Summary

Obstructive sleep apnea (OSA) is an increasingly common sleep disorder, which is of particular concern to anesthesiologists as it is associated with increased perioperative morbidity and mortality. The prevalence of OSA is estimated to be 5-25%; However with aging and obesity, it is expected to increase significantly. Most importantly, OSA is undiagnosed in an estimated 70-80 % of patients. Preoperative identification of OSA should alter anesthesiologist to the potential concern included difficulty in securing and maintaining the upper airway as well as postoperative airway obstruction and respiratory depression.

During rapid eye movement (REM) sleep, there is loss of upper airway muscle tone, which can lead to increased pharyngeal resistance, generate negative pharyngeal pressure during inspiration and cause upper airway collapse. The factors that contribute to upper airway narrowing and subsequent collapse during sleep including; obesity, large neck circumference, upper airway abnormalities (e.g. anatomical or craniofacial abnormalities affecting the airway), and age.

Hypoxia and hypercarbia resulting from obstructive apnea lead to arousal from sleep followed by restoration of muscle tone and airflow. Resumption of airflow is usually followed by hyperventilation, which may cause hypocapnea and loss of respiratory drive, and further predispose to apnea. Frequent arousal results in sleep disruption and excessive daytime somnolence. In addition, oxygen desaturation, sympathetic hyper activity, and systemic inflammatory response may contribute to cardiovascular co-

morbidities including systemic hypertension, cardiac arrhythmias, myocardial ischemia, pulmonary hypertension, and heart failure.

The growing OSA managemnt problems is almost certainly fueled by the growin obesity epidemic; the large majority of these patients (70-90 %) are obese (BMI >35 kg/m2). the number of people with clinically significant OSA in 1993 was approximately 18 million and is certainly much higher in 2009.

OSA and anesthesia creats a multidimintional problem: The litrature indicates that at the present time desastrous respiratory out comes during the perioperative management of patients with OSA are major and increasing problems for the anesthesia community.

The disastrous outcomes are due to: - 1) faluire to secure the airway during the induction of anesthesia, (the specific data for OSA patients is not known but its likely the data similar to, or worse than, for all comers) 2) respiratory obstruction sooner after extubation and 3) respiratory arrest after the adminstration of opioides and/or sedation to postoperative extubated patients (personal observation).

Sedatives-hypnotics, opioids, and muscle relaxants impair neural input to the upper airway muscle and therefore may worsen or even induce upper airway obstruction and apnea. In addition, these drugs also decrease the ventilatory response to hypoxemia and hypercarbia further exaggerating OSA. In contrast to natural sleep, in which OSA patients arouse due to

asphyxia, drug-induced airway obstruction and apnea lack the ability to arouse and respond adequately to asphyxia. This may have life-thereating consequences.

Postoperative sleep disturbances appear to be related to the location and invasiveness of the surgical procedure. Fewer sleep disturbances occur after mild-to-moderately invasive surgery, commonly performed on an outpatient basis than with major inpatient surgical procedures.

The severity of OSA may be determined from a sleep study using the apnea-hypopnea index (AHI), which measures the frequency of apnea (cessation of breathing for >=10 seconds despite continuing ventilation efforts) and hypopnea (more than 50% diminished airflow for>= 10 seconds) events per hour, is used to characterize the severity of OSA. An AHI of 6-20 indicates mild OSA, AHI 21-40 indicates moderate OSA, AHI more than 40 indicates severe OSA.

Sleep laboratories differs in their criteria for defining severity of OSA. If asleep study is not available, patients should be treated as though they have moderate OSA if they demonstrate two or more symptoms and signs in two or more of the categories described in **(Table :1)**, and patients should be treated as though they have severe OSA if one or more of the symptoms or signs is severely abnormal. Importantly, when in doubt, it is prudent to treat the patients as though they have severe OSA.

Table 1: Identification and Assessment of OSA in Adults (Modified From ASA Practical Guidelines)

History of apparent airway obstruction during sleep • Frequent /Loud snoring (heard through closed doors) • Witnessed apnea • Frequent arousals from sleep • Awakens from sleep choking or grasping
Somnolence (one or more of the following present) • Excessive somnolence or fatigue despite adequate "sleep" • Falls asleep easily in non stimulating environment (e.g., watching TV) despite adequate "sleep"
Clinical signs suggesting OSA • Obesity (BMI > 35 kg/m2) • Increased neck circumference (> 17 inches in males and > 16 inches in females) • Craniofacial abnormalities affecting the airway (micrognathia, retrognathia) • Nasal obstruction (mouth breathing) • Large tonsils nearly touching or touching in the midline