risk on elderly participant health, aging, and body composition study. Participants were randomly chosen from zip codes in Pittsburgh, PA, and Memphis, Tennessee, from March 1997 to July 1998 (Bauer et al., 2012). The researchers utilized 2,125 participants over the age of 70 years, 54.2% whites, and 46.8% of minorities (Bauer et al., 2012). Researchers also indicated that of the participants, 221 were current smokers, and 739 were past smokers. Data has indicated after median 9.4 years of follow up, there was a higher significance rate in those that currently smoked, who had incidences of heart attacks than any other group. Those who currently smoked had 21.9 events per 1,000 persons of heart failure (Bauer et al., 2012). Additionally, smoking has also been shown to cause vascular wall inflammation, which may contribute to plaque buildup in cardiovascular diseases, including heart failure (Bernhard & Messner, 2014).

Benjamin et al. (2018) revealed that for blacks, there was a significant association with cardiac dysfunction. The researcher's focus in this study was to assess black participants who did not have a history of heart failure for coronary heart disease at the Jackson Heart Study (Taylor (2005) as cited in Benjamin et al., 2018). Benjamin et al. (2018) indicated that smoking was linked to carbon monoxide exposure and reported that

it increased the oxidative stress resulting in mitochondrial function, inflammation, damage endothelial function. Researchers have indicated that smoking was associated with higher BNP, inflammatory cytokines, and cellular death (Benjamin et al., 2018). Lastly, Benjamin et al. (2018) identified that in relation to blacks, cigarette smoking was a significant risk factor for heart failure symptoms such as left ventricular hypertrophy and systolic dysfunction.

Balfour et al. (2015) focused on identifying a risk prediction equation to utilize in primary care to identify patients at high risk for heart failure. The researchers used 6814 participants, between the ages of 48 to 84 years old, from 2000 to 2002, that were from various ethnicities, including African Americans (Balfour et al., 2015). After a 7.1-year follow-up, 176 participants developed heart failure. Those who developed heart failure had significant characteristics, including being male, elderly, African American, current smokers, diabetic, hypertensive, had high BMI, left ventricular ejection fraction and increased Nt-proBNP (Balfour et al., 2015). In sum, though the direct evidence for linking heart failure and smoking was unknown in various studies, there was a significant risk factor associated with smoking as it relates to heart failure. Therefore, the covariate of smoking should be assessed in this study.

Oral Devices and OSA

Obstructive sleep apnea is an epidemic that affects over 12% of adults in the United States (Watson, 2016). This disease causes gasps in breathing during sleep, and the repeated slowing down or stopping of oxygen from getting to the lungs due to a collapse of the airway (ALA, 2018). As a result of this lack of oxygen to the body, it was

associated with heart failure as a condition of significant OSA (Badr et al., 2013; Booth et al., 2014). OSA is a disease that has severe implications for those affected, and there have been several measures used to address this health concern to avoid cardiovascular disease, motor vehicle accidents, poor neurocognitive performance, and decreased mortality (Booth et al., 2014).

Oral appliance efficacy was chiefly defined as successful by apnea-hypopnea index reduction (Cistulli et al., 2014a). Apnea-hypopnea indexes in patients that are <5 (identified as OSA resolution), < 10 (very mild disease), or a percentage lessening that was clinically significant from baselines of typically 50% are generally considered successful (Cistulli et al., 2014a). Oral devices are developed as non-custom-made devices (Cistulli et al., 2014a). Custom made devices are cast created from dentition and bite registrations by a dentist (Cistulli et al., 2014a; United States Department of Veterans Affairs [USDVA], 2018). Long-term effectiveness of oral appliances has been shown to stabilize AHI after 1 to 4 years of use, and in those who continue use after five years, adherence increase to over 90% (Cistulli et al., 2014a). Researchers have shown that efficacy in some studies that gradual titration advancement occurs in three levels that consist of 2, 4, 6 mm of overnight oximetry occurring in 25%, 48%, and 65 %, while those with severe OSA have shown improvement by 75% (Cistulli et al., 2014a). The researchers have also revealed that from a review of collected studies that various oral appliance designs have yielded similar effects treating OSA and that two-piece appliance considered to improve comfort and wear ability, while more than 82% have chosen customized oral devices (Cistulli et al., 2014a). Cistulli et al. (2014a) indicated that data

has shown that CPAP and OSA testing yields similar results in trials, though there was higher adherence related to the oral appliance. Additionally, researchers suggest that there was a benefit in observation and randomized controlled trials that address cardiometabolic outcomes as a marker of cardiovascular risk with the use of both oral appliance and continuous positive airway pressure (Cistulli et al., 2014a).

OSA treatments have been used to address both OSA and heart failure (Kasai et al., 2014). Oral devices are a relatively new method of non-implantable non-invasive medical devices geared to reduce OSA symptoms such as oxygen saturation levels, blood pressure, and heart rate (Booth et al., 2014; Chervin et al., 2015). The gold standard method for treating OSA was the CPAP (Cistulli et al., 2014a). The CPAP was normally used independently during sleep in patients with OSA (Cistulli et al., 2014a). Though the CPAP was considered efficacious, it does not consistently result in better health outcomes (Cistulli et al., 2014a). Oral appliances have primarily been used as a secondary and often tertiary method in combination with CPAP use for non-compliant patients with sleep-disordered breathing. Oral devices are available in custom or non-custom made, and they can be controlled mechanically or remotely (Cistulli et al., 2014a). Oral appliances are divided into three categories: MAD, tongue restraining devices (TRD), and combination therapies. In sum, the above studies indicate the need for further and observational evaluation of oral devices such as MAD to be considered as a first-line treatment for those with OSA and possibly those with heart failure.

MAD

Mandibular attachment devices are oral devices similar to sporting mouth guards (Booth et al., 2014). Mandibular attachment devices are removable noninvasive devices fitted around the teeth by a dentist in an effort to move the lower jaw and tongue forward to increase airflow (Booth et al., 2014). The MAD has been recommended as a second-line of treatment for those who have mild to moderate OSA, or those who prefer MAD than a CPAP. In addition, MAD was also the recommended choice for those who have more severe disease and do not comply with the CPAP regimen (Marklund et al., 2012). The mandibular attachment device has often been called mandibular attachment splint, mandibular repositioning appliance, as well as mandibular repositioning device (Booth et al., 2014; Finkel Dental Forum 2015). This treatment has a history of effectiveness on the apnea-hypopnea index in this with OSA and displays up to 64% more compliance than the CPAP in some studies (Booth et al., 2014). The MAD has been indicated to improve vascular functioning, blood pressure, and reduce daytime sleepiness (Booth et al., 2014).

Tongue restraining devices are similar to the MAD and implanted in the user's mouth orally (Bajaj et al., 2017). The tongue restraining devices has a tongue suppression piece used to control and pull the tongue forward and are known to be less comfortable than MAD (Bajaj et al., 2017). Combination therapies consist of a custom-made MAD that was attached to a CPAP, however, was often used at a much lower pressure setting than regular CPAP use (AADSM, 2011). The mandibular attachment device is a commonly utilized sleep device, to open the airways by promoting breathing (Bajaj et al., 2017)

The study by Baldini, Ballanti, Cozza & Ranieri (2015) aimed to evaluate a 48-month long-term efficacy study of MAD in an adult patient with mild to moderate an OSA. The researchers used 28 (6 females and 22 male) participants from the Department of Orthodontics of the University of Rome Tor Vergata in Rome, averaging 52.2 years of age (Baldini, Ballanti, Cozza & Ranieri, 2015). Data was evaluated by using the polysomnography and anamnesis results. Baldini et al. (2015) indicated significant efficacy using MAD on those with OSA and that 80% of patients displayed a reduction in sleepiness, snoring, and sleep discomfort. Researchers have yielded stable effects and significant improvement in the long term 48-month study for patients suffering mild to moderate OSA.

The Cistulli et al. (2009) study aimed to assess the mechanism of action in a mandibular attachment appraising the outcome on patients who have obstructive sleep apneas' upper airway. This prospective design utilizes 69 mandibular attachment splint patients with OSA from a sleep disorder clinic performed during wakefulness. Cistulli et al. (2009) indicated that those who participated in the use of mandibular attachment use a titratable form, of which acclimation occurred over 6-8 weeks. In those that used the mandibular attachment, there was a significant reduction in the apnoea-hypopnea index from 27.0+14.7 events/h to 12.5 events/h. There was also an increased distance between the hyoid and the posterior nasal spine, and significant increases in total airway volume (Cistulli et al., 2009). Due to an increased volume of velopharynx, as well as lower anterior facial height hyoid, and a para pharyngeal fat pads being relocated away from the airway. Cistulli et al. (2009) revealed x-rays were used to gauge the change in airway

opening. Researchers in this study have shown the potential for improved airway by using MAD and the basis for potential biomarker improvement.

In the randomized crossover study by Bishop, Girvan &Verrett (2014), the focus was on determining treatment outcomes between two different mandibular appliances and patient preferences. Bishop et al. (2014) have carried out this study in the San Antonio Texas Veteran Affairs Hospital, using 24 subjects with OSA and compared two popular titratable MAD devices in the Klearway vs. TAP3. Researchers have revealed that 18 participants completed the study, and outcomes were measured using AHI and respiratory disturbance index (RDI), oxygen saturation, heart rate levels, and subjective feedback (Bishop, Girvan &Verrett, 2014). Bishop et al. (2014) indicated that there were no individual statistical differences between the two MAD devices. Nevertheless, TAP 3 means including scales such as the respiratory disturbance index, and oxygen saturation appeared lower than the Klearway device (Bishop et al., 2014). The TAP 3 elite results related to RDI improvement were the closest of the two devices to statistical significance, with $p = 0.559$ (Bishop et al., 2014). Bishop et al. (2014) have also shown that neither appliance proved significantly more effective over one another in either the AHI category of mild, moderate, or severe. There were 72.2% of participants displayed a preference for TAP 3 elite versus the Klearway device, indicating that its choice due to its comfort from minimal bulk and lack of hardware against the palate (Bishop et al., 2014).

Researchers have shown that the Veteran Health Care facilities have utilized the TAP 3 elite OSA device (Bishop et al., 2014; Department of Veteran Affairs [DVA], 2015b). In sum, MAD was an oral device used as a secondary treatment on those with

OSA or in combination with CPAP, if there was CPAP non-compliance. Researchers in multiple studies have shown that the Mandibular attachment devices were beneficial on OSA. There have been scarce studies in excess Akinnusi et al. (2013) that explored the benefit of MAD on cardiovascular disease and shown it to be beneficial. Of the mandibular attachment devices used, TAP3 has been shown to the most used and requested by patients with OSA.

Marklund et al. (2012) suggested there was limited information on the type of custom MAD that was most effective on OSA, in excess of titratability. Researchers further indicated that there was a greater need for knowledge about MAD outcomes related to cardiovascular issues and a need for longer-term studies (Marklund et al., 2012). Marklund et al. (2012) identified patients that may benefit from MAD treatment and its comorbidities to provide patients with optimal alternative therapies. In sum, the researchers displayed the purpose of the MAD, its functionality, the significance efficacy using MAD on those with OSA. Researchers have also shown that TAP 3 was the MAD most preferred by patients and was used by the Veterans Affairs (Marklund et al., 2012). Additionally, the gap in the literature suggests the possible benefit of MAD on cardiovascular diseases. Therefore, the information provided by the researcher supports that a quantitative design was needed to show MAD effectiveness.

CPAP

The CPAP is an electronic positive airway pressure device for addressing sleep apnea (Pinto & Sharma, 2018). This device forces air through the patient's airways, keeping the air passages to maintain constant breathing during sleeping hours (Pinto &

Sharma, 2018). This device was used in patients who do not require invasive ventilation; however, they have BMI and OSA concerns or in patients with heart failure (Pinto & Sharma, 2018). It was used to successfully extubate patients that might still benefit from positive pressure but who may not need invasive ventilation, such as obese patients with OSA or patients with congestive heart failure (Pinto & Sharma, 2018). Significant complications with CPAP consist of its long acclimation period resulting in irritation and uncomfortableness, induced claustrophobic tendencies, or patient embarrassment resulting in removal during use and noncompliance (Anderson et al., 2016; Pinto & Sharma, 2018). Compliance has also been found to be significantly less in African Americans regarding CPAP use more than whites (Anderson et al., 2016). Other concerns result in the participants experiencing congestion, nasal drainage, dry mouth, or nasal hemorrhages (Pinto & Sharma, 2018).

CPAP versus MAD

The Akinnusi et al. (2013) study aimed to assess long-term cardiovascular mortality in participants with OSA that utilize CPAP versus a MAD. Akinnusi et al. (2013) performed a non-concurrent study that used 570 participants from the Erie County Medical Center between February 2002 and January 2004. Researchers utilized four comparison groups consisting of a control group without obstructive sleep apnea, an untreated MAD group, a CPAP group, and the MAD group. Results indicated cumulative cardiovascular mortality in those treated with MAD ($P = 0.21$). CPAP ($P = 0.29$) was similar, and there was no significant difference between those treated with either MAD or CPAP (Akinnusi et al., 2013). However, the use of MAD did result in an AHI, mean

titration, and adherence that was higher than that of CPAP users. Akinnusi et al. (2013) showed that MAD was comparable and related to treatment. Moreover, the MAD has the potential for significant compliance, possibly resulting in the need for reconsideration as a primary method. The researchers assessed long-term cardiovascular mortality in participants with OSA that utilize CPAP versus MAD. Akinnusi et al. (2013) has also shown that MAD was a comparable OSA method, was potentially significant for compliance, and has a possible need for reconsideration as a primary method.

The article by Bratton et al. (2015) focuses on providing a meta-analysis comparing MADs to CPAP, placebo, and no placebo groups with alterations in both SBP and DBP. Data were gathered from several databases until August 2015, collecting a randomized clinical trial of 872 studies, containing over 4,888 patients. There were 51 reviews used in the final analysis. Treatment comparison was held for CPAP versus inactive control, MAD versus inactive control, and CPAP versus MAD while assessing biomarkers of the apnea-hypopnea index, oxygen-desaturation index, baseline blood pressure, length of follow-up, type of control and blood pressure measurement (Bratton et al., 2015). The researcher's results displayed that of the participants, both MADs and CPAP use showed a significant non-discriminatory reduction in blood pressure for both SBP and DBP. The Bratton et al. (2015) report displayed a meta-analysis of CPAP usage with sleep apnea related to a heart failure marker in blood pressure via BNP levels. Bratton et al. (2015) also reported that MAD (reducing PSBO by 2.1 mmHg, DBP by 1.9 mmHg) was comparable when affecting blood pressure versus CPAP (reducing SBP by 2.5 mmHg, DBP by 2.0 mmHg). Information provided by the researchers has suggested

that MAD was as effective as CPAP usage with Sleep Apnea related to a blood pressure via BNP levels.

Cistulli et al. (2013) focus was to assess if the effect of mandibular attachment therapy was similar to the health outcomes of CPAP in a short-term study. The researchers used a randomized crossover open-label non-inferiority trial with 126 participants in three Sleep Centers from Sydney, Australia, over the course of 1 month. The participants were 20 years old and older, familiarized with using both CPAP and MAD treatments after a two-week wash and re-acclimation phase. Cistulli et al. (2013) indicated that the use of MAD was neither inferior nor superior to CPAP results, neither treatment lessened baseline blood pressure, self-reported quality of life, nor daytime sleepiness. The researchers have provided information that is similar and favorable to results related to health outcomes such as neurobehavioral and quality of life within participants who suffer from mild to moderate OSA from both CPAP and mandibular attachment therapy and a need for further long-term comparative analysis. Cistulli et al. (2013) have also shown that not all patients with severe OSA respond well to MAD, though a sizeable minority does. Thus, MAD may be an appropriate strategy for severe OSA; it may require reconsideration and follow-up studies to assess its efficacy (Cistulli et al., 2013). The researchers have verbalized a need for a long-term review, more than one month, related to cardiovascular issues with mandibular attachment therapy (Cistulli et al., 2013).

CPAP use has been supported by the benefit of respiration leading to cardiovascular outcomes (Bauman et al., 2016; Eisele et al., 2015; Kasai et al., 2014).

Limited reports outside of Akinnusi et al., (2013), have supported the use of MAD relating to the potential of cardiovascular improvement. Akinnusi et al. (2013) aimed was to assess the long-term cardiovascular mortality in participants with severe OSA with CPAP or MAD. Over 570 participants with severe OSA were used in this study. Akinnusi et al. (2013) provided a non-concurrent cohort study that gathered data from 2002 to 2004 at the Erie County Medical Center in Buffalo, New York. The researchers indicated that after 3-month total participants dwindled to 208 participants, 177 used CPA, and 72 used fitted with MAD (Akinnusi et al., 2013). Among those treated with CPAP or MAD, the lower cardiovascular death rate was associated with users of the CPAP (0.56 per 100 person-years) closely followed by MAD (0.61 per 100 person-years), indicating no true difference in cardiovascular death rate (Akinnusi et al., 2013). Therefore, the Akinnusi et al. (2013) report has shown that MAD and CPAP may be equally effective in reducing the risk of cardiovascular events in those with severe OSA, and further studies are needed to affirm the results.

Information gathered throughout several studies discussed have indicated that CPAP use has been shown as an effective initial treatment for OSA when there was compliance. However, other methods such as MAD, which have a history of high compliance, have not explicitly been assessed for patients with OSA and heart failure (Abraham et al., 2013). In sum, the comparable measure between the CPAP and the MAD and the potential for a need for a longer duration to identify the effect MAD may have on cardiovascular disease. Therefore, the researcher provided by Akinnusi et al. (2013) and Cistulli et al. (2013) should be identified as a reason for the MAD to be

considered a testable variable to possibly reduce the symptoms of heart failure in those with OSA in this study.

CPAP versus MAD Economic Cost

Cistulli & Sutherland (2011) indicated that various oral appliance designs are simple to use, inexpensive, and have available techniques that can be applied in office-based settings. Side effects of oral appliances include increased salivation, mouth dryness, tooth pain, gum irritation, headaches, and temporomandibular joint discomfort, which decrease around the second month (Cistulli et al., 2014a). Bajaj et al. (2017) has shown that MAD was the cheapest treatment modality to address Sleep Apnea at a rate of 50-160 times cheaper than CPAP but equally as effective. The researchers have also suggested the cost of MAD internationally was approximately $1000 and was economically more efficient for both populations without electricity and those who cannot afford the high cost of CPAP (Bajaj et al., 2017). The American Sleep Association [ASA] (2018) has indicated that the CPAP cost an average of approximately $850 to $3000, in excess of continual electricity usage.

Gillespie et al. (2015) provided a comprehensive literature review of 106 studies on the healthcare, workplace, societal consequences, cost, and economic burden of prolonged OSA that goes untreated. Gillespie et al. (2015) also yielded data from different research studies that have noted that OSA has been associated with various health diseases, including heart failure. The researchers have also indicated that if OSA was left untreated, it can be medically severe and economically expensive (Gillespie et al., 2015). Though CPAP was considered the gold standard, compliance remains an issue

as well as the unit's utility usage cost in addition to the need for insurance coverage (Camacho, Certal & Riaz, 2015; Gillespie et al., 2015). Surgeries to address OSA via tracheostomies are useful; however, they have significant drawbacks relating to high morbidity, long recoveries, and little patient interest (Gillespie et al., 2015).

Untreated OSA may lead to a more reduced quality of life affecting the patient, their family at home, and potentially resulting in a 10% greater chance of workplace disability. However, the quality of life level was difficult to calculate or assess due to reduced productivity, absenteeism, health care cost, and potential liability in relation to the occurrence of accidents (Gillespie et al., 2015). Untreated OSA may lead to transportation consequences such as excessive daytime sleepiness, impaired awareness, focus, and potential accidents (Gillespie et al., 2015). Gillespie et al. (2015) have shown that 810,000 motor vehicle accidents resulted in 1400 deaths that cost approximately $15.9 billion, if 70% of those with OSA were treated and were compliant with an airway device like CPAP, 500,000 collisions would have been avoided (Davidson et al. (2004) as cited in Gillespie et al., 2015). This level of avoidance would have resulted in 1,000 lives saved, and the cost would be lowered by 11.1 billion (Gillespie et al., 2015). Undiagnosed patients with OSA are estimated to cost $1,950 to $3,899 per patient, per year times, the extrapolated number of 29.5 million people from the Wisconsin study. This study indicated that even if 60% remained undiagnosed, it would place a 34 to 69-million-dollar economic burden annually on the health care system (Dempsey, Palta, &Young, 1993 as cited in Gillespie et al., 2015). Those diagnosed with OSA, but who are untreated, have higher medical costs versus those receiving OSA treatment.

Those diagnosed who are treated are estimated to cost $2,700 to $5,200 less than those not receiving OSA treatment (Gillespie et al., 2015). Gillespie et al. (2015) has indicated that in some studies, CPAP has been more efficacious in addressing AHI than MAD. However, MAD has greater compliance. Additionally, CPAP is covered by insurance. However, MAD devices are not covered by insurance, leaving the patient to take the brunt of the cost as a deterrent due to the initial cost of the device versus its poor suggested efficacy (Gillespie et al., 2015). This approach possible extinguishing hope in care or alternative care options. The CDC (2016) indicated that heart failure occurs when the heart is unable to pump enough blood and oxygen to support an individual's other organs adequately, though severe, it does not mean your heart has stopped working. In sum, OSA and untreated OSA are economically costly and that CPAP can be both expensive and continual. However, MAD may be more reasonable and cost-efficient overall due to not requiring insurance, maintenance, or utilities.

Summary and Conclusion

In this chapter, I have found that higher a BMI level among African Americans leads to increased mortality and are found increasingly in African Americans in recent decades in the United States. Obstructive Sleep Apnea affects a significant number of the population, specifically African Americans, resulting in 38,000 annual heart failure deaths (Bergehenryent, 2017). Obstructive Sleep Apnea causes an increased AHI, which leads to oxygen desaturation, eventually altering heart rate and blood pressure and causing heart failure (Booth et al., 2014). Heart failure continues to rise and is affecting African Americans more than Whites and Asians (Colvin et al., 2014). The evidence

provided in the literature on the racial disparity regarding the mortality from OSA and heart failure affecting African Americans has shown to be significant (Badr et al., 2013). Therefore, smoking and BMI should be considered as covariates in this study.

Body mass index was significantly associated with an increase in heart failure (Clark et al., 2014; Del Gobbo et al., 2015). Heart failure was affected considerably by both past and current smokers causing impaired endothelial function and oxidative stress, which increases heart failure risk (Bauer et al., 2012). Obstructive sleep apnea treatments historically have been used to address heart failure (Kasai et al., 2014). Newer treatments to address OSA include oral appliances, which reduce oxidative stress levels, blood pressure, and heart rates (Booth et al., 2014). The oral appliance has used a lower pressure setting than CPAP; however, are similarly effective regarding AHI and compliance. Researchers have had limited studies that have addressed oral appliance effectiveness on heart failure, and few are suggesting its possible need for further study (Akinnusi et al., 2013; Cistulli et al., 2013). The MAD has been well received by patients and effective in the respiratory disturbance index. Researchers have been conflicted in various studies indicating that MAD was as effective in relation to CPAP use on OSA patients (Al-Shorman & Shydfat, 2015; Berge, Gjerde, Lehmann, Johansson, A. & Johansson, A. K., 2015; Cistulli et al., 2013). Biomarkers used to identify the effectiveness of heart failure symptoms have been indicated as left ventricular ejection function, SBP, DBP, oxygen saturation, BNP, NT-proBNP, and troponin levels (Bratton et al., 2015; Gaggin & Januzzi, 2015). Cost of MAD appears cheaper overall versus the CPAP due to requiring electricity usage. Those diagnosed with heart failure are likely to

be male, elderly, African American, smokers, and have high BMI (Balfour et al., 2015). The results of the demographics and characteristics of those with heart failure present the need for this study's African American male population over age 20 and its covariates. Chapter 3 provides information on the research method, research design and rationale, methodology, data collection, and threats to validity.

Chapter 3: Research Method

Introduction

Obstructive sleep apnea is a public health concern in the United States that repeatedly blocks the airway during sleep (NIH, 2017). The prevalence rate for OSA is approximately "10% among those age 30-49 and 17% among those 50-70" (Barnet et al., 2013, p. 1006). Surkin (2013) revealed that 71% of those diagnosed with cardiovascular disease have OSA. Obstructive Sleep Apnea has a direct association with heart failure (NIH, 2017; Surkin, 2013).

Heart failure is a common and highly fatal disease if left untreated, and mortality rates are 20 times higher for African Americans than any other ethnicity for those under the age of 50 (Blair, Huffman & Shah, 2013). The purpose of this study was to determine whether treatment using the MAD effectively impacts symptoms of heart failure in African American male veterans over 20 years of age who have been diagnosed with OSA and whether treatment efficacy differs after controlling for BMI and smoking status. In Chapter 3, the design and rationale are discussed. This chapter addresses the study variables and justification for research questions and the repeated measures design. The operationalization of the conceptual framework focused on translating the oxidative stress theory into a measurable outcome within the study. The methodology section includes the target population, sampling procedure methods, tools, and software used for analysis, handling of secondary archival data, and the operationalization of constructs. Chapter 3 introduced the data analysis plan and identified threats to validity and ethical concerns that may arise, as well as procedures applied in the study to address them.

Research Design and Rationale

This study used a quantitative nonexperimental longitudinal design. The study was to evaluate the level of mean change in terms of symptoms of heart failure as a result of MAD use, and therefore a quantitative design was most appropriate. A quantitative analysis was used to identify if there was a statistically significant impact on the study's dependent variables (ejection fraction, SBP, diastolic blood pressure, oxygen saturation, brain natriuretic peptide (BNP), n-terminal pro-brain natriuretic peptide (NT-proBNP), and troponins levels) in order to indicate if there was a correlation between MAD use and the effect on heart failure symptoms. This study also used a nonexperimental research method due to it not requiring the manipulation of the independent variable, there being no need for random assignment, and primarily relying on observation and interpretation of data from archived files (Hughes, Matt & O'Reilly, 2014; Price, Jhangiani & Chiang, 2013. This study employed the nonexperimental research method by using observations to identify if there were correlations between MAD and symptoms of heart failure. The longitudinal design was appropriate in this study due to it allowing for the dependent variables in a repeated measures design in a particular case to be followed over a period of time (Caruana et al., 2015). This design was most appropriate for this study due to it allowing for the assessment of relationships between risk factors and the progress of disease and effects of treatment over variable lengths of time (Caruana et al., 2015).

The repeat measures test relates to the study questions design when identifying if there was a significance in MAD use on the main risk factors of heart failure in African Americans when controlling such as BMI and smoking over time. The independent

variable in this study was the use of the MAD. The dependent variables in this study were symptoms of heart failure (ejection fraction, SBP, DBP, oxygen saturation, BNP, NT-proBNP, and troponins levels). Body mass index and smoking were covariates to indicate how they affect the outcome of the study. Wludyka (2012) has revealed that repeated measures studies have been used historically by epidemiologists to comprehend correlations between risk factors and the development of diseases as well as treatment outcomes over time. The repeated measures design was used as an effective way to assess samples of deidentified records involving MAD treatment and create a hypothesis regarding why a product or service delivery may or may not appear to correlate.

Epidemiologists have also used repeated measures studies to assess interventions and multiple measurements over various intervals of time for an independent subject (Wludyka, 2012). The quantitative repeated measures design can be useful to advance knowledge forward in epidemiology as it relates to the MAD as an alternative first-line treatment for those with heart failure. The repeated measures design was used because it required fewer records for the overall study and could detect effect size within a study with a smaller sample size (Jeyabalasingham et al., 2011; Statisticshowto, 2018b). This design was also chosen due to its high statistical power and quicker and more efficient results (Statisticshowto, 2018b). The rationalization for this design has been based on the purpose of the study to examine whether MAD therapy impacts symptoms of heart failure in African American male veterans diagnosed with OSA over a six-month timeframe. This study attempted to evaluate the level of mean change in symptoms of

heart failure as a result of MAD use, and therefore a quantitative study was most appropriate.

Researchers have recommended to further review the use of observational study designs to more closely analyze variables including MAD, BMI, and smoking (Arynchyn et al., 2009; Babson et al., 2013; Brenner et al., 2015). Additionally, past researchers have used cohort studies to assess a form of heart failure variable risk factors associated with mortality impact on patients (Akinnusi et al., 2103; Balfour et al., 2015; Bauer et al., 2012). Though a cohort study can be used to assess cardiovascular disease, it was not beneficial to this study due to there being no need for expensive follow-up of a large number of subjects for an extended time to address research questions (LaMorte, 2016). Berge et al. (2015) used a retrospective study to assess outcome developments of patient's MAD recipients, and the data provided was beneficial relating to significant effect at baseline from comorbidities. LaMorte (2016) also revealed that retrospective studies frequently have a lack of data on confounding factors if recorded in the past, create difficulty identifying the exposed cohort and comparison group, were subject to selection bias and if older records were used that have not been maintained the available data may be of poor quality. Popat and Sainani (2011) have also indicated that retrospective studies have possible low data quality and a lower level of generalizability in relation to cross-section studies due to the limited volume of hospitals, clinics, and the potential for a limited level of participation.

Other experimental study designs were not chosen because the research does not require participants or the altering of independent or dependent variables. Price et al.

(2013) have revealed that a quasi-experimental design allows for the manipulation of the independent variable or control the assignment of the treatment conditions. Quasi-experimental designs were not chosen due to there not being a need to manipulate variables or covariates. Experimental designs were not chosen because there being was no need for random assignment in this study. Moreover, an experimental design was not selected due to the study not requiring an overly expensive design for an issue that can be inexpensively addressed (Popat and Sainani, 2011). The experimental design was also not chosen due to it not always being generalizable, ethical, or feasible (Popat and Sainani, 2011). A repeated measures design was chosen because of its capability of assessing the prevalence of risk factors and outcomes over time (Chambers, Graeme, Hickey, Mokhles & Kolamunnage, 2018). Other benefits of this study may include its inexpensiveness, short turnover, output clarity, increased generalizability, and valid evaluations of the prevalence of risks (Chambers et al., 2018; Popat and Sainani, 2011). Additionally, the repeated measures design was linked to the research questions in this study due to its ability to assess changes in values of dependent variables and to evaluate if treatment with MAD effectively reduces heart failure symptoms in African American veterans with OSA and whether treatment efficacy differs by BMI and smoking status.

Methodology

In this section, I indicated how the study was implemented. This section includes a description of the population, sampling, sampling procedure, the sampling frame including the inclusion and exclusion criteria, power analysis, data collection, use of archival data, data analysis plan, threats to validity, and ethnic procedures.

Population

As of 2016, there were approximately 20 million veterans who were served nationally by the VHA Population Projection Model (DVA, 2016b). Data has shown that over 50% of Veterans live in 10 states within the United States, of which Florida was within the top three. Within the state of Florida, there were approximately 1.6 million veterans, including nearly 91% or 1,452,024 males and 09% or 142,193 females (DVA, 2016a). Of the Florida veteran population, there were approximately 180,315 black or African Americans (DVA, 2016a). The target population of this study was African Americans over the age of 20 years living in the United States. Study data from archived files include those registered at the VHA and diagnosed with OSA and heart failure. Data from archived files consisted of those who have been issued MAD and have polysomnography results indicating sleep apnea within the past year. The approximate Veteran population size consisted of 23 de-identified archived records.

Sampling and Sampling Procedures

Sampling is an essential method of selecting a representative part of a population for the purpose of determining characteristics of the entire population (Emerson, 2015; Statistics Solutions, 2017). Purposeful sampling was used to screen deidentified medical records for: African Americans registered with the VHA who are diagnosed with OSA and heart failure and were 20 years of age and older. Data regarding dependent and independent variables were assessed from medical problem lists, primary care doctor, nurse, respiratory, dental, cardiology notes, and labs. Those sampled were ages twenty and older, using MAD and have polysomnography results indicating sleep apnea within

the past year and six-month polysomnography test after receipt of the MAD. Other inclusion criteria consisted of de-identified records data from archived files on those who have been seen by VHA respiratory clinics and been referred to dental services from a MAD. Records were excluded if they did not meet the inclusion criteria. The approach of using non-probability sampling can be viewed as economical, though there were practical reasoning for its application (Dever and Valliant, 2014).

A benefit of the purposeful sampling strategy was its ability to target a population that meets the inclusion criteria, its accessibility, geographical proximity, and the fact that it has the secondary data needed for the study (Alkassim, Etikan & Musa, 2016). Variable data was downloaded from the VA VINCI onto a password encrypted isolated hard drive for storage. Study data to address mean changes in variables was computed using the VINCI's Statistical Package for the Social Sciences (SPSS)/PSPP (USDVA, 2016).

Sample Size/Power Analysis

Ergin, Kibar & Konak (2016) have shown that a G*Power analysis serves as a free and independent power analysis to conduct an estimated requisite sample size. A G*Power analysis incorporates multiple testing types, including an f-test, which was used in this analysis to appropriately calculate a generalizable sample (Statistics Solutions, 2018). According to G*Power, an appropriate significance level or (α) alpha error probability of 0.05, indicates that there was a 5% or less chance of a type I error rejecting the null hypothesis (G Power, 2017). Additionally, the lowest power probability of 0.80 indicates that there was an 80% chance or better that there would be no chance of type I error (G Power, 2017; Lakens, 2013; Taylor, 2011). The effect size was used to measure

the distance between the null and alternative hypothesis and in an f-test would be between the lower limit of 0.10, the medium limit of .25, and the largest limit of .40 (G Power, 2017). A commonly used effect size would be located at the middle limit, which would be an f of 0.25 (G Power, 2017).

Without appropriate Veterans Affairs data, it may be inappropriate to identify an actual expected number of records that meet the eligibility criteria for the study. Aaronson et al. (2015) using MANOVA and MANCOVA relating to OSA, and cardiac status issues have yielded effect sizes ranging from as low as .20 to as high as .80. Therefore, historically used values that were placed into the G power software to identify an appropriate sample size consisted of using a medium effect size of 0.25, an alpha error probability of 0.05, a power probability of 0.80, number of groups of 1, number of measurement (dependents) of 7 and correlation among measures 0.5 resulting in approximately 23 de-identified archived files to have at least an 80% power probability.

Table 1

Power Analysis

Options: F tests Manova: Repeated measures, within factors

Analysis: A priori: Compute required sample size

Input	Identified G-Power Measurement
Effect size f	0.25
α err prob	0.05
Power (1-β err prob)	0.80
Number of groups	1
Number of measurements	7
Corr among rep measures	0.5
Output	

(table continues)

Non-centrality parameter λ	20.1250000
Critical F	2.6986599
Numerator df	6.0000000
Denominator df	17.0000000
Total sample size	23
Actual power	0.8175114
Pillai V	0.4666667

Data Collection

Data were gathered via nonprobability purposeful sampling from archived from the Veterans Health Care Administration Dental Clinics records that met the diagnosis and inclusion criteria. Archived records from the Veterans Affairs agency were chosen due to the agency's ability to address Veteran needs. The Veterans Affairs mission is related to putting veterans first, by honoring the commitment of President Abraham Lincoln to care for the Veteran, his widow, and orphan, and balancing resources and the patients' needs (Dasheiff & Finn, 2009; DVA, 2015a). This study does not require or involve the recruitment of subjects as all information was collected from existing archived secondary data. The archived data utilized in this study from the Veterans Affairs electronic record was scrubbed of Patient Health Information (PHI) identifiers by the agency prior to receipt and used by the researcher.

The Veterans Affairs electronic record was the Veterans Integrated System and Technology Architecture (VISTA) based interface, which was used by VA clinicians called the Computerized Patient Record System (CPRS) (Brandt et al., 2017; Espadas et al., 2011). The electronic record included veterans who were served at VHA hospitals and Community-Based Outpatient Clinics (CBOCs) throughout the U.S. (Department of

Veterans Affairs Office of Inspector General, 2015). Computerized patient record system files incorporate all Veterans serviced within Veterans Affairs Hospitals. The Computerized Patient Record System (CPRS) has been used since the 1980s by the VA and identifies patients serviced in the various independent disciplines, units, and clinics within the local veteran affairs agency (Agha et al., 2014; Espadas et al., 2011). Storage of this electronic patient data was updated nightly from VISTA/CPRS and was contained in VA VINCI, which was an integrated national database and toolkit for the analysis of sensitive healthcare data in a secure computer environment (Brandt et al., 2017; USDVA, 2014).

Data for this study were captured after receiving approval from the Walden University Internal Review Board (IRB) #09-11-18-0235165. Once approval was gained, this study was submitted to the Gainesville, Florida Veterans Affairs office of Human Research Protection Process (HRPP). I have contacted the Veterans Affairs office of HRPP and have been notified that I was unable to receive a signed letter of cooperation from the VA granting authority to this researcher to access the needed study information until the proposal was completed and it had been approved by Walden University IRB. This researcher completed document requirements then submitted the request for review by the Veterans Affairs office of HRPP. The HRPP encompassed approvals by VA identified Internal Review Boards, Research and Development, and National Data Systems. Requirements to gain access to the veteran affairs data consisted of completing documents such as the: conflict of interest, abstract form, responsible request and project information sheet, research and development information system investigator data, safety

cover sheet, medical center support form, new study submission coversheet, data management and access plan, protocol form, scope of practice for research staff form, data access request tracker application, VHA research addendum form, authorization for use and release of individually identifiable health information collected for VHA research, and the research protocol safety evaluation form.

Once approval was provided by the Veterans Affairs HRPP, this researcher logged onto the secured VINCI site on a dedicated North Florida South Georgia VA computer and specified the search under the dental department. De-identified data sets were accessed from VINCI by performing a query on records that meet the inclusion criteria. Data from collected records were observationally screened for appropriate inclusion criteria indicated in the study. Collected data was then uploaded, and statistical analysis was performed via VINCI's SPSS/PSPP. The analysis for the study research questions were addressed through the rMANOVA and rMANCOVA tests. Data highlighted and accessed was primarily based on the independent variable, the use of MAD on African American male veterans. The dependent variables in this study were the symptoms of heart failure (ejection fraction, SBP, DBP, oxygen saturation, BNP, NT-proBNP, and troponins levels). Identified covariates in this study include BMI and Smoking status. Additionally, as a result of the accessing data from archived secondary data, informed consent was not needed, nor was there a need to provide follow-up or exit interviews. Storage and analysis of data were housed in a VINCI workspace. Once the statistical analysis has been completed, statistical reports would be downloaded to this researcher's password encrypted external hard drive and was stored in a secured room

within the VA, behind a locked door, and inside of a locked file cabinet of which this researcher has the only key. Data results were transferred from this secured password encrypted hard drive and displayed as usable research analysis within this study to be potentially displayed in a medical journal (i.e., PubMedCentral) or a Veterans centered journal within one year of the studies completion.

Use of Archival Data

Secondary data is a reanalysis of data that has been collected for an alternative purpose (Cheng & Phillips, 2014). Data were accessed from the North Florida South Georgia Veterans Affairs hospital after I obtained VHA IRB and Walden University IRB approval. Data were provided by the VHA Office of Research and Development's VINCI. Data were downloaded onto an isolated and password encrypted computer hard drive, then uploaded into SPSS/PSPP. Time to collect secondary data and the cost of acquiring data was minimal in comparison to primary data collection methods, and the data was usually cleaned by professional staff.

Instrumentation and Operationalization of Constructs

The secondary archival data used in this study to ascertain de-identified veteran's medical data was collected by the VINCI database from the VA. The VINCI has collected data from the Veterans Affairs agency since 2000 (Agha et al., 2014). The VINCI was a database warehouse that allows research to facilitate the analysis of VHA data in a private and data secured setting (USDVA, 2014). The VINCI can be accessed remotely through a desktop from anywhere within the VA network inside of its own project site (USDVA, 2014). This database program allows the use of datasets through

structured queries language (SQL) queries and extractions from VISTA/CPRS and was compatible with SPSS/PSPP (USDVA, 2014). This database was not a published instrument and was not publicly available and requires approval from the Veterans Affairs Internal Review Board.

BNP. BNP levels have been indicated in three scale levels, from those <100pg/ml were considered ordinary, ruling out heart failure (Mangla, 2014). Those between 100pg/ml to 400pg/ml warranted further investigation, due to there being a 95% chance of heart failure and in those greater than 400pg/ml, heart failure was likely (Mangla, 2014).

Diastolic blood pressure. Pressure levels have been indicated in three scale levels: normal < 80 mmHg, at-risk (prehypertension) 80-89 mmHg, and high ≥90mmHg (CDC, 2017d).

Ejection fraction. The left ventricle is the main chamber pumping oxygenated blood through to the body. Ejection fractions were typically measured only in the left ventricle and have been indicated in two scale levels 55 percent < were considered normal, and 50 percent > were considered reduced/borderline (Mayo Clinic, 2018b).

NT-proBNP. NT-proBNP levels have been indicated in scale levels including those who were <125/ pg ml aged 0-74 years old, and <450pg/ml aged 75-99 years old were considered normal. Those over 450pg/ml for those under 50 years old, and > 900pg/ml for those over 50 years old were considered unstable (Cleveland Clinic, 2018b).

Oxygen saturation. Normal oxygen saturation scale levels were considered

between 95%-100%, those between 94%- 91% require evaluation and treatment, and those who have less than 90% were considered to have a low oxygen saturation level leading to hypoxemia indicating the patient was having a clinical emergency (WHO, 2011).

Systolic blood pressure. Pressure levels have been indicated in three scale levels: normal < 120 mmHg, at-risk (prehypertension) 120-139 mmHg, and high ≥140mmHg (CDC, 2017c).

Troponins. Those with troponin levels < 0.03 were considered normal, those with levels between 0.03-0.09 were considered to have a medium risk or unstable angina, and those 0.1 were thought to indicate a myocardial infarction (Alosert et al., 2009).

Instrumentation and Operationalization of Variables

Heart failure. Heart failure symptoms were assessed as dependent variables by reviewing significant heart failure markers. Heart failure markers consisted of left ventricular ejection function (LVEF), SBP, DBP, oxygen saturation, BNP, NT-proBNP, and troponins levels for appropriate measurement indications (Balfour et al., 2015; Gaggin & Januzzi, 2015; Gilliespie et al., 2015). These markers were observationally assessed from test results of secondary data using observed medical notes from a physician, nursing, and cardiology notes, labs, and consults.

Table 2

Symptoms of Heart Failure

Symptom	Level of Measurement	Source of data
left ventricular ejection function	Ratio	(Anavekar et al., 2012)

(table continues)

systolic blood pressure	Ratio	(Chin, 2016)
diastolic blood pressure	Ratio	(Chin, 2016)
oxygen saturation	Ratio	(Gastel et al., 2016)
BNP	Ratio	(Clavel et al., 2016)
NT-proBNP	Ratio	(Enseleit et al., 2012)
troponins levels	Ratio	(Enseleit et al., 2012)

OSA. Obstructive sleep apnea was observationally assessed from secondary data by verifying OSA ICD-10 code diagnosis found in the medical problem list of the VA electronic medical record, which has been inputted as a diagnosis by a licensed physician.

BMI. Body mass index levels within archived files were observationally assessed from secondary data using medical notes. BMI categories were assigned according to guides established by the CDC. The CDC (2017a) has identified those less than 18.5kg/ m^2 as underweight, those 18.5-24.9 kg/ m^2 as normal, those 25-29.9 kg/ m^2 as overweight and those equal to or greater than 30kg/ m^2 as obese. BMI level data from archived files were observationally assessed in research question two as a covariate using medical notes. The rMANCOVA was used due to its ability to potentially meet all the assumptions, therefore, increasing validity and reliability.

Smoking. Smoking status data within archived files was observationally assessed in research question three. This study used archived records that included nicotine labs from secondary medical data. The covariate smoking was coded using 0 for no and 1 for yes. The rMANCOVA was used due to its ability to potentially meet all the assumptions, therefore, increasing validity and reliability (Lund and Lund, 2013b).

Data Analysis Plan

A correlational design using repeated measures within factor study was obtained from secondary data collected through VHA, due to the inexpensiveness, accessibility, availability of the data. Benefits of the repeated measures within factor was that it eliminates individual differences and has a test type that has high sensitivity (Nimmo-Smith, 2009). Other benefits of this study design include the ability to conduct research with few records available while allowing statistical inference to be made with data from archived files, and its ability to allow for the study to be conducted quicker and with greater efficiency (Jeyabalasingham et al., 2011). Lastly, this study type allowed for the researcher to monitor the changes in records over time.

The data analysis for this study consisted of descriptive statistics and pre-data screening procedures to eliminate and address all missing data. Descriptive statistics were used to describe the sample data obtained from VHA records. The reported statistics included frequency counts, measures of variability, and measures of dispersion.
The pre-data screening consisted of assessing for missing data and testing statistical assumptions. Missing data was assessed through visual scanning of the data and frequency counts by this researcher. The amount of missing data determines what actions to take with regard to the missing data. Screening for missing data allows for this researcher to identify missing values in a dataset to reduce the possible hindrances in the outcome of a study (Kang et al., 2013). Screening for missing data was important because the lost data could cause bias in the estimation of parameters (International Business Machines, n.d.a.; Kang et al., 2013). It was important to screen for missing data as

incomplete study values can provide misleading results (International Business Machines, n.d.a.; Kang et al., 2013). Missing data could also reduce statistical power (Hawkes, Mendelson, Su & Yan, 2016; Kang et al., 2013). Lastly, missing data can complicate theories by not being able to use complete cases (International Business Machines, n.d.a.; Kang et al., 2013). Testing statistical assumptions were important before conducting statistical procedures because it validates the test being used to ensure accurate results (Lund & Lund, 2013a; Lund & Lund, 2013b).

The rMANOVA analysis was deemed to be valid when the seven assumptions were met, which include that two or more dependent variables were continuous. Variables were assessed as continuous variables if they were interval or ratio (Lund and Lund, 2018). Another measure was that the independent variable possesses two or more categorical related groups (Lund and Lund, 2018). Groups were considered related when they were measured at all time points or all receive treatment (Lund and Lund, 2018). This assumption was addressed by the measurement of MAD use at the three different monthly intervals.

Additional measures were to have an average sample size. This assumption was addressed by using a Gpower analysis to determine the appropriate sample size. This assumption was addressed by having a larger number of records in each group than the number of dependent variables (Lund and Lund, 2018). Other fitting measures of appropriateness were that there were no significant univariate or multivariate outliers. This assumption was assessed by using z-scores of $\pm$3.29 to identify extreme univariate outliers (Steyn, 2018). The Mahalanobis distance calculation was used to suggest

possible multivariate outliers (Garson, 2012; Lund and Lund, 2013a; Pennsylvania State University, 2019). Also, a measure of appropriateness was that there was multivariate normality. This assumption was assessed by using the Q-Q plots to assess the differences in the data from a normal distribution and Shapiro-Wilk test of normality on each dependent variable for each of the group's independent variable (Lund and Lund, 2018; Statistics Solutions,2019a). This test was recommended for studies with small sample sizes up to n=2000 (Garson, 2012).

Moreover, another measure was that there was a linear relationship between each dependent variable for each group of the independent variable (Lund and Lund, 2018; Statistics Solutions, 2013). This assumption was assessed by using a Pearson correlation r table to assess the linear relation between variables (Kent State University, 2019; StatTrek, 2019). Another measure was that there were one or more continuous covariates. Variables were assessed as continuous variables if they were interval or ratio (Lund and Lund, 2013b). Lastly, another measure was that there was no multicollinearity. This assumption was assessed by evaluating the Pearson correlation. A study by Lee et al. suggests that values between 0.5 and 0.8 suggest no multicollinearity. Several researchers have proposed that greater than 0.8 were considered to have multicollinearity (Lee et al., 2016; Negreiros, 2018). As a result, VIF values greater than 0.9 were considered evidence of multicollinearity between variables.

The rMANCOVA analysis was a form of the RM analysis of variance (ANCOVA) considered appropriate when the four assumptions were met, which include that there was homogeneity of variance and covariance (Griffith, 2015; Mertler &

Vannatta, 2002; Statistics Solutions, 2013). The homogeneity of covariance was assessed by using a Box M test of equality of covariance to test whether two or more covariances were homogenous (Garson, 2012; Statisticshowto, 2018a). Homogeneity of variances was tested with Mauchly's test of sphericity (Horn, n.d.). Moreover, another measure was that there was a linear relationship between each dependent variable for each group of the covariate variable (Lund and Lund, 2018; Statistics Solutions, 2013). This assumption was assessed by a Pearson correlation r table to assess the linear relationship between variables (Kent State University, 2019; StatTrek, 2019). Lastly, another appropriate measure was that there must be multivariate normality (Griffith, 2015). This assumption was assessed by using the Q-Q plots to assess the differences in the data from a normal distribution and the Shapiro-Wilk test of normality on each dependent variable for each of the group's independent variable (Lund and Lund, 2013b). This test was recommended for studies with small sample sizes up to n=2000 (Garson, 2012).

RQ1: Does use of MAD therapy have a statistically significant effect on the symptoms of heart failure in male African American veterans diagnosed with OSA?

$H_0 1$: Use of MAD therapy does not have any statistically significant effect on the symptoms of heart failure in male African American veterans diagnosed with OSA.

$H_a 1$: Use of MAD therapy does have a statistically significant effect on the symptoms of heart failure in male African American veterans diagnosed with OSA.

The independent variable for this research question was the use of the MAD. The dependent variables were measures of heart failure symptoms (ejection fraction, SBP, DBP, oxygen saturation, BNP, NT-proBNP, and troponins levels). Changes in symptoms

of heart failure were assessed by looking at the changes in the data at the second, fourth, and sixth month follow up after treatment with the MAD began. The rMANOVA procedure was used to test the follow-up measures of heart failure and determine if the use of MAD has a statistically significant effect on heart failure symptoms in male African American Veterans with OSA. The use of the rMANOVA allowed me to assess the magnitude of changes in values for the seven dependents variables while controlling for the inflation of Type I error. Independent and dependent data from archived files were evaluated using scatterplots and correlation tables. A scatterplot was used to test the assumption that there was a linear relationship between each dependent variable and the independent variables (Lund & Lund, 2013a). Correlation tables were used to identify the significance of the change in dependent variables means.

RQ2: What is the moderating effect of BMI on the use of MAD therapy for treating symptoms of heart failure in male African American veterans diagnosed with OSA?

$H_0$2: BMI levels do not have a statistically significant moderating effect on the use of MAD therapy for treating symptoms of heart failure in male African American veterans diagnosed with OSA.

H_a2: BMI levels do have a statistically significant moderating effect on the use of MAD therapy for treating symptoms of heart failure in male African American veterans diagnosed with OSA.

The independent variable in the study was the use of the MAD. The dependent variables were the measures of heart failure symptoms (ejection fraction, SBP, DBP,

oxygen saturation, BNP, NT-proBNP, and troponins levels). The covariate was the level of BMI. The rMANCOVA was used to examine whether BMI moderates the impact of MAD therapy on symptoms of heart failure. The rMANCOVA allowed me to assess the magnitude of changes in values for the seven dependent variables while controlling for the inflation of type I error. Incremental analysis reviews were performed at the end of the second, fourth, and sixth months of the study to compare study results. Independent and dependent data from archived files were evaluated using a scatterplot and correlation table. A scatterplot was used to test the assumption that there was a linear relationship between dependent variables, independent variables, and between the covariate BMI, as well as each of the dependent variables, and the independent variable (Lund & Lund, 2013b). Correlation tables were used to identify the significance of the change in dependent variables.

RQ3: Does smoking status have a statistically significant moderating effect on the use of MAD therapy for treating symptoms of heart failure in male African American veterans diagnosed with OSA?

$H_0 3$: Smoking status does not have a statistically significant moderating effect on the use of MAD therapy for treating symptoms of heart failure in male African American veterans diagnosed with OSA.

$H_a 3$: Smoking status does have a statistically moderating effect on the use of MAD therapy for treating symptoms of heart failure in male African American veterans diagnosed with OSA.

The independent variable in the study was the use of the MAD. The dependent variables were the outcomes of heart failure symptoms (ejection fraction, SBP, DBP, oxygen saturation, BNP, NT-proBNP, and troponins levels). The covariate was smoking. The rMANCOVA was used to examine whether smoking status moderates the impact of MAD therapy on symptoms of heart failure levels at the end of the second, fourth, and sixth months of the study to compare study results. The rMANCOVA allowed me to assess the magnitude of changes in values for the seven dependent variables while controlling for the inflation of Type I error. Independent and dependent data from archived files were evaluated using a correlation table and scatterplot format. A scatterplot was used to test the assumption that there was a linear relationship between dependent variables, independent variables, and between the covariate smoking, as well as each dependent variable and the independent variable (Lund & Lund, 2013b). Correlation tables were used to identify the significance of the change in dependent variables.

Threats to Validity

External validity describes the ability for the findings to be generalizable to the target population (Crawford and Khorsan, 2014). Heart failure affects 5.7 million people in the United States (CDC, 2016). Casey et al. (2012) has shown that OSA affects between 26-36% of veterans, due to there being a relative sample size it may aid in the external validity of the study. This study was a repeated measures study using a purposeful convenience sample of African Americans over the age of 20, using MAD, who have been diagnosed with OSA and heart failure within the past year. Threats to the

external validity may include the fact that there cannot be a cause-effect relationship in this study design, however, that there may be correlation pending the sample was large enough to be representative (Carlson and Morrison, 2009). Another threat to external validity may include purposeful convenience sampling due to the potential selection of records (Dudovskiy, 2019). Though the number of African American veterans using MAD who have heart failure in this sampling method was unknown, theoretically, to minimize this threat, the best technique was to focus on the distinctive characteristics of the target population. Therefore, to minimize these threats, there was one researcher to review the data to verify that the actual data needed for the study was present and that to ensure no missing data values and to identify possible misclassification.

Patten (2012) has revealed that threats to external validity applying to the secondary data in this study include: (a) interaction effects of selection and experimental variables, (b) specificity of variables, (c) and multiple treatment interferences. Interaction effects of selection and experimental variables indicate that certain groups may be more affected by treatment due to the makeup of the group (Rommel-Esham, 2010). The study results regarding African American Veterans may not be applicable to other demographics or females as a result of other patients not having the benefits of VA healthcare access, and the opportunity for alternative treatment coverage. Specificity of variables occurs when the variables were so unique to the study that it lessens the ability to generalize (Georgia Southern University, n.d.). De-identified data used in the study from archived files may be affected due to the receipt of MAD in a sterile healthcare environment, possibly being quite different from the natural home environment in which

the device was used during sleep. Patten (2012) has indicated that multiple treatment interferences occur when there was more than one treatment given to a group, due to it potentially having the opportunity to effect each other. Multiple treatment interferences may consist of records having other medical impairments or carryover effects to the treatment being offered in this study that cause counter interactions that make it hard for results to be associable. Additionally, archived records used in the study from the target population may also involve other health conditions (diabetes, cancer, etc.) or treatments that may cause a simultaneous form of impairment to the study. However, it was hard to generalize the conflict of simultaneous forms of health condition or treatments being received by the study population because the researcher has no control and cannot control for extraneous variables.

Threats to internal validity include maturation and mortality (Flannelly et al., 2018). Maturation results in bodily changes that occur over time within the study (Flannelly et al., 2018). Throughout the course of the study, results may indicate a maturation change in BMI, smoking, and level of severity of heart failure symptoms (Flannelly et al., 2018). Mortality indicates any failure of data from archived files that do not to complete the study (Flannelly et al., 2018). Due to the longevity of the study, rates of attrition may occur as a result of death, non-compliance, and drop out. To address this concern, queries in VINCI were ran to identify the number of records at the beginning of the study and to determine the percentage of attrition within the target population.

Threats to conclusion validity may be due to the random heterogeneity of respondents, which addresses diversity among respondents and determining the

likelihood if a relationship exists. This validity concern may be present due to only having African American Veterans in this study, which may result in it not being generalized to the overall population. In sum, this study does not address the female sex or other ethnicities in excess of African Americans. An effort to improve conclusion validity has been implemented by having good statistical power in the study over .80. The current study power in this study was 0.8175114.

Ethical Procedures

Ethical epidemiological practice was maintained in this study. Data collected from secondary data provided by the VA agency did not require informed consent. I, as the primary data collector of secondary data, required approval from both the Walden University IRB and VA identified IRBs. Additionally, VA HRPP face-to-face training, VA via University of Florida's IRB-01 Health Insurance Portability and Accountability Act (HIPAA) for Research, local IRB training, the NIH Office of Extramural Research Protecting Human Research Participants, and the Collaborative Institutional Training Initiative (CITI) training certificates were completed by me before handling any personal healthcare information as a measure of data security. Once approval was gained, I was able to review de-identified data in VINCI. All data, however, was scrubbed and redacted for personal identifying information to make anonymous in relation to name, social security number, address, phone numbers, physician names, and other safeguards for those records who were utilized. Linkage of data was associated via a codex, followed by gender, ethnicity, and categorical diagnosis. Data collected was housed on a password encrypted isolated hard drive and only displayed and calculated via the VINCI's

SPSS/PSPP. Due to the use of de-identified data, I was the only researcher in this secondary data study, and there was no need for dissemination of PHI due to no identifying marks or interventions activities for refusal or withdrawal. Therefore, upon completion and publication of this study, data will be stored for a minimum of five years before being deleted and destroyed to prevent any accidents or lapses in security that would allow any PHI to escape (University of Florida, 2014).

Summary

This study used a quantitative repeated measures design to assess if there was a significant reduction in heart failure symptoms through MAD use among African American Veterans with OSA who were age 20 or older and whether treatment efficacy differs in terms of BMI and smoking status. This study uses secondary data collected from the VHA to address the three research questions. Chapter 4 includes the statistical results of the data for this study using SPSS/PSPP.

Chapter 4: Results

Introduction

This quantitative correlational study was conducted to determine whether MAD therapy impacts symptoms of heart failure among African American male veterans diagnosed with OSA. The independent variable consisted of the use of the MAD. The dependent variables were symptoms of heart failure (SBP, DBP, oxygen saturation, BNP, NT-proBNP, and troponins levels). Data analyzed in this study were obtained from the VA VINCI database and performed using a correlational design. Data analysis was conducted through a repeated measures test and calculated using SPSS version 25. The rMANOVA and rMANCOVA analysis were used to test the hypotheses in this study. The research questions and associated hypotheses were:

RQ1: Does use of MAD therapy have a statistically significant effect on the symptoms of heart failure in male African American veterans diagnosed with OSA?

$H_0 1$*:* Use of MAD therapy does not have any statistically significant effect on the symptoms of heart failure in male African American veterans diagnosed with OSA.

$H_a 1$*:* Use of MAD therapy does have a statistically significant effect on the symptoms of heart failure in male African American veterans diagnosed with OSA.

RQ2: What is the moderating effect of BMI on the use of MAD therapy for treating symptoms of heart failure in male African American veterans diagnosed with OSA?

$H_0 2$*:* BMI levels do not have a statistically significant moderating effect

on the use of MAD therapy for treating symptoms of heart failure in male African American veterans diagnosed with OSA.

H_a2: BMI levels do have a statistically significant moderating effect on the use of MAD therapy for treating symptoms of heart failure in male African American veterans diagnosed with OSA.

RQ3: Does smoking status have a statistically significant moderating effect on the use of MAD therapy for treating symptoms of heart failure in male African American veterans diagnosed with OSA?

H_03: Smoking status does not have a statistically significant moderating effect on the use of MAD therapy for treating symptoms of heart failure in male African American veterans diagnosed with OSA.

H_a3: Smoking status does have a statistically moderating effect on the use of MAD therapy for treating symptoms of heart failure in male African American veterans diagnosed with OSA.

The rMANCOVA analysis was used to examine whether the use of MAD therapy impacted measures of symptoms of heart failure levels in the second, fourth, and sixth months of the study. The rMANCOVA allowed me to assess the magnitude of changes in values for the dependent variables while controlling for the inflation of type I error. Chapter 4 includes the results of the completed data analysis. This chapter addresses the data collection process, results of data analysis, discussion of results, evaluation of statistical assumptions, and discussion of the statistical analysis.

Data Collection

Data used in this quantitative nonexperimental longitudinal study were supplied by the VHCA's VINCI program. In this study, secondary data was used, and it did not require recruitment or response rates. Data were initially requested for this study from the VA VINCI on April 9, 2019. The VINCI program data was provided to me by an assigned VA approved and certified principal investigator. The assigned VA principal investigator completed all VA-required training between July 24 and August 17, 2018, to certify her ability to handle secondary data from the federal government. Initially, I requested data on the Orlando Veterans Affairs Healthcare system and the North Florida/ South Georgia Veterans Affairs Healthcare system from the North Florida/South Georgia Veterans Affairs Healthcare systems and was notified by the VA that this request was too specific, and would not yield more than a small number of results due to the MAD not being provided at every station. Therefore, I was provided data from veterans' healthcare facilities across the United States of America that used the MAD between 2005 and2019. Data has been queried by VINCI from SQL, converted to Excel, sent to the assigned VA approved principal investigator, and supplied to me on the initial date of June 5, 2019. I was provided access to the data through a designated VA computer within a designated workspace, using a VA- encrypted username and password login so that I could review the data related to the study.

The data I received from the VA principal investigator was in Excel format and contained the following information: station location, race, sex, date of birth, OSA diagnosis, OSA diagnosis date, MAD receipt date, dependent variable dates collected and

dependent variables data from the second, fourth, and sixth months relating to ejection fraction, SBP, DBP, oxygen saturation, BNP, NT-proBNP, and troponins levels. Data by the VA approved principal investigator did contain PHI in the form of a unique identifier via the patient-internal control numbers (ICN) with no real or scrambled social security numbers. The data set was supplied to me without any code to connect the ICN to the patient. Once I received the data set, I immediately analyzed it for the appropriate population, geographic location, correct focus of study, and appropriate variables. I then scrubbed it of its PHI relating to the Patient ICNs using the anonymization technique of suppression. Suppression is a technique to reduce the risk of identification for participants by removing identifying markers and assigning it an appropriate variable (Benschop, Machinguata & Welch, 2018; Nayaki, Simi & Sudheep, 2017). The ICN data was suppressed by deleting the ICN column and providing each set of data in that column with an individual variable identification number in SPSS to de-identify the data set. Data collected was then observationally screened for accuracy and appropriate inclusion criteria relating to study variables. All data that did not meet the study criteria were excluded from the study, including non-African Americans, OSA diagnosis dates, heart failure diagnosis dates, dependent variables dates, and cases containing insufficient data due to not being relevant to the study questions.

There were 1634 cases initially provided in the data sample. Of 1634, 23 cases were stricken from the study due to not meeting the criteria related to ethnicity. Within the 1611 cases remaining, 1582 cases were removed, due to insufficient data across the variables and intervals of interest for the study.

Within the 29 cases, I utilized the compute variables box and a syntax code of COMPUTE age = CTIME.DAYS(formdate-birthdate)/365.25 to convert the given date of birth with the date the data was received to calculate a given age for each case. Verification of age conversion uniformity consisted of going into the variable view tab and verifying that the date box was set to date, month, and year (dd.mm.yyyy). Calculated ages were used to represent cases of African American male veterans who used the MAD device. A means imputation was performed within the 29 cases, due to each case having variations of missing data within the dependent variables and the covariate of BMI. This method was used to assign a scale mean on the missing data within dependent variables to "maximize the data collected and minimize the effects of the missing data" (Harris, 2013, p. 90). According to Harris (2013), this approach does not modify the value of the data because it inputs scale means that do not alter the general mean; however, it reduces the number of dropped cases from the analyses.

Repeated measures MANOVA assumptions were assessed observationally for adequate sample size, dependent variables measured as ratio levels, and the independent variable consisted of two or more categorical independent groups. Other rMANOVA assumptions were assessed through SPSS statistical analysis testing. The rMANOVA assumptions consisted of identifying if there was multivariate normality and determining if there were linear relationships between each pair of dependent variables within each group of the independent variables. Additional rMANOVA assumptions were comprised of verifying there were no univariate or multivariate outliers, and that there was no multicollinearity.

The rMANCOVA assumptions were assessed by observationally inspecting that two or more dependent variables that were measured in interval or ratio levels, and that the one independent variable consists of two or more categorical, independent groups. Further rMANCOVA assumptions consisted of visually assessing that one or more covariates were continuous variables and that there was independence of observations. Other rMANCOVA assumptions were assessed through SPSS statistical analysis testing which included determining if there were linear relationships between each pair of dependent variables within each group of the independent variable, and determining if there were linear relationship between the covariate and each dependent variables within each group of the independent variable. Lastly, additional rMANCOVA assumptions consisted of assessing the homogeneity of variances and covariances, multivariate normality, determining there were no significant univariate outliers in the independent variable groups in term of each dependent variable and no multivariate outliers in the groups of the independent variables in terms of each dependent variable. Data that met the study's inclusion criteria were then statistically analyzed in SPSS using the rMANOVA to assess the magnitude of changes in values for the seven dependent variables relating to heart failure while controlling for the inflation of type I error. Data that met the study's inclusion criteria were also statistically analyzed in SPSS using the rMANCOVA to examine whether BMI and Smoking moderates the impact of MAD therapy on symptoms of heart failure and to assess the magnitude of changes in values for the seven dependent variables while controlling for the inflation of type I error.

Discrepancies

The discrepancies in the data collection plan differed from the method identified in chapter 3 in several ways as a result of the VA requirements, access to data, and access to data analysis tools. Initially, in Chapter 3, I did not include the use of a principal investigator due to being notified by the VA that one may not be needed. However, a VA trained, and certified principal investigator was indeed required, assigned, and provided by VA. The VA principal investigator was educated on the study, its structure, and its needed data components and provided monitoring of the study data. Initially, my study criteria included data gathered within the past year. Thought the initial data review timeframe was from 2016-2017, as a result of a combination of data not being available, limited sample size, and need for inclusion criteria to be met, I was provided data from the last 14 years from 2005-2019. Within this data, useable data from the timeframe of 2016-2017 was not sufficient or applicable. There were a few populations' discrepancies, which were included in the study. A discrepancy was that I was not able to gather data primarily from the Veterans Affairs Healthcare systems due to being notified by the VA that the request was too specific and would not yield more than a significantly limited set of results due to the device not being widely used at every station. I, however, was provided data from Veterans Health Care facilities across the country that were trained and able to utilize this device.

Other discrepancies related to polysomnography results indicating sleep apnea within the past year was changed as a result of notification from VA that these tests were utilized in several ways including community outsourcing through community care,

watches, and overnight polysomnography test all would be difficult to capture longitudinally, and therefore the OSA diagnosis of ICD-9 code 327.23 and ICD-10 code G47.33 were used. Data containing PHI in the form of Patient ICN with no real or scrambled social security numbers and no connecting code were provided to me from the VA Principal Investigator; however, it was anonymized via suppression. Originally, I indicated that archived data utilized in this study would be scrubbed of Patient Health Information (PHI) identifiers by the agency prior to my receipt and used by the researcher. Additionally, a password encrypted hard drive was not needed due to VA providing an isolated password encrypted computer that logged onto a secured site of which data was not able to be removed, printed, downloaded, or saved from the VINCI workstation. Moreover, the PSPP software was not able to be used in this study as an analysis tool in VINCI due to it not being able to perform rMANOVA or the rMANCOVA analysis. Therefore, SPSS was provided by the VA.

Further discrepancies were related to several additional forms, processes, and documents needed to gain access to the data from the HRPP. The HRPP encompassed approvals by Gainesville, Florida, VA, and identified the following entities: the Internal Review Board from the University of Florida, the VA WOC program, VA Research and Development, and the VA National Data Systems. Requirements to gain access to the Veteran Affairs data consisted of completing forms such as the: Gainesville's VA academic affiliation agreement with Walden University, face-to-face VA research SharePoint training, and the completion of the North Florida / South Georgia SharePoint form. Additional VA requirements consisted of completing training such as the:

Collaborative Institutional Training Initiative (CITI) on human research for good clinical practice, CITI on VA human subject's protection training, CITI on VA office of research and development biosecurity, government ethics training, ethics most wanted, VA privacy and information security awareness and rules of behavior, and VA privacy and HIPAA.

VA documents required to gain access to data consisted of completing forms such as the: VA abstract, authorization form for use and release of individually identifiable health information collected for VHA research, certification of licensure form, coversheet subcommittee on research safety, data management and access plan (DMAP), data use agreement (DUA) between VHA and federal entity, privacy, confidentiality, and information security source checklist for research. Additional documents that required completion in order to gain access to the VA data were the declaration for federal employment form, DUA VHA limited data set, education verification form, HRPP memo research staff only, HRPP new study submission sharepoint introduction training, human research subject protection, human subject research pre-review checklist. Continual documents needed per VA to gain data access were the employment verification form, new investigator form, privacy review, privacy security data, protocol form, research and development information system investigator data, research financial conflict of interest statement, research protocol safety evaluation form, safety cover sheet, scope of practice, VA10-9012 if protocol used a drug, and VA and Walden affiliation agreement.

Requirements from the Gainesville, Florida VA Without Compensation (WOC) program also consisted of completing: a packet checklist, appointment position

description, appointment patient contact statement, appointment experience working with animals, appointee intellectual property agreement, appointment mandatory training instruction, appointment information manager and personal identity verification sponsor. Additional required documents needed to gain WOC status needed to gain access to the VA data were the certification of licensure, VA declaration of federal employment, ethics most wanted training, intellectual property agreement, information system security officer (ISSO) review/approval letter, myIRB study submission form, personal resume, real social security number access form, registration or bar membership form, scope of practice for research staff, scope of practice for CPRS access, and scope of work form. Continual documents needed for WOC were the VA clinical studies center education verification form, VA national rules of behavior form, verification of license and certifications, VA talent management system training, VA assigned VA principal Investigator, VA privacy HIPAA training, VA privacy and information security awareness and rules of behavior training, without compensation application, and the Department of Homeland Security employment eligibility verification.

Training needed from the University of Florida consisted of the completion of the myIRB SharePoint form and training that included CITI human research -good clinical practice, CITI VA human subjects protection, CITI mandatory IRB training-biomed, CITI VA ORD biosecurity training, completing virtual private network (VPN) access, completed associate affiliation assurance, IRB01 local training, IRB01 local training refresher, NIH extramural education and UF HIPAA and privacy – research, and UF gatorlink registration. The following documents were needed to gain access to VINCI:

the ISSO referral letter, VINCI registration, complete data access request tracker (DART) application, research request memo, research study IRB approval letter, research and development committee approval letter. Additional completed documents needed for VINCI were the IRB approval of waiver of HIPAA-compliant authorization, research protocol, corporate data warehouse (CDW)-domain checklist, real SSN access request, vital status rules of behavior, national data system (NDS) data use agreement, research privacy review checklist, receive VA VINCI link and password, VINCI assigned sequel assistant, and CDW request forms prior to receiving access to data.

After approval to access the data was provided by the VA office HRPP, I logged onto the provided secured VINCI site on a dedicated VA computer. Additional changes in this study consisted of the study sample changing from de-identified data to a limited data set with no real or scrambled social security numbers that have limited PHI in the form of non-identifiable ICN. Data was accessed from VINCI by performing a query on records that meet the inclusion criteria. Additional data was excluded from the study data set due to not meeting the inclusion criteria and not being relevant to the study analysis. Excluded data that did not meet the study criteria included the following: dates of diagnosis for OSA and heart failure, and dates of collection for symptoms of heart failure, BMI, and smoking status. Furthermore, several dependent variables were removed from the study due to not having sufficient data across the 2^{nd}, 4^{th}, and 6^{th} monthly intervals, including ejection fraction, BNP, proBNP, and troponin. The remaining three dependent variables include SBP, DBP, and oxygen saturation. Lastly, within the covariate of smoking status, a means imputation was not able to be performed using this variable due

to inconsistent data, there being no uniform depiction of smoking type within the variable and the mean imputation yielding undistinguishable half measures across the 2nd, 4th, and 6th month interval.

Demographics

Table 4 includes the archived records of 29 African American male Veterans, from 16 VA health care systems, nationally, who were between the ages of 43 to 84 years old. The independent variables consisted of data collected at the 2nd, 4th, and 6th month on the use of the MAD. The dependent variables consisted of symptoms of heart failure (SBP, DBP, oxygen saturation). The covariate was BMI.

Results

Descriptive Statistics

Demographic data. Table 3 presents a summary of descriptive statistics for the demographic data. The mean age documented in the records was 62.83 years, and the standard deviation as 8.97 years. The ages ranged from 43 to 84 years of age.

Frequency counts were performed to acquire baseline descriptive statistics on the demographic data and data provided within the sample data set. There were 29 valid list wise and archived African American male Veterans records. The frequency counts in Table 4 shows that the most frequently occurring age was 66 years and older.

Table 3

Descriptive Statistics

(table continues)

Black or African American	N	Range	Minimum	Maximum	Sum	Mean	Std. Dev.
age	29	41	43	84	1822	62.83	8.969
Valid N (listwise)	29						

Table 4

Frequency Statistics for Age

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	under 45	2	6.9	6.9	6.9
	46-55	2	6.9	6.9	13.8
	56-65	10	34.5	34.5	48.3
	66-and older	15	51.7	51.7	100.0
	Total	29	100.0	100.0	

Table 5 presents a summary statistic for the data collected on dependent variables at the 2nd, 4th, and 6th months after the use of the MAD began. Data collected in the 2nd month shows the range of SBP levels were from 107 mmHg to 165 mmHg, with an average SBP level amount of 127.32 (SD = 14.597. Diastolic blood pressure levels ranged from 58 mmHg to 121 mmHg, with an average DBP level of 76.95 (SD = 11.159). Oxygen saturation levels ranged from 93% to 99%, with an average oxygen saturation level of 97.00 (SD = 1.035). The BMI levels ranged from 26 kg/m2 to 46 kg/m2, with an average BMI level amount of 34.89 (SD = 4.028).

Also exhibited in Table 5, data collected in the 4th month, SBP levels ranged from 105 mmHg to 171 mmHg, with an average SBP level amount of 133.18 (SD = 12.084). The most frequent SBP level amount was 133 mmHg. Diastolic blood pressure levels

ranged from 60 mmHg to 95 mmHg, with an average DBP level amount of 81.53 (SD = 7.026. The range of oxygen saturation levels were from as low as 86% to as high as 100%, with an average oxygen saturation level amount of 96.92 (SD = 2.337. Systolic blood pressure levels ranged from 114 mmHg to 156 mmHg, with an average SBP level amount of 136.73 (SD = 9.155). Diastolic blood pressure levels ranged from 51 mmHg to 97 mmHg, with an average DBP level amount of 80.60 (SD = 8.451. Oxygen saturation levels ranged from 94% to 100. Lastly, the range of BMI levels was from 27 kg/m2 to 46 kg/m2, with an average BMI level of 34.07 (SD = 3.360).

Table 5

Summary Descriptive Statistics for Dependent Variables across Bimonthly Intervals for 6 months

Variable	N	Min	Max	Sum	Mean	SD
Systolic Blood Pressure						
2 months	29	107	165	3692	127.31	14.59
4 months	29	105	171	3862	133.17	12.08
6 months	29	114	156	3965	136.73	9.15
Diastolic Blood Pressure						
2 months	29	58	121	2231	76.94	11.15
4 months	29	60	95	2364	81.52	7.02
6 months	29	51	97	2337	80.60	8.45
Oxygen Saturation						
2 months	29	93	99	2813	97.00	1.03
4 months	29	86	100	2811	96.91	2.33
6 months	29	94	100	2824	97.36	.93
BMI						
2 months	29	26	46	1012	34.88	4.02
4 months	29	20	46	998	34.39	4.04
6 months	29	27	46	988	34.07	3.35

Testing Statistical Assumptions

Sample size. Within both the rMANOVA and rMANCOVA assumptions, 23 cases were considered to have an adequate sample size according to the G*Power analysis, which was calculated on a medium effect size of f = 0.25, α err prob = 0.05, Power (1-β err prob) = 0.80. This sample size was met due to there being 29 cases utilized in the study, with no missing cases.

Independent variables. The independent variable in this study was the use of the MAD, which was present in two or more independent groups, i.e., in the 2nd, 4th, and 6th month, therefore, this assumption was met.

Dependent variables. All the dependent variables in this study were on a ratio level of measurement; therefore, this assumption was met.

Covariates. This study did have one covariate in BMI. The covariate BMI was presented as a continuous ratio variable in this study in three different monthly interval measurements in the 2nd, 4th, and 6th months. Therefore, this assumption has been met.

Outliers. SPSS produced a z-score table used in assessing the z-scores of $\pm$3.29 to identify extreme univariate outliers (Steyn, 2018). In Appendix A, the SPSS produced a z-score table, that shows five outliers identified ranging from as low as -4.67 to as high as 3.96. Due to there being no universal best choice to address the outliers that exist in the Z-score table, I performed a visual inspection to determine the nature of the outliers (Pum, 2019). Results from the visual inspection that all suspected values were truly possible values for the variables included in the data set. Therefore, the univariate outlier assumption was met.

In addition, mahalanobis distance was used to test for multivariate outliers in the data (Statistics Solutions, 2019b). The mahalanobis distance statistic was a common, sensitive, and robust method for detecting multivariate outliers (Deng, Jiang, Li, L. & Li, X., 2019). It was based on the Rocke estimator, by using the threshold set in the chi-square distribution using a $p<.001$ to identify a critical value (Deng et al., 2019). According to the chi-square value table, the maximum allowable critical value for the Mahalanobis distance for 3 variables was 16.266 (Palay, 2016). It can be suggested that there were outliers within the set of data if the identified critical value result was more than the maximum critical value of 16.266.

In Appendix B, the residuals statistics table shows that the Mahalanobis distance mean was 8.690 (SD=7.004), while the maximum critical value was 23.021. Due to the outlier assumption being violated, therefore, a commonly used correction was executed. I performed a visual inspection of the dependent variables in the data for appropriateness (Beaton et al., 2019). Results have shown that all possible outliers were within the normal range for dependent variable data. Therefore, the assumption for no multivariate outliers was met.

Linear relationships. According to Lund and Lund (2018), the linear relationship between each dependent variable for each group of the independent variable was needed for the assumption to be valid. Due to the correlation tables' ability to assess the degree to which a relationship is linear, a scatterplot was not used in this linear relationship analysis (Lund & Lund, 2018). The test assessed linear relationships between heart failure symptoms, i.e., SBP, DBP, oxygen saturation, and MAD use via the use of a

Pearson Correlation table (Kent State University, 2019; StatTrek, 2019). The Pearson correlation *r* was used when both variables were measured on an interval or ratio scale to indicate the magnitude and direction of a linear relationship between variables (Kent State University, 2019; StatTrek, 2019). According to NAME OF AUTHOR (YEAR), Pearson correlation r values were between -1 and 1. Additionally, the further the Pearson correlation *r* value was from zero, the stronger the linear relationship between two variables was considered (University of Texas, 2015).

In Table 6, a Pearson's r test was computed to assess the relationship between the independent variable of MAD use at 2^{nd}, 4^{th}, 6^{th} months on the dependent variables of SBP, DBP, and oxygen saturation. The sample size for all variable samples were n=29. There appears to be a statistically significant, positive linear relationship between 2-month SBP and 2-month DBP (r=.661, p=.000), 4-month SBP and 4-month DBP (r= .693, p=.000), 4-month SBP and 4-month oxygen saturation (r= .576, p=.001), 4-month DBP and 4-month oxygen saturation (r= .607, p=.000), and 6-month SBP and 6-month DBP (r=.662, p=.000). Overall, there appears to be a significantly strong linear relationship between MAD use and SBP, DBP, and oxygen saturation. Therefore, indicating that as one variable increased, the other increased.

Table 6

Correlations

		@2moSystolic_BP	@2moDiastolic_BP	@2moO2	@4moSystolic_BP	@4moDiastolic_BP
@2moSystolic_BP	Pearson Correlation	1	.661**	.118	.105	.096
	Sig. (2-tailed)		.000	.541	.586	.620
@2moDiastolic_BP	Pearson Correlation	.661**	1	.164	-.171	.007
	Sig. (2-tailed)	.000		.396	.376	.970
@2moO2	Pearson Correlation	.118	.164	1	-.093	-.069
	Sig. (2-tailed)	.541	.396		.630	.721
@4moSystolic_BP	Pearson Correlation	.105	-.171	-.093	1	.693**
	Sig. (2-tailed)	.586	.376	.630		.000
@4moDiastolic_BP	Pearson Correlation	.096	.007	-.069	.693**	1
	Sig. (2-tailed)	.620	.970	.721	.000	
@4mo_O2	Pearson Correlation	-.121	-.074	.031	.576**	.607**
	Sig. (2-tailed)	.532	.702	.874	.001	.000
@6moSystolic_BP	Pearson Correlation	.191	.068	.118	.274	.238
	Sig. (2-tailed)	.320	.724	.543	.150	.213
@6moDiastolic_BP	Pearson Correlation	.083	.090	.079	.170	.213
	Sig. (2-tailed)	.668	.644	.683	.378	.266
@6mo_O2	Pearson Correlation	.166	.176	.198	-.208	-.009
	Sig. (2-tailed)	.390	.362	.304	.278	.962

(table continues)

		@4mo_O2	@6moSystolic_BP	@6moDiastolic_BP	@6mo_O2
@2moSystolic_BP	Pearson Correlation	-.121	.191	.083	.166
	Sig. (2-tailed)	.532	.320	.668	.390
@2moDiastolic_BP	Pearson Correlation	-.074	.068	.090	.176
	Sig. (2-tailed)	.702	.724	.644	.362
@2moO2	Pearson Correlation	.031	.118	.079	.198
	Sig. (2-tailed)	.874	.543	.683	.304
@4moSystolic_BP	Pearson Correlation	.576**	.274	.170	-.208
	Sig. (2-tailed)	.001	.150	.378	.278
@4moDiastolic_BP	Pearson Correlation	.607**	.238	.213	-.009
	Sig. (2-tailed)	.000	.213	.266	.962
@4mo_O2	Pearson Correlation	1	.019	.003	-.082
	Sig. (2-tailed)		.923	.990	.673
@6moSystolic_BP	Pearson Correlation	.019	1	.662**	.360
	Sig. (2-tailed)	.923		.000	.055
@6moDiastolic_BP	Pearson Correlation	.003	.662**	1	-.095
	Sig. (2-tailed)	.990	.000		.625
@6mo_O2	Pearson Correlation	-.082	.360	-.095	1
	Sig. (2-tailed)	.673	.055	.625	

**. Correlation is significant at the 0.01 level (2-tailed).
*. Correlation is significant at the 0.05 level (2-tailed).
a. Cannot be computed because at least one of the variables is constant.
b. *All Dependent Variables Sample N=29*

In Table 7, among the 2-month BMI correlation, there appears to be a positive significant linear relationship with 2-month SBP (r= .359, p= .056). In the 4-month BMI correlation, there appears to be a positive linear relationship with 4-month SBP (r=.601, p=.001), 4-month DBP (r=.492, p=.007), and 4-month oxygen saturation (r= .660, p=.000). Lastly, in the 6-month BMI correlation, there appears to be a positive linear relationship 2-month SBP (r= .428, p= .020), 4-month SBP (r=.461, p=.012), and 6-month SBP (r=.470, p=.010). Overall, due to all values being between -1 and 1, there appears to be a significant linear relationship between MAD use and BMI on SBP, DBP, and oxygen saturation. Overall, the linear assumption was met.

Table 7

Correlations

		@2moBMI_1_1	@4mo_BMI_1_1	@6mo_BMI_1_1
@2moSystolic_BP_1	Pearson Correlation	.359	.195	.428*
	Sig. (2-tailed)	.056	.310	.020
@2moDiastolic_BP_1	Pearson Correlation	.160	.001	.024
	Sig. (2-tailed)	.406	.995	.901
@2moO2_1_1	Pearson Correlation	-.028	.157	.088
	Sig. (2-tailed)	.886	.416	.652
@4moSystolic_BP_1	Pearson Correlation	.175	.601**	.461*
	Sig. (2-tailed)	.364	.001	.012
@4moDiastolic_BP_1	Pearson Correlation	-.051	.492**	.096
	Sig. (2-tailed)	.792	.007	.620
@4mo_O2_1_1	Pearson Correlation	.045	.660**	.011
	Sig. (2-tailed)	.817	.000	.954
@6moSystolic_BP_1	Pearson Correlation	.320	.205	.470*
	Sig. (2-tailed)	.090	.285	.010
@6moDiastolic_BP_1	Pearson Correlation	.242	.163	.361
	Sig. (2-tailed)	.205	.397	.054

(table continues)

@6mo_O2_1_1	Pearson Correlation	.027	-.082	-.082
	Sig. (2-tailed)	.889	.673	.672
@2moBMI_1_1	Pearson Correlation	1	.368*	.632**
	Sig. (2-tailed)		.050	.000
@4mo_BMI_1_1	Pearson Correlation	.368*	1	.486**
	Sig. (2-tailed)	.050		.008
@6mo_BMI_1_1	Pearson Correlation	.632**	.486**	1
	Sig. (2-tailed)	.000	.008	

Multicollinearity. In Tables 7 and 8, I assessed the lack of multicollinearity assumption by evaluating the Pearson Correlation. Research has shown that Pearson correlation values up to 0.8 suggest no multicollinearity, and those greater than 0.8 were considered to have multicollinearity (Lee et al., 2016; Lund and Lund, 2013a; Negreiros, 2018). According to the data in correlations tables, there were no correlations 0.8, indicating no multicollinearity. Overall, the assumption of lack of multicollinearity has been met.

Multivariate normality. Due to having a sample size under n=2000, the test used on this assumption was the Shapiro-Wilk test of normality on each dependent variable for each of the group independent variable (Garson, 2012; Lund & Lund, 2013a). In Table 8, according to the Shapiro-Wilk test for normality of the three dependent variables and one covariate, all have a significance level less than .05, indicating that there was a non-normal distribution and that the assumption was violated (Northern Arizona University, n.d.). Due to the violation of multivariate normality, a known correction has been performed. According to Mordkoff (2016), it has been suggested that "N's of 10 or more were almost always enough to correct for any problems," such as outliers. This correction

was considered sufficient even though the unproven sample sizes around 30 have been suggested to be a rule of thumb to meet the central limit theorem (Mordkoff, 2016). Therefore, multivariate normality was appropriate.

Table 8

Tests of Normality

	Kolmogorov-Smirnov[a]			Shapiro-Wilk		
	Statistic	df	Sig.	Statistic	df	Sig.
@2moSystolic_BP_1	.259	29	.000	.820	29	.000
@2moDiastolic_BP_1	.257	29	.000	.792	29	.000
@2moO2_1_1	.362	29	.000	.693	29	.000
@4moSystolic_BP_1	.253	29	.000	.873	29	.002
@4moDiastolic_BP_1	.259	29	.000	.877	29	.003
@4mo_O2_1_1	.431	29	.000	.507	29	.000
@6moSystolic_BP_1	.296	29	.000	.833	29	.000
@6moDiastolic_BP_1	.328	29	.000	.822	29	.000
@6mo_O2_1_1	.362	29	.000	.707	29	.000
@2moBMI_1_1	.305	29	.000	.806	29	.000
@4mo_BMI_1_1	.293	29	.000	.736	29	.000
@6mo_BMI_1_1	.293	29	.000	.777	29	.000

a. Lilliefors Significance Correction

Due to the conservative nature of the Shapiro-Wilks test, it has been indicated that it should not be the lone determining factor of normality (Northern Arizona University, n.d.). Additionally, understanding what multivariate normality looks like was important. Quantile-quantile (Q-Q) plots, located in Appendix C-N, were used to address the differences that the variables may have in relation to a normal distribution (Statistics Solutions, 2019a). Research suggests that Q-Q plot curve interpretations should be identified as one of six categories: normally distributed, convex (skewed left), concave

(skewed right), convex-concave (heavy-tailed), concave-convex (light-tailed), or a bimodal (separated cluster) distribution (Ruppert, 2011).

Another Q-Q plot displayed in Appendix C at 2-months SBP appears to be a positive skewed concave-convexed distribution with several outliers between 120-140 and 160-170. Within the Q-Q plots in Appendix D at 2-months, diastolic blood pressure appears to be a positively skewed convex-concaved distribution with one outlier between 120-125. The Q-Q plots in Appendix E at 2-months oxygen saturation appears to be a positively skewed convex-concaved distribution with one outlier between 93-94.

The Q-Q plots in Appendix F at 4-months SBP appear to be a positively skewed convex-concaved distribution with several outliers between 100-105 and 165-175. Also, the Q-Q plot in Appendix G, at four months diastolic blood pressure, appears to be a positively skewed convex-concaved distribution with several outliers between 60-67. Within the Q-Q plot in Appendix H, at 4^{th} months oxygen saturation appears to be a positively skewed convex-concaved distribution with several outliers between 80-85 and possible 93-95.

Also, within the Q-Q plot in Appendix I, at 6-months SBP appears to be a positively skewed convex-concaved distribution with several outliers between 115-120, 120-125, and 135-140. Furthermore, the Q-Q plot in Appendix J at 6-months diastolic blood pressure appears to be a positively skewed convex-concaved distribution with several outliers between and 50-53. Lastly, the Q-Q plot in Appendix K at 6-months oxygen saturation appears to be a positive skewed convex-concaved distribution with two outliers between 94, 97, and 100.

In Appendix L, the Q-Q plot at 2-month BMI appears to be a positively skewed convex-concaved distribution with several outliers between 25-28, 33-35, 35-38, and 44-46. Within Appendix M, the Q-Q plot at 4-month BMI appears to be a positively skewed convex-concaved distribution with several outliers between 19-20, 30-35, 35-38, and 45-47. Lastly, in Appendix N, the Q-Q plot at 6-month BMI appears to be a positively skewed convex-concaved distribution with several outliers between 25-27, 34-38, and 46-48. Overall, multivariate normality does appear violated. Therefore, the corrective action of performing a visual inspection was implemented, and all assumed outlier values were identified as possible values and included in the data set (Ramzan, Ramzan, & Zahid, 2013). Therefore, the multivariate normality assumption was met.

Homogeneity of Variance and covariance. The test was initially noted to be evaluated by using the Box's M test of Equality of Covariance Matrices, however, due to it not computing and there being fewer than two non-singular cell covariance matrices, the Levene's test of equality of error variances was used. According to Garson, p-values in the Levene's test was considered significant at <.05 (Garson, 2012). The Levene's test of equality of error variances result has shown a non-statistically significant result for all the dependent variables indicating homogeneity of variance and covariance except for 4 months diastolic blood pressure p-value of .004, and 4-months oxygen saturation which had a p-value of .005 (Northern Arizona University, n.d.). Overall, results show that two variables indicate statistically significant outcomes. Therefore, the significance of these results has shown a noticeable difference between the variable variances. Due to multivariate normality being violated, commonly used corrections have been reviewed,

and studies have suggested changing to a robust Levene's significance level of <.001 (Allen & Bennett, 2008; Gastwirth, Gel, & Miao, 2009; Han & Zhu, 2014). Therefore, correcting the number of significant results to zero, meeting the homogeneity of variance and covariance assumption.

Table 9

Levene's Tests of Equality of Error Variance

	F	df1	df2	Sig.
@2moSystolic_BP_1	.861	3	25	.474
@2moDiastolic_BP_1	1.145	3	25	.350
@2moO2_1_1	1.547	3	25	.227
@4moSystolic_BP_1	.980	3	25	.418
@4moDiastolic_BP_1	5.656	3	25	.004
@4mo_O2_1_1	5.496	3	25	.005
@6moSystolic_BP_1	2.391	3	25	.093
@6moDiastolic_BP_1	1.485	3	25	.243
@6mo_O2_1_1	2.375	3	25	.094

Tests the null hypothesis that the error variance of the dependent variable is equal across groups.[a]

a. Design: Intercept + @2moBMI_1_1 + @4mo_BMI_1_1 + @6mo_BMI_1_1

Results

RQ1: Does use of MAD therapy have a statistically significant effect on the symptoms of heart failure in male African American veterans diagnosed with OSA?

The independent variable in this study was the use of the MAD. The dependent variables were data collected using secondary data at the 2nd, 4th, and 6th month on the results of SBP, DBP, and oxygen saturation. Overall, within the dependent variable demographics, there was an increase in the SBP mean values while using the MAD device over the three interval months. There was also an increase in the DBP mean value

from the 2nd to the 4th month and a slight decrease in the 6th month. Additionally, there was a decrease in the oxygen saturation mean value from the 2nd to the 4th month, followed by a slight increase in the 6th month. Lastly, there was a very slight mean decrease in the covariate BMI from the 2nd month through the 6th month. I ran the rMANOVA test to assess the magnitude of the change in the mean values for measures related to symptoms of heart failure as a result of MAD use across the time span of 6 months. Results related to this test were provided below.

The first symptom to be reported were the values for SBP. Results from the Mauchly's test of sphericity indicated that sphericity assumption was not violated as the result was not statistically significant at $\chi^2(2) = 3.72$, $p = .155$. It was noted that the variances dated across the three time periods were equal. Therefore, values for the sphericity assumed line were used to assess the statistical significance of the results. A summary of the rMANOVA for SBP was presented in Table 10. The data revealed a statistically significant result [F (2, 56) = 5.341, p = .008]. The observed power statistic reveals that the differences in the mean scores were large enough to be detected with an 82.00% probability. A review of the descriptive statistics revealed that mean SBP while using MAD increased from 127.31mmHg in the 2nd month, to 133.17 mmHg in the 4th month to 136.73 mmHg in the 6th month. Therefore, indicating that there was a significant difference in the effect of MAD use on SBP.

Table 10

Test of Within-Subjects Effects for Systolic Blood Pressure

(table continues)

Measure: MEASURE_1

Source		Type III Sum of Squares	Df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
factor1	Sphericity Assumed	1311.660	2	655.830	5.341	.008	.160	10.681	.820
	Greenhouse-Geisser	1311.660	1.772	740.405	5.341	.010	.160	9.461	.784
	Huynh-Feldt	1311.660	1.882	696.769	5.341	.009	.160	10.054	.802
	Lower-bound	1311.660	1.000	1311.660	5.341	.028	.160	5.341	.607
Error(factor 1)	Sphericity Assumed	6876.690	56	122.798					
	Greenhouse-Geisser	6876.690	49.603	138.634					
	Huynh-Feldt	6876.690	52.710	130.463					
	Lower-bound	6876.690	28.000	245.596					

a. Computed using alpha = .05

The second symptom to be reported were the values for diastolic blood pressure. Results from the Mauchly's test of sphericity indicated that sphericity assumption was not violated as the result was not statistically significant at $\chi^2(2) = 3.76$, $p = .152$. It was noted that the variances in the data across the three time periods were equal. Therefore, values for the sphericity assumed line was used to assess the statistical significance of the results. A summary of the rMANOVA for diastolic blood pressure was presented in table 11. The data revealed a non-statistically significant result [F (2, 56) = 2.28, p = .111]. The observed power statistic reveals that the differences in the mean scores were large enough to be detected with a 44.50% probability.

Table 11

Test of Within-Subjects Effects for Diastolic Blood Pressure

(table continues)

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
factor1	Sphericity Assumed	340.273	2	170.136	2.282	.111	.075	4.565	.445
	Greenhouse-Geisser	340.273	1.770	192.269	2.282	.118	.075	4.039	.416
	Huynh-Feldt	340.273	1.880	180.957	2.282	.115	.075	4.292	.430
	Lower-bound	340.273	1.000	340.273	2.282	.142	.075	2.282	.309
Error(factor1)	Sphericity Assumed	4174.245	56	74.540					
	Greenhouse-Geisser	4174.245	49.554	84.237					
	Huynh-Feldt	4174.245	52.652	79.281					
	Lower-bound	4174.245	28.000	149.080					

a. Computed using alpha = .05

The third symptom to be reported was the values for oxygen saturation. Results from the Mauchly's test of sphericity indicated that the sphericity assumption was violated as the result was statistically significant at $\chi^2(2) = 16.78$, $p = .000$. It was noted that the variances dated across the three time periods were not equal. Subsequently, values for the Greenhouse-Geisser correction were used to assess the statistical significance of the results. A summary of the rMANOVA for oxygen saturation was presented in table 12. The data revealed a non-statistically significant result [F (1.36, 38.27) = .671, p = .462]. The observed power statistic revealed that the differences in the mean scores were detected with a 13.70% probability.

Table 12

Test of Within-Subjects Effects for Oxygen Saturation

(table continues)

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared	Noncent. Parameter	Observed Power[a]
factor1	Sphericity Assumed	3.277	2	1.638	.671	.515	.023	1.342	.157
	Greenhouse-Geisser	3.277	1.367	2.397	.671	.462	.023	.918	.137
	Huynh-Feldt	3.277	1.413	2.318	.671	.467	.023	.949	.139
	Lower-bound	3.277	1.000	3.277	.671	.420	.023	.671	.124
Error(factor 1)	Sphericity Assumed	136.682	56	2.441					
	Greenhouse-Geisser	136.682	38.278	3.571					
	Huynh-Feldt	136.682	39.578	3.453					
	Lower-bound	136.682	28.000	4.881					

a. Computed using alpha = .05

Results from the Pairwise Comparison test were provided below. Due to the small sample size, the Bonferroni test was used to limit Type 1 error across the pairwise comparison test (Horn, n.d.). The data revealed a statistically significant difference between the 2nd month SBP and 6th month SBP, p =.009. There were no statistically significant differences between either of the three periods among DBP or oxygen saturation.

Table 13

Pairwise Comparison Test

Measure	(I) factor1	(J) factor1	Mean Difference (I-J)	Std. Error	Sig.[b]	95% Confidence Interval for Difference[b] Lower Bound	Upper Bound
SBP	1	2	-5.861	3.332	.268	-14.345	2.623
		3	-9.418*	2.911	.009	-16.830	-2.005
	2	1	5.861	3.332	.268	-2.623	14.345

(table continues)

		3	-3.557	2.415	.456	-9.707	2.593
	3	1	9.418*	2.911	.009	2.005	16.830
		2	3.557	2.415	.456	-2.593	9.707
DBP	1	2	-4.582	2.441	.213	-10.797	1.633
		3	-3.653	2.485	.458	-9.980	2.675
	2	1	4.582	2.441	.213	-1.633	10.797
		3	.929	1.814	1.000	-3.690	5.549
	3	1	3.653	2.485	.458	-2.675	9.980
		2	-.929	1.814	1.000	-5.549	3.690
O2sat	1	2	.083	.469	1.000	-1.111	1.278
		3	-.364	.232	.386	-.955	.228
	2	1	-.083	.469	1.000	-1.278	1.111
		3	-.447	.481	1.000	-1.671	.777
	3	1	.364	.232	.386	-.228	.955
		2	.447	.481	1.000	-.777	1.671

Based on estimated marginal means

*. The mean difference is significant at the .05 level.

b. Adjustment for multiple comparisons: Bonferroni.

In conclusion, the rMANOVA was conducted to test the mean change in symptoms of heart failure as a result of MAD use. The results have shown that there was a significant mean difference between the intervention and the dependent variable. The dependent variable SBP was found to be significantly affected.

RQ2: What is the moderating effect of BMI on the use of MAD therapy for treating symptoms of heart failure in male African American veterans diagnosed with OSA?

The independent variable in this study was the use of the MAD. The dependent variables were data collected using secondary data at three different monthly interval measurements of the 2nd, 4th, and 6th month on the results of SBP, DBP, and oxygen

saturation. The covariate was BMI. I ran the rMANCOVA test to assess the magnitude of change in the mean values for measures related to symptoms of heart failure as a result of MAD use across the time span of 6 months while controlling for the covariates BMI. Results related to this test were provided below.

The first symptom to be reported were the values for SBP. Results from the Mauchly's test of sphericity indicated that sphericity assumption was not violated as the result was not statistically significant at $\chi^2(2) = 4.27$, $p = .118$. It was noted that the variances dated across the three time periods were equal. Therefore, values for the sphericity assumed line were used to assess the statistical significance of the results. A summary of the rMANCOVA for SBP was presented in Table 14. The data revealed no statistically significant results within SBP [F (2, 50) = .640, p = .531], SBP *2moBMI [F(2, 50) = .944, p = .396], SBP *4moBMI [F(2, 50) = 2.516, p = .091], and SBP *6moBMI [F(2, 50) = .025, p = .976].

Table 14

Test of Within-Subjects Effects for Systolic Blood Pressure and the Covariate of BMI

Measure: MEASURE_1

Source		Type III Sum of Squares	Df	Mean Square	F	Sig.	Partial Eta Squared	Noncent Parameter	Observed Power[a]
SBP	Sphericity Assumed	154.748	2	77.374	.640	.531	.025	1.280	.151
	Greenhouse-Geisser	154.748	1.719	90.005	.640	.509	.025	1.101	.143
	Huynh-Feldt	154.748	2.000	77.374	.640	.531	.025	1.280	.151
	Lower-bound	154.748	1.000	154.748	.640	.431	.025	.640	.120

(table continues)

SBP * @2moBMI_1_1	Sphericity Assumed	228.137	2	114.068	.944	.396	.036	1.888	.204
	Greenhouse-Geisser	228.137	1.719	132.689	.944	.385	.036	1.623	.191
	Huynh-Feldt	228.137	2.000	114.068	.944	.396	.036	1.888	.204
	Lower-bound	228.137	1.000	228.137	.944	.341	.036	.944	.154
SBP * @4mo_BMI_1_1	Sphericity Assumed	608.143	2	304.072	2.516	.091	.091	5.032	.481
	Greenhouse-Geisser	608.143	1.719	353.710	2.516	.100	.091	4.326	.443
	Huynh-Feldt	608.143	2.000	304.072	2.516	.091	.091	5.032	.481
	Lower-bound	608.143	1.000	608.143	2.516	.125	.091	2.516	.332
SBP * @6mo_BMI_1_1	Sphericity Assumed	5.937	2	2.968	.025	.976	.001	.049	.053
	Greenhouse-Geisser	5.937	1.719	3.453	.025	.962	.001	.042	.053
	Huynh-Feldt	5.937	2.000	2.968	.025	.976	.001	.049	.053
	Lower-bound	5.937	1.000	5.937	.025	.877	.001	.025	.053
Error(SBP)	Sphericity Assumed	6042.598	50	120.852					
	Greenhouse-Geisser	6042.598	42.983	140.580					
	Huynh-Feldt	6042.598	50.000	120.852					
	Lower-bound	6042.598	25.000	241.704					

a. Computed using alpha = .05

The second symptom to be reported was the values for diastolic blood pressure (DBP). Results from the Mauchly's test of sphericity indicated that sphericity assumption was not violated as the result was not statistically significant at $\chi^2(2) = 5.15$, $p = .076$. It was noted that the variances dated across the three time periods were equal. Therefore, values for the sphericity assumed line was used to assess the statistical significance of the results. A summary of the rMANCOVA for diastolic blood pressure was presented in

table 15. The data revealed no statistically significant results within DBP [F (2, 50) = .310, p = .735], DBP*2moBMI [F(2, 50) = 1.156, p = .323], DBP*4moBMI [F(2, 50) = 1.980, p = .149], and DBP*6moBMI [F(2, 50) = 1.015, p = .370].

Table 15

Test of Within-Subjects Effects for Diastolic Blood Pressure and the Covariate BMI

Measure: MEASURE_1

Source		Type III Sum of Squares	Df	Mean Square	F	Sig.	Partial Eta Squared	Noncentrality Parameter	Observed Power[a]
DBP	Sphericity Assumed	44.981	2	22.491	.310	.735	.012	.621	.097
	Greenhouse-Geisser	44.981	1.676	26.834	.310	.696	.012	.520	.093
	Huynh-Feldt	44.981	1.998	22.508	.310	.734	.012	.620	.097
	Lower-bound	44.981	1.000	44.981	.310	.582	.012	.310	.084
DBP * @2moBMI_1_1	Sphericity Assumed	167.566	2	83.783	1.156	.323	.044	2.312	.243
	Greenhouse-Geisser	167.566	1.676	99.964	1.156	.317	.044	1.938	.223
	Huynh-Feldt	167.566	1.998	83.847	1.156	.323	.044	2.311	.243
	Lower-bound	167.566	1.000	167.566	1.156	.293	.044	1.156	.179
DBP * @4mo_BMI_1_1	Sphericity Assumed	286.972	2	143.486	1.980	.149	.073	3.960	.390
	Greenhouse-Geisser	286.972	1.676	171.196	1.980	.157	.073	3.319	.355
	Huynh-Feldt	286.972	1.998	143.595	1.980	.149	.073	3.957	.390
	Lower-bound	286.972	1.000	286.972	1.980	.172	.073	1.980	.273
DBP * @6mo_BMI_1_1	Sphericity Assumed	147.085	2	73.542	1.015	.370	.039	2.030	.217
	Greenhouse-Geisser	147.085	1.676	87.745	1.015	.359	.039	1.701	.200
	Huynh-Feldt	147.085	1.998	73.598	1.015	.370	.039	2.028	.217

(table continues)

	Lower-bound	147.085	1.000	147.085	1.015	.323	.039	1.015	.163
Error (DBP)	Sphericity Assumed	3623.161	50	72.463					
	Greenhouse-Geisser	3623.161	41.907	86.457					
	Huynh-Feldt	3623.161	49.962	72.518					
	Lower-bound	3623.161	25.000	144.926					

a. Computed using alpha = .05

The third symptom to be reported were the values for oxygen saturation (O2sat). Results from the Mauchly's test of sphericity indicated that the sphericity assumption was violated as the result was statistically significant at $\chi^2(2) = 6.936$, $p = .031$. It was noted that the variances dated across the three time periods were not equal. Therefore, values for the Huynh-Feldt correction were used to assess the statistical significance of the results. A summary of the rMANCOVA for oxygen saturation was presented in table 16. The data revealed a statistically significant result O2sat *4moBMI [F (1.89, 47.39) = 16.83, p = .000]. The observed power statistic reveals that the differences in the mean scores were large enough to be detected with a 99.80% probability. Data revealed no statistically significant results within O2sat [F (1.89, 47.39) = 2.61, p = .087], O2sat *2moBMI [F (1.89, 47.39) = .222, p = .790], and O2sat *6moBMI [F (1.89, 47.39) = 2.60, p = .087].

Table 16

Test of Within-Subjects Effects for Oxygen Saturation and the Covariate BMI

Measure: MEASURE_1

(table continues)

Source		Type III Sum of Squares	Df	Mean Square	F	Sig.	Partial Eta Squared	Noncentrality Parameter	Observed Power[a]
O2Sat	Sphericity Assumed	8.452	2	4.226	2.611	.083	.095	5.223	.497
	Greenhouse-Geisser	8.452	1.599	5.287	2.611	.097	.095	4.175	.439
	Huynh-Feldt	8.452	1.896	4.458	2.611	.087	.095	4.950	.482
	Lower-bound	8.452	1.000	8.452	2.611	.119	.095	2.611	.343
O2sat * @2moBMI_1_1	Sphericity Assumed	.718	2	.359	.222	.802	.009	.444	.083
	Greenhouse-Geisser	.718	1.599	.449	.222	.752	.009	.355	.080
	Huynh-Feldt	.718	1.896	.379	.222	.790	.009	.421	.082
	Lower-bound	.718	1.000	.718	.222	.642	.009	.222	.074
O2sat * @4mo_BMI_1_1	Sphericity Assumed	54.490	2	27.245	16.834	.000	.402	33.669	1.000
	Greenhouse-Geisser	54.490	1.599	34.083	16.834	.000	.402	26.914	.998
	Huynh-Feldt	54.490	1.896	28.742	16.834	.000	.402	31.915	.999
	Lower-bound	54.490	1.000	54.490	16.834	.000	.402	16.834	.976
O2sat * @6mo_BMI_1_1	Sphericity Assumed	8.445	2	4.223	2.609	.084	.095	5.218	.496
	Greenhouse-Geisser	8.445	1.599	5.282	2.609	.097	.095	4.171	.439
	Huynh-Feldt	8.445	1.896	4.455	2.609	.087	.095	4.946	.482
	Lower-bound	8.445	1.000	8.445	2.609	.119	.095	2.609	.342
Error(O2sat)	Sphericity Assumed	80.920	50	1.618					
	Greenhouse-Geisser	80.920	39.969	2.025					
	Huynh-Feldt	80.920	47.395	1.707					
	Lower-bound	80.920	25.000	3.237					

a. Computed using alpha = .05

In conclusion, the rMANCOVA was conducted to test the mean change in symptoms of heart failure as a result of MAD use while isolating for BMI. The result showed that there was a significant mean difference between the intervention and dependent variable oxygen saturation when controlling for BMI at the 4-month.

Summary

The focus of my research was to assess change in the mean values for dependent variables effected by the use of MAD therapy while isolating for BMI. Based upon the analysis of the data, for RQ1, the alternative hypothesis was accepted due to there being a positive mean change that was specifically indicated in the dependent variable SBP. Additionally, in RQ2, the alternative hypothesis was accepted due to results indicated that MAD use was shown to have a significant negative effect on oxygen saturation in the 4th month when controlling for BMI. Therefore, the alternative hypothesis was accepted. Lastly, within the RQ3, data was observationally assessed to be insufficient due to having significant amounts of missing data and, therefore was not able to be determined.

Chapter 5: Discussions, Conclusions, Recommendations

Introduction

The purpose of this quantitative correlational study was to determine whether MAD therapy impacted symptoms of heart failure in African American male veterans over 20 years old diagnosed with OSA by using the rMANOVA. This study determined that treatment efficacy differs after controlling for patient BMI levels by using the rMANCOVA. Within the rMANOVA, it was revealed there was a mean change in SBP while using the MAD. This study's findings indicate that SBP was affected by the use of MAD among those with OSA and may benefit African American male veterans with OSA. Within the rMANCOVA, it was determined that MAD use treatment efficacy was negatively moderated after controlling for an obese BMI after 4 months pertaining to the oxygen saturation level. In Chapter 5, I discuss interpretations of the findings, limitations of the study, recommendations, implications, and provide a conclusion.

Interpretation of the Findings

The results of my study should be considered when trying to relay information to improve the heart failure biomarkers of oxygen saturation and SBP. The archival data in my study did provide comparable information as shown by Dudley and Patel (2016), which argued that BMI among male African Americans was significantly high. Within the study by Cistulli et al. (2009), reports indicated that the upper airway was affected by the use of oral mandibular devices on patients. Results of my study yielded a statistically significant negative result on the dependent variable oxygen saturation in male African American veterans who used the MAD, when controlling for an obese BMI.

According to Booth et al. (2014), the MAD has been suggested to possibly improve blood pressure. Throughout my study, changes in the mean values of SBP were seen in African Americans male veterans while using MAD therapy. The result of my study extended knowledge to the public health and healthcare disciplines that was previously not available by providing information that aids in analyzing the effect that MAD therapy has on heart failure symptoms among the African American populations related to those with OSA, heart failure, and BMI. Furthermore, my study allowed for information to be gained regarding the effects MAD therapy has on heart failure symptoms and identified which biomarkers were susceptible to change when using the MAD device within a 6-month timeframe.

In relation to the oxidative stress theory, study results have suggested that oxidative damage to lipids, DNA, and proteins may be reduced as a result of MAD therapy. The possible change in oxygen flow may be evidence of the statistical changes in SBP levels, which may suggest reductions in attacks on mitochondria by potentially limiting ROS. This potential change may allow for increased cardiovascular function resulting in decreased heart failure symptoms as a result of a decrease in BMI.

Limitations of the Study

Limitations in this study may have been due to the use of secondary data as well as the lack of complete data provided in the initial study sample. Cheng and Phillips (2014) have indicated that limitations may consist of secondary data not always having been collected by the same person, which may have led to inconsistent routines or knowledge bases to verify data. Secondary data usage may have also served as a

limitation in the study due to the initial medical provider who collected the data possibly having biases, brain fatigue, or brain overload, resulting in possible inaccurate information being recorded (Cheng and Phillips, 2014). Within the initial queried sample from VINCI there was a significant amount of missing data for many of the study variables resulting in data being limited. Limited data present in my study resulted in removing 1582 African American archived records that may have been used to provide more validity to the study if not for missing data. The limitation in archived records may have affected the potential generalizability of the study, even though the study did display adequate power and sample size (Davis-Kean, Jager & Maslowsky, 2015). Additionally, limitations due to missing data resulted in having RQ3 removed. These limitations were difficult to address due to having no control over extraneous variables.

In addition to the possible limitations involved in purposeful convenience sampling, there were no cause-effect relationships between variables, several of the assumptions for both rMANOVA and rMANCOVA were violated, and there was a possible decrease in validity and reliability. Limitations in purposeful sampling included susceptibility to judgment error, low reliability, high potential for bias, and the inability of findings to be generalized (Dudovskiy, 2019). Due to the use of purposeful sampling, there may be a limitation in external validity due to the lack of sample randomization (Crawford and Khorsan, 2014). Another limitation of the study may be its correlational design. Correlational designs may show that there is a relationship between variables; however, they cannot imply causal effect (Rohrer, 2018). Causal effect indicates that one action causes another (Australian Bureau of Statistics, 2013). As a result of the use of

secondary data, a causal effect may not be clear due to data possibly having uncontrollable variables, including human error, exposure to unknown factors that may alter results, and the potential for biases before receiving.

Some initial dependent variables in the study displayed missing data, resulting in a means imputation; however, if missing data was initially provided at the beginning of the study, it may have enhanced the study validity, and possibly increased the low generalizability within this study (Davis-Kean, Jager, & Maslowsky, 2015). Therefore, the results of the study may not be generalizable to the larger African American male veteran study population due to the small sample size. This study may not be generalizable due to having used only African American male veterans who were over 20 years old, which may make it difficult to generalize results to any other group, gender, or race. Conclusion validity was the level to which a reasonable deduction was reached in terms of relationships of variables through statistical analysis observation. Conclusion validity must also be considered as a limitation due to having a violation of multivariate normality assumptions that may have led to an incorrect relationship inference (Indiana, n.d.). Conclusion validity may have been mitigated by using an appropriate statistical power of at least 80% (Hoare & Hoe, 2012). Within this study, there were possible unknown extraneous variables in the data that may have included the possible attrition of participants in the form of non-compliance or drop out of the study. According to Amankwaa (2016), trustworthiness involves credibility, transferability, dependability, or confirmability. The National Coordination Office for Networking and Information Technology Research and Development (2016), indicated the trustworthiness of data by

federal agencies are required by the federal government due to the Information Quality Act of 2001. Due to the data being securely gathered, maintained, and provided by the VA, there were no concerns involving a lack of trustworthiness.

Recommendations

This study began to bridge the gap in the literature regarding the effect the MAD has on heart failure symptoms as well as the moderating effect of BMI. This study analysis did not, however, answer all the questions pertaining to the effects that MAD has on the scope of heart failure. I recommend further research be done to confirm the results of this study. Research may also be needed that uses larger sample sizes with more access to heart failure biomarkers. Further investigation should be done as a comparative analysis on the effect that MAD therapy has on heart failure symptoms across studies. Future research might include identifying how the MAD effects heart failure symptoms through a pre and posttest. Recommendations for practice might also include a bimonthly analysis of how MAD use effects heart failure symptoms in African American veteran women vs. men. Another recommendation would be to study the effect that the MAD has on different levels of BMI in African American veterans over age 20.

Additionally, another recommendation would be to perform this study on non-veteran African Americans, other minorities, and races. This study was primarily focused on African American male veterans in the U.S. and may benefit from further research abroad on other minority foreign military veterans, such as India who one of the world's largest military services (Statista, 2019). Lastly, additional research might include an exploration into why the MAD is currently not covered by insurance companies for OSA

patients. A higher number of African Americans suffer from OSA, more than any other race (Clark et al., 2016).

Implications

The potential impact from this study to create positive social change may be felt at the family and individual levels for caregivers and patients with OSA and heart failure patients wanting information on the benefits of MAD use. Organizationally this data can be used for health agencies and departments wanting to know the benefits of MAD use OSA in relation to heart failure symptoms prior to the prescription of the other devices or medications. Societally, the results of this study on possible SBP improvement may be used by the general public to help promote the need for an alternative cost-effective OSA health solution that does not require electricity usage or excess equipment. This study may also benefit society due to the information it has obtained on MAD use over the extended six month time period. Results from this study may also be beneficial, providing information as to what interval measure the device was most useful in relation to symptoms of heart failure. Information captured in this study may also be beneficial societally by knowing the potential limitations that can be caused when using the MAD and having a high BMI. Regarding policy, this study adds more information to the policymaking arena on the uses of MAD and its effectiveness on OSA as well as its benefit on symptoms of heart failure, i.e., SBP. Results from this study can be utilized by both the public health and medical field to further indicate the benefits of MAD treatment on OSA and heart failure. Currently, insurance companies do not cover the MAD as an alternative treatment for OSA, and results from this study can be used as a reference for

policies to be written by agencies and lawmakers at the local, state level, and federal levels regarding insurance coverages.

Conclusion

Heart failure symptoms in those with OSA were found to be a destructive epidemic that affects 24.9 million Americans and 4.1 million African Americans every year, of all ages, genders, and ethnicities but especially those who are African American, males, and Veterans (Capaldi, Guerrero & Killgore, 2011; Dudley & Patel, 2016; United States Census Bureau, 2017; Watson, 2016). This epidemic has many different parameters and has historically occurred involuntarily, gone unnoticed for the better part of a century and in some cases, continues to go untreated due to lack of knowledge, finances, insurances, or resources (ALA, 2018; Gillespie et al., 2015). This gap in the literature has guided me to inspect whether MAD therapy impacts the symptoms of heart failure in African American male veterans diagnosed with OSA. This study has also determined whether MAD treatment efficacy differs after controlling for patient BMI levels, however, it was unable to assess the MAD effects of those who smoke. This study sought to shine a light on specific questions regarding the use of the MAD. My overall goal was to better understand the effect the MAD therapy has on heart failure symptoms in African American Veterans in hopes of extending the public health knowledge base regarding this non-invasive treatment for the benefit of those most affected. Though there remains much more research to be done, I hope the results provided in this study can lay the groundwork for others to continue research and to further understand the benefit of the MAD.

References

Aaronson, J. A., Hofman, W. F., van Bennekom, C. A., van Bezeji, T., van den Aardewg, J. G., Groet, E… Schmand, B. (2015). Obstructive sleep apnea is related to impaired cognitive and functional status after stroke. *Sleep, 38*(9), 1431-1437. doi:10.5665/sleep.4984

Abraham, P., Gagnadoux, F., Meslier, N., Racineux, J., Rousseau, P… Trzepizur, W. (2009). Microvascular endothelial function in obstructive sleep: Impact of continuous positive airway pressure and mandibular advancement. *Sleep Medicine, 10*(7), 746-752. doi:10.1016/j.sleep.2008.06.013

Abraham, W. T., Clark, L., Gocke, B., Khayat, R., Krueger, S., Rathman, L. …Yamokoski, L. (2013). Sleep-disordered breathing in heart failure: Identifying and treating an important but often unrecognized comorbidity in heart failure patients. *Journal of Cardiac Failure, 19*(6), 431-444. doi:10.1016/j.cardfail.2013.04.005

Adams, R. J., Antic, N., Appleton, S. L., Catcheside, P. G., Grant, J. F., Lang, C. J., Martin, S. A. …Wittert, G. A. (2017). Associations of undiagnosed obstructive sleep apnea and excessive daytime sleepiness with depression: An Australian population study. *Journal of Clinical Sleep Medicine, 13*(4), 575-582. doi:10.5664/jcsm.6546

Agha, Z., Anderson, N., Armstead, L., Bell, D.S., Berman, D., Burns, J.C., Cooper, D….Westerman, D. (2014). pSCANNER: patient-centered scalable national network for effectiveness research. *Journal of the American Medical Informatics*

Association, 21(4), 621-626. Retrieved from https://academic.oup.com/jamia/article/21/4/621/2909301

Akinboboye, O., & Cuyjet, A. B. (2014). Acute heart failure in the African American patient. *Journal of Cardiac Failure, 20*(7), 533-540. doi: 10.1016/j.cardfail.2014.04.018

Akinbobye, O., Jean-Louis, G., Mitchell, J., Ogedegbe, G., & Olafiranye, O. (2013). Obstructive sleep apnea and cardiovascular disease in blacks: A call to action from association of black cardiologist. *American Heart Journal, 165*(4), 468-476. doi:10.1016/j.ahj.2012.12.018

Akinnusi, M., Anandam, A., El-Solh, A. A., Jaoude, P. & Patil, M. (2013). Cardiovascular mortality in obstructive sleep apnoea treated with continuous positive airway pressure or oral appliance: An observational study. *Respirology, 18*(8), 1184-1190. doi: 10.1111/resp.12140.

Akinnusi, M., Churder, P. M., El-Solh, A. A., Lafornara, A. M., & Moitheennazima, B. (2011). Combined oral appliance and positive airway pressure therapy for obstructive sleep apnea: A pilot study. *Sleep and Breathing, 15*(2), 203-208. doi: 10.1007/s11325-010-0437-1

Allen, P. & Bennett, K. (2008). Statistical package for the social sciences for the health and behavioural sciences. Southern Melbourne, Australia: Cengage Learning Australia.

Albert, M. A., Anderson, C. A. M., Bertoni, A. G., Carnethon, M. R. Howard, G., Mujahid, M. S., Palanippan, L… Yancy, C. (2017). Cardiovascular health in

African Americans: A scientific statement from the American Heart Association. *Circulation, 136*(21), e393-e423. doi:10.1161/CIR.0000000000000534

Albert, M. A., Bhatt, D. L., Fonarow, G. C., Kebede, S., Lu, D., Ngwa, J., Powell-Wiley, T. M… Yancy, C. (2018). Impact of body mass index on heart failure by race/ethnicity from the get with the guidelines- Heart failure (GWTG-HF) registry. *Journal of American College of Cardiology, 6*(3), 233-242. doi: 10.1016/j.jchf.2017.11.011

Alkassim, R. S., Etikan, I., & Musa, S. A. (2016). Comparison of convenience sampling and purposive sampling. *American Journal of Theoretical and Applied Statistics, 5*(1), 1-4. doi:10.11648/j.ajtas.20160501.11

Alosert, M., Butros, V., Haj, A. A., Fikree, M., Mohammed, I., Mohammed, S., & Naroo, G. Y. (2009). Elevated heart-type fatty acid-binding protein predicts early myocardial injury and aids in the diagnosis of non-st elevation myocardial infarction. *Hong Kong Journal of Emergency Medicine, 16*(3), 141-147. doi: 10.1177/102490790901600303

Al-Shorman, I. S. & Shydfat, N. (2015). Oral device therapy for obstructive sleep apnea. *Pakistan Oral & Dental Journal, 35*(1), 70-73,4P. Retrieved from http://eds.b.ebscohost.com/

Amankwaa, L. (2016). Creating protocols for trustworthiness in qualitative research. *Journal of Cultural Diversity, 23*(3), 121-127. Retrieved from http://eds.a.ebscohost.com/eds/pdfviewer/pdfviewer?vid=1&sid=51224771-9665-4712-bd98-18a354ddd894%40sessionmgr4008

Ambrosy, A. P., Butler, J., Chioncel, O., Fornarow, G. C., Gheorghiade, M., Greene, S. J. …Vaduganthan, M. (2014). The global health and economic burden of hospitalizations for heart failure: Lessons learned from hospitalized heart failure registries. *Journal of the American College of Cardiology, 63*(12), 1123-1133. doi:10.1016/j.jacc.2013.11.053

American Academy of Dental Sleep Medicine (2011). *Study finds that combination therapy reduces pauses in breathing caused by obstructive sleep apnea*. Retrieved from http://www.aadsm.org/articles.aspx?id=2296

American Academy of Sleep Medicine (2016). *Hidden health crisis costing America billions: Underdiagnosing and undertreating obstructive sleep apnea draining healthcare system*. Retrieved from https://aasm.org/resources/pdf/sleep-apnea-economic-crisis.pdf

American Academy of Sleep Medicine (2018). *Rising prevalence of sleep apnea in the united states threatens public health*. Retrieved from: https://aasm.org/rising-prevalence-of-sleep-apnea-in-u-s-threatens-public-health/

American Heart Association. (2017a). Causes and risks of heart failure. Retrieved from http://www.heart.org/HEARTORG/Conditions/HeartFailure/CausesAndRisksForHeartFailure/Causes-and-Risks-for-Heart-Failure_UCM_002046_Article.jsp#.WRJXzHk2yM8

American Heart Association (2017b). Know your risk factors for high blood pressure. Retrieved from http://www.heart.org/HEARTORG/Conditions/HighBloodPressure/UnderstandSy

mptomsRisks/Know-Your-Risk-Factors-for-High-Blood-Pressure_UCM_002052_Article.jsp#.WxqjUKQvwdU

American Heart Association (2018). Sleep apnea and heart disease, stroke. Retrieved from https://www.heart.org/en/health-topics/consumer-healthcare/what-is-cardiovascular-disease/sleep-apnea-and-heart-disease-stroke

American Lung Association (2018). Obstructive sleep apnea symptoms, causes & risk factors. Chicago, IL. Retrieved from http://www.lung.org/lung-health-and-diseases/lung-disease-lookup/sleep-apnea/osa-symptoms-causes-risk.html

American Sleep Apnea Association (2017). Sleep apnea information for. Washington, District of Columbia. Retrieved from https://www.sleepapnea.org/learn/sleep-apnea-information-clinicians/

American Sleep Association (2018). Continuous positive airway pressure machines and masks. Devices reviews and costs. Lititz, PA. Retrieved from https://www.sleepassociation.org/sleep-treatments/cpap-machines-masks/

Anand, I., Boer, R.A., Bozkurt, B., Chow, S.L., Felker, G. M., Fonarow, G.C., Greenberg, B…. Zile, M.R. (2017). Role of biomarkers for the prevention, assessment, and management of heart failure: A scientific statement from the american heart association. *Circulation, 2017*(135), e1054-e1091. doi: 10.1161/CIR.0000000000000490

Anavekar, N.S., Araoz, P.A., Bonnichsen, C.R., Foley, T.A., Mankad, S.V., Morris, M.F., Miller, T.D. (2012). Measuring left ventricular ejection fraction- techniques

and potential pitfalls. *European Cardiology, 8(2)*, 108-114. doi: 10.15420/ecr.2012.8.2.108

Anderson, W.M., Foulis, P.R., Iannacone, M.R., Rosas, J., Schwartz, S.W., Sebastiao, Y. (2016). Racial disparity in adherence to positive airway pressure among US Veterans. *Sleep Breathing, 20*(3), 947-955. doi: 10.1007/s11325-016-1316-1

Antoni, M.H., Dietz, N.A., Hooper, W., Okuyemi, K.S., Resniscow, K., & Tan, M. M. (2018). Association between smoking cessation and weight gain in treatment-seeking African Americans. *Addiction Behaviors, 81*(1), 84-90. doi: 10.1016/j.addbeh.2018.02.002

Arnett, D.K., Benjamin, E.J., Blaha, M.J., Cushman, M., de Ferranti, S., Despres, J., Fullerton, H.J…. & Yeh, R.W. (2015). Heart disease and stroke statistics-at-a-glance-2015 update: A report from the American heart association. *Circulation, 2015*(131), e29-e322. doi: 10.1161/CIR.0000000000000152

Arynchyn, A., Bibbins-Domingo, K., Gardin, J.M., Hulley, S.B., Lewis, C., Lin, F…. Williams, O.D. (2009). Racial differences in incident heart failure among young adults. *The New England Journal of Medicine, 360*(12), 1179-1190. doi: 10.1056/NEJMoa0807265

Atlasti. (2018). Qualitative Research. Berlin, Germany. Retrieved from http://atlasti.com/qualitative-research/

Australian Bureau of Statistics (2013). Correlation and causation. Retrieved from

https://www.abs.gov.au/websitedbs/a3121120.nsf/home/statistical+language+-+correlation+and+causation

Aune, D. Janszky, I., Norat, T., Romundstad, P., Sen, A., Tonstad, S., & Vatten, L.J. (2016). Body mass index, abnormal fatness and heart failure incidence and mortality: A systematic review and dose-response meta-analysis of prospective studies. *Circulation, 133*(7)*,* 639-649. doi:10.1161/CIRCULATIONAHA.115.016801

Ayas, N., Badran, M. & Laher, I. (2014). Cardiovascular complications of sleep apnea: Role of oxidative stress. *Oxidative medicine and Cellular Longevity, 201*(985258)*, 10*. doi: 10.1155/2014/985258

Ayo-Yusuf, O. A., Motloba, D. P., Solomons, Y. F., & Sethusa, M.P.S. (2015). Obstructive sleep apnoea: Epidemiology, quality of life, and management - implications for dentists. A review. *South African Dental Journal*, *70*(5), 190-195. Retrieved from http://www.scielo.org.za/scielo.php?script=sci_arttext&pid=S0011-85162015000500003&lng=en&tlng=en.

Babineau, D.B., Bhatt, D.L., Blumenthal, R.S., Gottlieb, D.J., Lewis, E.F., Mehra, R., Patel, S.R. …. Tracy, R.P. (2014). CPAP versus oxygen in obstructive sleep apnea. *The New England Journal of Medicine*, *370*(24), 2276–2285.doi: 10.1056/NEJMoa1306766

Babson, K.A., Bonn-Miller, M.O., Del Re, A.C., & Woodward, S.H. (2013). The Comorbidity of sleep apnea and mood, anxiety, and substance use disorders

among obese military veterans within the veterans' health administration. *Journal of Clinical Sleep Medicine, 9*(12), 1253-1258. doi: 10.5664/jcsm.3262

Badr, M.S., Pranathiageswaran, S., Rowley, J.A., & Severson, R. (2013). The influence of race on the severity of sleep disordered breathing. *Journal of Sleep Medicine*, *9*(4), 303-309. doi: 10.5664/jcsm.2572

Bahammam, A. (2011). Obstructive sleep apnea: From simple upper airway obstruction to systemic inflammation. *Annals of Saudi Medicine*, *31*(1), 1–2. doi: 10.4103/0256-4947.75770

Bajaj, D.K., Dubey, A., Kant, S. & Singh, B.P. (2017). Prospects of mandibular advancement device as a preferred treatment of obstructive sleep apnea in India: A systematic review. *Annals of Tropical Medicine and Public Health, 10*(1), 1-6. doi: 10.4103/1755-6783.205552

Bakker, J.P., Patel, S., Punjabi, N.M., Redline, S., Wang, R., Weng, J., (2015). Associations between obstructive sleep apnea, sleep duration, and abnormal fasting glucose. The multi-ethnic study of atherosclerosis. *ATS Journals*, *192*(6), 745-753. doi: 10.1164/rccm.201502-0366OC

Baldini, A., Ballanti, F., Cozza, P. & Ranieri, S. (2015). Long term therapeutic efficacy of a soft monobloc mandibular advancement device in adults with obstructive sleep apnea. *The Scientific World Journal, 2015*(1), 408469. doi: 10.1155/2015/408469

Balfour, P., Bluemke, D.A., Burke, G., Chahal, H., Folsom, A., Herrington, D. …Wu, C.O. (2015). Heart failure prediction in the multi-ethnic study of atherosclerosis. *BMJ Journals, 101*(1), 58-64. doi: 10.1136/heartjnl-2014-305697

Barbe, F., Drager, L.F., Lorenzo-Filho, G., McEvoy, R.D., & Redline, S. (2017). Sleep Apnea and Cardiovascular Disease. Lessons from recent trials and needs for team science. *American Heart Journal*, *136*(19), 1840-1850. doi: https://www.ahajournals.org/doi/10.1161/CIRCULATIONAHA.117.029400

Bahrami, H., Bluemke, D. A. & Kronmal, R. (2008). Differences in the incidence of the congestive heart failure by ethnicity: The multi-ethnic study of atherosclerosis. *Archives of Internal Medicine, 168*(19), 2138-2145. doi: 10.1001/archinte.168.19.2138

Barnet, J.H., Hagen, E.W., Hla, K.M., Palta, M., Peppard, P. E. & Young, T. (2013). Increased prevalence of sleep-disordered breathing in adults. *American Journal of Epidemiology*, *177*(9), 1006-1014. doi: 10.1093/aje/kws342.

Bauer, D.C, Gopal, D.M., Kalogeropoulos, A.P., Georgiopoulou, V.V., Smith, A.L., Newman, A … Butler, J. (2012). Cigarettes smoking exposure and heart failure risk in older adults: The health, aging and body composition study. *American Heart Journal*, *164(2)*, 236-242. doi: 10.1016/j.ahj.2012.05.013

Baumann, G., Glos, M., Penzel, T., Schoebel, C., Nitzsche, G.-R., Zimmermann, S., Rudolph, C. … Fietze, I. (2016). Comparison of effects of obstructive sleep apnea treatment by mandibular attachment device and by continuous positive airway

pressure on cardiac autonomic function during daytime. *Sleep & Breathing*, *20*(2), 635–646. doi: 10.1007/s11325-015-1265-0

Beaton, D., Binns, M. A., Fraser, J., Kwan, D., Mclaughlin, P. M., Montero-Odesso, M., Peltsch, A.J., Pieruccini-Faria, F., Sahlas, D.J., Strother, S.C., & Swartz, R.H. (2019). The utility of multivariate outlier detection techniques for data quality evaluation in large studies: an application within the ONDRI project. *BA Medical Research Methodology, 19,102,* (2019). Retrieved from https://doi.org/10.1186/s12874-019-0737-5

Becker, D.M., Becker, L.C., Bis, J.C., Boerwinkle, E., Buyske, S., Carty, C.L. … Yanek, L.R. (2014). Prospective associations of coronary heart disease loci in African Americans using the metabochip: The PAGE study. *PLoS, 9*(12), e113203. doi: 10.1371/journal.pone.0113203

Benjamin, E.J., Bhatnagar, A., Blaha, M. J., Butler, J., Cain, L. R., Correa, A., Fox, E.R.… Winniford, M.D. (2018). Cigarette smoking and incident heart failure: Insight from jackson heart study. *Circulation, 137*(23), 00-00. doi: 10.1161/CIRCULATIONAHA.117.031912

Benjamin, E.J., Blaha, M.J., Chiuve, S.E., Cushman, M., Das, S.R., Deo. R. …. Wu, J.HY. (2017). Heart disease and stroke statisitics-2017 update. *American Heart Association, 135*(1), 00-00. doi: 10.1161/CIR.0000000000000485

Bennett, M., Cameron, M., Chadwick, R., Clutterbuck-James, A., Glover, M., Davis, M.…Quinnell, T. *(2014).* Clinical and cost-effectiveness results from the randomized controlled trial of oral mandibular advancement devices for

obstructive sleep apnoea-hypopnoea and long-term economic analysis of oral devices and continuous positive airway pressure. *Health Technol Assess, 18*(67), 1-296. doi: 10.3310/hta18670

Benschop, T., Machingauta, C., & Welch, M. (2018). Anonymization methods. Retrieved from https://sdcpractice.readthedocs.io/en/latest/anon_methods.html

Berge, M., Gjerde, K., Lehmann, S., Johansson, A.K., Johansson, A. (2015). Oral appliance treatment in moderate and severe obstructive sleep apnea patient's non-adherent to CPAP. *Journal of Oral Rehab, 42*(4), 249-58. doi: 10.1111/joor.12376

Bergerhenryent. (2017). Sleep apnea statistics and fact 2016. East Norrington, PA. Retrieved from http://www.bergerhenryent.com/sleep-apnea-statistics-2016/

Bernhard, D. and Messner, B. (2014). Smoking and cardiovascular disease mechanisms of endolethial dysfunction and early atherogenesis. *Arteriosclerosis, Thrombosis, and Vascular Biology, 2014* (34), 509-515. doi: 10.1161/ATVBAHA.113.300156

Bethea, T.N., Black, A., Blot, W.J., Boggs, D.A., Cohen, S.S., de Gonzalez, A.B. ….Singh, P. (2014). A pooled analysis of body mass index and mortality among African Americans. *PLoS. 9*(11), e111980. doi: 10.1371/journal.pone.0111980

Birch-Machin, M.A., Bowman, A., & Kandola, K. (2015). Oxidative stress – a key emerging impact factor in health, ageing, lifestyle and aesthetics. *International Journal of Cosmetic Science. 37*(52), 1-8. doi: 10.1111/ics.12287

Bishop, B., Girvan, T., & Verrett, R. (2014). A randomized crossover study comparing two mandibular repositioning appliances for treatment of obstructive sleep apnea. *Sleep Breath, 18*(1), 125. doi: 10.1007/s11325-013-0859-7

Bisht, S. & Dada, R. (2017). Oxidative stress: Major executioner in disease pathology, role in sperm dna damage and preventive strategies. *Frontiers in Bioscience, 9*(1), 420-447. doi: 10.2741/495

Blair, J. E. A., Huffman, M., & Shah, S. J. (2013). Heart failure in north America. *Current Cardiology Reviews, 9*(2), 128-146. doi: 10.2174/1573403X11309020006

Bobak, M., Brenner, H., Gardiner, J., Holleczek, B., Jansen, E. H. J. M., Kubinova, R.…Topor-Madry, R. (2015). Evidence for the free radical/oxidative stress theory of ageing from the chances consortium: A meta-analysis of individual participant data. *BMC Medicine, 13*(1), 300. doi: 10.1186/s12916-015-0537-7

Bokov, A., Ikeno, Y., Mele, J., Perez, V.I., Ran, Q., & Richardson, A. (2009). Is the oxidative stress theory of aging dead? *Biochim Biophys Acta, 1790*(10), 1005-1014. doi:10.1016/j.bbagen.2009.06.003

Booth, A.J., Djavadkhani, Y. &. Marshall, N.S. (2014). A critical review of the treatment options available for obstructive sleep apnea: an overview of the current literature available on treatment methods for obstructive sleep apnea and future research directions. *Bioscience Horizons, 7*(1), hzu011. doi: 10.1093/biohorizons/hzu011

Brandt, C.A., Charpentier, P., Fried, T.R., Justice, A., Levin, F.L., Miller, P., Niehoff, K.M.... Rajeevan, N. (2017). Utilizing patient data from the veterans administration electronic health record to support web-based clinical decision support: Informatics challenges and issues from three clinical domains. *BMC Med Inform Decis Mak, 17*(1), 111. doi: 10.1186/s12911-017-0501-x

Bratton, D.J., Gaisl, T., Kohler, M. & Wons, A.M. (2015). Continuous positive airway pressure vs mandibular advancement devices and blood pressure in patients with obstructive sleep apnea: A systematic review and meta-analysis. *JAMA, 314*(21), 2280-93 doi: 10.1001/jama.2015.16303

Brenner, A., Bruce, M., Diez-Roux, A.V., Dubbert, P., Gebreab, S.Y., Hickson, D. … Taylor, H. (2015). Perceived discrimination is associated with health behaviours among African Americans in jackson heart study. *BMJ Journals, 70*(2), 187-194. doi: 10.1136/jech-2015-206390

Brown, J.J. (2018). BMI and heart disease have a cause-and-effect connection, not just a link. Everyday Health. Hudson, New York. Retrieved from https://www.everydayhealth.com/heart-failure/living-with/bad-news-if-your-bmi-is-high-fat-causes-heart-failure/

Brown, N.R., Christy, K., Jensen, J.D., Jones, C.L., Scherr, C.L., & Weaver, J. (2015). The health belief model as explanatory framework in communication research: Exploring parallel, serial and moderated mediation. *Health Communication, 30*(6), 566-576. doi: 10.1080/10410236.2013.873363

Bush, T. Catz, S.L., Deprey, M, Jack LM, Javitz HS, McAfee T, McClure JB… Zbikowski SM. (2011) Utilization of services in a randomized trial testing phone and web-based interventions for smoking cessation. *Nicotine Tob Res, 13*(5), 319–27. doi: 10.1093/ntr/ntq257

Camacho, M., Certal, V., & Riaz, M. (2015). Portable power supply options for positive airway pressure devices. *Rural and Remote Health, 15*(3), 1-8. Retrieved from http://www.rrh.org.au

Capaldi, V.F., Guerrero, M.L., & Killgore, W.D.S. (2011). Sleep disruptions among returning combat veterans from Iraq and Afghanistan. *Military Medicine, 176*(8), 879-888. doi: 10.7205/MILMED-D-10-00440

Caruana, E. J., Hernández-Sánchez, J., Roman, M…. & Solli, P. (2015). Longitudinal studies. *Journal of Thoracic Disease, 7*(11), E537–E540. doi: 10.3978/j.issn.2072-1439.2015.10.63

Carlson, M.A. & Morrison, R.S. (2009). Study design, precision, validity in observation studies. User's guide to research in palliative care. *Journal of Palliative Medicine, 12*(1), 77-82. doi: 10.1089/jpm.2008.9690

Casey, K.R., Knepler, J., Panos, R.J., & Samson, P. (2012). Clinical characteristics, comorbidities, and response to treatment of veterans with obstructive sleep apnea Cincinnati veterans affairs medical center, 2005-2007. Center for Disease Control. *Preventing Chronic Disease, 9*(E46), 110-117. doi: 10.5888/pcd9.110117

Center for Disease Control (2015a). Body mass index. Division of nutrition, physical activity, and obesity. Atlanta, Georgia. Retrieved from https://www.cdc.gov/healthyweight/assessing/bmi/index.html

Center for Disease Control. (2016). Heart failure fact sheet. National center for chronic disease prevention and health promotion. Atlanta, Georgia. Retrieved from https://www.cdc.gov/dhdsp/data_statistics/fact_sheets/fs_heart_failure.htm

Center for Disease Control. (2017a). About adult body mass index. Atlanta, Georgia. Retrieved from https://www.cdc.gov/healthyweight/assessing/bmi/adult_bmi/index.html

Center for Disease Control. (2017b). Health of black or African American non-Hispanic population. Atlanta, Georgia. Retrieved from https://www.cdc.gov/nchs/fastats/black-health.htm

Center for Disease Control. (2017c). Heart disease fact sheet. Atlanta, Georgia. Retrieved from https://www.cdc.gov/dhdsp/data_statistics/fact_sheets/fs_heart_disease.htm

Center for Disease Control. (2017d). High blood pressure. Atlanta, Georgia. Retrieved from https://www.cdc.gov/bloodpressure/measure.htm

Center for Disease Control (2018a). Leading causes of death in males, 2015. Health Equity. Atlanta, Georgia. Retrieved from https://www.cdc.gov/healthequity/lcod/men/2015/black/index.htm

Center for Disease Control (2018b). Smoking and tobacco use. Atlanta, Georgia. Retrieved from https://www.cdc.gov/tobacco/disparities/african-americans/index.htm

Chambers, D.J., Graeme, L., Hickey, G.L. Mokhles, M. M., Kolamunnage-Dona, R. (2018). Statistical primer: performing repeated-measures analysis. *Interactive CardioVascular and Thoracic Surgery, 26*(4), 539–544. doi: 10.1093/icvts/ivy009

Chan, J. Z., Chan, M.Y., Chandra, S., Chen, S., Drager, L. F., Furlan, S. F.... Zhang, W. (2016). Intervention obstructive sleep apnea and cardiovascular events after

percutaneous coronary. *Circulation American Heart Association, 137*(4), 86-100. doi: 10.1161/CIRCULATIONAHA.115.019392

Chaturvedi, S. and Marulanda-Londono, E. (2017). The interplay between obstructive sleep apnea and atrial fibrillation. *Frontiers in Neurology*, *8(DEC)*, 668. doi: 10.3389/fneur.2017.00668

Chen, J., Dong, M., Li, Z. Luo, Y., Wang, Y., Wen, N., Zhang, B. (2017). Reoxygenation reverse hypoxic pulmonary arterial remodeling by inducing smooth muscle cell apoptosis via reactive oxygen species-mediated mitochondrial dysfunction. *Journal of the American Heart Association, 6*(6), e005602. doi: 10.1161/JAHA.117.005602

Cheng, H.G. & Phillips, M.R. (2014). Secondary analysis of existing data: opportunities and implementation. *Shanghai Arch Psychiatry, 26*(6), 371-375. doi: 10.11919/j.issn.1002-0829.214171

Cheriyath, P., Guduru, S. S., Jadhao, Y., Komanduri, S., & Wert, Y. (2016). Prevalence and risk factors of heart failure in the USA: NHANES 2013-2014 epidemiological follow-up study. *Journal of Community Hospital Internal Medicine Perspectives, 7*(1), 15-20. doi: 10.1080/20009666.2016.1264696

Chervin, R.D., Dort, L.C., Harrod, C.G., Katz, S.G., Lettieri, C.J., Ramar, K., & Thomas, S.M. (2015). Clinical practice guideline for the treatment of OSA and snoring with oral appliance therapy: An update for 2015. *Journal of Clinical Sleep Medicine, 11*(7), 773-827. doi: 10.5664/jcsm.4858

Chin, C. (2016). High blood pressure: Understanding blood pressure readings. National Heart Centre Singapore Department of Cardiology. Dallas, Texas. Retrieved from https://www.healthxchange.sg/high-blood-pressure/essential-guide-high-blood-pressure/high-blood-pressure-BP-reading

Christensen, H.M., Faber, J., Flyvbjerg, A., Frystyk, J., Kistrop, C. & Schou, M. (2013). Body mass index in chronic heart failure: Association with biomarkers of neurohormonal activation, inflammation and endothelial dysfunction. *BMC Cardiovascular Disord., 13*(1), 80. doi: 10.1186/1471-2261-13-80

Cistulli, P.A., Chan, A.S.L., Darendeliler, M.A., Lee, R.W.W., Petocz, P., Schwab, R. … Zeng, B. (2009). The effect of mandibular advancement on upper airway structure in obstructive sleep apnoea. *Thorax,* 65(8), 726-32. doi:10.1136/thx.2009.131094.

Cistulli, P.A., Darendeliler, M. A., Grunstein, R. R., Marks, G.B., Mihailidou, A.S., Phillips, C.L.…Yee, B.J. (2013). Health outcomes of cpap versus oral appliance treatment for obstructive. *American Journal of Respiratory and Critical Care Medicine*, *187*(8), 879-87. doi: 10.1164/rccm.201212-2223OC

Cistulli, P.A., Gagnadoux, F., Kushida, C.A., Marklund, M., Sutherland, K., Tsuda, H., & Verbaecken, J. (2014a). Oral appliance treatment for obstructive sleep apnea: An update. *Journal of Clinical Sleep Medicine, 10*(2), 215-227. doi: 10.5664/jcsm.3460

Cistulli, P.A., Lowe, A. A., Pantino, D., Prinsell, J., Remmers, J., & Rogers, R. R. (2014b). History of dental sleep. *Journal of Dental Sleep Medicine, 1*(1), 67-74. Retrieved from https://aadsm.org/docs/JDSM.1.1.67.pdf

Cistulli, P.A. & Sutherland, K. (2011). Mandibular advancement splints for the treatment of sleep apnoea syndrome. *Swiss Medical Weekly, The European Journal of Medical Sciences, 141*(w13276), 1-10. doi:10.4414/smw.2011.13276

Clark, A.L., Fonarow, G.C., & Horwich, T.B. (2014). Obesity and the obesity paradox in heart failure. *Progress in Cardiac Disease, 56*(4), 409-414. doi: 10.1016/j.pcad.2013.10.004

Clark, K. P., Ehlen, J. C., & Paul, K. N. (2016). Race and gender disparities in sleep-disordered breathing. *Journal Sleep Disorder Treat Care 6*(1), 00-00. doi: 10.4172/2325-9639.1000185

Clavel, M.A., Tribouilloy, C., & Vanoverschelde, J.L. (2016). Association of b-type natriuretic peptide with survival in patients with degenerative mitral regurgitation. Journal of American *College of Cardiology, 68*(12), 1297-1307. doi: 10.1016/j.jacc.2016.06.047

Cleveland Clinic (2018a). Do you snore? How sleep apnea can hurt your heart. Cleveland, Ohio. Retrieved from https://health.clevelandclinic.org/do-you-snore-how-sleep-apnea-can-hurt-your-heart/

Cleveland Clinic (2018b). NT-proB-type natriuretic peptide (BNP). Cleveland Clinic Foundation. Cleveland, Ohio. Retrieved from https://my.clevelandclinic.org/health/diagnostics/16814-nt-prob-type-natriuretic-peptide-bnp

Colvin-Adams, M., Sharma, A., Yancy, C.W. (2014). Heart failure in African Americans: disparities can be overcome. *Cleveland Clinic Journal of Medicine, 81*(5), 301-311. doi:10.3949/ccjm.81a.13045

Crawford, C. and Khorsan, R. (2014). External validity and model validity: A conceptual approach for systematic review methodology. *Evidence-Based Complementary and Alternative Medicine, 2014*(694804), 12. doi: 10.1155/2014/694804

Dallas, P., Holty, J.C., Owens, D.K., Qaseem, A., Shekelle, P. (2014). Diagnosis of obstructive sleep apnea in adult: A clinical practice guide from the American college of physicians. *Annals of Internal Medicine, 161*(3), 210-220. doi: 10.7326/M12-3187

Dare, S., Mackay, D. F., & Pell, J. P. (2015). Relationship between smoking and obesity: A cross-sectional study of 499,504 middle-aged adults in the United Kingdom general population. *PLoS ONE*, *10*(4), e0123579. doi: 10.1371/journal.pone.0123579

Dasheiff, R.M. & Finn, R. (2009). Efficient treatment of obstructive sleep apnea syndrome. *Journal of Oral Maxillofac Surg, 67*(1), 2171-2182. Retrieved from http://www.breathesleepandbewell.com/articles/osa-thornton.pdf

Davis-Kean, P., Jager, J. & Maslowsky, J. (2015). Answering development questions using secondary data. *Child Development Perspectives, 9*(4), 256-261. doi: 10.1111/cdep.12151

Del Gobbo, L.C., Imamura, F., Kalantarian, S., Lemaitre, R., Mozaffarian, D., Psaty, B.M., & Siscovick, D.S. (2015). Contribution of major lifestyle risk factors for

incident heart failure in older adults: The cardiovascular health study. *JACC: Heart Failure, 3*(7), 520-528. doi: 10.1016/j.jchf.2015.02.009

Dempsey, J., Palta, M., & Young, T. (2019). The occurrence of sleep-disordered breathing among middle-aged adults. *New England Journal of Medicine, 328*(17), 1230-1235. Retrieved from https://www.ncbi.nlm.nih.gov/pubmed/8464434

Deng, S., Jiang, Y., Li, L., & Li, X. (2019). Outlier detection based on robust mahalanobis distance and its application. *Open Journal of Statistics, 9*(01), 15-26. doi: 10.4236/ojs.2019.91002

Department of Veterans Affairs Office of Inspector General (2015). Review of community-based outpatient clinics and other outpatient clinics of north Florida/ south Georgia. Report No. 15-00143-372. Washington, D.C. Retrieved from https://www.va.gov/oig/pubs/VAOIG-15-00143-372.pdf

Department of Veteran Affairs (2015a). About VA. Mission, vision, core values & goals. Retrieved from: http://va.gov/about_va/Mission.asp

Department of Veteran Affairs (2015b). Thornton adjustable positioner. North Texas dental laboratory product Lineup. Washington, District of Columbia. Retrieved from: http://vaww.va.gov/CDL/NTDL/NTDLTAP.asp

Department of Veterans Affairs. (2016a). National center for veteran analysis and statistics. Washington, District of Columbia. Retrieved from: https://www.va.gov/vetdata/veteran_population.asp

Department of Veterans Affairs. (2016b). Veterans population projections 2017-2037. Washington, District of Columbia. Retrieved from:

https://www.va.gov/vetdata/docs/Demographics/.../Vetpop_Infographic_Final31.pdf

Dever, J.A. and Valliant, R. (2014) Estimation with non-probability surveys and the question of external validity. Statistics Canada Symposium. Ontario, Canada. Retrieved from https://www.statcan.gc.ca/sites/default/files/media/14288-eng.pdf

Dudley, K. A., & Patel, S. R. (2016). Disparities and genetic risk factors in obstructive sleep apnea. *Sleep Medicine*, *18*(1), 96–102. doi: 10.1016/j.sleep.2015.01.015

Dixon-Williams, S., Krishnan, V., & Thornton, J. D. (2014). Where there is smoke…there is sleep apnea: Exploring the relationship between smoking and sleep apnea. *Chest, 146*(6), 1673–1680.doi: 10.1378/chest.14-0772

Downs, J.W., Langton, R.S., Neyra, J., & Niebuhr, D.W. (2016). The relationship between enlistment body mass index and the development of obstructive sleep apnea in the United States military. *Military Medicine*, *181*(8), 913–919. doi: 10.7205/MILMED-D-15-00295

Dudovskiy, J. (2019). Purposive sampling. [Blog Post]. Research Methods. Retrieved from https://research-methodology.net/sampling-in-primary-data-collection/purposive-sampling/

Dunlay, S. M., & Roger, V. L. (2014). Understanding the epidemic of heart failure: Past, present, and future. *Current Heart Failure Reports*, *11*(4), 404–415. http://doi.org/10.1007/s11897-014-0220-x

Duran, N., George, S., & Norris, K. (2014). A systematic review of barriers and facilitators to minority research participation among African Americans, Latinos,

Asian Americans and Pacific Islanders. *American Journal of Public Health, 104*(2), e16-e31. doi: 10.2105/AJPH.2013.301706

Eisele, H. Markart, P. & Schulz, R. (2015). Obstructive sleep apnea, oxidative stress, and cardiovascular disease: Evidence from human studies. *Oxid Med Cell Longev, 2015*(608438), 1-9. doi: 10.1155/2015/608438

Emerson, R.W. (2015). Convenience sampling, random sampling, and snowball sampling: How does sampling affect the validity of research? *Journal of Visual Impairment & Blindness, 109*(2), 164. Retrieved from https://search.proquest.com/openview/957d4455fad5e340326811abffe0e088/1?pq-origsite=gscholar&cbl=2027465

Emory Healthcare (2018). Heart failure statistics. Atlanta, GA. Retrieved from https://www.emoryhealthcare.org/heart-vascular/wellness/heart-failure-statistics.html

Enseleit, F., Frohlich, G.M., Keller, P., Luscher, T.F., Noll, G., Ruschitzka, F., Schoch, B.… Sudano, I. (2012). Takotsubo cardiomyopathy has a unique cardiac biomarker profile: Nt-probnp/myoglobin and nt-probnp/troponin t ratios for the differential diagnosis of acute coronary syndromes and stress induced cardiomyopathy. *International Journal of Cardiology, 154*(3), 328-332. doi: 10.1016/j.ijcard.2011.09.077

Ergin, E.S., Kibar, S. & Konak, H. E. (2016). The effect of single-task and dual-task balance exercise programs on balance performance in adults with osteoporosis: A

randomized controlled preliminary trial. *Osteoporosis International, 27(11)*, 3271-3278. doi: 10.1007/s00198-016-3644-1

Espadas, D., Esqivel, A., Hysong, S., Paul, L. A., Singh, H., Singh, S. & Sitting, D. F. (2011). Towards successful coordination of electronic health record based-referrals: A qualitative analysis. *Implementation Science, 6*(1), 84. doi: 10.1186/1748-5908-6-84

Farinde, A. (2015). Lecture on sleep apnea. American Sleep Association. Lititz, Pennsylvania. Retrieved from https://www.sleepassociation.org/sleep-disorders/sleep-apnea/intro-lecture-on-sleep-apnea/

Farmakis, D., Papingiotis, G., & Parissis, J. (2017). Acute heart failure: Epidemiology and socioeconomic burden. *Continuing Cardiology Education, 3*(3), 88-92. Retrieved from https://doi.org/10.1002/cce2.61

Davidson, T.M., Findley, L.J., George, C., Goldlust, E., Kryger, M. & Sassani, A. (2004). Reducing motor-vehicle collisions, costs, and fatalities by treating obstructive sleep apnea syndrome. *Sleep, 27*(3), 453-458. doi: 10.1093/sleep/27.3.453

Finkel Dental Forum. (2015, April 23). *Obstructive Sleep Apnea Part 10: Oral Appliances, Appliance Selection* [Video File]. Retrieved from https://www.youtube.com/watch?v=DyO3bg1Dwoc

Flannelly, K.J., Flannelly, L.T. & Jankowski, K.R. (2018). Threats to the internal validity of experimental and quasi-experimental research in healthcare. *Journal of Health Care Chaplaincy, 24*(1), 1-24.doi: 10.1080/08854726.2017.1421019

Floras, J.S. (2017). Ambulatory apnea monitoring heart failure. Journal of the American College of Cardiology. *Circulation, 70* (11), 1365-1367. doi: 10.1016/j.jacc.2017.07.761

Food and Drug Administration (2017). Code of federal regulations Title 21. Dental services. Department of Health and Human Services subchapter h. *Code of federal regulations, 21*(8), 872. Retrieved from https://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfcfr/cfrsearch.cfm?fr=872.5570

Gaggin, H.K. & Januzzi, J.L. (2015). Cardiac biomarkers and heart failure. American College of Cardiology. Washington, District of Columbia. Retrieved from http://www.acc.org/latest-in-cardiology/articles/2015/02/09/13/00/cardiac-biomarkers-and-heart-failure

Gapstur, S.M., Hildebrand, J.S., & Patel, A. V. (2014). Body mass index and all-cause mortality in a large prospective cohort of white and black United States adults. *PLoS ONE, 9*(10), e109153. doi: 10.1371/journal.0109153

Garson, G.D. (2012). Testing statistical assumptions. North Carolina State University School of Public and International Affairs. Asheboro, North Carolina. Retrieved from file:///C:/Users/Tracey/Downloads/testing%20statistical%20assumptions.pdf

Gastel, M., Hann, G., & Stuijk, S. (2016). New principle for measuring arterial blood oxygenation, enabling motion-robust remote monitoring. *Scientific Reports, 6*(1), *38609*. doi: 10.1038/srep38609

Gastwirth, J.L.,Gel, Y.R., & Miao, W. (2009). The impact of levene's test of equality of

Variances on statistical theory and practice. *Statistical Science 24*(3), 343-360. doi: 10.1214/09-STS301

Georgia Southern University (n.d.). Experimental research: Control procedures, experimental designs, internal and external validity. Statesboro, Georgia. Retrieved from http://www.bwgriffin.com/gsu/courses/edur7130/content/experimental_research.htm

Gillespie, M.B., Knauret, M., & Naik, S. (2015). Clinical consequences and economic costs of untreated obstructive sleep apnea syndrome. *World Journal of Otorhinolaryngology-Head and neck Surgery, 1*(1), 17-27. doi: 10.1016/j.wjorl.2015.08.001

Gladyshev, V. N. (2014). The free radical theory of aging is dead. Long live the damage theory! *Antioxidants & Redox Signaling*, *20*(4), 727–731. doi: 10.1089/ars.2013.5228

G Power (2017). Power analysis and sample size estimations. Düsseldorf, Germany. Retrieved from www.gpower.hhu.de/fileadmin/redaktion/Fakultaeten/.../gpower/GPowerManual.pdf

Goff, D.M., Schwartz, B.M. & Wilson, J.H. (2018). Repeated measures: Everybody plays. [Adobe Digital Editions version]. Retrieved from https://us.sagepub.com/sites/default/files/upm-

binaries/91135_Chapter_8_Pages_from_Schwartz__An_EasyGuide_to_Research_Design_%26_SPSS_2e_2.pdf

Gordon, T., Kannel, W.B., & Schwartz, M.J. (1971). System versus blood pressure and risk of coronary heart disease. The framingham study. *American Journal of Cardiology, 27*(4), *335-346.* doi: 10.1016/0002-9149(71)90428-0

Goswami, S. K., Maulik. S. K. (2015). Oxidative stress in cardiovascular diseases. *Journal of the Practice of Cardiovascular Sciences, 1*(1), *15-18.* doi: 10.4103/2395-5414.157555

Greenberg, B. (2017). Use of the wearable cardioverter defibrillator in cardiac patients at high risk of sudden arrhythmic death. Heart failure 2017- An update on therapy. San Diego, California. Retrieved from https://pdfs.semanticscholar.org/presentation/174e/6a1e8ea410f161fbc1cfb3e270e420245cb1.pdf

Han, J. & Zhu, Q. (2014). Effects of different sport participations on prosocial and antisocial behaviors. Korean Institute of Sports Science. Retrieved from http://ijass.sports.re.kr/_common/do.php?a=current&b=21&bidx=1595&aidx=20056

Harris, S. M. (2013). Development of the perceptions of mentoring relationships survey: A mixed method approach. *International Journal of Multiple Research Approaches. 7*(1), 83-95. Scholarworks. Retrieved from https://search-ebscohost-com.ezp.waldenulibrary.org/login.aspx?direct=true&db=ir00976a&AN=wldu.sp.pubs.1086&site=eds-live&scope=site

Hawkes, W., Mendelson, A., Su, Z. & Yan, X. (2016). Missing data in observational studies. Quintiles. Durham, North Carolina. Retrieved from https://www.ispor.org/Event/GetReleasedPresentation/716

Heron, M. (2017). Deaths: Leading causes for 2015. National Vital Statistics Reports, 66(5) -75. Hyattsville, Maryland. Retrieved from https://www.cdc.gov/nchs/data/nvsr/nvsr66/nvsr66_05.pdf

Hoare, Z & Hoe, J. (2012). Understanding quantitative research: part 1. *Nursing Standard, 27*(15-17), 52-57. Doi: 10.7748/ns2012.12.27.15.52.c9485

Horn, R. A. (n.d.). Sphericity in repeated measures analysis of variance. Northern Arizona University. Retrieved from: http://oak.ucc.nau.edu/rh232/courses/EPS625/Handouts/RM-ANOVA/Sphericity.pdf

Huang, B., Ray, P.D., Tsuji, Y. (2012). Reactive oxygen species (ROS) homeostasis and redox regulation in cellular signaling. *Cell Signal, 24*(5), *981-990.* doi: 10.1016/j.cellsig.2012.01.008

Hughes, A.L., Matt, J.J. & O'Reilly, F.L. (2014). Principal support is imperative to the retention of teachers in hard-t-o-staff-schools. *Journal of Education and Training Studies, 3*(1), 129-134. doi: 10.11114/jets.v3i1.622

Hwang, D., Mereddy, S., Parthasarthy, S., Pepin, J.L., & Tamisier, R. (2017). Does remote monitoring change obstructive sleep apnea management and continuous positive airway pressure adherence? *Respiratory Sleep Disorders, 22*(8), 1508-1517. doi: 10.1111/resp.13183

International Business Machines (n.d.a.). Missing analysis values. International Business Machines Knowledge Center. Armonk, New York. Retrieved from https://www.ibm.com/support/knowledgecenter/en/SSLVMB_22.0.0/com.ibm.spss.statistics.help/spss/mva/idh_miss.htm

Institute of Medicine (2015). Public health implications of raising the minimum age of legal access to tobacco products. The National Academies Press. Washington, District of Columbia. doi: 10.17226/18997

Jarolim, P., and Mahajan, V. (2011). How to interpret elevated cardiac troponin levels. American Heart Association. *Circulation,* 124(21), 2350-2354. doi: 10.1161/CIRCULATIONAHA.111.023697

Javaheri, A., Javaheri, S., & Javaheri, S. (2013). Sleep apnea, heart failure, and pulmonary hypertension. *Current Heart Failure Reports*, *10*(4), 315–320. doi:10.1007/s11897-013-0167-3

Jeyabalasingham, A., Lanka, S., Ragavan, A. (2011). Repeated measures analysis of correlated data with multiple responses using SAS. SAS Global Forum. Reno, Nevada. Retrieved from http://support.sas.com/resources/papers/proceedings11/231-2011.pdf

Kang, H. (2013). The prevention and handling of the missing data. *Korean Journal of Anesthesiology, 64*(5), 402-406. doi: 10.4097/kjae.2013.64.5.402

Kasai, T., Kato, T., & Suda, S. (2014). Positive airway pressure therapy for heart failure. *World Journal of Cardiology, 6*(11), 1175-1191. doi: 10.4330/wjc.v6.i11.1175

Kent State University (2019). Spss Tutorials: Pearson Correlation. Retrieved

https://libguides.library.kent.edu/SPSS/PearsonCorr

Kimmenade, R.R.J. and Januzzi, J. L. (2011). Emerging biomarkers in heart failure. *Clinical Chemistry, 58*(1), 127-138. doi: 10.1373/clinchem.2011.165720

Lagouge, M. and Larsson, N. G. (2013). The role of mitochondrial DNA mutations and free radicals disease and ageing. *Journal of Internal Medicine, 273*(6), 529-543. doi:10.1111/joim.12055

Lakens, D. (2013). Calculating and reporting effect sizes to facilitate cumulative science: A practical primer for t-tests and ANOVAs. *Frontiers in Psychology, 4*(1), 863. doi: 10.3389/fpsyg.2013.00863

LaMorte, W.W. (2016). Diffusion of innovation theory. Behavioral change models. Boston University School of Public Health. Boston, Massachusetts. Retrieved from http://sphweb.bumc.bu.edu/otlt/MPH-Modules/SB/BehavioralChangeTheories/BehavioralChangeTheories4.html

Lee, M., McCormick, J.B., Rahbar, M.H., & Vatcheva, K.P. (2016). Multicollinearity in regression analyses conducted in epidemiologic studies. *Epidemiology, 6*(2), 227. doi: 10.4172/2161-1165.1000227

Levy, D., Mahmood, S.S., Vasan, R.S., & Wang, T.J. (2013). The framingham heart study and the epidemiology of cardiovascular diseases: A historical perspective. *Lancet, 383*(9921), 999-1008. doi: 10.1016/S0140-6736(13)61752-3

Lombardi, C., Losurdo, A., Montano, N., Parati, G., & Tobaldini, E. (2017). Obstructive sleep apnea syndrome and cardiovascular system. *Medicina del Lavoro, 108*(4), 276-282. doi: 10.23749/mdl.v108i4.6427

Lund, A. and Lund, M. (2013a). One-way MANOVA in SPSS statistics. Laerd Statistics. Lund Research LTD. Derby, United Kingdom. Retrieved from https://statistics.laerd.com/spss-tutorials/one-way-manova-using-spss-statistics.php

Lund, A. and Lund, M. (2013b). One-Way MANCOVA in SPSS statistics. Laerd Statistics. Lund Research LTD. Derby, United Kingdom. Retrieved from https://statistics.laerd.com/spss-tutorials/one-way-mancova-using-spss-statistics.php

Lund, A. and Lund, M. (2018). One-Way repeated measures MANOVA in SPSS statistics. Laerd Statistics. Lund Research LTD. Derby, United Kingdom. Retrieved from https://statistics.laerd.com/spss-tutorials/one-way-repeated-measures-manova-using-spss-statistics.php

Lung, L.H. & Saverese, G. (2017). Global public health burden of heart failure. *Cardiac Failure Review, 3*(1), 7-11. doi: 10.15420/cfr.2016:25:2

Mangla, A. (2014). Brain-type natriuretic peptide. Reference range. Medscape. New York City, New York. Retrieved from https://emedicine.medscape.com/article/2087425-overview

Marklund, M., Randerath, W., & Verbaecken, J. (2012). Non-continuous positive airway pressure therapies in obstructive sleep apnoea: mandibular advancement device therapy. *European Respiratory Journal, 39*(5), 1241-7. doi: 10.1183/09031936.00144711

Mayo Clinic (2018a). Heart failure. Scottsdale, Arizona. Retrieved from https://www.mayoclinic.org/diseases-conditions/heart-failure/symptoms-causes/syc-20373142

Mayo Clinic. (2018b). Ejection fraction: What does it measure? Rochester, Minnesota. Retrieved from https://www.mayoclinic.org/ejection-fraction/expert-answers/faq-20058286

Mertler, C.A. &Vannatta, R.A. (2002). Advanced and multivariate statistical methods Practical application and interpretation. (2nd ed). Los Angeles:Pyrczak Publishing

Mordkoff, T. (2016). The Assumptions of Normality. Iowa City, Iowa. Retrieved from www2.psychology.uiowa.edu

National Institute of Health (2017). Sleep apnea. Bethesda, Maryland. Retrieved from https://www.nhlbi.nih.gov/health-topics/sleep-apnea

National Institute of Health (2020). Cigarettes and other tobacco products. Bethesda, Maryland. Retrieved from https://www.drugabuse.gov/publications/drugfacts/cigarettes-other-tobacco-products

Nayaki, S., Simi, M., & Sudheep, M. (2017). An extensive study on data anonymization algorithms based on k-anonymity. *IOP Conf. Series: Materials Science and Engineering, 225*, 012279. Retrieved from https://iopscience.iop.org/article/10.1088/1757-899X/225/1/012279/pdf

Negreiros, J.G.M (2018). Spatial analysis techniques using mygeoffice. Ch.4 Spatial Regression, Pg. 89. Retrieved from https://books.google.com

Nimmo-Smith, I. (2009). Repeated measures & mixed model anova within & between-subject design. University of Cambridge, England. Retrieved from imaging.mrc-cbu.cam.ac.uk/statswiki/StatsCourse2009?action...do...Repeated...

Northern Arizona University (n.d.) Understanding the one-way anova. Retrieved from http://oak.ucc.nau.edu/rh232/courses/EPS525/Handouts/Understanding%20the%20One-way%20ANOVA.pdf

Ohn, K.M. & Tin, W. (2016). Modalities of treatment for sleep disordered breathing. *Asian Journal Medical and Biological Research, 2*(3), 361-369. doi: 10.3329/ajmbr.v2i3.30105

Palay, R. M. (2016). The chi-squared critical values table. Washtenaw Community College. Ann Arbor, Michigan. Retrieved from http://courses.wccnet.edu/~palay/math160r/chisqtable.htm

Patten, M.L. (2012). Understanding research methods: An overview of essentials. [Adobe Digital Editions version]. Retrieved from: http://web.mnstate.edu/malonech/Psy633/Articles/PreExperimental%20and%20IntExt%20Validity.pdf

Pennsylvania State University (2019). 4.4 – multivariate normality and outliers. Retrieved from: https://newonlinecourses.science.psu.edu/stat505/lesson/4/4.4

Pinto VL and Sharma S. (2018). Continuous positive airway pressure. Treasure Island, FL. Retrieved from: https://www.ncbi.nlm.nih.gov/books/NBK482178/

Plurphanswat, N. and Rodu, B. (2014). The association of smoking and demographic characteristic on mass index and obesity among adults in the United States, 1999-2012. *BMC Obes, 1*(1), 18. doi: 10.1186/s40608-014-0018-0

Popat, R.A. & Sainani, K.L. (2011). Understanding study design. Statistically speaking. *American Academy of Physical Medicine and Rehabilitation, 3*(6), 573-577. doi: 10.1016/j.pmrj.2011.04.001

Price, P. C., Jhangiani, R.S. & Chiang, I-C. A (2013). Overview of non-experimental research-research methods in psychology. [Adobe Digital Editions version]. Retrieved from https://opentextbc.ca/researchmethods/chapter/overview-of-nonexperimental-research/

Pum, J. (2019). A practical guide to validation and verification of analytical methods in the clinical laboratory. *Advances Clinical Chemistry, 90*(1), 215-281. Retrieved from https://www.sciencedirect.com/topics/engineering/outlier-detection

Ramzan, S., Ramzan, S. & Zahid, F.M. (2013). Evaluating multivariate normality: A graphical approach. *Middle-East Journal of Scientific Research, 13*(2), 254-263. doi: 10.5829/idosi.mesjr.2013.13.2.1746

Resmedica (2011). Thirty years of CPAP. Resmedica newsletter. San Diego, California Retrieved from *https://www.resmed.com/au/dam/documents/articles/clinical.../resmedica14.pdf*

Richardson, A.G. & Schadt, E. E. (2014). The role of macromolecular damage in aging

and age-related disease. *The Journals of Gerontology: Series A, 69*(1), S28-S32. Retrieved from https://academic.oup.com/biomedgerontology/article/69/Suppl_1/S28/587011

Rohrer, J.M. (2018). Thinking clearly about correlations and causation: Graphical causal models for observations data. Advances in Methods and Practices in Psychological Science, 1(1), 27-42. doi:10.1177/25152459917745629

Rommel-Esham, K. (2010). Designing quantitative research. Geneseo, New York. Retrieved from https://www.geneseo.edu/~rommel/educ504/ch_9.pdf

Ruppert, D. (2011). Statistics and data analysis for financial engineering. Ch2, pg 15. Retrieved from https://faculty.washington.edu/ezivot/econ424/RuppertChapter2.pdf

Sanz, A. (2016). Mitochondrial reactive oxygen species: Do they extend or shorten animal lifespan. *Biochimica et Biophysica Acta- Bioenergentics, 1857*(8), 1116-1126. doi: 10.1016/j.bbabio.2016.03.018

Simon, M.K. (2011). Dissertation and scholarly research: Recipes for success. Seattle, Washington. Retrieved from http://dissertationrecipes.com/wp-content/uploads/2011/04/AssumptionslimitationsdelimitationsX.pdf

Statista (2019). The largest armies in the world based on active military personnel in 2019. Retrieved from https://www.statista.com/statistics/264443/the-worlds-largest-armies-based-on-active-force-level/

Statisticshowto (2018a). Box's m test. Greencove, Florida. Retrieved from http://www.statisticshowto.com/boxs-m-test/

Statisticshowto (2018b). Repeated measures design/ crossover design. Greencove, Florida. Retrieved from http://www.statisticshowto.com/repeated-measures/

Statistics Solutions. (2013). Data plan analysis: Repeated measures ANCOVA. Clearwater, Florida. Retrieved from https://www.statisticssolutions.com/data-analysis-plan-repeated-measures-ancova/

Statistics Solutions. (2017). Quantitative research approach. Clearwater, Florida. Retrieved from http://www.statisticssolutions.com/quantitative-research-approach/

Statistics Solutions. (2018). Take the guess work of out sample size: Why and how to conduct a power analysis. Clearwater, Florida. Retrieved from https://www.statisticssolutions.com/take-the-guesswork-out-of-sample-size-why-and-how-to-conduct-a-power-analysis/

Statistics Solutions. (2019a). Testing multivariate normality in SPSS. Clearwater, Florida. Retrieved from https://www.statisticssolutions.com/testing-multivariate-normality-in-spss/

Statistics Solutions. (2019b). Univariate and multivariate outliers. Clearwater, Florida. Retrieved from https://www.statisticssolutions.com/univariate-and-multivariate-outliers/

StatTrek (2019). Correlation coefficient. Retrieved from https://stattrek.com/statistics/correlation.aspx

Steyn, P. (2018, June 26). Outlier cases-univariate outliers [Blog post]. Retrieved from http://www.introspective-mode.org/univariate-outliers/

Study.com (2018). Non-experimental and experimental research differences, advantages & disadvantages. Mountain View, California. Retrieved from https://study.com/academy/lesson/non-experimental-and-experimental-research-differences-advantages-disadvantages.html

Surkin, L.A. (2013). The role of the cardiologist in sleep disordered breathing management: "Opportunity or obligation?" Washington, District of Columbia. Retrieved from http://www.acc.org/latest-in-cardiology/articles/2014/07/22/08/27/the-role-of-the-cardiologist-in-sleep-disordered-breathing-management-opportunity-or-obligation

Taylor, A. (2011). JMASM31: MANOVA procedure for power calculations (SPSS). *Journal of Modern Applied statistical Methods, 10*(2), 33. doi: 10.22237/jmasm/1320121920

Taylor, H. A. (2005). The jackson heart study: an overview. *Ethnicity & Disease, 15*(S6), 1-3. Retrieved from htpps://www.ncbi.nlm.nih.gov/pubmed/16317981

The National Academic Press (2017). The state of health disparities in the United States. Communities in action: Pathways to health equity. [Adobe Digital Editions version]. Retrieved from NAP.edu/read/24624/chapter/4

The National Coordination Office for Networking and Information Technology Research and Development (2016). The federal big data research and development strategic plan. Executive Office of the President. Washington, District of Columbia. Retrieved from https://bigdatawg.nist.gov/pdf/bigdatardstrategicplan.pdf

Trochim, M.K. (2006). Quasi-experimental design. Social research methods knowledge base. New York City, New York. Retrieved from https://www.socialresearchmethods.net/kb/quasiexp.php

United States Census Bureau (2017). Annual estimates of the resident population by sex, race alone or in combination, and Hispanic origin for the united states, states and counties. Washington, District of Columbia. Retrieved from https://factfinder.census.gov/faces/tableservices/jsf/pages/productview.xhtml?src=bkmk

United States Census Bureau (2019). Annual estimates of the resident population by sex, race alone or in combination, and Hispanic origin for the united states, states: April 1, 2010 to July1, 2018. Washington, District of Columbia. Retrieved from https://factfinder.census.gov/faces/tableservices/jsf/pages/productview.xhtml?pid=PEP_2018_PEPALL5N&prodType=table

United States Department of Veterans Affairs (2014). VA informatics and computing infrastructure. Health services research & development. North Florida South Georgia Veteran Health System. Washington, District of Columbia. Retrieved from https://www.hsrd.research.va.gov/for_researchers/vinci/default.cfm

United States Department of Veterans Affairs (2016). VA clinical database research. Malcom Randall VA Medical Center. Washington, D.C. Retrieved from https://www.northflorida.va.gov/NORTHFLORIDA/Research/files/VINCIIntro1.2.docx

United States Department of Veterans Affairs (2018). Sleep medicine. North Florida South Georgia Veteran Health System. Washington, D.C. Retrieved from https://www.northflorida.va.gov/NORTHFLORIDA/services/sleep_center.asp

University of Florida (2014). Investigator requirements for retaining research data. Gainesville, Florida. Retrieved from http://irb.ufl.edu/index/data/investigator-requirements-for-retaining-research-data.html

University of Texas (2015). Pearson correlation and linear regression. Retrieved from http://sites.utexas.edu/sos/guided/inferential/numeric/bivariate/cor/

Watson, N. F. (2016). Health are savings: The economic value of diagnostic and therapeutic care for obstructive sleep apnea. *Journal of Clinical Sleep Medicine*, *12*(8), 1075–1077. doi: 10.5664/jcsm.6034

World Health Organization (2011). Pulse oximetry training manual. Patient safety. Geneva Switzerland. Retrieved from http://www.who.int/patientsafety/safesurgery/pulse_oximetry/who_ps_pulse_oxymetry_training_manual_en.pdf

Wludyka, P. (2012).Study design and their outcomes. Epidemiology for Advanced Nursing Practice. [Adobe Digital Editions version]. Retrieved from http://samples.jbpub.com/9780763789961/89961_CH03_Macha.pdf

Appendix A: Z-score Univariate Outliers

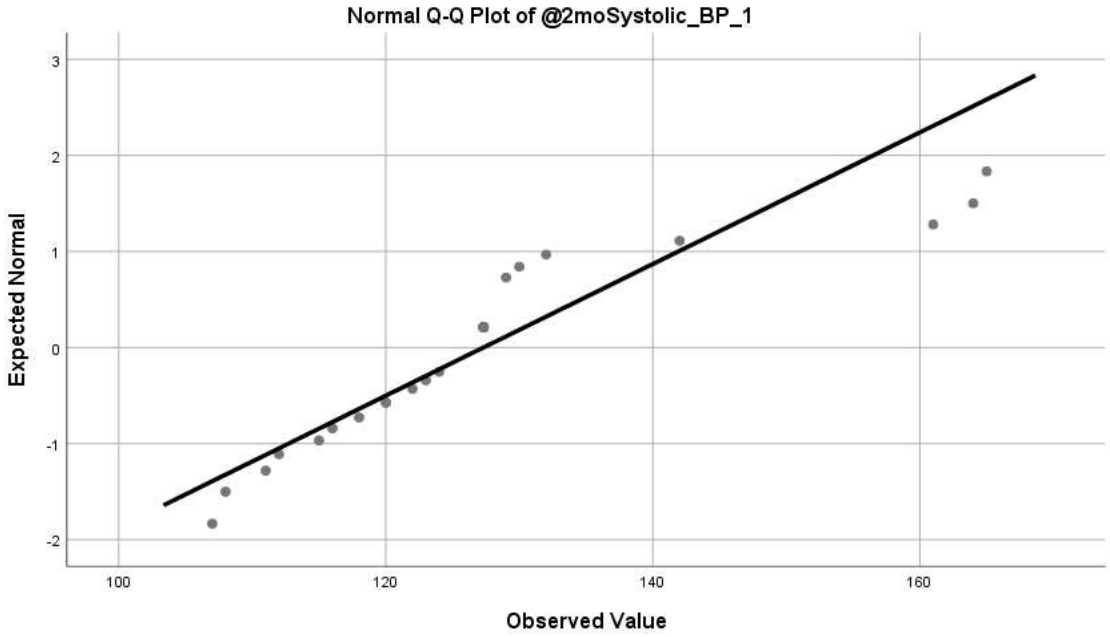
Z@2mo SBP	Z@2mo DBP	Z@2mo O2	Z@4mo SBP	Z@4mo DBP	Z@4mo_O2	Z@6mo SBP	Z@6mo DBP	Z@6mo_O2
.11538	.72160	1.93218	-.92492	-.50233	.00000	.13835	.75733	.00000
-.36417	-1.07060	-.96609	.73020	-.92931	.89148	-2.48309	-1.84600	-3.59255
-.50118	-.71216	.00000	.00000	.00000	.00000	-2.37386	-3.50267	.67967
.00000	.00000	.00000	.56469	1.34792	.03566	-1.50005	.40233	-.38838
.00000	.00000	.00000	.06815	.06698	-.82016	.79371	.40233	.00000
.18389	.27355	.00000	.00000	.00000	.00000	.00000	.00000	.00000
-.50118	-.08489	.00000	-.51114	.35163	.00000	.00000	.00000	.00000
.32090	.99043	.00000	-.01460	.63629	.00000	.00000	.00000	.00000
2.30760	3.94755	.00000	.00000	.00000	.00000	.00000	.00000	.00000
-1.04924	-.98099	.00000	3.13012	.92094	1.31939	1.12139	.40233	.67967
.00000	.00000	.00000	.23366	.77861	.46357	.79371	.87567	.00000
2.58162	.00472	.96609	1.80602	.35163	.00000	2.10443	.87567	.67967
.00000	.00000	.00000	-2.33177	-3.06422	-4.67134	.00000	.00000	.00000
-1.39177	-1.69787	.96609	.00000	.00000	.00000	.00000	.00000	.00000
.00000	.00000	.00000	.73020	1.91723	.00000	.02913	-.66267	.00000
-.22715	-1.16021	.00000	.00000	.00000	.00000	.00000	.00000	.00000
1.00597	.27355	.00000	.00000	.00000	.00000	-.08010	-.78100	.00000
-.77521	.81121	1.93218	.06815	.35163	1.31939	.00000	.00000	.00000
.00000	.00000	.00000	-1.33870	-.21768	.46357	.79371	-1.37267	2.81578
.00000	.00000	.00000	-1.33870	-.78699	.03566	-1.71850	.04733	.67967
-.63819	-.98099	-.96609	1.14398	1.91723	.89148	1.44907	1.94067	-1.45644
.00000	.00000	.00000	.00000	.00000	.00000	-.18933	1.23067	-1.45644
.00000	.00000	.00000	.00000	.00000	.00000	.00000	.00000	.00000
-1.11774	.00472	.96609	-.92492	-1.07164	.03566	.00000	.00000	.00000
-1.32326	-.71216	.00000	-1.09043	-2.06793	.03566	.00000	.00000	.00000
.00000	.00000	.00000	.00000	.00000	.00000	1.12139	1.23067	.67967
2.51312	1.16965	.00000	.00000	.00000	.00000	.00000	.00000	.67967
-.84372	-.17450	-3.86437	.00000	.00000	.00000	.00000	.00000	.00000
-.29566	-.62255	-.96609	.00000	.00000	.00000	.00000	.00000	.00000

Appendix B: MAD Residuals Statistics

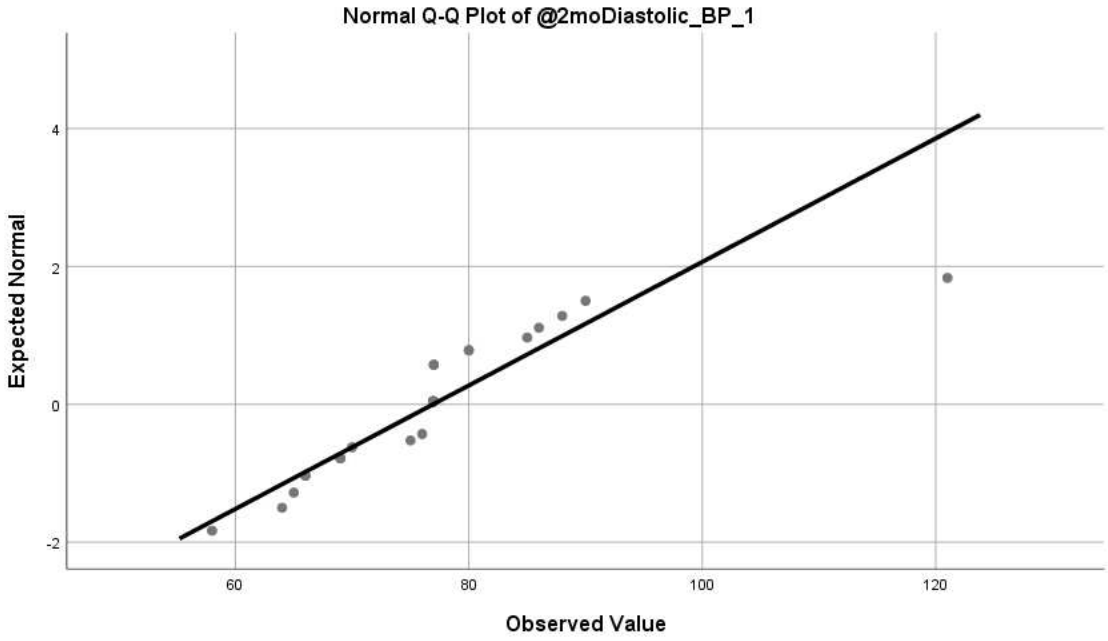
	Minimum	Maximum	Mean	Std. Deviation	N
Predicted Value	45.098	81.485	62.833	7.181	29
Std. Predicted Value	-2.470	2.597	.000	1.000	29
Standard Error of Predicted Value	1.211	6.036	3.553	1.455	29
Adjusted Predicted Value	41.686	88.335	62.373	8.792	29
Residual	-11.059	9.278	.000	5.372	29
Std. Residual	-1.696	1.423	.000	.824	29
Stud. Residual	-2.084	1.904	.011	1.059	29
Deleted Residual	-20.696	26.907	.460	10.079	29
Stud. Deleted Residual	-2.310	2.060	.000	1.112	29
Mahal. Distance	.000	23.021	8.690	7.044	29
Cook's Distance	.001	1.340	.123	.280	29
Centered Leverage Value	.000	.822	.310	.252	29

a. Dependent Variable: Factor1

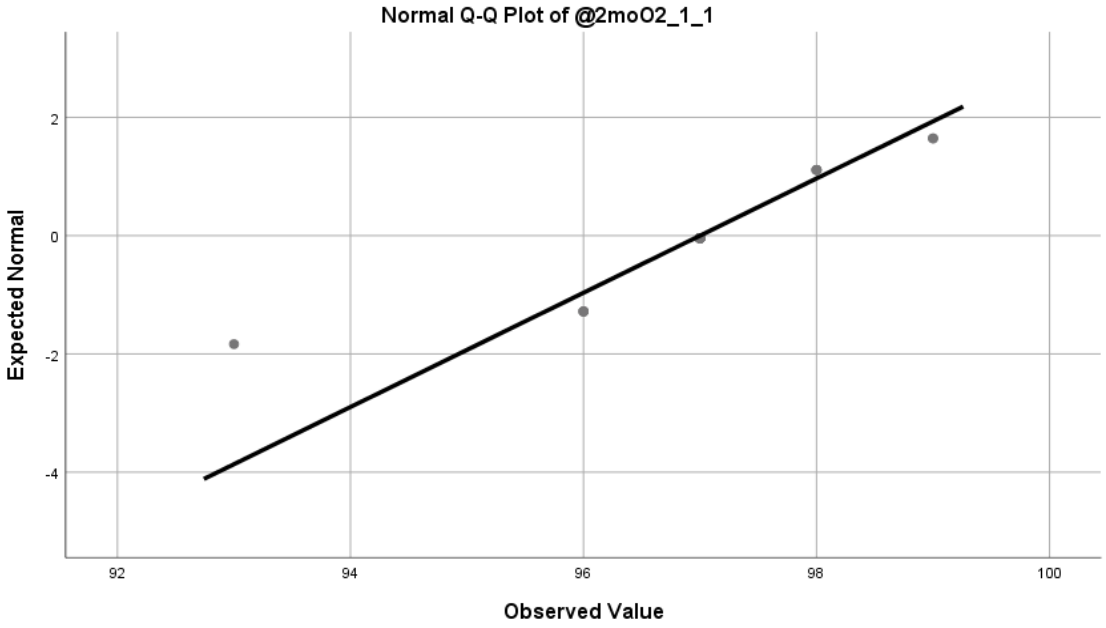
Appendix C: rMANOVA Q-Q Plot for SBP @ 2-months



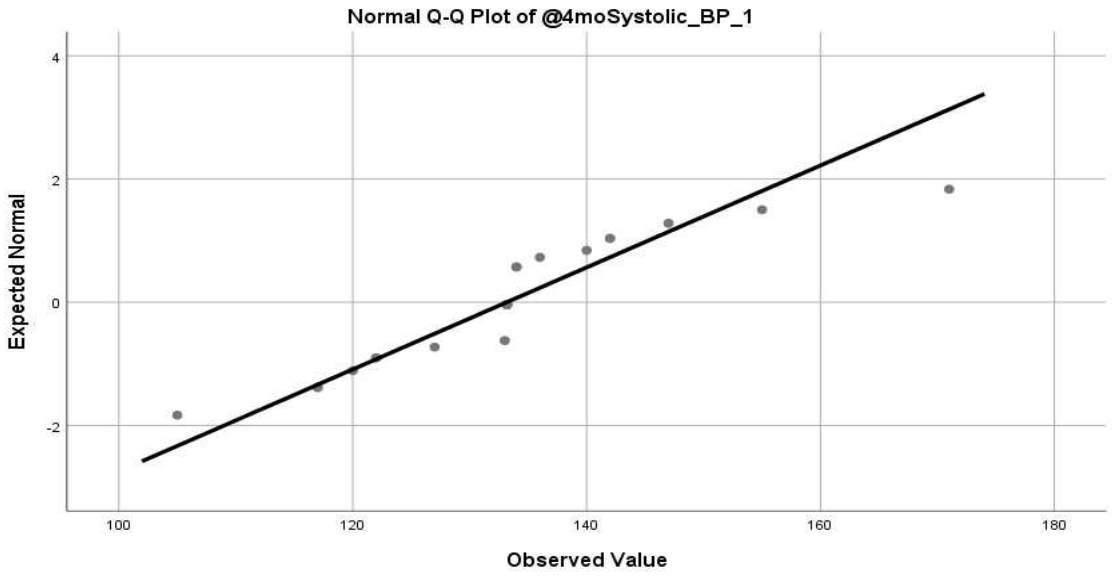
Appendix D: rMANOVA Q-Q Plot for DBP @ 2-months



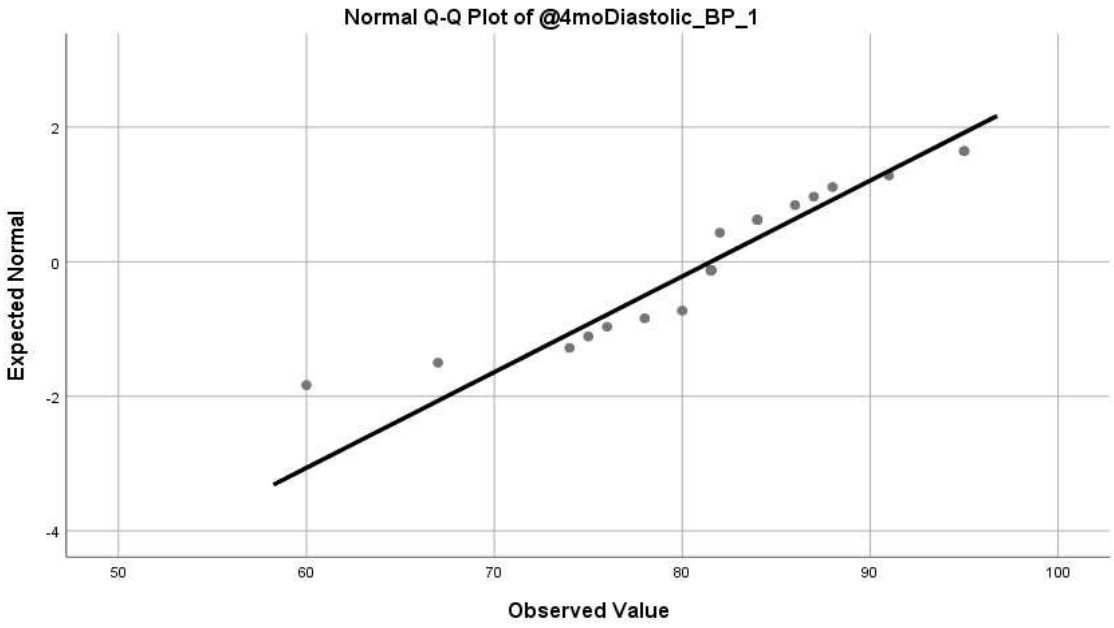
Appendix E: rMANOVA Q-Q Plot for O2sat @ 2-months



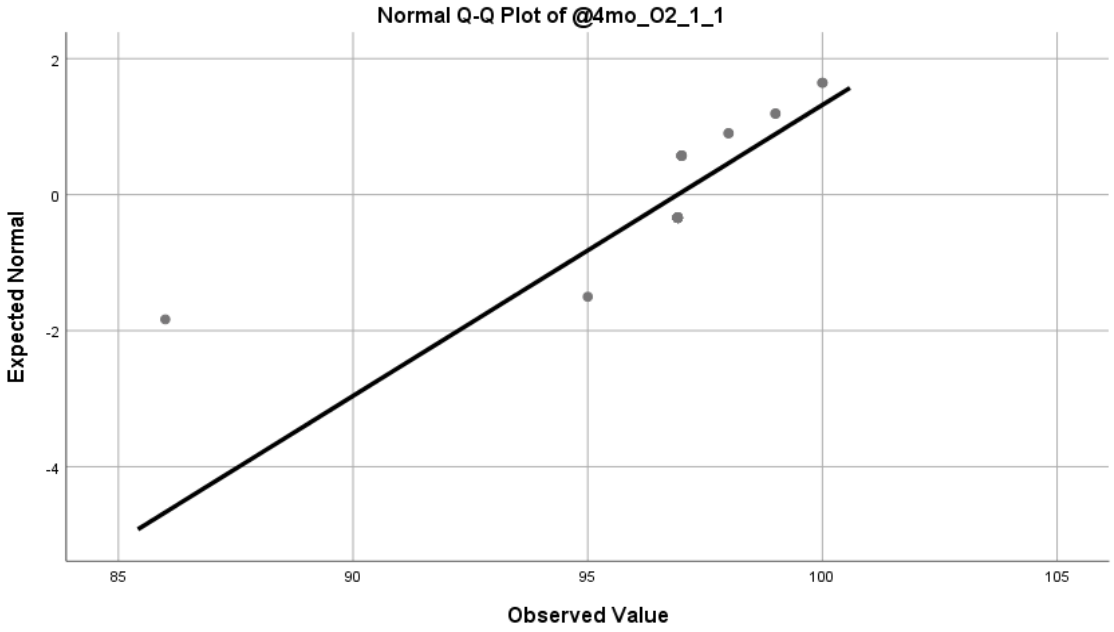
Appendix F: rMANOVA Q-Q Plot for SBP @ 4-months



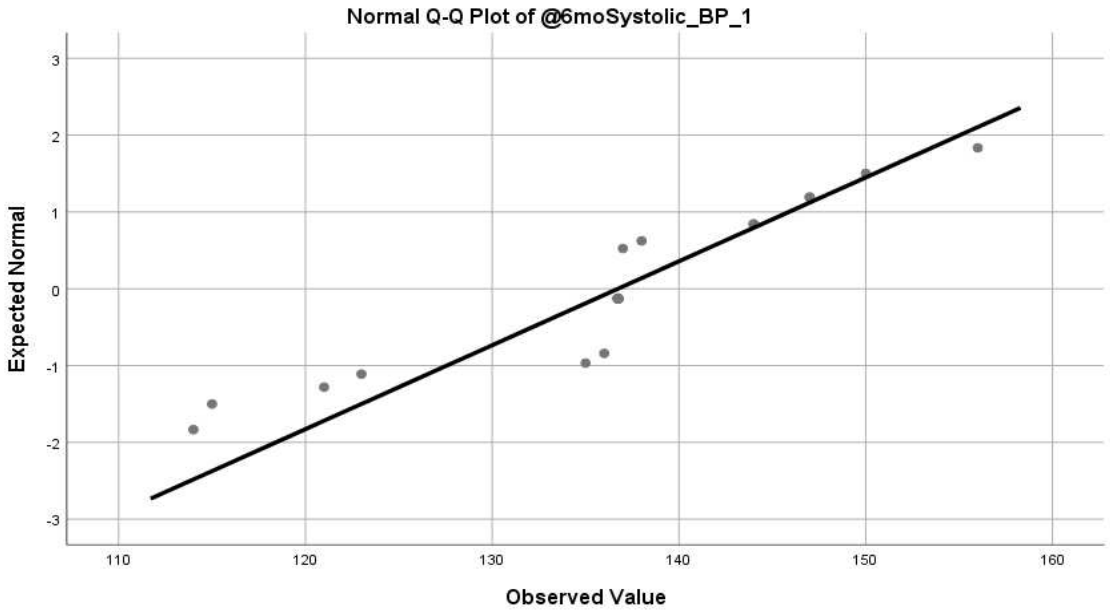
Appendix G: rMANOVA Q-Q Plot for DBP @ 4-months



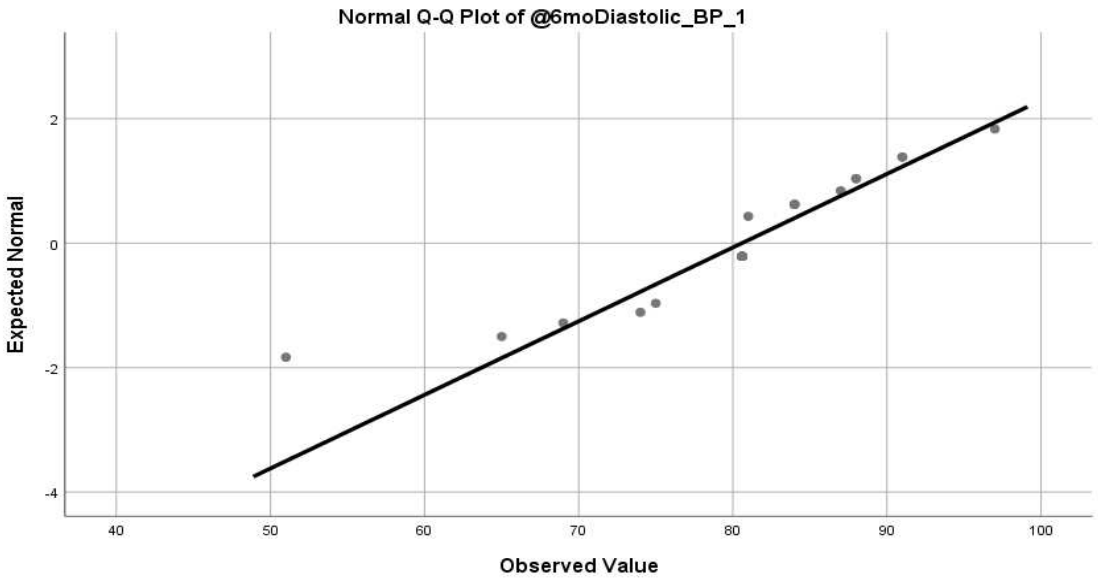
Appendix H: rMANOVA Q-Q Plot for O2sat @ 4-months



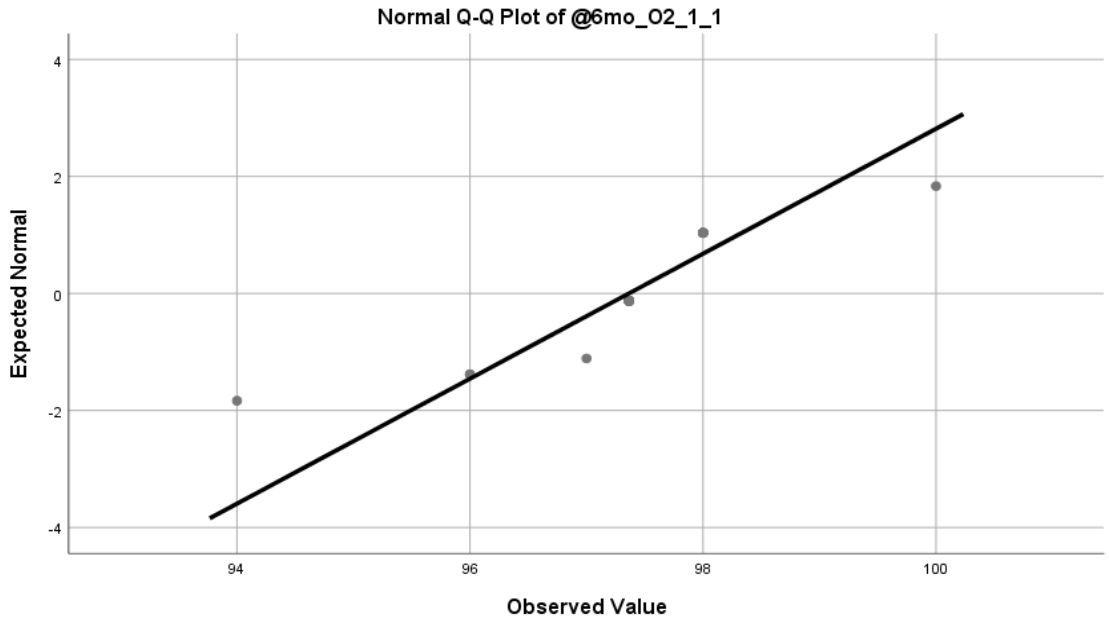
Appendix I: rMANOVA Q-Q Plot for SBP @ 6-months



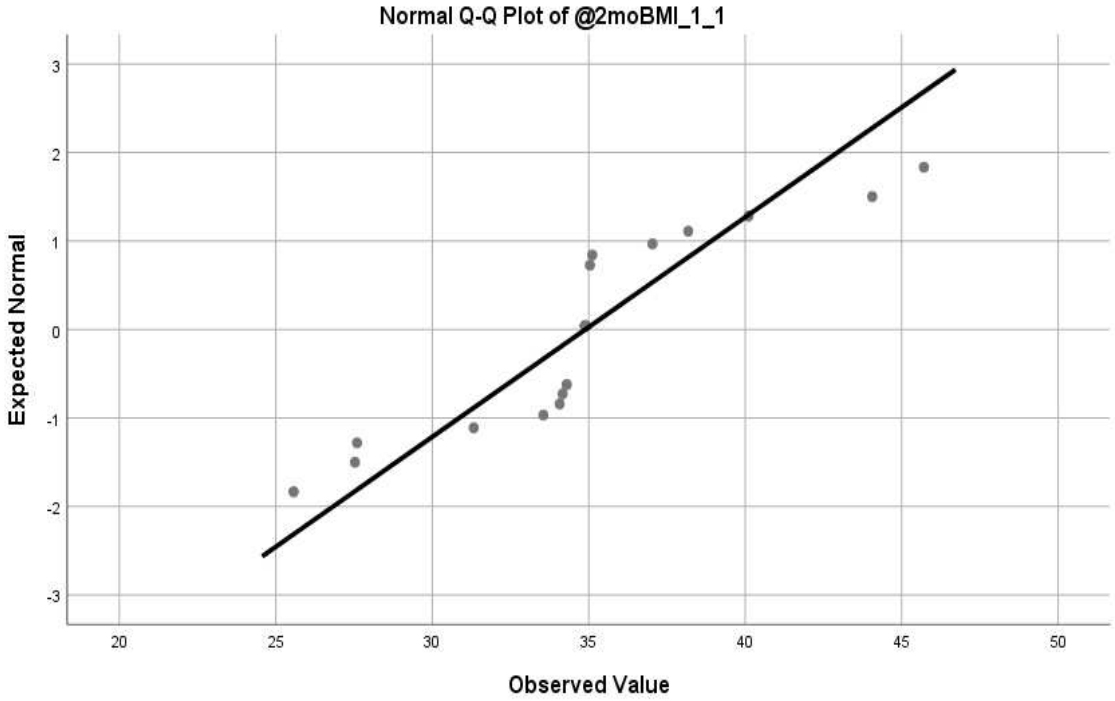
Appendix J: rMANOVA Q-Q Plot for DBP @ 6-months



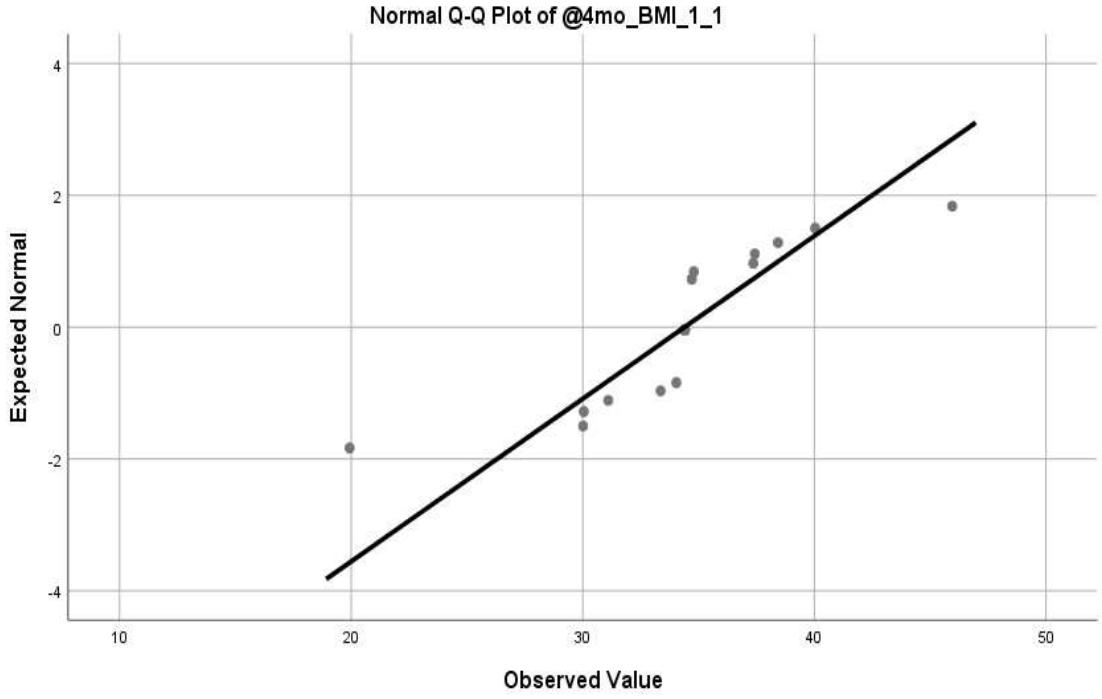
Appendix K: rMANOVA Q-Q Plot for O2sat @ 6-months



Appendix L: rMANCOVA Q-Q Plot for BMI @ 2-months



Appendix M: rMANCOVA Q-Q Plot for BMI @ 4-months



Appendix N: rMANCOVA Q-Q Plot for BMI @ 6-months