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UNIVERSITY OF SAN DIEGO

Hahn School of Nursing and Health Sciences

DOCTOR OF PHILOSOPHY IN NURSING

OBSTRUCTIVE SLEEP APNEA AND THE EFFECTS OF CO-EXISTING FACTORS
ON PACU STAY TIME

By

Shari F. Jones, CRNA, PhD

A dissertation presented to the

FACULTY OF THE HAHN SCHOOL OF NURSING AND HEALTH SCIENCES

In fulfillment of the requirements for the degree

DOCTOR OF PHILOSOPHY IN NURSING

August 2012

Dissertation Committee

Dr. Joseph Burkard, DPNC, CRNA, Chairperson

Dr. Cynthia D. Connelly, PhD, RN, FAAN

Dr. Darren Couture, PhD, MSN, CRNA

DEDICATION

This study is dedicated to my father, a veteran of twenty-three years of military service, and to all those like him. To my patients and colleagues, active duty, retired and dependents of the armed forces community who have served or are currently serving in varied roles of support to this great country. There is no greater sacrifice than submitting personal freedoms to protect, support and preserve the freedom for us all.

ACKNOWLEDGMENT

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OBSTRUCTIVE SLEEP APNEA EFFECT COFACTOR ON PACU RECOVERY

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OBSTRUCTIVE SLEEP APNEA EFFECT COFACTOR ON PACU RECOVERY

ABSTRACT

Significance/Background: Obstructive Sleep Apnea (OSA) is the most common disturbance during sleep affecting 30% of the population. The occurrence of moderate/severe OSA is 11.4% in men and 4.7% in women. OSA is caused by repetitive partial or complete obstruction of the upper airway with apnea episodes. OSA is prevalent in western society, yet is frequently undiagnosed due to lack of knowledge of the disorder. This population presents a challenge during the peri-operative period due to increased risk for several complications. Of particular interest to Nurse Anesthetists, are respiratory complications that result in delayed emergence from anesthesia, delayed post-operative recovery, increased apneic episodes, hypoxemia, and death. Examining duration of recovery periods between the two OSA groups will help determine if patients with additional cofactors benefit from longer post-operative monitoring or modification of anesthetic technique.

Methods: An observational correlation design to include a non-randomized convenience sample of two groups. Comparisons were made between groups regarding cofactors, complications and duration of postoperative recovery times. Patients having elective abdominal surgery under general anesthesia were identified. OSA status was determined based upon Sleep Studies, or STOP-BANG tool criteria. Medical, surgical, and anesthesia data was reviewed. Total recovery times and pre-existing disease documented. Multiple Logistic and Regression testing compared effects of cofactors on postoperative recovery times. Chi square analysis evaluated relationship of individual cofactors with

OSA and Non-OSA patients. T-test analysis compared demographic information. P score of $< .05$ was considered significant.

Results: This study indicated significant correlation with higher number of cofactors amongst patients diagnosed with OSA ($p < 0.012$). Increased incidence of higher ASA classification in OSA diagnosed patients; ($p < .017$) extended PACU stay time in OSA diagnosed patients; ($p = 0.05$) and unplanned admissions in OSA patients; ($p = .007$).

Patients with OSA use higher number of prescribed medications compared to non-OSA patients have a 3.36 greater chance of hypertension as an additional cofactor ($p < .05$) and were 8.75 times more likely to experience airway difficulties or complications with general anesthesia when compared to non-OSA patients ($p < .05$). Multiple logistic regression testing demonstrated increased incidence for both airway difficulties ($p = .003$) and Hypertension ($p = .054$). Multiple linear regression results indicate one cofactor HTN as a significant predictor for PACU stay time. ($R^2 = .179$, $R^2_{adj} = .136$, $F(1)$)

Implications: Findings will lead to optimum monitoring, management; recovery measures and anesthesia techniques that will prevent extended postoperative periods and reduce or eliminate postoperative complications of OSA.

Key Words: Obstructive Sleep Apnea, Cofactors, STOP-BANG

CHAPTER 1

Introduction

Obstructive Sleep Apnea (OSA) is the most common disturbance during sleep affecting 2-26% of the general population. (Chung et al, 2008) The occurrence of moderate to severe OSA is estimated to be 11.4% in men and 4.7% in women. (Seet et al, 2010) OSA is a prevalent problem in Western Society estimated to occur between 1 to 9% in patients presenting for surgery, yet it is frequently undiagnosed due to lack of awareness or knowledge of the disorder. (Kaw et al, 2006)

OSA is caused by repetitive partial or complete obstruction of the airway during sleep. Diagnosis of OSA is determined by the occurrence of airway obstruction during sleep with apnea periods lasting greater than 10 seconds for five or more times per hour with a resultant decrease in arterial oxygen saturation (SaO₂). (Seet et al, 2010) These events are known as apnea/hypopnea episodes and the measurement of episodes is referred to as the Apnea-Hypopnea Index [AHI]. The degree of severity of this disorder is determined by the number of AHI's per hour (AHI: 5-15 mild, ≥ 15 -30 moderate, and > 30 severe).

A Polysomnography study otherwise known as a Sleep Study is recognized as the "Gold Standard" for the diagnosis of OSA. Polysomnography data defines the degree of severity of the condition by the number of apneic episodes. Unfortunately polysomnography testing is time consuming; patients are reluctant to participate in them,

they require an extended stay in the sleep lab; and are expensive. Medical professionals have become more aware of OSA as a significant disorder with resulting adverse effects for their patients. More physicians are consulting to polysomnography specialists for sleep studies to ascertain occurrence and severity of OSA, and the availability of appointments have become more limited. Unfortunately for the OSA patient scheduled for surgery, polysomnography cannot be obtained rapidly and this may be the cause of OSA going undetected. (Chung, 2008) Subsequently, the OSA patient population presents a unique challenge during the peri-operative period due to an increased risk for lack of identification and subsequent life threatening complications. Of particular interest to the Nurse Anesthetist treating a OSA patient, are the respiratory complications that can result in delayed emergence from anesthesia, delayed post operative recovery, increased apneic episodes, hypoxemia, cardiac disturbances, and ultimate death.

Obesity, gender, snoring, and age are strong indicators in the occurrence for OSA. Hypertension, cardiovascular disease, diabetes, and stroke are prevalently reported as confounding and coexisting factors. (Dykes et al, 1996) Netzer et al. (2003) surveyed OSA patients in 26 US primary care clinics and 14 European primary care clinics. Comparison of the results indicated similar symptoms regardless of age, gender, and hypertension. Obesity was common between both sets of OSA patients although the incidence was much higher in the United States than in Europe. In a Spanish study, Duran et al. (2001) reported linkages between OSA and hypertension after interviewing over 2,000 patients.

The predominant cardiovascular effects of OSA are hypertension and refractory bradycardia followed by abrupt tachycardia. (Vanninen et al, 1996). The hemodynamic instability seems to be related initially to hypoxemia from the airway obstruction, and then sudden arousal as the patient awakens to breath. The rapid fluctuations of vaso-activity is thought to be responsible for the incidence of cardiac arrhythmias, the development of chronic arterial hypertension, the occurrence of myocardial infarction, and sudden cardiac death (Vanninen et al, 1996)

The severity and incidence of documented post surgical complications from patients with moderate to severe OSA is significant. Yet, most of the data on incidence, occurrence, surgical complications and treatment of the OSA patients come from case reports and retrospective reviews not empirical studies. (Chung et al, 2008)

The American Society of Anesthesiologist (ASA) developed guidelines in 2006, based on Level IV evidence and expert consensus reports to address the identification and treatment needs of the OSA patient. The ASA consultants reached agreement on treatment criteria for most simple outpatient and minor orthopedic surgical procedures but could not find consensus regarding more invasive surgeries. The guidelines emphasized that certain factors needed to be considered when determining both preoperative and postoperative risk for the OSA patient: status of sleep apnea, (treated or untreated), level of comorbidities (hypertension, cardiovascular disorders, diabetes are they appropriately managed), type of surgery, and anesthesia modality.

(Chung et al, 2008) The key experts in the diagnosis and treatment of the OSA patients acknowledge the poor quality of evidence. A citation from the American Academy of

Sleep Medicine stated that the scientific literature regarding perioperative risk and best management techniques for OSA patients was scanty and of limited quantity, more empirical studies are needed. (Meoli et al, 2003). The ultimate goal for all Nurse Anesthetists is to provide the safest, highest quality anesthetic management and care to their patients. If Nurse Anesthetists can identify and understand the coexisting factors creating the greatest risk for perioperative complications in the OSA patient, they can better prepare themselves to manage those risks and provide optimum care. Nurse Anesthetists can select anesthetic practices that minimize risk and maximize safer outcomes. Development of more concise and accurate techniques to eliminate the factors producing increased risk for this population could be incorporated. Valid criteria for preoperative identification of the OSA patient could be developed and preoperative management techniques could be identified. Established guidelines and protocols could be created to decrease or eliminate most if not all risk factors. The complete elimination of incidence and occurrence of morbidity and mortality for the OSA patient in the perioperative environment would be the ultimate goal.

Purpose and Specific Aims

The purpose of this study is to identify preexisting factors that significantly impact postoperative recovery times for patients undergoing surgical procedures.

Specific aims of this study include:

Aim 1. To examine the difference between the postoperative stay times of OSA and Non- OSA patients.

Aim 2. Compare OSA and Non OSA patients in regards to existing cofactors and medical treatments

Aim 3. Assess the relationship between OSA and airway management difficulties and complications

Aim 4. Assess the relationship between the pre-existing cofactors of: Hypertension, Cardiovascular disease, diabetes and GERD.

Aim 5. Examine which of the pre-existing cofactors account for the explained variance in postoperative recovery time.

CHAPTER TWO

Literature Review

The purpose of this study is to identify preexisting factors that significantly impact postoperative recovery times for Obstructive Sleep Apneic patients undergoing surgical procedures. Obstructive sleep apnea (OSA) both diagnosed and undiagnosed is one pre-existing factor identified as increasing medical and surgical risks and ultimately increased morbidity and mortality for individuals during the surgical, and perioperative environment. Yagg and associates (2005) argue untreated OSA significantly and independently increased the incidence of stroke or death from any other underlying cause. Patients with undiagnosed OSA pose greater risk of airway compromise and require additional measures and management regimes to ensure safe anesthesia care and decreased PACU length of stay, information related to the existence of co-morbidities is of great significance. The effects of OSA and coexisting disease in the perioperative setting have not been fully explained. Does OSA have a significant impact on underlying disease processes that lead to perioperative complications and increased length of PACU stay? A review of the relevant literature was conducted to identify the current state of the science on the effects of OSA and coexisting factors on perioperative care. In this chapter the literature review is presented to include OSA, OSA and comorbidities, assessment of OSA, and the impact on health outcomes. The theoretical framework “Startling Resistor Theory” guiding the study will also be presented.

Obstructive Sleep Apnea

Obstructive sleep apnea (OSA) is a disorder becoming much more prevalent within the population of the United States and Europe. Obstructive sleep apnea is a disorder that occurs during the normal sleep cycle of certain individuals and can be elicited during surgery or medical procedures using sedation and general anesthesia. Obstructive sleep apnea is caused by repetitive partial and complete obstruction of the upper airway by relaxed pharyngeal muscle and tissues during normal sleep. It is also facilitated by implementation of vaporized anesthetic agents during surgery and the introduction of narcotics and analgesic medications for deep sedation (Chung et al, 2008).

The etiology of OSA is not completely understood but there is a general consensus that the negative pressure created by the inspiratory muscles exceeds the capacity of the upper airway dilator muscles to maintain airway patency, thus obstruction of the nasopharynx, oropharynx, and laryngopharynx are obstructed (Chung et al, 2008). The tensor palatine, genioglossus, and hyoid muscles contract to dilate and elevate the nasopharynx, oropharynx, and laryngopharynx respectively. Action of the tensor palatine is mainly tonic activity and this action decreases during sleep leading to upper airway resistance. The action of the genioglossus is phasic with inspiration; contraction of this muscle advances the tongue forward and is essential in keeping the tongue from blocking the oropharynx. Genioglossus function also decreases with sleep and has been shown to cease completely during rapid eye movement (REM) sleep. Studies have noted that OSA patients may have a primary myopathy of their genioglossus muscle leading to complete

loss of function at the onset of an apneic event and returning at the termination of obstruction (Chung et al., 2008; White, 1995).

Patencies of these three collapsible regions of the pharynx are dependent on the inspiratory function of the three-dilator muscles. The larger the negative inspiratory pressure during respiration the stronger the force of pharyngeal contraction required from these dilator muscles to maintain patency of the airway. Pressure sensitive airway receptors are responsible for instigating the negative pressure reflex response. This is elucidated by the fact that the implementation of anesthesia effectively abolishes this reflex (White, 1995). Finally a group of three individual muscles grouped together as the hyoid muscles are responsible for enlarging and opening the laryngopharynx by elevation of the hypo- epiglottic ligament. The activities of these muscles are also phasic with respiration. Neck positioning and facial abnormalities can greatly affect the function of the hyoid muscles leading to narrowing of the laryngopharynx (Pierce et al, 1999; White, 1995) Function of pharyngeal muscles and airway patency are critical during sleep. More specifically during REM sleep. Rem sleep is a very deep sleep and is characterized by a general loss of muscle tone as which has been demonstrated by electromyelographic imaging. It is during REM sleep that rhythmic activity of the upper airway muscles can cease completely, and upper airway resistance increases maximally. It has been noted that the most important region of collapse is within the lateral pharyngeal muscle walls. In the OSA patient apnea occurs when complete closure of the lateral pharyngeal walls occur. Gradations of weakness within the lateral pharyngeal walls during Non REM sleep results in audible snoring. Fluttering of weakened pharyngeal walls causes it. In general

the weaker the walls the louder the snoring and complete closure results in absence of sound (Wilson et al., 1999). Obesity has been found to be a direct cause of OSA due to increased fat deposition in the pharynx; which has been demonstrated by magnetic resonance imaging, to decrease airway patency (Mortimer et al., 1998). Furthermore, the fat deposition in OSA patients has been found to predominate in the lateral pharyngeal walls. The volume of pharyngeal wall fat correlates directly with the severity of OSA (Horner et al., 1989; Shelton et al., 1993). The deposition of fat not only narrows the airway in the OSA patient but it also changes the shape transforming the normal elongated lateral wall with an elliptical shape to a more anterior posterior axis with a rounded lateral wall and circular shape (Pierce et al., 1999). Studies denote that anterior-posterior axis leads to less efficient function of the pharyngeal airway dilator muscles than an elongated elliptical axis (Leiter, 1996).

Overall patency of the pharyngeal airway is determined by varying pressure exerted on the walls of the airway. The nasopharynx, oropharynx and laryngopharynx act like compressible tubes and both internal and external wall pressures affect the opening of such tubes. Transmural airway pressure is the difference between external and internal airway wall pressures. If this pressure is equal and constant then the airway remains open as long as the tissues remain compressible. In the obese patient external wall pressure are increased due to the increased fat deposits within the submandibular tissue. OSA patients typically present with short fat necks and “sagging jowl’s”. Davies et al, 1990-1992) found significant correlation with increased neck circumference and severity of OSA.

John L. Benumof (2002) defines obstructive sleep apnea as “cessation of airflow for more than 10 seconds despite continuing ventilatory effort five or more times per hour of sleep which is usually associated with a decrease in oxygen saturation of more than 4 percent “ (p.790) A series of respiratory events occur during an obstructive apnea episode. As the duration of apnea increases pulmonary arterial oxygen tension (PaO₂) decreases. Decreased PaO₂ that in turn augments carotid body output causing vasoconstriction and increased heart rate causes arousal.

Concurrently, pulmonary artery carbon dioxide tension (PaCO₂) increases, which stimulates central nervous receptors similarly eliciting arousal state. Ventilatory effort is promoted due to the fluctuations of PaO₂ and PaCO₂ and intra-airway pressures become increasingly negative, increasing likelihood of airway closure. In this instance arousal may be initiated by pressure sensitive receptors in the upper airway (Benumof, 2002). The four mechanisms discussed lead to increased neural activity in the reticular activating system and arouse the individual. Snoring, sputtering, gasping, twitching or extremity movement may preclude the manner of arousal. The muscles of the pharyngeal airway open in response to the arousal and ventilation reoccurs (Benumof, 2002, p.796).

The physiological events necessary for adequate ventilation are essential for the OSA patient to support life. The repetitive recurrence of these essential physiological processes have been found to adversely influence and enhance the occurrence of other disease disorders such as hypertension, cardiac arrhythmia, myocardial infarction, diabetes, and coronary vascular incidents (Chung et al., 2008). According to Seet et al. (2010) Obstructive sleep apnea has been associated with several co-morbidities cardiovascular

disease, including MI, heart failure, arrhythmias, hypertension, cerebrovascular disease, metabolic syndrome, obesity, and gastroesophageal reflux. (P.200)

The occurrence of diagnosed obstructive sleep apnea patients has been estimated to be 1 in 4 men (11.4%) and 1 in 10 women (4.7%). The statistics are even greater in the obese population; 7 of 10 obese individuals have been diagnosed with obstructive sleep apnea. Due to the increase of obesity within our nation, it is estimated that within the American population today a significant number of individuals; as many as 90%, may be undiagnosed with obstructive sleep apnea. (Benumof, 2002; Chung et al, 2008; Seet et al, 2010) The presumption is the numbers will only increase, as the incidence of obesity remains unchanged and in fact growing statistically. It is estimated that the number of diagnosed and clinically presumptive sleep apnea patients will increase five to ten times by 2012 and continue to escalate thereafter. (Benumof, 2002)

The implications for both a diagnosed and undiagnosed obstructive sleep apneic population of such vast numbers is increased medical and surgical risks and ultimately increased morbidity and mortality for these individuals during medical, surgical and perioperative management. Untreated obstructive sleep apnea significantly and independently increased the incidence of stroke or death from any other underlying cause. (Yaggi et al, 2003p. 2040)

For Nurse Anesthetists and anesthesiologists; patients with undiagnosed obstructive sleep apnea pose greater risk of airway compromise and require additional measures and management regimes to ensure safe anesthesia care.

Obstructive sleep apneic patients have statistically difficult airways for intubation, increased sensitivity to anesthetic agents and a propensity for increased postoperative adverse events and complications. (Seet et al., 2010)

Recent studies have demonstrated that patients with obstructive sleep apnea undergoing upper airway surgery, joint replacement surgery and cardiac surgery exhibit increased risk of postoperative complications. (Gupta et al, 2001; Kim et al., 2005; Liao et al., 2009; Pang et al., 2006)

These findings are alarming and do not even represent the number of surgical or sedation patients who may arrive for anesthesia with undiagnosed obstructive sleep apnea. It is important that the OSA patient be preoperatively identified so that the anesthesia provider may provide the necessary treatment precautions to eliminate operative and postoperative complications and adverse events.

The established gold standard for diagnosing obstructive sleep apnea is Polysomnography more commonly known as a sleep study. The purpose of polysomnography is not only to establish a diagnosis of OSA but also to determine the degree of severity of the obstructive airway disorder. The apnea hypopnea index (AHI) is the number of adverse respiratory events occurring per hour of sleep. The American Academy of Sleep medicine defines for mild OSA as 5- 10 AHI, moderate OSA as 15-30 AHI and severe OSA as more than 30 AHI within one hour of sleep. Differing classification for OSA prevail for example the United States Medicare guidelines define OSA as an AHI of 15, or an AHI of 5 with 2 comorbidities and in Canada the accepted criteria is establishment of daytime sleepiness, without other definitive cause, or two

other symptoms of OSA. (i.e. choking/gasping during sleep, recurrent awakenings from sleep, daytime fatigue, impaired concentration) Duration of oxygen desaturation, rate of desaturation, adequacy of ventilator recovery, and level of arousal are other factors that have been incorporated to evaluate OSA. (Seet et al., 2006) (p.200)

Even with the accepted gold standard of diagnosis “polysomnography” there exist problems with implementation of the test as well as accurate and consistent diagnosis of the degree of severity of this disorder. Polysomnography is time consuming (8 hour to 2 consecutive night study), expensive, resource intensive, and not at all suitable as a short term screening device. (Chung et al, 2007)

Eastwood et al (2010) cite an example of various studies in which the categorizing of AHI of 5 within a 1993 Wisconsin Study in the New England Journal of Medicine was the equivalent to a Chicago definition of a category AHI of 15. The absence of a global consensus of standardized criteria for the definition of the severity of OSA has significant impact on identification and comparative findings for research and methodologies. Lack of standardization has significant effects on OSA prevalence estimates, treatment decisions and the establishment of links between OSA and comorbidities. (Eastwood et al, 2010)

In 2006, the American Society of Anesthesiologist attempted to provide consensus for the diagnosis of OSA as well as provide management guidance. A task force made up of a panel of 12 members including anesthesiologists, a bariatric surgeon, an otolaryngologist and two methodologists developed “Practice guidelines for the perioperative management of patients with obstructive sleep apnea”. (Para. 3) The

purpose of this publication was to aid in the identification of the OSA patient within the preoperative interview and provide guidelines for perioperative management of their care. The establishment of these guidelines resulted in a format of fourteen categorized questions comprising a checklist of physical characteristics, identification of airway obstruction during sleep, and complaints of somnolence. All criteria established to be present and contributing to obstructive sleep apnea. The sensitivity of the task force's checklist was 79% to 87% as compared to the "gold standard" of Polysomnography. (Seet et al, 2010) Subsequently a more efficient, concise screening tool was developed called the STOP-BANG questionnaire. A meta-analysis of clinical screening measures for obstructive sleep apnea identified and evaluated 26 different clinical prediction tests, 8 of these were survey questionnaires, and 18 algorithms, regression models or neural networks. The findings supported and recommended the STOP-BANG as a preoperative screening tool on the basis of ease of use and the excellence of predictability of severe OSA (AHI>30) with 96% sensitivity. The simple acronym STOP BANG and linear scale make the tool practical and very user friendly in the perioperative setting (Ramachandran et al., 2009).

Additional information related to the existence of comorbidities is of great significance to the anesthesia provider. It is well established that untreated OSA results in greater morbidity and mortality in the perioperative period. (Bahammam et al., 1999; Bixler et al., 2000; Dykes et al., 1996; He et al., 1988;Hunge et al., 1990). Yet, the effects of OSA and coexisting disease in the perioperative setting have not been fully explained. Does OSA have a significant impact on underlying disease processes that lead to perioperative

complications? Several studies have drawn associations between patients with OSA, diabetes, cardiovascular disease, obesity, arrhythmias and stroke.

Obstructive Sleep Apnea and Diabetes

A high association between OSA and metabolic disorders would seem very likely since the link with severe OSA and obesity as a risk factor are so strong. There is growing evidence that OSA serves as an independent entity of risk for adverse glucose metabolism. Does OSA have a definitive role in the onset of diabetes? The mutual connections between obstructive sleep apnea and diabetes are still not fully understood. It is known that the glucose sensing process uses the same neurotransmitter pathways as the hypoxic sensing process. The carotid body glomus is the polymodal receptor that detects low blood glucose. Rasche et al. (2010) identified that the hypoxic events associated with obstructive sleep apnea combined with diabetes all signal responsive elements in the carotid body. This carotid gluco-sensor malfunction could be challenged and unable to initiate the regulatory process necessary to counter increased blood glucose. In this manner sleep apnea may increase the propensity for the development of diabetes, independent of other metabolic related processes. (Rasche et al, 2010)

Chasens (2003) examined some of the theories that might explain the relationship of OSA and diabetes. Of definitive agreement among the theories is the centroid pattern of obesity occurring in persons with insulin resistant or diabetes Type 2. The existence of extra body weight increases the resistance on respiratory structures and leads to narrowing and collapse of upper airway tissue in OSA. Yet Stahl (2002) offered a

coexisting theory, that the hypoxia and hypercapnea that occur in OSA provoke sympathetic nervous system stimulation that results in hyperinsulinemia. The mechanism proposed for increased insulin resistance and glucose regulatory dysfunction is the result of repetitive hypoxia and hypercapnea, which occurs with apneic events, triggers the stress response invoking chemoreceptors in carotid bodies, aortic arch and in the medulla of the brain.

The results of these processes are the release of catecholamines: epinephrine, norepinephrine and cortisol. OSA interferes with glucose management by insulin deregulation (Chasens et al., 2003) (p.89). A study by Mahmood et al. (2009) also supports the sympathetic response theory citing “severe OSA results from increased neurogenic sympathetic activity and circulating levels of norepinephrine” which predisposes to increased glycogenolysis, lipolysis, and insulin resistance. (P.219)

Another study by Eastwood et al. (2010) described obesity (excess adipose tissue) as a” cofounder or active partner in crime” when evaluating the impact of OSA on metabolic disorders (p. 590). If this were fact then you would expect that a relationship with OSA and the development or existence of diabetes would depend on the amount of adipose or fat cells the individual with OSA exhibits. Normal or lean BMI OSA individuals would be unlikely to have diabetes. Speculation would seem to favor the fact that body fat would be the greater modulator of diabetes rather than OSA since body fat has direct impact on cause of diabetes. Yet, published studies suggest that OSA may impair insulin secretion, leading to glucose intolerance or diabetes. This physiological manifestation of an insulin- resistant state has little to do with obesity. Ultimately the

“lean” OSA individual may still suffer from diabetes. (Ip et al, 2002; Punjabi et al, 2009) Steiropoulos and colleagues (2009) conducted a study of 31-year-old males newly diagnosed with OSA without diabetes and found significant correlations between glycolated hemoglobin levels and AHI as well as arousal index and percentage of sleep time with hemoglobin saturation less than 90%. The correlations suggest that OSA posed a significant risk factor for glucose dysmetabolism irrespective of the presence of diabetes. The authors suggest the need for addressing preexistence of OSA in both diabetic and non-diabetic patients to effectively assess glycemic control. Additional studies have shown strong associations between OSA, glucose intolerance, insulin resistance and diabetes independent of obesity. A 2010 study by Arhanson indicated a high prevalence of OSA in patients with diabetes Type-2. The existence of a graded and inverse relationship between OSA severity and glucose control was demonstrated in patients with Type 2 diabetes, after controlling for the degree of obesity (BMI), age, and gender. Furthermore the study may have significant implications to support a hypothesis that reducing the severity of OSA may improve overall glycemic control in diabetes Type 2 patients and prevent onset for the non-diabetic OSA patient.

A pilot study completed at Grenoble University in France consented 40 subjects with Type 1 diabetes to be screened for obstructive sleep apnea. Sixteen of the 40 subjects were determined by Polysomnography to have OSA. In addition, 10 of the 16 subjects were classified with severe OSA. A total of 40% of the Type 1 diabetic subjects were determined to have obstructive sleep apnea; this is an exceptionally high percentage. The subjects were all of normal body habitus, not obese and BMI was not associated with

pathological oximetry values. The pilot studies suggest a linkage between pharyngeal neuropathy and high prevalence of OSA in Type 1 diabetic subjects. In a similar study Ficker et al. 1998 demonstrated that obstructive sleep apnea was associated with autonomic neuropathy in Type 2 diabetics (Borel et al, 2010).

In a United Kingdom a sample of 938 subjects with diabetes were recruited from a tertiary hospital clinic and five primary care clinics within the community. Sleep studies were obtained and prevalence of OSA was found in 23% of the respondents as compared to 6% of the non-diabetics within that general community (Foster et al., 2009). A Canadian study of 2,149 subjects; 8.1% were diagnosed with diabetes and a twofold risk of OSA was found in the diabetes group when compared to the non-diabetic group, despite adjustment for potential confounders (Ronskley et al., 2009). Elmasry (2001) conducted a longitudinal study in Sweden looking at the relationship of diabetes, hypertension and OSA. One hundred sixteen hypertensive subjects with OSA were selected. They found that although obesity is strongly associated with the coexistence of diabetes mellitus, severe OSA seems to add significantly to this association, Their comparative study included 4 groups of hypertensive men (Non-OSA: non-obese, Non-OSA: obese, OSA: non-obese and OSA: obese) the highest percentages of diabetes occurred in the subjects with OSA: Obese. But of significance to the authors, 35% the second highest incidence of diabetes was in the OSA: non-obese group. Elmasry et al. conclude that following linear regression their data supported significant association between AHI and fasting blood glucose, fasting serum insulin, and HbA1c levels. Using multiple regression desaturation index also demonstrated significant association with

fasting serum insulin, fasting blood glucose and glycohemoglobin -HbA1c. (p.158) This data supports the evidence that obstructive sleep apnea has independent and significant adverse effects on fasting serum insulin, blood glucose and HbA1c when adjustments are made for central obesity, even in normoglycemic hypertensive males (Elmasry et al., 2001)

Associations of OSA and glucose disorders are not limited to western culture alone. In a 2009 Japanese longitudinal study; subjects with nocturnal intermittent hypoxia exhibiting AHI <5 and lowest SpO₂ during the night were associated with an increased risk of type 2 diabetes independent of the traditional risk factors of age, gender and BMI (Muraki et al., 2010). Another study conducted in Japan 2010 examined the prevalence of OSA associated with Type 2 diabetes. Of the 165 subjects that underwent Polysomnography 89 were found to have clinically diagnosed OSA. The prevalence of associative OSA and Diabetes in the Asian population was 53.9%, with 18% having severe OSA and 15% classified as moderate OSA. In Japanese, 40 to 60 years old, the study denotes a threefold increase in men (32% vs. 10%) and a twofold increase in women (14% vs. 6%) with associative OSA and diabetes as compared to the general population. The actual numbers of OSA and diabetes occurrence are more representative of findings in the United Kingdom and Canada (Lam et al., 2010).

When examining the incidence of OSA and diabetes within different ethnic and age subgroups it was determined that Hispanics had a high prevalence of Type 2 diabetes. It was also observed that the occurrence of Type 2 diabetes in middle-aged subjects with OSA was 2.8 times higher than in younger or middle aged subjects without OSA. Older

subjects had higher odds of Type 2 diabetes regardless of OSA status. This may allude to a genotype component responsible for diabetes disorders regardless of OSA (Mahmood et al., 2009).

The Center of Disease Control estimates the number of individuals in the United States with diagnosed and undiagnosed diabetes to be around 24 million. The prevalence of diagnosed obstructive sleep apnea in patients with Type 2 diabetes ranges from 73 to 86 percent, this suggest a staggering number of individuals; approximately 17 million people with diabetes are unaware of a significant co-morbidity. This is a major concern for those in the medical profession, and more specifically for the anesthesia provider whose delivery and management of care is directly influenced by the associated effects of obstructive sleep apnea and diabetes. (Pamidi et al, 2010)

Obstructive Sleep Apnea and Cardiovascular Disease

A growing body of evidence supports obstructive sleep apnea as an important risk factor for hypertension related cardiovascular disease and dysrhythmias. Lavie and associates (2001) estimated that approximately 40 to 60 percent of individuals diagnosed with obstructive sleep apnea in sleep labs had concurrent hypertension. This rate of hypertension was two to three times greater than the general population. Since OSA prevalence has increased to an estimated 34% (diagnosed and undiagnosed) of the population in the last ten years; the increase in hypertension will also have grown exponentially.

A number of studies have elicited theories describing the physiological processes for OSA's contribution to hypertension and arrhythmia disorders. Ryan et al (2009) suggest that inflammatory processes leading to endothelial disturbances play an essential role in the pathogenesis. The repetitive hypoxia –hypopnea events that occur in OSA create short cycles of desaturation followed by rapid re-oxygenation, initiates the inflammatory process. This intermittent re-oxygenation resembles the same characteristics of reperfusion injury and is proposed to result in the development of atherosclerosis.

The inflammatory markers known as cytokines and chemokine's (adhesion molecules) are precursors in the pathogenesis of atherosclerosis and are potential cardiovascular risk markers. These inflammatory markers have been identified as independent risk factors for cardiovascular diseases (Ryan et al., 2009). The cytokine Tumor Necrosis Factor Alpha (TNF- α) is recognized as important in the development of atherosclerosis. Circulating levels of TNF- α have been indicators for early signs of atherosclerosis in middle aged men and are predictive for coronary heart disease and congestive heart failure. Studies examining levels of TNF- α with obstructive sleep apnea syndrome have consistently shown elevated levels of the cytokine in OSA subjects in comparison to controls and independent of obesity. These same studies have demonstrated significant reduction in TNF- α following implementation of continuous positive airway pressure (CPAP) therapy. So, TNF- α levels could be the best predictors of cardiovascular risk for the individual with obstructive sleep apnea (Ryan et al., 2009).

Another chemokine, Interleukin- 8 (IL-8) has been found to be associated with OSA. IL-8 is another precursor to atherosclerosis; it plays a major role in plaque destabilization

and atherogenesis. IL-8 levels were notably increased with OSA and similar to TNF- α response; IL-8 levels demonstrated a significant reduction when CPAP treatment was initiated within the study. Both the cytokine TNF- α and the chemokine IL-8 are modulated by the same transcription factor Nuclear Factor Kappa B (NF- κ B). This transcription factor plays a predominate role in initiating the inflammatory process and its effect on the other mediators supports the role of OSA in inflammatory response and cardiovascular disturbances (Ryan et al., 2009). Yamauchi et al (2008) identified similar processes as the Ryan study and cite the contribution of the inflammatory process and immune response to the development of atherosclerosis. Atherosclerosis is identified as the “common pathological factor for cardiovascular disease” (p.756) This study similarly identifies the hypoxia-re-oxygenation as the component of OSA causing the inflammatory process. The study also demonstrated that OSA induced intermittent hypoxia activated NF- κ B and that this activated transcription factor induced the inflammatory responses in the arterial walls that impairs endothelial function leading to cardiovascular consequences. The re-oxidation and reperfusion phase results in the production of reactive oxygen species (ROS). ROS reactive molecules damage intimal cells and promote oxidative stress. A number of studies have reported markers of ROS are associated with OSA. These two articles found similar conclusions: inflammatory response and endothelial dysfunction lead to the pathogenesis of atherosclerosis. Transcription factors, chemical mediators: cytokines, chemokines, markers of oxidative stress are likely activated by OSA – related oxidative stress (hypoxia-reperfusion) and may define the pathway in the cardiovascular consequences of OSA (Yamauchi et al.,

2008). The first direct evidence at the cellular and molecular level, of the impact of oxidative stress on the endothelial cells was determined by Padelletti et al (2008). They found that the endothelial nitric oxide synthase (eNOS) and its phosphorylated active form (p-eNOS) were weakened in the OSA patient and that the oxidative stress marker nitrotyrosine, which is an indicator of nitric oxide inactivation by oxidative stress, was elevated. (p. 3469)

Mansfield and Naughton (2005) described evidence of OSA's causation and progression of congestive heart failure and cardiovascular disease including hypertension. Cardiac preload and afterload are affected by intra-thoracic pressure. The fluctuations of intrathoracic pressure (exaggerated falls of intrathoracic pressure) created by repetitive ventilator efforts against closed airway occurring during the hypoxic- re-oxygenation episodes of OSA create conditions of increased left ventricular afterload and decreased preload. The result is overall reduction in stroke volume and cardiac output which over time progresses to congestive heart failure (CHF) (Mansfield et al., 2005). Kato et al (2000) and Gami et al (2005) studies found a relationship between OSA syndrome, sudden death, and nocturnal myocardial infarction. Repetitive cycles of bradycardia and tachycardia are commonly documented during sleep studies of the OSA patient. Severe sinus bradyarrhythmias are prevalent in OSA patients with severe hypoxemia, especially in rapid eye movement (REM) sleep. Severe hypoxemia during REM sleep may predispose OSA patients to abnormal heart rhythms such as extreme bradycardia, sinus pauses, or conduction blocks. Evidence from previous studies also supports an independent association of OSA and atrial fibrillation (Lavie et al., 2008).

The effects of hypoxia, hypercapnea, baroreceptor desensitization, and arousal responses are all factors that contribute to both the acute and chronic augmentation of the sympathetic nervous system. Sympathetic nervous system activation remains the most likely and consistently identified explanation for the development of chronic hypertension (Mansfield et al., 2005). It has been demonstrated that patients with OSA do not exhibit the normal nocturnal “dipping” of blood pressure during sleep. Non-dipping nocturnal blood pressure remains a characteristic factor in underlying OSA distinct from the hypertensive population without OSA, which may prove valuable as a diagnostic measure (Portaluppi et al., 1997).

Various studies have encouraged the incorporation of CPAP to OSA patients as restorative treatment for hypertension and CHF. Malone et al (date) found a 12% improvement in left ventricular ejection fraction (LVEF) when CPAP via nasal cannula was introduced for 4 weeks to their subjects diagnosed with idiopathic dilated cardiomyopathy. In another study, 24 patients with CHF and OSA were randomized to one month CPAP or no treatment groups. The CPAP treatment group demonstrated an 8.8% improvement in their left ventricular ejection fraction and a fall in mean systemic blood pressure of 10mm hg was also reported. (Kaneko et al., 2003)

A controlled paralleled trial of 118 patients with hypertension and OSA underwent CPAP treatment and mean systemic blood pressure fell by 2.5mmhg over 4 weeks. (Peperell et al, 2002) The result was more pronounced in the more severe OSA group with hypertension; mean systemic blood pressure dropped by 6.6mmhg. In these studies CPAP appears to lower blood pressure and LVEF by eradicating obstructive sleep apnea-

hypopnea. In a study of untreated hypertension in middle aged men with undiagnosed mild OSA ($AHI \geq 5$) and without OSA the mean change in nocturnal blood pressure during and after 3 weeks of nightly CPAP was significantly greater in the mild OSA patients adjusted for age and BMI. The CPAP treatment did not affect the blood pressure of non-apneic subjects (Marrone et al., 2003). The effectiveness of CPAP therapy in improving cardiovascular disorders such as hypertension, and idiopathic cardiomyopathy provides strong evidence that obstructive sleep apnea and its resultant hypoxic-hypopnea events have a direct effect on cardiovascular function. The association of obstructive sleep apnea events and endothelial dysfunction, oxidative stress, inflammatory pathways, and sympathetic nervous system response warrants direct concerns for physicians and anesthesia providers alike. The high prevalence of OSA and its close association with cardiovascular dysfunctions lead too much greater risk for morbidity and mortality. Adverse outcomes will definitely result if these associative disorders are not identified, addressed, and corrected.

Post-operative Complications with OSA

The risk of serious postoperative complications has been previously addressed in regards to obstructive sleep apnea alone. Most OSA patients have sleep disorders involving both REM and Non REM sleep. Nocturnal apneic events coupled with preoperative anxiety; common prior to surgery, leads to overall poor sleep quality, and sleep deprivation prior to surgical events. This causes many patients to experience post-operative REM rebound characterized by increased episodes of airway atony and

subsequently airway collapse. This phenomenon can occur up to the third or fourth postoperative day when patients have returned home (Pang, 2006). Benumof (2004) found supporting evidence that in the postoperative setting, sleep mechanics were disturbed due to the requirement for increased analgesia for surgical pain. Analgesia through the first three days after surgery tended to incur a life threatening apnea from drug-induced sleep. He also described rebounds in REM sleep originating from poor quality sleep this was believed to increase the danger of a detrimental natural deep sleep-induced apnea. He suggested that patients with OSA are at risk of prolonged apnea during sleep for up to one week in the postoperative period.

Correlations between hypoxic events and episodes of recurrent oxygen desaturations associated with adverse outcomes have been reported. Gupta et al (2001) examined procedure specific complications in the post operative period and found that OSA patients experience significantly more occurrence of complications than the group without OSA. Complications included acute hypercapnea, $\text{PaCO}_2 > 45$, episodic desaturations reflected by $\text{SpO}_2 < 90\%$ with witnessed apneic events; arrhythmias, MI, or infarction, and delirium.

In the United Kingdom a mailed survey was provided to a group of anesthetists to define perioperative care of adults with OSA: Perioperative complications included 34% difficult intubations, 47% difficult ventilations, 24% cardiovascular instability, difficult analgesia in 41%, difficult extubation 13%, postoperative apnea 46%, required reintubation 27%, unplanned ICU admission 23%, and acute cardiac events 25% (Bamgbade et al., 2009).

These findings support the need for accurately assessing the postoperative status of OSA patients and determining criteria for risk assessment. Studies have shown strong relationships between the numbers of oxygen desaturations episodes within the post anesthesia care unit to occurrence of adverse events. Questions could be posed regarding the causes of the postoperative complications. OSA attributed events such as: Hypoxia, Hypopnea, Hypercapnea, snoring etc. Are they independently caused by the OSA disorder? Or is their synergistic component with other cofactors (HTN, Diabetes, CVD) that creates the adverse events? Does the OSA patient with one or more cofactors have more adverse events and do they require a longer PACU stay?

There currently is no clear explanation. It would be valuable information for the anesthesia provider and other health care providers to know the most significant cause of adverse events in the OSA patient.

Preoperative assessment of all surgical patients using validated instruments would aid in the identification of those patients with both known OSA and more importantly identify those that are unknown. Cofactors can be identified and appropriate optimization measured can be applied. Intraoperative management could be streamlined and specified for the OSA patient in a manner that minimizes complications. Postoperative measures and protocols could be developed to improve the care for the OSA patient; eliminating the risk factors and provision for safe analgesia that inhibits heavy sedation while promoting optimum oxygenation.

Theoretical Framework

An English physiologist Ernest Starling identified a theoretical phenomenon of vessel capacitance as influenced by flow, forces, and pressure. The Starling Resistor Theory (Stalford, 2004) explains the function of fluid flow through the coronary arteries. The Starling Resistor Theory is offered as a theoretical framework to compare and contrast airway physiology and function within the normal adult airway and in the OSA airway.

The Starling Resistor Model (Stalford, 2004) can be readily applied to describe flow in the upper airways. The concept provides an excellent explanation of airway dynamics as influenced by changing forces or pressures. A resistor is described as a collapsible tube passing through a chamber that is capable of having its pressure changed. For application purposes we can identify the oropharynx as the tube and the surrounding tissue of the neck as the chamber. If the oropharynx remains open then airflow will be determined by the pressure differences from the two sides of tissue within the neck; from the pressure above; the proximal region of the nose or mouth and from the pressure below; the distal portion of the trachea.

The main characteristic dysfunction of OSA is obstruction of airflow. The patient with OSA has recurrent apneic events that are the result of a closed or “blocked” airway that occurs during sleep. To understand the co-existing factors that may contribute to the adverse affects of OSA we must first understand the anatomic and physiologic process that defines OSA. The airway of the OSA patient presents differently than that of a typical adult airway. An OSA patient’s airway is smaller and narrower in contrast. The

OSA airway has a more lateral axis while the typical adult airway is more horizontal. This variant is thought to be caused by an excess of redundant tissue. In fact a definitive feature of OSA patients is an increased neck circumference or “thick neck” (Benumof, 2004). When sleeping the oropharyngeal muscles of the adult airway are relaxed and pharyngeal narrowing occurs and there is increased negative inspiratory pressure especially during rapid eye movement (REM) sleep. Additional negative inspiratory pressure is generated by diaphragmatic excursion and pharyngeal dilator muscle activity is incorporated as a compensatory mechanism, which maintains airway patency in the normal adult. In the OSA patient, pharyngeal dilator muscle activity maintains patent during awake periods but the muscle activity is lost when the OSA patient is asleep. Excessive adipose tissue in the neck of the OSA patient surrounds the oropharynx and compresses the airway making muscular activity less effective. Increased negative inspiratory pressure along with snoring, which can cause vibratory trauma can further enhance upper airway narrowing (Woodson et al., 1991).

The tissue pressure (P_{tissue}) corresponds directly to the pressure surrounding the tissues of the oropharynx that constrict the upper airway. A high opening pressure (P_{proximal}) will assist in overcoming the collapsing pressure of tissue (P_{tissue}). A low or negative pressure below the vocal cords (P_{distal}) that occurs as a result of the inspiratory effort will contribute to airway collapse if P_{tissue} is elevated. When sufficient muscular tone is operative in tissues surrounding the collapsible tube, the effect is a “lifting of the tissue” away from the collapsible portion. Thus P_{tissue} does not contribute to airway collapse. (Stalford, 2004)

Figure 1. Starling Resistor Model

P_d (distal)=upstream pressure, P_p (proximal)=downstream pressure and P_t (tissue)=pressure in the chamber (Stalford, 2004).

In the OSA patient, arousal is essential for airway patency and survival. A lack of oxygenation leads to hypoxemia and hypercarbia. Increased levels of CO₂ stimulate chemoreceptors within the CNS, which in turn release neurotransmitters promoting arousal. Reflex response activates pharyngeal dilator muscles and airways open, ventilation is returned. (Kaw et al, 2006)

Figure 2. Sleep Apnea Cycle

Starlings Resistor Theory was formulated to explain the properties of vascular compliance and resistance forces. The theory has already been established and accepted as defining the properties of fluid flow through blood vessels, and vascular circulation.

Although the coexisting factors of hypertension and cardiac disturbances in the OSA patient are not fully understood, an association with flow, increased pressure, and collapsibility may offer an explanation using Starling's Resistor Theory (Stalford, 2004). Applying this theoretical framework for both airway and hemodynamic pressures is appropriate and could indicate symbiotic relationships

CHAPTER 3

METHODOLOGY

The purpose of the study is to identify preexisting factors that significantly impact postoperative recovery times for patients undergoing surgical procedures. The research is informed by the Starling Resistor Theory (Stalford, 2004). The relationship and associations of pre-existing disorders such as hypertension (HTN), diabetes (DM-II), cardiovascular disease, and body mass index (BMI) in obstructive sleep apnea (OSA) patients will be investigated as co-factors in PACU recovery times. Hypertension, cardiovascular disease, and diabetes are prevalently reported as confounding and coexisting factors in obstructive sleep apnea patients. (Dykes et al, 1996). Yet there is no evidence as to whether these cofactors have any deleterious affect on post surgical recovery times of sleep apnea patients. This chapter presents the research methodology including design, sample and sampling, data collection procedures with instrumentation, and data analytic procedures. The protection of human subjects is also discussed.

Design

A quantitative observation correlational design method will be utilized for the proposed study. Determining a relationship of postoperative ambulatory care unit (PACU) stay times between obstructive sleep apnea patients and those without sleep apnea following elective abdominal surgery. The relationship and associations of pre-existing disorders such as hypertension (HTN), diabetes (DM-II), cardiovascular disease and GERD in

obstructive sleep apnea (OSA) patients will be investigated as co-factors in PACU recovery times. Munro (2005) states correlational techniques are used to study relationships. They may be used in exploratory studies, when the intent is to determine whether relationships exist, or in hypothesis testing studies, in which we test a hypothesis about a particular relationship. (p. 239)

Based upon a review of the literature the relationship between OSA and the cofactors of hypertension, diabetes, and cardiovascular disease have been identified. Data findings have reported each cofactor's independent relationship with OSA. It is beneficial to this study to investigate the correlations of several cofactors and their relationship with OSA. Additionally, to provide the body of evidence as to which cofactors more significantly affect the occurrence, prevalence and adverse effects of OSA.

SAMPLE

A purposive sample of active duty, retired, or family members of military service members who have consented to elective surgery at the Naval Medical Center San Diego (NMCSD) and received a score of 5 or greater from preoperative evaluation using the STOP-BANG screening measurement will provide data for the proposed study.

Inclusion Criteria consists of males and females 18 to 65 years, who have elected to have general anesthesia, surgery, category ASA I - III, able to read and comprehend English. Exclusion Criteria consisted of members above who had pre-existing pulmonary disorders (COPD, etc.), neurological dysfunction, delayed or decreased cognitive function and recent MI.

SAMPLE SIZE CALCULATION

For the purpose of this study which is to determine if identified obstructive sleep apnea (OSA) patients have longer PACU recovery times as compared with non-OSA patients undergoing elective surgery and if there is an association between OSA patients with pre-existing factors (HTN, Diabetes, Cardiovascular Disease, BMI, type of surgery) and extended PACU recovery times. A multiple linear regression will be used to analyze the data. There is no consensus on mechanisms determining samples; several mathematical formulas exist along with computer program power analysis software to help determine appropriate sample size. These formulas and software programs assure that the number of participants selected for the study provide enough information to establish a valid correlation of event and outcome. Computer programs give an adjusted R^2 (correlation) as well as an actual R^2 (Munro, 2005). This type of computed formula is referred to as a shrinkage formula because of the relationship between the predictive values of actual vs. adjusted R^2 . Calculated math formulas vary, Cohen for example establishes effect size; Nunnally and Bernstein 1994 suggest that 10 subjects per predictor is a minimum “in order to even hope for a stable prediction equation” (p.201).

Published studies have cited occurrence of OSA in western populations within the range of 23-38%. (Benumof, 2004 Seet et al 2010, Eastwood, 2010 Chung et al 2008) More specific approximation of incidence of OSA in surgical patients falls within 24-26%. (Chung et al, 2008) The sample size for this study will be based on the consensus of data establishing a 26% occurrence rate. Utilizing Nunnally and Bernstein (1994) and Katz (2006) guidelines of ten subjects per predictor, 50 subjects with OSA would be a

minimum. Considering the incidence of OSA in the surgical population of 26% the estimated sample size for this study is 100 subjects.

SETTING

Data collection for this research will take place in the preoperative clinic, main operating room, and post-operative acute care unit of the Naval Medical Center San Diego (NMCS D). NMCS D is the largest naval military medical center within Naval Medicine West Region. The navy medical center averages 1,340 surgical cases a month, performing a variety of surgical procedures for numerous specialty medical services. It provides treatment and procedures for the largest number of military beneficiaries in the Western United States.

MEASUREMENT

Obstructive Sleep Apnea is caused by physiological closure of the oropharyngeal airway during sleep and will be assessed and defined by utilizing the STOP-BANG screening parametric, a concise and simple to use screen specifically developed to assess OSA criteria. Use of a previously established database evaluating OSA using the Stop-Bang Measure will be incorporated. The database was generated in a phase one portion of this cumulative research. Stop-Bang is an 8-question (item) assessment that has been implemented and validated for use in defining OSA criteria in a surgical patient

population (Chung, 2008). OSA is defined by a greater than 5 score determined from responses to the 8 questions.

The STOP Bang Questionnaire is made up of 8 questions related to:

- 1). Snoring
- 2). Tiredness
- 3). Observed Apnea during sleep
- 4). High Blood Pressure.
- 5). BMI (Body Mass Index)
- 6). Age
- 7). Neck Circumference
- 8). Gender

It is a self- report, short answer /choice (yes/no) paper and pencil scale that takes an estimated 1 minute to complete.

RELIABILITY AND VALIDITY

Factor Analysis was completed and 4 underlying factors emerged for the 14 questions related to snoring, tiredness, apnea and hypertension. Four questions were chosen that best represented the factor criteria in the simplest report format. Validity was determined via a pilot study comparing results from the STOP questionnaire to the established OSA measurement of polysomnography (sleep study). The sensitivity of the STOP questionnaire was 72%, the specificity was 33.3%, the positive predictive value

75%, and the negative predictive value was 30%. Reliability was established via test-retest. Fifty-five patients answered the STOP questionnaire twice during a time interval of 1-27 days (median 8 days); 53 (96.4%) of the patients had the same score upon retesting. It was also demonstrated that the positive predictive value greatly increased in groups with: BMI > 35 kg/m², age > 50 y/o, neck circumference > 40cm, and male gender. Thus a STOP-BAG measurement has been adapted for even greater reliability that includes B- BMI, A- age, N-neck circumference, and G-gender. (Chung, 2008)

ALDRETE SCORE

Protocols are utilized to immediately assess a patient's recovery from anesthesia after surgery. PACU guidelines govern different phases of the anesthesia recovery process these include early recovery, intermediate recovery, and late recovery. In Phase I, following the anesthesia providers report, PACU nurses implement a scoring system or scale to continue assessment of the patients. The Aldrete Score is a universally used scale, which scores the patient on mobility, respiratory status, circulation, consciousness, and pulse oximetry. The Aldrete score was formulated in 1970 and modified in 1992. Aldrete uses a rating scale of 0 thru 2 to score the five physiological criteria previously listed. A score of 2 in each category is considered best in all areas. The patient is discharged from the PACU when he/she meets the established criteria. A patient must attain an overall score of at least 9 to be considered for progression and discharge.

DATA COLLECTION PROTOCOL

Patients scheduled for elective surgery are directed to the preoperative clinic to complete a preoperative questionnaire. This questionnaire identifies patient having any pre-existing disease factors such as hypertension, diabetes, suspected, or diagnosed obstructive sleep apnea. These patients are flagged and required to have a more in depth assessment by an anesthesia provider prior to their surgery, this could occur anytime between 1 and 14 days prior to their elected procedure. The preoperative nursing staff hand out the STOP- BANG measurement questionnaire along with the standard preoperative questionnaire. The preoperative score the STOP-BANG tool; patients with a score of 5 or greater will receive further assessment from an anesthesia provider. Any other preexisting factors will be identified and assessed by the anesthesia provider at this time.

Phase I. A computerized data query comprised of research study criteria was completed. The primary investigator reviewed query results and compiled a purposive list of participant medical records containing the required data criteria for the study. A purposive sample of medical records was pulled and all data was obtained and recorded by the primary investigator. Data management and security was maintained in compliance with HIPAA standards and those of the Clinical Investigations Department (CID) at NMCSO.

Phase II. Data collection will consist of anesthesia assessment record review, Innovian electronic anesthesia intra-op and post-op record review, essentris electronic record documentation review from PACU and SDS environments. During record review demographic data will be recorded to include age, gender, BMI. In addition PACU recovery time, medications, existing cofactors, type and length of surgical procedure and complications will be recorded. Aldrete and PADSS information and score will be reviewed. STOP BANG measurement questionnaire reviewed and evaluated. CPAP use determined. EKG's will be verified if subjects have HTN, cardiovascular disease or cardiac arrhythmias. Baseline blood glucose will be recorded from patients with DM-II. Time frame for this period was approximately 20-30 minutes per medical record review.

Area Specific Information includes:

- a. Pre-Op Hold Area: existing cofactors, day of surgery medications, NPO status, surgical procedure, review of systems, pain assessment.
- b. Intra-Operative Data: Information obtained from electronic anesthesia record (Innovian) includes intraoperative anesthetic, pain medications, muscle relaxants and use or non-use of relaxant reversal medications.
- c. Post Operative Data: Aldrete scores, PADSS scores, recovery times, incidence of desaturations (O2 Sat < 90%), pulse oximetry measurements, CPAP use, FS blood glucose (DM-II pts.), hypertension documentation, medication treatments for pain and any other co-existing factors (essentris computer record). Additional Data: Any admissions to ICU will be followed and study data will continue to be documented until discharge from ICU. (Electronic record)

The Data collection protocol can be viewed in Appendix C.

STATISTICAL ANALYSIS

Specific Aims:

Aim 1. To examine (or characterize) sleep disturbed breathing (OSA), hypertension, cardiovascular disease, diabetes, and postoperative recovery time, in a cohort of patients undergoing surgical procedures.

Aim 2. To describe the relationships between sleep disturbed breathing (OSA), hypertension, cardiovascular disease, diabetes, and postoperative recovery time among a group of patients undergoing surgical procedures.

Aim 3, 4, 5- Analyze the covariance of independent variables within the two separate surgical groups.

Aim 6, 7 - Examine the relationships that the independent variables (HTN, DM-II, CVD) have on postoperative recovery time. The dependent variable of postoperative recovery time (PORT) will be examined as a categorical variable (PORT longer or not)

Research Questions:

- 1). Examine the relationship of OSA patients with incidence of HTN, DM-II, Cardiac-arrhythmia, CVD, and BMI having elective surgery under general anesthesia.
- 2). Examine the relationship of Non-OSA patients with incidence of HTN, DM-II, Cardiac arrhythmia, CVD, and BMI having elective surgery under general anesthesia.

- 3). Examine the relationships between patients with OSA and prevalence of HTN, Cardiovascular Disease, DM-II, GERD, gender, age, BMI, surgical procedure compared to a group of non- OSA patients after elective surgery under GETA.
- 4). Characterize postoperative recovery time of OSA patients with incidence of HTN, DM-II, Cardiovascular Disease, BMI and GERD having elective surgery under general anesthesia.
- 5). Characterize postoperative recovery time of Non- OSA patients with incidence of HTN, DM-II, Cardiovascular Disease, BMI and GERD having elective surgery under general anesthesia.
- 5). Examine which predictors (HTN, Cardiovascular Disease, DM-II, BMI, GERD, surgical procedure) account for explained variance in Post Operative Recovery Time.
- 6). Examine the relationship between severity or degree of OSA (mild, moderate and severe) and length of postoperative recovery time.

Descriptive statistics were computed for demographic data such as age, gender, marital status, medications, and incidence of sleep study. Descriptive statistics were incorporated to examine pain occurrence, type of surgery and length of surgery.

PROTOCOL

The primary investigator (PI); a privileged ad working staff member in the preoperative,main operative, and postoperative areas of NMCSD; with granted permitted access to the medical records of study participants for the duration of the data-collecting period. IRB approved co-investigators had authority to review pre-operative,

intraoperative, and postoperative data from identified subjects. Incorporating co-investigators ensured maximum enrollment, accuracy and accountability of the data. Study integrity is vital for data accuracy and validity.

PROTECTION OF HUMAN SUBJECTS

The collection of non-invasive medical record data involves a minimal risk to subjects. “Minimal risk” is defined as risks no greater than those experienced in everyday life. An inclusion criterion for this particular study involved the subjects electing to have a surgical procedure requiring general anesthesia. The surgeon and anesthesia provider had already obtained the subjects consent for surgery with general anesthesia. Therefore no additional risk is incurred as a participant of this study. All safety precautions, procedures, and requirements ensured for the preoperative, intraoperative and postoperative portions of this study were maintained. Direct consent for record review was not required; IRB approval was obtained and documented as phase II component of a Phase I preliminary study. Phase I: “STOP BANG TOOL TO DETECT OBSTRUCTIVE SLEEP APNEA FOR THE PERIOPERATIVE PATIENT”

SAFETY PRECAUTIONS AND EMERGENCY PROCEDURES

The data for this study will be obtained from medical records from surgical participants from the Naval Medical Center San Diego, a large Naval Medical Training Facility. The principal investigator is a Naval Officer all HIPPA mandates and security measures were incorporated to protect the privacy of all medical information obtained for

this study. To ensure confidentiality all data was labeled with a unique subject identifier that linked the subject with the data, and only the investigators involved in the study had access to this link. No personal health information was attached.

DESCRIPTION OF THE SYSTEM FOR MAINTENANCE OF RECORDS

Sources of data for this study included the STOP-BANG, Aldrete protocol, and Anesthesia Information Management System (AIMS). All instruments were coded using each subject's assigned number. The master-coding list was kept in a locked file in an assigned office in the anesthesia department at NMCSO and only the investigators involved in the study had access to this data. Data was entered into a password-protected computer. The only link to the subject's identity was by unique subject identification number. No protected health information was on any of the data collection forms or computer files.

Research protocols were maintained in a computer data password protected. Completed protocols were stored in compliance with SECNAVINST 3900.39C and BUMEDINST 3900.6B. Only the investigators had access to the master list. No protected health information was placed on any of the psychometric instruments. These measures ensure compliance with HIPAA standards and those of the Clinical Investigations Department (CID) at NMCSO.

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TABLES

Table 1. Pre-Post STOP-BANG & DX OSA

PRESTOP DATA	7.50%	92.50%	NS
POST STOP DATA	37.60%	62.40%	P< 0.001
COFACTOR DATA	32.60%	67.40%	P< 0.001

Table 2. OSA vs. Non-OSA & Amount Medication

COFACTOR	2.07	1.53	1.91	41	P< 0.05
MEDICATIONS	1.92	1.13	2.64	40	P< 0.01
TIME	283.76	235.75	1.76	40	P< 0.05

Table 3. Logistic Regression: Cofactors and effects on OSA

Variables in the Equation							95% C.I. for EXP(B)	
	B	S.E.	Wald	df	Sig.	Exp(B)	Lower	Upper
Step 1 ^a AirWayProblem s	1.884	.638	8.714	1	.003	6.582	1.884	23.001
HTN	1.017	.528	3.709	1	.054	2.766	.982	7.787
CAD	-.132	.536	.060	1	.806	.877	.307	2.505
Diabetes	-.045	.618	.005	1	.942	.956	.284	3.212
GERD	.201	.469	.183	1	.669	1.222	.488	3.064
Constant	-4.537	1.634	7.708	1	.005	.011		

a. Variable(s) entered on step 1: AirWayProblems, HTN, CAD, Diabetes, GERD.

Table 4. Linear Regression: Cofactors effect PACU Time.

Model	Unstandardized Coefficients		Standardized Coefficients	t	Sig.
	B	Std. Error	Beta		
1 (Constant)	475.705	140.381		3.389	.001
HTN	-202.568	48.910	-.453	-4.142	.000
CAD	56.731	47.692	.125	1.190	.237
Diabetes	63.708	56.032	.115	1.137	.258
GERD	-15.944	42.381	-.036	-.376	.708
Air Way Problems	-27.213	55.520	-.047	-.490	.625

a. Dependent Variable: Total PACU Time

DIAGRAMS

FIGURE 1. STARLING RESISTOR MODEL

FIGURE 2. SLEEP ANEA CYCLE.

APPENDICIES

Appendix A. Stop Bang Tool

Appendix B. Aldrete Score

Appendix C. Data Sheet

APPENDIX A.

STOP-Bang Scoring Model

1. Snoring

Do you snore loudly (louder than talking or loud enough to be heard through closed doors)? Yes No

2. Tired

Do you often feel tired, fatigued, or sleepy during daytime? Yes No

3. Observed

Has anyone observed you stop breathing during your sleep? Yes No

4. Blood pressure

Do you have or are you being treated for high blood pressure? Yes No

5. BMI BMI more than 35 kg/m²? Yes No

6. Age Age over 50 yr. old? Yes No

7. Neck circumference Neck circumference greater than 40 cm? Yes No

8. Gender Gender male? Yes No

High risk of OSA: answering yes to two or more items.

APPENDIX B.

Modified Aldrete Scoring

Activity Able to move four extremities voluntarily on command 2

Able to move two extremities voluntarily on command 1

Able to move no extremities voluntarily on command 0

Respiration Able to breathe deeply and cough freely 2

Dyspnea or limited breathing 1

Apneic 0

Circulation BP $\pm$ 20% of pre-anesthetic level 2

BP $\pm$ 20 – 49% of pre-anesthetic level 1

BP $\pm$ 50% of pre-anesthetic level 0

Pulse Rate Pulse $\pm$ 20 beats of pre-sedation rate 2

Pulse $\pm$ 50 to 21 beats of pre-sedation rate 1

Pulse $>\pm$ 51 beats of pre-sedation rate 0

Consciousness fully awake 2

Arousable on calling 1

Not responding 0

O2 Saturation Maintains baseline saturation on room air 2

Needs O2 to maintain $>90\%$ saturation 1

O2 saturation $<90\%$ with O2 supplement 0

- Patients with an Aldrete score of two (2) points or more below baseline must be referred to the physician responsible for the procedure for further evaluation.
- ** Patients equal to baseline post-procedure will be discharged from the procedure area as in 5.0 of the Sedation/Analgesia Policy

APPENDIX C.

Hospital Stay Date: _____		1
OSA /COFACTOR DATA SHEET		
ID # _____		
SEX _____	AGE _____	WT _____ BMI _____
STOP/BANS SCORE: YES _____ NO _____		
AHI _____ MILD _____ MOD _____ SEVERE _____		
COFACTORS: DM TYPE 1 _____ DM TYPE 11 _____ MEDS: _____		
CAD YES _____ NO _____ MEDS: _____		
HTN YES _____ NO _____ S/P _____ MEDS: _____		
STROKE YES _____ NO _____ MEDS: _____		
OTHER MEDS: _____		
MDC:		SDC:
1. _____	1. _____	
2. _____	2. _____	
3. _____	3. _____	
4. _____	4. _____	
5. _____	5. _____	
SURGICAL PROCEDURE: _____		
GETA: ETT SIZE: _____		
AGENT _____		
VENT: VC _____ MAC _____		
PC _____		
PSVPRO _____		
SIMV _____		
O2 % _____ AIR _____ N2O _____		
PRE-OP VS: S/P _____ HR _____ O2 Sats % _____ RR _____		
SURGICAL CASE: START: _____ END: _____ DURATION: _____		
NARCOTICS: TYPE 1. _____		TOT AMT 1. _____
2. _____		2. _____
3. _____		3. _____
OTHER MEDS: _____		
Surgical Complications: _____		
Airway Complications: _____		
PACU: Arrival Time: _____ Discharge Time: _____ Total PACU Time: _____		
O2 % _____ ROOM AIR _____		
POST-OP VS: S/P _____ HR _____ O2 Sats % _____ RR _____		
NARCOTICS: TYPE & Time 1. _____		TOT AMT 1. _____
2. _____		2. _____
3. _____		3. _____
OTHER MEDS: _____		
Airway Complications: _____		
ADMIT: Planned: _____ Reason: _____		
Unexpected: _____ Reason: _____		

Manuscript One

**STOP-BANG TOOL TO DETECT OBSTRUCTIVE SLEEP APNEA FOR THE
PERIOPERATIVE PATIENT**

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Conflict of Interest

The views expressed in this article are those of the authors and do not necessarily reflect the official policy or position of the Department of the Navy, Department of Defense, University of San Diego, or the United States Government. This work was prepared while the author was a student at the University of San Diego and as part of the author's official duty. Title 17 U.S.G. 105 provides 'Copy-right protection under this title is not available for any work of the United States Government.' Title 17 U.S.C. 101 defines a United States Government work as a work prepared by a military service member or employee of the United States Government as part of that person's official duties

Abstract

Introduction: According to the American Heart Association, fifteen million adults are affected by obstructive sleep apnea (OSA).¹ Chung et al.⁵⁻⁶ developed, piloted, and validated the STOP-BANG questionnaire as an OSA screening tool for surgical patients at preoperative clinics. This questionnaire is used to assist the perioperative team with routine screening for OSA. The national incidence of sleep apnea is 25% for men and 10% for women. At Naval Medical Center San Diego (NMCS D) the current incidence of sleep apnea is 11%. This study was designed to evaluate the effects of implementing the STOP BANG tool in preoperative clinics to identify undiagnosed OSA surgery patients.

Design and Methods: Pre and post quasi-experimental time series design project to include two phases: Phase One: pre-data collection; Phase Two: pre-operative assessment form and post-data collection. Five hundred and five charts were reviewed in phase one and phase two for a total same size of 1010. Pre and post means were evaluated using a student t-test; Chi-Square analyses was used to compare significance of OSA with snoring, hypertension, age and BMI. A correlational analysis was utilized to see which of the above variables had the most impact on sleep apnea. NMCS D IRB approved.

Results: There was no difference in demographic data. Pre identification of OSA patients was 7.5%. Post identification of OSA patients was 37.6 %. Use of the STOP-BANG tool increased OSA detection by 30.1%. Males and higher ASA classification were correlated with OSA ($p < 0.00$). The chance of identifying patients with OSA by using the STOP-BANG tool increased by 75% ($p < 0.001$).

Conclusions and Implications: Anesthesia and surgery can affect sleep in combination with the post-operative effects of anesthesia. Clinical suspicion of OSA may not be clear to the anesthesia provider. Use of the STOP-BANG tool in anesthesia pre-screening will increase patient safety.

Keywords: Obstructed Sleep Apnea, obese, STOP-BANG, apnea, hypercapnia, hypoxemia, hypertension.

Introduction

According to the American Heart Association, fifteen million adults are affected by OSA with a higher prevalence in obese men and patients suffering from cardiovascular disease.¹ OSA is a syndrome characterized by periodic, complete or partial obstruction of the pharyngeal airway during sleep.² Currently, an OSA screening tool is not used in the perioperative setting to detect undiagnosed OSA in patients. The current incidence of sleep apnea at NMCSO is 11% as indicated by quarterly quality assurance benchmark indicators.

The OSA sufferer has complete cessation of airflow lasting longer than 10 seconds during sleep despite continued respiratory efforts resulting in hypercapnia [increased carbon dioxide (CO₂) levels] and hypoxemia.² Usually the patient's apnea is terminated when his or her CO₂ levels build up enough to stimulate the central nervous system (CNS). Stimulation of the CNS arouses the individual enough to overcome the airway

obstruction.³ Often the inspiratory effort is a forceful opening of the pharyngeal airway and the airway is reestablished by loud snoring.⁴ Such respiratory efforts may be noted by the individual's family as a loud snort or snoring. The apnea causes repetitive arousal from sleep to restore airway patency, which may result in a poor sleep pattern with daytime sleepiness or hypersomnolence.³

According to the National Sleep Foundation, one in four Americans are at high risk for OSA, making the diagnosis the most prevalent sleep disorder.¹ The "Gold Standard" for OSA diagnosis is by polysomnography (PSG) to ascertain an apnea-hypopnea index (AHI). PSG is conducted during a sleep study.⁵ The patient wears a sensor attached to the head and body to record brain waves, eye movements, muscle activity and cardiovascular activity while sleeping overnight in the sleep laboratory. AHI is the calculated sum of apneic and hypopnea events experienced during each hour of sleep. OSA is diagnosed by the presence of a least five obstructive apneas, hypopneas, or both, per hour while the patient is sleeping. OSA is graded into three levels of severity: mild (AHI greater than 5 but less than 15 events per hour); moderate (AHI 15 to 30 events per hour); and severe (AHI of greater than 30 events per hour).⁶ PSG requires highly trained personnel, complex equipment, and is costly to perform. According to Chung⁷, most sleep study centers have long waiting lists requiring patients to wait for months for OSA diagnosis and treatment. Due to patients' inability to have a PSG performed, there has been a renewed clinical interest in finding a simple, easy tool to identify undiagnosed OSA patients.

The American Society of Anesthesiologist published guidelines recommending that patients be screened for OSA risk prior to entering the perioperative suite.² Typically, surgical patients have their preoperative clinic visit a few weeks prior to surgery. OSA has been linked to hypertension, heart failure, atrial fibrillation, stroke, myocardial infarction, and nocturnal sudden death.¹ Chung⁷ also suggested that OSA is a serious co-morbidity problem and needs to be evaluated for patients with a BMI greater than 40. ASA guideline recommendations have produced a number of predictive clinical models, but they usually require computer assistance and are not suitable for clinical practice.⁷ As a result, several different questionnaires have been designed for OSA screening. These questionnaires are the ASA checklist, Berlin questionnaire, STOP questionnaire (snororing, tiredness during daytime, observed apnea, and high blood pressure) and STOP-BANG Tool (which includes BMI, age, neck circumference, and gender).⁷ The purpose of this project is to introduce the STOP-BANG tool into our preoperative clinic to help identify undiagnosed OSA patients prior to surgery. The utilization of the STOP-BANG tool in the pre-operative clinic will aid the perioperative team in developing an appropriate anesthesia plan following ASA practice guidelines. This should lead to an increase in OSA detection for undiagnosed patients thus increasing patient safety.

Chung et al.⁵⁻⁶ developed, piloted, and validated the STOP-BANG questionnaire to be used for surgical patients to assist the anesthesia provider with OSA screening. The STOP questionnaire consists of four questions:

S: 'Do you snore loudly, louder than talking, or loud enough to be heard through closed doors?'

T: 'Do you feel tired, fatigued or sleepy during the daytime?'

O: 'Has anyone observed that you stop breathing during your sleep?'

P: 'Do you have or are you being treated for high blood pressure?'

The questionnaire uses a yes/no format to keep it concise and easy to use.⁷ If patients answer yes to two or more questions, they are at high risk of suffering from OSA. If patients have less than two positive answers, they are at low risk of having OSA. The STOP questionnaire sensitivity for an AHI of more than 5, more than 15 and more than 30 events was 65.6%, 74.3%, and 79.3%, respectively.⁷

An alternative scoring tool described by Chung et al.⁵⁻⁶ is called the STOP questionnaire and BANG-BMI tool. The BANG-BMI portion consists of B - BMI, A - age, N - neck circumference, and G - gender. The addition of these patient characteristics to the STOP questionnaire improves the sensitivity of the tool, detecting most patients with OSA; particularly those with severe and moderate OSA.⁷ In the BANG portion of the tool, BMI greater than 35, age older than 50 years, male gender, and neck circumference greater than 40 cm are scored positively. Patients answering yes to three or more items are considered at high risk for OSA.⁷

The BANG addition to the STOP questionnaire increased its sensitivity using an AHI greater than 5 (mild), more than 15 (moderate), and more than 30 (severe) events to 83.6%, 92.9%, and 100%, respectively.⁷ A moderately high level of sensitivity and specificity in surgical patients was demonstrated by the STOP questionnaire, and it was

more sensitive for OSA detection in patients with severe (AHI > 30) or moderate (AHI > 15) OSA. A 100% predictive level could be found with patients with certain clinical characteristics: male over 50, BMI greater than 35 and neck circumference greater than 40 cm.⁷ Chung et al.⁵⁻⁶ reported that incorporating BMI, age, neck circumference, and gender (BANG) into the STOP questionnaire increases tool sensitivity for the undiagnosed OSA patient. If the STOP-BANG questionnaire ranks a patient as low risk, the patient can be excluded from having moderate to severe OSA.⁷

The STOP questionnaire is an easy to use screening tool to identify patients with OSA. Incorporating BMI, age, neck size and gender into the STOP questionnaire (STOP-BANG) further increases the predictive value for patients with severe to moderate OSA. The STOP-BANG questionnaire will aid the perioperative team in evaluating the patient for OSA and in developing an appropriate anesthesia plan following ASA practice guidelines. The purpose of this study is to see if the STOP-BANG tool can increase the identification of patients at risk for OSA, which will decrease their risk for complications during anesthesia and improve patient safety.

Framework

The Iowa Model of evidence-based practice to promote quality preoperative care gives a basic outline to help implement the STOP-BANG tool. The OSA diagnosis is the clinical problem to be addressed, but the process of assessment also needs to be improved within the clinical setting. Since current research supports utilization of an OSA screening tool to identify potential OSA patients, the STOP-BANG tool will aid the

anesthesia provider in following ASA practice guidelines for the peri-operative management of these patients.

In reviewing the literature, there are many case reports of difficult tracheal intubation and cardio-respiratory consequences of OSA but very little research on patients with OSA and their peri-operative mortality and morbidity.⁷ In a recent systematic review by Chung et al.⁵⁻⁶, the evidence of the potential deleterious effects of anesthesia, sedatives and analgesics are compounded for the undiagnosed OSA patient. The increased risk of peri-operative adverse occurrences implies that the anesthetic management plan needs to be patient specific. It is imperative for the anesthesia provider to meet the challenge of maintaining an unobstructed patient airway and preventing perioperative complications.⁵⁻

⁶ The ASA guidelines emphasize the significance of evaluation, detection and preparation within the preoperative visit for the OSA patient when going for a surgical procedure.²

Methods / Design

This study used a pre and post quasi-experimental time series design. One thousand and ten patients were enrolled in two phases. Phase One was done before implementation of the STOP-BANG tool. Phase Two was completed after implementation of the tool. Subjects were recruited in the preoperative clinic. Inclusion criteria consisted of the following: 18 years or older, scheduled to undergo elective procedures in general surgery, urology, plastic surgery, orthopedics, gynecology, ophthalmology and neurosurgery. The subject had to be able to read and

write English, alert and oriented. Exclusion criteria consisted of the following: subjects who were unwilling or unable to fill out the STOP –BANG questionnaire, patients previously diagnosed with OSA or any other sleeping / breathing disorder, or patients expected to have abnormal neurological functioning. While filling out their anesthesia paperwork, preoperative nurses asked all patients who met inclusion criteria to complete the STOP-BANG questionnaire. The STOP-BANG tool is displayed in figure 1.

- ✦ Do you have sleep apnea?
- ✦ Do you use CPAP at home?
- ✦
- ✦ Scored Questions
- ✦
- ✦ 1. Do you snore loudly? (louder than talking or loud enough to be heard through closed doors)
- ✦ 2. Do you often feel tired, fatigued or sleepy during the daytime?
- ✦ 3. Has anyone observed you stop breathing during your sleep?
- ✦ 4. Do you have or are you being treated for high blood pressure or have you been advised to be treated for high blood pressure?
- ✦
- ✦ High risk of OSA: Yes to 2 or more questions
- ✦ Low risk of OSA: Yes to fewer than 2 questions
- ✦
- ✦ Additional questions:
- ✦ Height cm _____ Weight kg _____ BMI kg/m^2 _____ (35 kg/m^2)
- ✦ Age _____ (50) Gender _____ (M) Neck Circumference cm _____ (40cm)
- ✦
- ✦ Values $\geq$ those in parentheses increase sensitivity and
- ✦ decrease specificity of test if included in determining likelihood of OSA

Figure 1. STOP-BANG. Adapted from ASA practice guidelines.²

Every surgery patient was provided a pre-operative packet that included the STOP-BANG tool. The pre-operative clinic nurse performed the final review of the patient's paperwork. The nurse insured that the form was completed and measured the patient's neck circumference. Two reviewers performed the baseline data collection before and after tool implementation. The patient's characteristics were documented for baseline data to assess for the current patient population total. Baseline data consisted of: gender, age, weight (BMI), snoring, hypertension, tiredness, and obstruction; if the patient suffers from OSA what is the severity; have they had a sleep study; if the patient denies OSA, is there a suspicion by the anesthesia provider.

After implementation of the STOP-BANG tool, statistical analysis was done to compare the percentage of pre and post OSA diagnosed patients and to establish a baseline for OSA at the facility. Baseline data collected before tool implementation and after patients completed the STOP-BANG questionnaire was compared using Chi-square to test for differences in gender, age, weight (BMI), snoring, hypertension, tiredness, and airway obstruction. The Chi-square test helps determine if the STOP-BANG tool detects the undiagnosed OSA sufferer in the elective surgery population.

To determine the number of undiagnosed OSA patients seen by the pre-operative clinic, a pre implementation chart review was performed using the facility's anesthesia records. After orientation to the tool for all pre-operative clinic nurses and anesthesia

providers, all elective surgery patients were asked to fill out the STOP-BANG questionnaire which was placed in a pre-designated position within the chart.

Data collection related to the incidence of OSA in the population occurred over a three-month period prior to program implementation and for several months post implementation. All post-operative charts were collated within the chart house. The charts remained within the chart room for 60 days or until completion by the providers. During tool implementation, team members addressed any questions that arose. The elective surgery population is a random cluster sample. The sample size was dependent on the elective surgery schedule for the month. Chung et al.⁵⁻⁷ and Chung⁷ articles were used to establish the sample size. Five hundred and five patient charts were evaluated in the Phase One and Phase Two for a total sample size of one thousand and ten. Ten percent was added to the sample size for lost data. The actual number fluctuated depending on the rotational basis of surgery residents and deployment schedule of staff surgeons. Actual tool performance was simple for patients and pre-operative nurses. Data was entered into SPSS 19 to aid in data analysis. Nurses were trained to measure all patients for neck circumference uniformly at the level of the cricoid cartilage. A regression analyses model was utilized to see which variables impacted the dependent variable post implementation sleep apnea. IRB approval was obtained at the primary clinical facility and at the university campus.

Table 1. Comparison of Baseline Data and Post STOP-BANG Tool Implementation
(N=1010)

	Pre- Implementation	Post- Implementation	Significance
Male	61%	65%	NS
Female	39	35%	NS
Age	47.85	51.50	NS
BMI	27.32	28.76	NS
OSA diagnosed	7.5%	37.6%	P<0.001
Hypertension	24%	43%	P<0.001
Snoring	2.6%	30.9%	P<0.001
Medications	1.13	1.92	P<0.01
PACU Time	235.75	283.77	P<0.05

Graph 1. Comparison of Positive and Negative Pre-Sleep Apnea Data vs. Post-Sleep
Apnea Data

Table 2. Correlation Analyses Model Results Comparing the Impact of HTN, BMI and Age on Sleep Apnea.

Coefficients ^a						
Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	1.698	.268		6.344	.000
	PoHTN	.321	.095	.328	3.391	.001
	PoBMI	-.110	.044	-.215	-2.478	.015
	PoAge	-.107	.042	-.243	-2.569	.012

a. Dependent Variable: PoSA

Results

This sleep apnea project was completed in two phases. In Phase one, five hundred and five post-surgical charts were retrospectively reviewed for demographic and STOP-BANG data. In Phase Two, five hundred and five surgical patients completed the STOP-BANG tool during their preoperative visit, and their charts were retrospectively reviewed for demographic and STOP-BANG data. Forty-five charts from each group

were dropped due to lost data points. There was no difference in demographic data. Pre identification of OSA patients was 7.5%. Post identification of OSA patients was 37.6% (Graph One). Use of the STOP-BANG tool increased OSA detection by 30.1% ($p<0.001$). Hypertension, snoring, patient medications and extended PACU times were significantly different between the pre and post implementation groups (Table 1). Males and higher ASA classification were correlated with OSA ($p<0.001$). There is a 75% increased chance of identifying patients with OSA if the STOP-BANG tool is used ($p<0.001$). A correlation analyses model was completed looking at the impact of predictors on post implementation sleep apnea. The correlation model was significant, and it appeared that hypertension was the most significant associated with sleep apnea ($p<0.001$). BMI and patient age were also associated with sleep apnea, but not as significant ($p<0.015, 0.012$) (Table Two).

Discussion, Conclusion and Implications

Chung et al.⁵⁻⁶ clearly states that the incidence of sleep apnea is 25% for men and 10% for women. Before this evidence-based project was undertaken, we were postoperatively identifying 11% of sleep apnea patients. During this project, we were able to identify 37.6% of our surgical population who had sleep apnea. This was a 26.6% improvement over our prior clinical assessment. We obtained results similar to Chung et al.⁵⁻⁶ in identifying 28.3% of men and 9.3% of women. This project also confirmed Sommers et al.¹ work that cardiovascular issues such as hypertension may be a predictor for sleep apnea as well as snoring and may lead to prolonged recovery time as shown in our work.⁸

Future research is needed to confirm which co-factors impact recovery time in sleep apnea patients. In conclusion, anesthesia and surgery can affect sleep apnea in combination with the post-operative effects of anesthesia.⁸ Clinical suspicion of OSA may not be clear to the anesthesia provider. The use of the STOP-BANG tool is an easy addition to anesthesia pre-screening and increases patient safety. OSA syndrome impacts each phase of the anesthesia plan.³ In the pre-operative phase, the nurse or anesthesia provider may be the one to assess the patient for OSA prior to surgery. Within the operative phase, the anesthesia provider in charge ultimately decides on the type of anesthesia and pain medicine the patient is provided during their procedure. Within the recovery phase, the patient needs balance in pain control, comfort, and airway patency.³ The recovery room nurse monitors the patient for depressed ventilatory responses that can occur after anesthesia. Typically, hypercarbia and hypoxemia triggers the patient to arouse after anesthetics and analgesics, but the OSA sufferer has a depressed response.² Using a screening tool can facilitate a diagnosis of OSA, especially for patients who are not aware they have it.³ The pre-tool and post-tool evaluation found that 26.6% of patients were undiagnosed within our pre-operative clinic. The ultimate long-term goals of utilizing the STOP-BANG tool is reducing prolonged recovery room stays by 50% and reducing unplanned over-night admissions by 75%. Whether or not this is accomplished will be determined in future studies. In a recent by Chung et al.⁹, patients with OSA were found to have an increase in postoperative complications.⁸ Ultimately, increasing patient safety by pre-screening potential OSA patients will decrease our institutional mortality rate for undiagnosed patients to zero. The use of a screening tool for OSA will aid the

anesthesia provider in the decision-making needed to safely treat these patients intra-operatively.²

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Manuscript Two

UNIVERSITY OF SAN DIEGO

Hahn School of Nursing and Health Sciences

DOCTOR OF PHILOSOPHY IN NURSING

OBSTRUCTIVE SLEEP APNEA AND THE EFFECTS OF CO-EXISTING FACTORS
ON PACU STAY TIME

By

Shari F. Jones, CRNA, PhD

A dissertation presented to the

FACULTY OF THE HAHN SCHOOL OF NURSING AND HEALTH SCIENCES

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DOCTOR OF PHILOSOPHY IN NURSING

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ON PACU STAY TIME

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ABSTRACT

Significance/Background: Obstructive Sleep Apnea (OSA) is the most common disturbance during sleep affecting 30% of the population. The occurrence of moderate/severe OSA is estimated at 11.4% in men and 4.7% in women. OSA is caused by repetitive partial or complete obstruction of the upper airway with apnea episodes. OSA is prevalent in western society, yet is frequently undiagnosed due to lack of knowledge of the disorder. This population presents a challenge during the peri-operative period due to increased risk for several complications. Of particular interest to Nurse Anesthetists, are respiratory complications that result in delayed emergence from anesthesia, delayed post-operative recovery, increased apneic episodes, hypoxemia, and death. Examining duration of recovery periods between the two OSA groups will help determine if patients with additional cofactors benefit from longer post-operative monitoring or modification of anesthetic technique.

Methods: An observational correlation design to include a non-randomized convenience sample of two groups. Comparisons will be made between groups regarding cofactors, complications and duration of postoperative recovery times. Patients having elective abdominal surgery under general anesthesia at a Military Treatment Facility will be identified. OSA status will be determined based upon Sleep Studies, or STOP-BANG tool criteria. Medical, surgical, and anesthesia data will be reviewed. Total recovery times and pre-existing disease documented. Multiple Logistic and Regression testing will compare the effects of cofactors on postoperative recovery times. Chi square analysis will evaluate relationship of individual cofactors with OSA and Non-OSA patients. T-test analysis will compare demographic information. P score of $< .05$ will be considered significant.

Results: This study indicated significant correlation with higher number of cofactors amongst patients diagnosed with OSA ($p < 0.012$). Increased incidence of higher ASA

classification in OSA diagnosed patients; ($p < .017$) extended PACU stay time in OSA diagnosed patients; ($p = 0.05$) and Unplanned admissions in OSA patients; ($p = .007$). Patients with OSA use a higher number of prescribed medications as compared to non-OSA patients, they have a 3.36 greater chance of having hypertension as an additional cofactor ($p < .05$) and they were 8.75 times more likely to experience airway difficulties or complications with general anesthesia when compared to non-OSA patients ($p < .05$). Multiple logistic regression testing demonstrated increased incidence for both airway difficulties ($p = .003$) and Hypertension ($p = .054$). Multiple linear regression results indicate one cofactor HTN as a significant predictor for PACU stay time. ($R^2 = .179$, $R^2_{adj} = .136$, $F(1)$)

Implications: Findings will lead to optimum monitoring, management; recovery measures and anesthesia techniques that will prevent extended postoperative periods and reduce or eliminate postoperative complications of OSA.

Key Words: Obstructive Sleep Apnea, Cofactors, STOP-BANG

Introduction

Obstructive Sleep Apnea (OSA) is the most common disturbance during sleep affecting 2-26% of the general population.¹⁻² The occurrence of moderate to severe OSA is estimated to be 11.4% in men and 4.7% in women. OSA is a prevalent problem in Western Society estimated to occur between 1 to 9% in patients presenting for surgery, yet it is frequently undiagnosed due to lack of awareness or knowledge of the disorder.³⁻⁴

OSA is caused by repetitive partial or complete obstruction of the airway during sleep. Diagnosis of OSA is determined by the occurrence of airway obstruction during sleep with apnea periods lasting greater than 10 seconds for five or more times per hour; with a decrease in arterial oxygen saturation (SaO₂). These events are known as apnea/hypopnea episodes and the measurement of episodes is referred to as the Apnea-Hypopnea Index [AHI]. The degree of severity of the disorder is determinate of the number of AHI per hour. AHI: 5-15 mild, ≥ 15 -30 moderate and > 30 severe.³⁻⁴

A polysomnography otherwise known as a sleep study is recognized as the “Gold Standard” for the diagnosis of OSA. Polysomnography data defines the degree of severity of the condition and number of apneic episodes. Unfortunately polysomnography is time consuming; patients are reluctant to participate in them; they require an extended stay in the sleep lab; and they are expensive. Medical professionals have become more aware of OSA as a significant disorder with resulting adverse effects for their patients. More physicians are consulting to sleep labs to ascertain severity and the availability of appointments have become more limited. Unfortunately for the OSA patient, polysomnography cannot be obtained rapidly and in the case of surgical interventions that may be required for urgent or emergent disorders OSA may go undetected.¹⁻²

Subsequently, the OSA patient population presents a unique challenge during the peri-operative period due to an increased risk for lack of identification and subsequent life threatening complications. Of particular interest to the Nurse Anesthetist, are the respiratory complications that can result in delayed emergence from anesthesia, delayed postoperative recovery, increased apneic episodes, hypoxemia, cardiac disturbances and ultimate death in this patient population.

Obesity, gender, snoring and age have demonstrated to be strong indicators of occurrence for OSA. Hypertension, cardiovascular disease, diabetes and stroke are prevalently reported as confounding and coexisting factors.⁵ In 2003 Netzer et al, surveyed OSA patients in twenty-six US primary care clinics and fourteen European primary care clinics comparison of the results demonstrated similar symptoms of age, gender distribution and hypertension.⁶ Obesity was common among both sets of OSA patients although the incidence was much higher in the United States than in Europe. Another study obtained in Spain by Duran et al, 2001 reported linkages between OSA and hypertension after interviewing over 2,000 patients.⁷ Elevated blood pressure and refractory bradycardia followed by abrupt tachycardia are cited as predominate cardiovascular effects of OSA. The hemodynamic instability seems to be related initially to hypoxemia from the airway obstruction, and then sudden arousal as the patient awakens to breath. The rapid fluctuation of vaso-activity is thought to be responsible for the incidence of cardiac arrhythmias, the development of chronic arterial hypertension, the occurrence of myocardial infarction and sudden cardiac death.⁸

The severity and incidence of documented post surgical complications from patients with moderate to severe OSA is significant. Yet, most of the informational data of

incidence, occurrence, surgical complications and treatment of the OSA patients come from case reports and retrospective reviews not evidenced based scientific studies.

The American Society of Anesthesiologist (ASA) developed guidelines in 2006, based on Level IV evidence and expert consensus reports to address the identification and treatment needs of the OSA patient. The ASA consultants reached agreement on treatment criteria for most simple outpatient and minor orthopedic surgical procedures but could not find consensus regarding more invasive surgeries. The guidelines propose that for more invasive surgery certain factors need to be considered when determining postoperative risk for the OSA patient these included: status of sleep apnea, (treated or untreated), level of comorbidities (hypertension, cardiovascular disorders, diabetes are they appropriately managed), type of surgery, and anesthesia modality.¹⁻²

Key experts in the diagnosis and treatment of the OSA patient acknowledge poor quality of evidence in an article from the American Academy of Sleep Medicine; “the scientific literature regarding perioperative risk and best management techniques for OSA patients was scanty and of limited quantity”.⁹

The ultimate goal for all Nurse Anesthetists is to provide the safest, highest quality anesthetic management and care to their patients. If Nurse Anesthetists can identify and understand the coexisting factors creating the most risks for perioperative complications in the OSA defined patient they can better prepare themselves to manage those risks and provide optimum care. They can select anesthetic practices that minimize risk and maximize safer outcomes.

Development of more concise and accurate techniques to eliminate the factors producing increased risk for this population could be incorporated. Valid criteria for

preoperative identification of the OSA patient could be developed and preoperative management techniques could be identified. Established guidelines and protocols could be created to eliminate most if not all risk factors. The complete elimination of incidence and occurrence of morbidity and mortality for the OSA patient in the perioperative environment would be the ultimate goal.

The purpose of this study is to identify the preexisting factors that significantly affect postoperative recovery times between patients with Obstructive Sleep Apnea compared to patients without Obstructive Sleep Apnea undergoing abdominal surgical procedures. Specific aims to determine the incidence and occurrence of pre existing cofactors: hypertension, coronary artery disease, and diabetes will be evaluated. Secondary aims are to determine if relationships of occurrence exist between the co-existing factors. If relationships of co-existing factors are determined can they be further categorized as positive or negative relationship?

OSA will be defined utilizing the STOP-BANG screening parametric, a concise and simple to use screening tool specifically developed to assess OSA criteria. Stop-Bang is a 12-question tool that has been implemented and validated for use in defining OSA criteria in a surgical patient population. Obstructive Sleep Apnea is defined by a greater than 5 score determined from responses to the 12 question measure.¹⁻²

An English physiologist Ernest Starling identified a theoretical phenomenon of vessel capacitance as influenced by flow, forces and pressure. This Theory became known as the Starling Resistor Theory and explains the function of fluid flows through the coronary arteries. The Starling Resistor Theory is offered as a theoretical framework to compare and contrast airway physiology and function within the normal adult airway and in the

OSA airway.

The starling resistor model can be readily applied to describe flow in the upper airways. The concept provides an excellent explanation of airway dynamics as influenced by changing forces or pressures. A resistor is described as a collapsible tube passing through a chamber that is capable of having its pressure changed. For adaptation purposes we can identify the oropharynx as the tube and the surrounding tissue of the neck as the chamber. If the oropharynx remains open then airflow through will be determined by the pressure differences from the two sides of tissue within the neck; from the pressure above; the proximal region of the nose or mouth and from the pressure below; the distal portion of the trachea.

The main characteristic dysfunction of OSA is obstruction of airflow. The patient with OSA has recurrent apneic events that are the result of a closed or “blocked” airway that occurs during sleep. To understand the co-existing factors that may contribute to the adverse affects of OSA we must first understand the anatomic and physiologic process that defines OSA. The airway of the OSA patient presents differently than that of a typical adult airway. An OSA patient’s airway is smaller and narrower in contrast. The OSA airway has a more lateral axis while the typical adult airway is more horizontal. An excess of redundant tissue causes this variant. In fact a definitive feature of OSA patients is an increased neck circumference or “thick neck”. When sleeping the oropharyngeal muscles of the adult airway are relaxed and pharyngeal narrowing occurs and there is increased negative inspiratory pressure especially during rapid eye movement (REM) sleep. Additional inspiratory negative pressure is generated by diaphragmatic excursion and pharyngeal dilator muscle activity is incorporated as a compensatory mechanism that

maintains airway patency in the normal adult. In the OSA patient pharyngeal dilator muscle activity maintains patency during awake periods but the muscle activity is lost when the OSA patient is asleep.

Excessive or adipose tissue in the neck of the OSA patient surrounds the oropharynx and compresses the airway making muscular activity less effective. Increased negative inspiratory pressure along with snoring, which can cause vibratory trauma, can further enhance upper airway narrowing.¹¹

The tissue pressure (P_{tissue}) corresponds directly to the pressure with which the surrounding tissues of the oropharynx constrict the upper airway. A high opening pressure (P_{proximal}) will assist in overcoming the collapsing pressure of tissue (P_{tissue}). A low or negative pressure below the vocal cords (P_{distal}) that occurs as a result of the inspiratory effort will contribute to airway collapse if P_{tissue} is elevated. When sufficient muscular tone is operative in tissues surrounding the collapsible tube, in effect “lifting the tissue” away from the collapsible portion P_{tissue} does not contribute to airway collapse.¹² In the OSA patient arousal is essential for airway patency and survival.

Theoretical Perspective

Starlings Resistor Theory was formulated to explain the properties of vascular compliance and resistance forces. The theory has already been established and accepted as defining the properties of fluid flow through blood vessels, and vascular circulation. Although the coexisting factors of hypertension and cardiac disturbances in the OSA patient are not fully understood an association with flow, increased pressure and collapsibility may offer an explanation using Starling’s Resistor Theory. Applying this

theoretical framework for both airway and hemodynamic pressures is appropriate and could indicate symbiotic relationships.

Fig 1. Starling Resistor Theory Diagram

P_d (distal)=upstream pressure, P_p (proximal)=downstream pressure and P_t (tissue)=pressure in the box. ¹²

Lack of oxygenation leads to hypoxemia and hypercarbia. Increased levels of CO_2 stimulate chemoreceptors within the CNS, which in turn release neurotransmitters promoting arousal. Reflex response activates pharyngeal dilator muscles and airways open, ventilation is returned. ⁴

supporting evidence in the postoperative setting, that sleep mechanics were disturbed due to the requirement for increased analgesia for surgical pain. Analgesia through the first three days after surgery tended to incur a life threatening apnea from drug-induced sleep. He also described rebounds in REM sleep originating from poor quality sleep this was believed to increase the danger of a detrimental natural deep sleep- induced apnea. He suggested that patients with OSA are at risk of prolonged apnea during sleep for up to one week in the postoperative period.¹⁴

Correlations between hypoxic events and episodes of recurrent oxygen desaturations associated with adverse outcomes have been reported. Gupta et al (2001) examined procedure specific complications in the post operative period and found that OSA patients experience significantly more occurrence of complications than the group without OSA. Complications included acute hypercapnea, PaCo₂ >45, episodic desaturations reflected by SpO₂ <90% with witnessed apneic events mmHg; arrhythmias, MI, or infarction, and delirium.¹⁵

In the United Kingdom a mailed survey was provided to a group of anesthetists to define perioperative care of adults with OSA: Perioperative complications included 34% difficult intubations, 47% difficult ventilations, 24% cardiovascular instability, difficult analgesia in 41%, difficult extubation 13%, postoperative apnea 46%, required reintubation 27%, unplanned ICU admission 23%, and acute cardiac events 25%.¹⁶

These findings support the need for accurately assessing the postoperative status of OSA patients and determining criteria for risk assessment. Studies have shown strong relationships between the numbers of oxygen desaturations episodes within the post anesthesia care unit to occurrence of adverse events.

Questions could be posed regarding the causes of the postoperative complications. OSA attributed events such as: Hypoxia, Hypopnea, Hypercapnea, snoring etc. Are these conditions independently caused by the OSA disorder? Or is their synergistic components with other cofactors (HTN, Diabetes, CAD) that creates the adverse events? Does the OSA patient with one or more cofactors have more adverse events and do they require a longer PACU stay? There currently is no clear explanation. It would be valuable information for the anesthesia provider and other health care providers to know the most significant cause of adverse events in the OSA patient.

METHODOLOGY

An observational correlational design method will be utilized for determining the relationship of postoperative ambulatory care unit (PACU) stay times and hypertension (HTN), diabetes (DM-II), coronary artery disease, and body mass index (BMI) in obstructive sleep apnea (OSA) patients.

Sample

A purposive sample of active duty, retired, or family members of military service members having consented to elective abdominal surgery at the Naval Medical Center San Diego (NMCS D) and received a score of 5 or greater from preoperative evaluation using the STOP-BANG screening measurement.

Inclusion Criteria consists of males and females 18 to 65 years, who have elected to have general anesthesia for abdominal surgery, category ASA I & II. Exclusion Criteria will consist of members above who have pre-existing pulmonary disorders (COPD, etc.),

neurological dysfunction, delayed or decreased cognitive function, recent MI.

Demographic data will include age, gender, BMI, medications and type of surgical procedure.

Sample Size Calculation

For the purpose of this study which is to determine if identified obstructive sleep apnea (OSA) patients have longer PACU recovery times as compared with non-OSA patients undergoing abdominal surgery and if there is an association between OSA patients with pre-existing factors (HTN, Diabetes, Coronary Artery Disease, BMI) and extended PACU recovery times. A multiple logistic regression will be used to analyze the data. Several mathematical formulas exist along with computer program power analysis software to help determine appropriate sample size. These formulas and software programs assure that the number of participants selected for the study provide enough information to establish a valid correlation of event and outcome. Published studies have cited occurrence of OSA in western populations within the range of 23-38%.^{1-2,3,14,17} More specific approximation of incidence of OSA in surgical patients fall within 24-26%.¹⁻² The sample size for this study will be based on the consensus of data establishing a 26% occurrence rate. Utilizing Nunnally and Bernstein (1994) and Katz (2006) guidelines of ten subjects per predictor, 50 subjects with OSA would be a minimum. Considering the incidence of OSA in the surgical population of 26% the estimated sample size for this study is 119 subjects.^{18,19} Data collection for this research will consist of a medical record review of patients from the Naval Medical Center San Diego who have undergone an elective abdominal surgery utilizing general anesthesia. Preoperative, intraoperative and postoperative data will be evaluated for study criteria.

Measure of Obstructive Sleep Apnea

OSA will be defined utilizing the STOP-BANG screening parametric, a concise and simple to use screening tool specifically developed to assess OSA criteria. The STOP Bang Questionnaire is made up of 4 yes/no questions related to:

1. Snoring 2. Tiredness 3. Observed Apnea during sleep 4. High Blood Pressure. It is a self report, choice (yes/no) paper and pencil scale that takes an estimated 1 minute to complete.¹

Aldrete Score Measurement

Protocols are utilized to immediately assess a patient's recovery from anesthesia after surgery. A universally used scale is the Aldrete scale, which scores the patient on mobility, respiratory status, circulation, consciousness, and pulse oximetry. The Aldrete score was formulated in 1970 and modified in 1992. Aldrete uses a rating scale of 0 thru 2 to score the five physiological criteria previously listed. A score of 2 in each category is considered best in all areas. The patient is discharged from the PACU when they meet the established criteria for discharge an overall score of 9. This measurement will be utilized to determine overall PACU stay times.

Results

Findings from our pilot study of 100 patients demonstrated that OSA patients were 8.75 times more likely to experience airway difficulties or complications with general anesthesia when compared to non-OSA patients ($p < .05$). We found a significant correlation with a higher number of cofactors (co-existing disease) amongst patients diagnosed with OSA ($p < 0.012$) as compared to non-OSA patients. An increased

incidence of higher ASA classification ($p < 0.017$); extended PACU stay time ($p < 0.05$); and unplanned admissions ($p < .007$) was also found in OSA patients when compared to non-OSA patients.

In addition patients with OSA had a higher number of prescribed medications as compared to non-OSA patients. OSA patients had a 3.36 greater chance of having hypertension as an additional cofactor ($p < .05$)

Column1	Column2	Column3	Column4
Correlation	OSA DX YES	OSA DX NO	SIGNIFICANCE
PRESTOP DATA	7.50%	92.50%	NS
POST STOP DATA	37.60%	62.40%	$P < 0.001$
COFACTOR DATA	32.60%	67.40%	$P < 0.001$

Table 1. Pre-Post STOP-BANG & DX OSA.

COFACTOR	2.07	1.53	1.91	41	$P < 0.05$
MEDICATIONS	1.92	1.13	2.64	40	$P < 0.01$
TIME	283.76	235.75	1.76	40	$P < 0.05$

Table 2. OSA vs. Non-OSA amount medications.

Multiple logistic regression testing demonstrated increased incidence for both airway difficulties ($p = .003$) and Hypertension ($p = .054$). Multiple linear regression results indicate one cofactor HTN as a significant predictor for PACU stay time. ($R^2 = .179$, $R^2_{adj} = .136$, $F(1)$)

Table 3. Logistic Regression: Cofactors and effects on OSA

Variables in the Equation							95% C.I. for EXP(B)	
	B	S.E.	Wald	df	Sig.	Exp(B)	Lower	Upper
Step 1 ^a AirWayProblems	1.884	.638	8.714	1	.003	6.582	1.884	23.001
HTN	1.017	.528	3.709	1	.054	2.766	.982	7.787
CAD	-.132	.536	.060	1	.806	.877	.307	2.505
Diabetes	-.045	.618	.005	1	.942	.956	.284	3.212
GERD	.201	.469	.183	1	.669	1.222	.488	3.064
Constant	-4.537	1.634	7.708	1	.005	.011		

a. Variable(s) entered on step 1: AirWayProblems, HTN, CAD, Diabetes, GERD.

Table 4. Incidence of cofactors with OSA.

Coefficients^a

		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
Model		B	Std. Error	Beta		
1	(Constant)	475.705	140.381		3.389	.001
	HTN	-202.568	48.910	-.453	-4.142	.000
	CAD	56.731	47.692	.125	1.190	.237
	Diabetes	63.708	56.032	.115	1.137	.258
	GERD	-15.944	42.381	-.036	-.376	.708
	Air Way Problems	-27.213	55.520	-.047	-.490	.625

a. Dependent Variable: Total PACU Time

Discussion

In Phase I of our study, utilizing the STOP-BANG measurement tool demonstrated high predictability in defining OSA in patients with both unknown and suspected OSA condition. Complications associated with OSA can be severe and life threatening. These complications include: hypoventilation, hypoxia, airway obstruction, intubation difficulties, and a higher incidence of myocardial infarction. These complications can occur intra-operatively, post operatively and during the PACU stay period. There currently exists wide diversity in the postoperative management of the OSA patient. There is a consensus that the OSA population requires more stringent anesthesia management during induction but even more critical is the post operative period while the OSA patient is still under the effects of residual anesthetic agents and narcotics. Practice guidelines vary widely within medical facilities in the anesthesia management of OSA patients postoperatively. The average PACU stay time for the non -OSA patient is under 1 hour. The duration of time that the OSA patient should be observed in the PACU is not well defined due to the physiologic changes that occur in the OSA patient airway and respiratory mechanics while under anesthesia and sedation.

The importance of our OSA studies are their validation of instruments that can provide valuable preoperative information that would aid identification of surgical patients with both known; and more importantly unknown OSA. Phase II study goes further identifying the incidence and significance of co-existing factors (cofactors) that influence the PACU recovery time of OSA patients as compared with non-OSA patients. Valuable information determined from our studies can provide guidelines and protocols streamlining and specifying the intraoperative management for the OSA patient in a manner that minimizes complications. Significant cofactors can be identified and

appropriate optimization measures can be applied. Postoperative measures and protocols could be developed to reduce the risk factors and provide for safe analgesia that inhibits heavy sedation while promoting optimum oxygenation to eliminate adverse events and extended recovery times.

Limitations

The obvious limitation in our study was our sample size of 110 subjects. The data for our retrospective study was facilitated by the use of an anesthesia information management system (AIMS) that was incorporated 18 months prior to this study. Although utilization of the AIMS enabled a concise and consistent source of all data elements for our study, the number of electronic anesthetic records was not as robust as we would have liked. Further studies involving a greater number of records that duplicate our results would not only validate our study but may also demonstrate additional cofactors influencing OSA. Additionally, we did not incorporate physiologic monitoring such as capnography or cerebral oxygen saturation. Physiologic measurements such as carbon dioxide (Pco₂) and cerebral oxygenation could be incorporated to further investigate the synergistic relationship of hypoxia and hypopnea in the OSA patient with hypertension, coronary artery disease and diabetes.

Implications

By better understanding factors that increase the adverse events of OSA, we can better prepare for or even prevent bad outcomes. Identifying characteristics inherently consistent in patients with OSA will enable anesthesia to alleviate risks, optimize outcomes by implementing supportive measures and anesthesia techniques that will

decrease the occurrence of hypoxemia, apnea/hypopnea, hypertensive and cardiac events. Preoperative assessment of all surgical patients using validated instruments would identify surgical patients with both known OSA and more importantly identify those that are unknown. Cofactors can be identified and appropriate optimization measures can be applied.

Future Research

Once all mitigating factors are better understood anesthesia providers can direct efforts for increased study. Additional studies related to the physiological and neurological components that affect hypopnea; hypoxia and vaso-activity could provide further insight into the obstructive processes that cause the complications in the OSA patient. Optimum anesthetic measures and techniques could be investigated and incorporated throughout the continuum of perioperative care. Protocols could be developed as guidelines to provide safer and consistent quality care. Intraoperative management could be streamlined and specified for the OSA patient in a manner that minimizes complications. Postoperative measures and protocols could be developed to improve the care for the OSA patient; eliminating the risk factors and provide for safe analgesia that inhibits heavy sedation while promoting optimum oxygenation and pressure if need be to support airways and eliminate adverse events. Patient can be advised regarding self-care measures that can enhance their quality of life. Most important for the anesthesia provider is the further identification of all contributing and co-existing factors.

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Manuscript Three: AANA Grant

Obstructive Sleep Apnea and the Effects of Co-Existing Factors on PACU Recovery Time

Applying for (indicate application is for which grant):

☒ General Grants ☐ Veterans Affairs ☐ Office Base ☐ Wellness

Title of Research: Obstructive Sleep Apnea And The Effects Of Co-Existing Factors On PACU Stay Time

Principle Investigator: **Shari F. Jones, PhD, CRNA, CDR, USN**

Credentials: PhD, MSN, CRNA

Co-Investigators: Joseph Burkard, DNSc, CRNA, Associate Professor,
Hahn School of Nursing, University of San Diego, San Diego, CA

Primary Affiliation: Hahn School of Nursing, University Of San Diego, San Diego, CA

Research Category

Please select only one of the following research areas that best describes your proposal:

☐ Education ☐ Leadership ☒ Practice ☐ Healthcare policy ☐ Science of anesthesia

Have you already completed the research for which you are seeking funds?

☒ Yes

☐ No

AANA # : 70547

Address: 3424 Park West Lane

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1. Submission of a progress report to the AANA Foundation every six months. Failure to submit a progress report will result in a request for the funding to be returned.
2. Upon completion of this research project, an abstract will be submitted for poster or oral presentation at an AANA meeting, with manuscript submission for publication in the AANA Journal.
3. AANA Foundation support for the research will be acknowledged in each presentation and publication. The following statement must be included:

“This research has been supported fully or in-part by the AANA Foundation.”

_____ Shari F. Jones _PhD, CRNA_____

_____ Shari F. Jones_____

Signature of Investigator/Applicant

Typed Name of Investigator/Applicant

Introduction

Obstructive Sleep Apnea (OSA) is the most common disturbance during sleep affecting 2-26% of the general population (Chung et. al, 2008). The occurrence of moderate to severe OSA is estimated to be 11.4% in men and 4.7% in women (Seet et. al, 2010) OSA is a prevalent problem in Western Society estimated to occur between 1 to 9% in patients presenting for surgery, yet it is frequently undiagnosed due to lack of awareness or knowledge of the disorder (Kaw et al, 2006).

OSA is caused by repetitive partial or complete obstruction of the airway during sleep. Diagnosis of OSA is determined by the occurrence of airway obstruction during sleep with apnea periods lasting greater than 10 seconds for five or more times per hour; with a resultant decrease in arterial oxygen saturation (SaO₂). (Seet et al, 2010) These events are known as apnea/hypopnea episodes and the measurement of episodes is referred to as the Apnea-Hypopnea Index [AHI]. The degree of severity of the disorder is determinate of the number of AHI per hour. AHI: 5-15 mild, ≥ 15 -30 moderate and > 30 severe.

A Polysomnography otherwise known as a Sleep Study is recognized as the “Gold Standard” for the diagnosis of OSA. Polysomnography data defines the degree of severity of the condition and number of apneic episodes. Unfortunately polysomnography is time consuming; patients are reluctant to participate in them; they require an extended stay in the sleep lab; and they are expensive. Medical professionals have become more aware of OSA as a significant disorder with resulting adverse effects for their patients. More

physicians are consulting to sleep labs to ascertain severity and the availability of appointments have become more limited. Unfortunately for the OSA patient, polysomnography cannot be obtained rapidly and in the case of surgical interventions that may be required for urgent or emergent disorders OSA may go undetected. (Chung, 2008) Subsequently, the OSA patient population presents a unique challenge during the peri-operative period due to an increased risk for lack of identification and subsequent life threatening complications. Of particular interest to the Nurse Anesthetist, are the respiratory complications that can result in delayed emergence from anesthesia, delayed postoperative recovery, increased apneic episodes, hypoxemia, cardiac disturbances and ultimate death in this patient population.

Literature Review

Obesity, gender, snoring and age have demonstrated to be strong indicators of occurrence for OSA. Hypertension, cardiovascular disease, diabetes and stroke are prevalently reported as confounding and coexisting factors (Dykes et al, 1996). In 2003 Netzer et al, surveyed OSA patients in 26 US primary care clinics and 14 European primary care clinics comparison of the results demonstrated similar symptoms of age, gender distribution and hypertension. Obesity was common among both sets of OSA patients although the incidence was much higher in the United States than in Europe. Another study obtained in Spain by Duran et al, 2001 reported linkages between OSA and hypertension after interviewing over 2,000 patients.

Elevated blood pressure and refractory bradycardia followed by abrupt tachycardia are cited as predominate cardiovascular effects of OSA. The hemodynamic instability seems to be related initially to hypoxemia from the airway obstruction, and then sudden

arousal as the patient awakens to breath. The rapid fluctuation of vaso-activity is thought to be responsible for the incidence of cardiac arrhythmias, the development of chronic arterial hypertension, the occurrence of myocardial infarction and sudden cardiac death. (Vanninen et al, 1996).

The severity and incidence of documented post surgical complications from patients with moderate to severe OSA is significant. Yet, most of the informational data of incidence, occurrence, surgical complications and treatment of the OSA patients come from case reports and retrospective reviews not evidenced based scientific studies. A retrospective review in 2008 involved a search of surgical procedures involving patient who were previously or subsequently diagnosed with OSA. A total of 8,897 articles were discovered from 1966 to 2007. Limiting of the search to English language, adult population as well as preferentially selected Level I and II categorized studies: 198 articles were included for analysis whereby the authors clarified further that 18 articles were level III and IV (lack of adequate blinding, control group or randomization) due to lack of higher level studies. (Chung et al, 2008)

The American Society of Anesthesiologist (ASA) developed guidelines in 2006, based on Level IV evidence and expert consensus reports to address the identification and treatment needs of the OSA patient. The ASA consultants reached agreement on treatment criteria for most simple outpatient and minor orthopedic surgical procedures but could not find consensus regarding more invasive surgeries. The guidelines propose that for more invasive surgery certain factors need to be considered when determining postoperative risk for the OSA patient these included: status of sleep apnea, (treated or untreated), level of comorbidities (hypertension, cardiovascular disorders, diabetes are they appropriately managed), type of surgery, and anesthesia modality. (Chung, 2008)

The key experts in the diagnosis and treatment of the OSA patient even acknowledge poor quality of evidence in a citation from the American Academy of Sleep Medicine stating that the scientific literature regarding perioperative risk and best management techniques for OSA patients was scanty and of limited quantity. (Meoli et al, 2003)

Purpose and Specific Aims

The purpose of this study is to identify the preexisting factors that significantly affect postoperative recovery times between patients with Obstructive Sleep Apnea compared to patients without Obstructive Sleep Apnea undergoing abdominal surgical procedures. Specific aims to determine the incidence and occurrence of pre existing cofactors: hypertension, coronary artery disease, and diabetes will be evaluated. Secondary aims are to determine if relationships of occurrence exist between the co-existing factors. If relationships of co-existing factors are determined can they be further categorized as positive or negative relationship?

Methods

An experimental correlational design method will be utilized for determining the relationship of postoperative ambulatory care unit (PACU) stay times and hypertension (HTN), diabetes (DM-II), coronary artery disease, and body mass index (BMI) in obstructive sleep apnea (OSA) patients.

Obstructive Sleep Apnea will be defined utilizing the STOP-BANG screening parametric, a concise and simple to use screening tool specifically developed to assess OSA criteria. Stop-Bang is a 12-question tool that has been implemented and validated for use in defining OSA criteria in a surgical patient population. (Chung, 2008)

A purposive sample of active duty, retired, or family members of military service members having consented to elective abdominal surgery at the Naval Medical Center San Diego (NMCSD) and received a score of 5 or greater from preoperative evaluation using the STOP-BANG screening measurement. Inclusion Criteria consists of males and females 18 to 65 years, who have elected to have general anesthesia for abdominal surgery, category ASA I & II. Exclusion Criteria will consist of members above who have pre-existing pulmonary disorders (COPD, etc.), neurological dysfunction, delayed or decreased cognitive function, recent MI. Demographic data will include age, gender, BMI, medications and type of surgical procedure.

The sample size for this study will be based on the consensus of data establishing a 26% occurrence rate. Utilizing Nunnally and Bernstein (1994) and Katz (2006) guidelines of ten subjects per predictor, 50 subjects with OSA would be a minimum. Considering the incidence of OSA in the surgical population of 26% the estimated sample size for this study is ---- subjects.

Data Collection Protocol

1. Patients scheduled for elective surgery are directed to the preoperative clinic to complete a preoperative questionnaire. This questionnaire identifies patients having any pre-existing disease factors such as hypertension, diabetes, suspected or diagnosed obstructive sleep apnea. These patients are flagged and required to have a more in depth assessment by an anesthesia provider prior to their surgery, this could occur anytime between 1 and 14 days prior to their elected procedure.
2. After informed consent is obtained demographic data will be obtained and Stop Bang Measurement questionnaire collected and a copy maintained in the patient record for the

anesthesia providers review on the day of surgery. CPAP use evaluated and instructions delivered.

3. Data reviewed within the patients' medical record will consist of:

a. Pre-operative Data: The anesthesia pre-operative assessment: (which includes: medications, NPO status, surgical procedure, review of prior surgeries, review of systems, allergies, pain assessment, tobacco and alcohol use, lab data: the anesthesia intraoperative record (innovian computerized electronic record), Essentris PACU electronic medical report.

b. Intra-Operative Data: Information obtained from electronic anesthesia record (Innovian) includes all intraoperative anesthetic activity anesthetic agent, pain medications, muscle relaxants and use or non-use of relaxant reversal medications, anti-emetics and patients vital signs throughout the procedure.

c. Post Operative Data: Aldrete scores, recovery times, respiratory rates, incidence of desaturations (O2 Sat < 90), pulse oximetry measurements, End tidal CO2, Cerebral CO2, CPAP use, hypertension documentation and treatment. (Benumof, 2004)

Data analysis plan and procedures.

V. Timeline: (Illustrate a timeline – including the estimated protocol completion and abstract/manuscript submission dates)

First Article – Manuscript: “Stop Bang Tool use to detect Obstructive Sleep Apnea for the anesthesia provider.” Submission July 2012

Second Article – Manuscript “Obstructive Sleep Apnea: and the effects of cofactors on PACU Stay Time. Bound and submitted September 2012

Third Article - AANA Grant Submission due OCT 1st, submission complete by August 1st 2012.

VI. Budget [List and justify individual items. Be as specific as possible. Equipment such as computers will not be funded. The AANA Foundation funds up to 15% indirect costs.]

1. Equipment: Cerebral CO₂ monitor equipment: 10 Thousand Dollars

Relevance to Nurse Anesthesia

The ultimate goal for all Nurse Anesthetists is to provide the safest, highest quality anesthetic management and care to their patients. If Nurse Anesthetists can identify and understand the coexisting factors creating the most risks for perioperative complications in the OSA defined patient they can better prepare themselves to manage those risks and provide optimum care. They can select anesthetic practices that minimize risk and maximize safer outcomes.

Development of more concise and accurate techniques to eliminate the factors producing increased risk for this population could be incorporated. Valid criteria for preoperative identification of the OSA patient could be developed and preoperative management techniques could be identified. Established guidelines and protocols could be created to eliminate most if not all risk factors. The complete elimination of incidence and occurrence of morbidity and mortality for the OSA patient in the perioperative environment would be the ultimate goal

Implications to Research, Education, Practice, Policy

By better understanding factors that increase the adverse events of OSA, we can better prepare or even prevent bad outcomes. Identifying characteristics inherently consistent in patients with OSA will enable anesthesia to alleviate risks, optimize outcomes by implementing supportive measures and anesthesia techniques that will decrease the occurrence of hypoxemia, apnea/hypopnea, hypertensive and cardiac events. Once all mitigating factors are better understood anesthesia providers can direct efforts for increased study. Optimum anesthetic measures and techniques could be investigated and incorporated throughout the continuum of perioperative care. Protocols could be developed as guidelines to provide safer and consistent quality care. Patient can be advised regarding self-care measures that can enhance their quality of life. Most important for the anesthesia provider is the further identification of contributing and co-existing factors. The development of accurate and efficient preoperative identification measure that could prevent adverse events related to OSA could reduce or eliminate life-threatening outcomes.

TABLES 1.

Table 1. Pre-Post STOP-BANG & DX OSA

Column	Column2	Column3	Column4
Correlation	OSA DX YES	OSA DX NO	SIGNIFICANCE
PRESTOP DATA	7.50%	92.50%	NS
POST STOP DATA	37.60%	62.40%	P< 0.001
COFACTOR DATA	32.60%	67.40%	P< 0.001

Table 2. OSA vs. Non-OSA & Amount Medications.

COFACTOR	2.07	1.53	1.91	41	P< 0.05
MEDICATIONS	1.92	1.13	2.64	40	P< 0.01
TIME	283.76	235.75	1.76	40	P< 0.05

TABLES 2.

Table 3. Logistic Regression: Cofactors and Effects on OSA

Variables in the Equation							95% C.I. for EXP(B)	
	B	S.E.	Wald	df	Sig.	Exp(B)	Lower	Upper
Step 1 ^a AirWayProblems	1.884	.638	8.714	1	.003	6.582	1.884	23.001
HTN	1.017	.528	3.709	1	.054	2.766	.982	7.787
CAD	-.132	.536	.060	1	.806	.877	.307	2.505
Diabetes	-.045	.618	.005	1	.942	.956	.284	3.212
GERD	.201	.469	.183	1	.669	1.222	.488	3.064
Constant	-4.537	1.634	7.708	1	.005	.011		

a. Variable(s) entered on step 1: AirWayProblems, HTN, CAD, Diabetes, GERD.

Table 4. Linear Regression: Cofactors effect PACU Time.

Model	Unstandardized Coefficients		Standardized Coefficients	t	Sig.
	B	Std. Error	Beta		
1 (Constant)	475.705	140.381		3.389	.001
HTN	-202.568	48.910	-.453	-4.142	.000
CAD	56.731	47.692	.125	1.190	.237
Diabetes	63.708	56.032	.115	1.137	.258
GERD	-15.944	42.381	-.036	-.376	.708
Air Way Problems	-27.213	55.520	-.047	-.490	.625

a. Dependent Variable: Total PACU Time

LIST OF DIAGRAMS

FIGURE 1. STARLING RESISTOR MODEL

FIGURE 2. OSA SLEEP CYCLE