# Obesity, Sleep Apnea, the Airway and Anesthesia

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## I. INTRODUCTION

Recent articles in the Anesthesia Patient Safety Foundation Newsletter indicate that at the present time disastrous respiratory outcomes during the perioperative management of patients with OSA are a major problem for the anesthesia community (2002; 17:24 and 2002; 17:39). A recent review of the literature indicates that the disastrous outcomes are due to either intubation failure, or respiratory obstruction soon after extubation, or respiratory arrest after narcotic and sedative medication (J Clin Anes 2001; 13:144-156).

The number of adult obese patients with obstructive sleep apnea (OSA) is very large. It has been estimated that 24% of men and 9% of women, if formally studied during sleep, would have a diagnosis of OSA, and 4% of men and 2% of women would have clinically significant symptomatic OSA **(1,2)**. Obesity is a very important independent causative/risk factor; 60 to 90% of persons with OSA are obese (defined as a body mass index [BMI] > 29 kg/m$^2$) **(3,4)** with all indices of obesity, including BMI, waist, hip and neck circumferences, and skin fold thickness strongly and directly related to the severity of OSA **(1,2,5)**. In nonobese patients and many pediatric patients (the minority of patients with OSA), causative risk factors are craniofacial and orofacial bony abnormalities, nasal obstruction and large tonsils and are not considered here. Nevertheless, excess neck fat may be present in nonobese snorers **(6)** and superimposition of obesity on any pre-existing bony abnormality may increase the severity of OSA.

Unfortunately, at present, 80% **(7)** to 95% **(8)** of persons with OSA are undiagnosed; thus, the vast majority of patients with OSA who require anesthesia and surgery now have neither a presumptive clinical and/or a sleep study diagnosis of OSA **(9)** and the anesthesiologist may be the last physician who has a chance to make a diagnosis of OSA and thereby be the gate keeper of the well being of a perioperative OSA patient.

## II. DEFINITION OF OSA TERMS

Obstructive sleep apnea (OSA) is defined as cessation of airflow for more than 10 seconds, despite continuing ventilatory effort, 5 or more times per hour of sleep, and is usually associated with a decrease in arterial oxygen saturation (SaO2) of more than 4% (***TABLE 1***) **(10)**. Obstructive sleep hypopnea (OSH) is defined as a decrease in airflow of more than 50% for more than 10 seconds, 15 or more times per hour of sleep, and is usually associated with snoring and may be associated with a decrease in SaO2 of greater than 4% (Table 1). Both OSA and OSH repeatedly disrupt sleep due to increased ventilatory effort-induced arousal which, in turn, causes daytime sleepiness and altered cardiopulmonary and cortical function (***TABLE 1***) **(10)**.

**Table 1. Definition of OSA and Obstructive Sleep Hypopnea (OSH).**

| Obstruction | ↓ in airflow >10 seconds | Times /hour | ↓ in $O_2$ saturation | Disrupted sleep | Daytime sleepiness |
|---|---|---|---|---|---|
| OSA | 100% | >5 | ≥4% | Yes | Yes |
| OSH | >50% | >15 | ≥4% | Yes | Yes |

## III. PATHOPHYSIOLOGY OF OSA IN THE ADULT OBESE PATIENT

### A. NORMAL PHARYNGEAL MUSCLE ACTIVITY

The contraction of the diaphragm against the high resistance offered by the nose during inspiration creates a subatmospheric intra-airway pressure which may narrow the collapsible segments in the pharynx. There are three pharyngeal segments; the retropalatal pharynx (velo- or nasopharynx, posterior to the soft palate), the retroglossal pharynx (oropharynx, posterior to the tongue from the tip of the uvula to the tip of the epiglottis) and the retroepiglottic pharynx (laryngo- or hypopharynx, posterior to the epiglottis) (***FIGURE 1***). These pharyngeal segments are collapsible because the anterior and lateral walls lack bony support. The human is the only mammal to have an oropharynx (in all other mammals the tip of the uvula touches the tip of the epiglottis) which enables singing and speech (the oropharynx between the uvula and epiglottis creates a chamber for resonance) but also causes OSA (the oropharynx is collapsible)**(11)**. The inspiratory patency of the retropalatal, retroglossal and retroepiglottic pharynx is caused by contraction of the tensor palatini, the genioglossus, and the hyoid bone muscles, respectively (***FIGURE 1***).

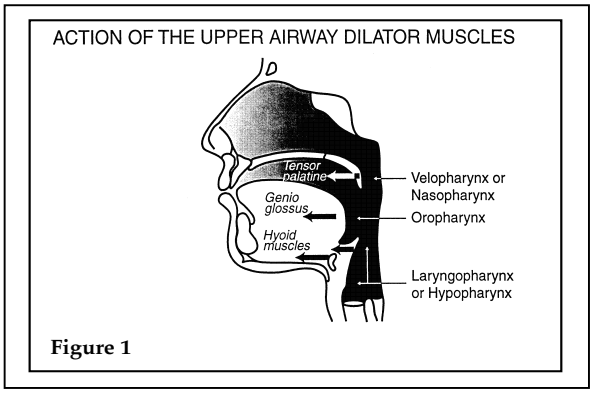



**Figure 1**

### B. NORMAL SLEEP

The relation between the anatomy and muscle function in the upper airway becomes critical during sleep. In adults, a typical night of sleep consists of 4 to 6 cycles of non-rapid eye movement (NREM) sleep followed by rapid eye movement (REM) sleep. There are four stages of NREM and one stage of REM sleep which represent progressively deeper sleep with progressive slowing of the electroencephalogram (EEG) waves. Deep NREM (Stage 3 and 4) and all of REM stages are called slow wave or

deep sleep, are restorative periods of sleep, and are characterized by a generalized loss of muscle tone. The loss of muscle tone results in pharyngeal collapse. The most important site of collapse is the compliant lateral pharyngeal walls, which is the major pharyngeal site of adipose tissue deposition in the obese patient (see below).

During NREM sleep, the rhythmic activity of the upper airway muscles decreases, upper airway resistance (UAR) increases significantly and can be twice that during the awake state **(12,13)**. In REM sleep, the activity of the upper airway muscles can disappear completely and UAR increases even further. As UAR increases, the pharyngeal subatmospheric pressure generated by a given diaphragmatic contraction increases **(14)**. As pharyngeal pressure becomes more negative, pharyngeal collapse increases. In approximately 50% to 75% of OSA cases, the obstruction occurs in at least two of the pharyngeal segments with the retropalatal nasopharynx plus one of the other pharynxes being the most common combination (**Principles and Practice of Sleep Med., Saunders, 1994, P. 706-721).** With respect to the oropharynx and laryngopharynx, magnetic resonance imaging, with and without nasal continuous positive airway pressure (N-CPAP) (used as an experimental mechanism to identify movement of various parts of the pharyngeal perimeter), shows that the most important site of collapse is the compliant lateral pharyngeal walls **(15)**. The fact that there are usually multiple sites of obstruction in OSA patients readily explains why the uvulopalatopharyngeoplasty operation is successful in decreasing the severity of OSA by > 50% only 40% to 50% of the time **(Sleep 1996; 19:156-177).**

**C. OBESITY AND OSA: PHARYNGEAL PATHOLOGY AND INCIDENCE**

There are three reasons why obesity *per se* may cause OSA and OSH. First, there is an inverse relationship between obesity and pharyngeal area **(16,17)**. Magnetic resonance imaging shows that the decreased pharyngeal area in obesity results from deposition of adipose tissue into pharyngeal tissues; the structures involved are the uvula, the tonsils, the tonsillar pillars, the tongue, aryepiglottic folds and, most importantly and predominantly, the lateral pharyngeal walls. Indeed, the volume of fat in the lateral pharyngeal walls correlates well with the severity of OSA **(6,18-22)**. The converse is also true; i.e., weight loss improves the pharyngeal and glottic function of OSA patients **(23)**. Increased fat deposition in the pharynx resulting in decreased patency of the pharynx increases the likelihood that relaxation of the upper airway muscles will cause collapse of the soft-walled oropharynx between the uvula and epiglottis**.**

Second, the patency of the collapsible pharynx is determined by the transmural pressure across its wall (the difference between the extraluminal and intraluminal pressure) and the compliance of the wall. If the compliance of the wall and intraluminal pressure (inspiratory airway pressure) are constant, then the remaining important determinant of upper airway patency is extraluminal pressure. In obese patients, extraluminal pressure is increased by superficially located fat masses **(24,25)**; i.e., the upper airway is compressed externally. Therefore, it is not surprising that the neck is significantly fatter in obese OSA patients compared to equally obese non-OSA patients **(26)** and that the incidence and severity of OSA correlates better with increased neck circumference than with general obesity **(27-29)**.

Third, Amer J Respir Crit Care Med 2002; 165:1239 describes 13 individual studies that used both dietary and surgical methods for weight reduction and <u>all</u> 13 studies showed that weight loss results in a significant reduction in OSA severity.

**D. AROUSAL**

Over the course of an apnea a number of important respiratory events occur. First, arterial oxygen tension (PaO2) decreases as a function of the initial PaO2, functional residual capacity (FRC), and the duration of apnea **(30,31)**. Second, arterial carbon dioxide tension (PaCO2) increases as a function of duration of apnea **(32)**. Third, ventilatory effort progressively increases as the apnea proceeds as a function of both the decreasing PaO2 and increasing PaCO2 **(33,34)**. Finally, as a function of the increased ventilatory effort, intra-airway pressure becomes progressively more negative **(35)**. Any or all of these four mechanisms could increase neural traffic in the reticular activating system and arouse the individual (expressed as increased EEG activity, vocalization, extremity twitching, turning, gasping or snorting on airway opening). Once arousal occurs, the muscles of the upper airway reactivate, and the pharyngeal airway opens. Ventilation then resumes, hypoxia and hypercapnia are corrected **(36)**, the individual returns to sleep and the cycle begins again. Obviously, the arousal response is necessary for survival. However, the physiological events (see below) that surround the arousal response, if repeated often enough, will ultimately result in serious systemic pathophysiologic consequence.

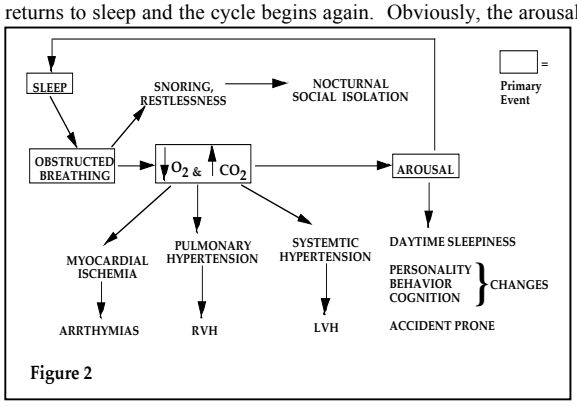


Figure 2

**E. SYSTEMIC PATHOPHYSIOLOGY OF OSA**

***<u>FIGURE 2</u>*** outlines the systemic effects of the sleep  arousal  sleep cycles that occur many times during every sleep. Decreases in PaO2 during apnea may cause bradycardia with return to baseline during and after arousal. In approximately half of patients with apneic events, long sinus pauses, second degree heart block and ventricular dysrhythmias occur **(37)**. When SaO2 decreases below 60% the severity of the bradycardia and the onset of ventricular ectopy increases markedly **(38)**. The high incidence of arrthymias in OSA patients may explain the higher incidence of nocturnal angina and myocardial infarction in these patients **(3)**. Proper treatment of OSA decreases the incidence of these arrthymias **(39)** and presumably decreases the incidence of myocardial ischemia.

Diurnal pulmonary and systemic hypertension (Ppa and Psa) in OSA patients is likely caused by the innumerable repetitive increases in sympathetic tone that occurs with each hypoxemic-hypercapnic arousal event **(10,40)**. Diurnal Psa hypertension is present in 50% of OSA patients **(41)** and is independent of obesity, age and sex **(4,10,39,42)**; proper treatment of OSA results in a decrease in Psa hypertension **(39)**.

Diurnal and nocturnal Ppa and Psa hypertension likely accounts for the 71% and 31% incidence of right and left ventricular hypertrophy, respectively, in OSA patients **(10,39,43)**. In view of the dual circulation hypertension, biventricular hypertrophy, increased incidence of arrthymias, myocardial infarction and stroke, it is not surprising that the cumulative 8-year mortality in patients with moderate untreated OSA is 37% compared to 4% in patients with mild OSA **(10,44-46)**.

The many brief periods of sleep fragmented by arousal diminishes restorative deep sleep. The sleep deprivation causes daytime sleepiness and fatigue, morning headaches, diaphoresis, nocturnal enuresis, decreased cognition and intellectual function, and personality and behavioral changes **(10)**. The excessive daytime sleepiness increases the risk of motor vehicle accidents for patients with OSA; the risk is reported to be six-to-seven times that of the general driving population **(47,48)** and independent of any other possible confounding factor **(47,49)**.

## IV. DIAGNOSIS OF OSA

A presumptive clinical diagnosis of OSA can be made in a patient with the classical signs and symptoms of obesity, snoring and/or apnea during sleep, periodic snorting and apparent arousal (extremity movement, turning, vocalization), and daytime sleepiness or fatigue. Increased neck circumference is associated with OSA **(27-29)**; specifically, neck circumference of OSA patients = 41.1 ± 3.5 cm vs neck circumference in patients without OSA = 38.0 ± 3.5 cm, $p<0.001$) **(50)**.

Obesity is best expressed quantitatively as body mass index (BMI):

$$BMI = mass/height^2 = kg/m^2 \text{ or } 703 X lbs/inches^2$$

where underweight, normal, overweight, obesity and morbid obesity equals < 19.0, 19.0-24.9, 25.0-29.9, 30.0-34.9, and > 35, respectively **(51)**. Ninety percent of OSA patients may have a BMI > 28 $kg/m^2$ **(3)**. While the principle limitation of use of the BMI is that it does not distinguish between fat or muscle, in the general population it is much more likely that an individual with a BMI > 30 $kg/m^2$ is obese rather muscular.

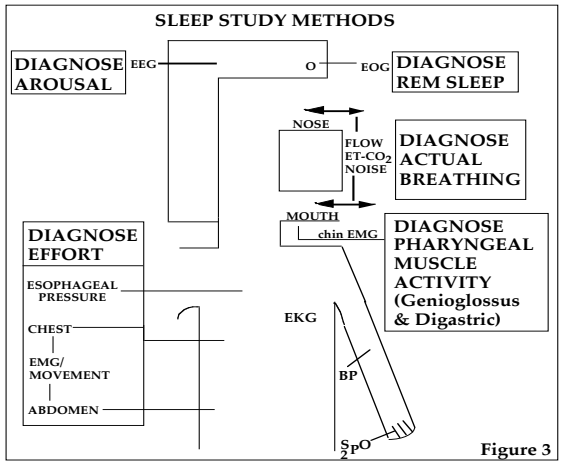


Figure 3

The definitive diagnosis of OSA and OSH, however, must be made by some form of sleep study. A full sleep study consists of monitoring the EEG (for stage of sleep and arousal), the EOG (electrooculogram, for NREM vs REM sleep), oral and nasal airflow sensors and capnography (for actual movement of air), noise (for snoring and snorting), esophageal pressure and chest and abdominal movements (for breathing effort), submental and extremity electromyography (for pharyngeal [genioglossus] muscle activity and extremity movement, respectively), oximetry (pulse, ear, transcutaneous) for SpO2 and noninvasive blood pressure and EKG for cardiovascular function (***FIGURE 3***). Rarely, direct systemic and pulmonary artery pressure monitoring is performed for more precise determination of cardiopulmonary function.

The breathing effort monitors, along with the airflow monitors, allow the diagnosis of obstructive versus central sleep apnea. OSA and OSH mean there is respiratory effort but no air flow and central sleep apnea means there is no effort and there is no air flow. The quantitative results of a sleep study are reported as events and indexes and as narration (see ***TABLE 2***) as follows: The total number of apneas and hypopneas per hour is called the apnea-hypopnea index (AHI) and is used to define the severity of OSA; values of 5-15, 16-30 and > 30 indicate mild, moderate and severe OSA, respectively. Certainly clinical management decisions are likely to be different for patients with AHI of 12 versus 52. The total number of arousals per hour is reported as a total arousal index (TAI, arousal/hour). The sum of AHI and TAI is called the respiratory disturbance index (RDI). Central sleep apneas (no flow, no effort) are usually reported separately. In morbidly obese patients who required treatment for OSA [mean (range) BMI=48.7 (32.4-78.6) and Age=41 (28-63)], the percent of central apneas out of the total AHI equaled only 5.8% **(52)**.

**Table 2. Understanding The Sleep Study Report**

| *EVENTS* | *INDEXES* |
|---|---|
| **Apnea = No airflow >10 Sec** | **Events/hour; AHI, ODI, AI** |
| **Hypopnea = TV<50% for >10 Sec** | **Severity of sleep apnea is f(AHI) with** |
| **Desaturation = $SpO_2$ ↓ >4%** | **5-15 = mild; 16-30 = moderate;** |
| **Arousal = clinical or EEG** | **>30 = severe** |

**$SpO_2$ data also # of events per 60-69%; 70-79%; 80-89%; and the lowest. EKG and Hemodynamics usually descriptive and the extremes reported . TV = tidal volume; AHI = apnea hypopnea index, ODI = oxygen desaturation, AI = Arousal index**

Oxygen data are reported in several ways. Most laboratories report the number of $SpO_2$ desaturations greater than 4%, the lowest $SpO_2$, and time spent at a certain range of $SpO_2$ (e.g., 89-80%, 79-70%, etc.). The cardiovascular manifestations of $SpO_2$ desaturation are variously described but always include maximum and minimum heart rate and blood pressure during the event and the occurrence of any arrthymias or EKG changes that are consistent with myocardial ischemia. If CPAP was used for part of the sleep period, then all of the above data will be reported with and

without CPAP. In increased BMI patients, room air ABGs are very desirable in order to diagnosis the Obesity Hyperventilation Syndrome (OSH); certainly, mechanical ventilation decisions are likely to be different for patients with a resting $PaCO_2$ of 44 versus 59 mmHg.

Severity of OSA can be expressed in terms of AHI (***TABLE 2***), as well as loudness of snoring and movement during sleep (social problem), risk of arrhythmia and myocardial ischemia (decreased $SpO_2$), dual circulation hypertension and biventricular hypertrophy, and risk of death from a motor vehicle accident (daytime somnolence).

## V. EFFECT OF ANESTHETICS ON AIRWAY PATENCY IN ADULT OBESE OSA PATIENTS

All central depressant drugs diminish the action of the pharyngeal dilator muscles in adult obese OSA patients thereby promoting pharyngeal collapse around a fat laden pharynx **(53-59)**. The commonly-used anesthetic drugs that have been demonstrated to cause pharyngeal collapse are propofol **(60)**, thiopental **(61,62)**, narcotics **(56,63,64)**, benzodiazepines **(56,65-67)**, small doses of neuromuscular blockers **(68-70)**, and nitrous oxide **(71)**. The anesthetic drug-induced pharyngeal collapse around the excessive airway tissue "makes the airway lumen resemble the interior of the intestine and the path of the airway easily becomes lost among the folds" **(72)**. Furthermore, if opioids cause airway obstruction, then the opioids may also cause a poor ventilatory response to the ensuing hypoxemia and hypercapnia **(73)**.

It is important to understand that in the postoperative setting, sleep architecture is disturbed. During the first three days after surgery, pain scores are highest and deep stage 3 and 4 NREM and REM sleep are often suppressed **(74)**. High levels of pain results in increased analgesic requirements during the first three postoperative days, and from this perspective, the danger of life-threatening apnea during drug-induced sleep is increased. In the next three days, deep REM sleep rebounds **(74,75)**. During this phase of recovery the danger of life-threatening natural deep sleep-induced apnea is increased. Thus, for separate in-series reasons (increased analgesic requirement followed by increased amount of REM sleep), the risk of prolonged apnea during sleep is increased for approximately one week for the postoperative OSA patient **(76,77)**.

Given that airway obstruction is more likely when either drug- or REM sleep-induced pharyngeal collapse occurs, it is not surprising that heavy snorers (identified preoperatively) have more severe decreases in SpO2 during sleep postoperatively than normal patients **(78)**. This consideration is highlighted by numerous reports of the need for rescue airway management in postoperative OSA patients **(79-86)**.

## VI. IMPLICATIONS FOR AIRWAY MANAGEMENT

### A. PREOPERATIVE EVALUATION: OSA AND AIRWAY STATUS

Since most adult obese patients with OSA are undiagnosed, many who presently require anesthesia and surgery have neither a presumptive clinical or sleep study diagnosis of OSA. Thus, the anesthesiologist may be the diagnostic gate keeper of OSA. The essential items on history that must be present for a presumptive clinical diagnosis of OSA in the adult obese patient are history of snoring and/or apnea during sleep, frequent arousals (twitching, turning, vocalization, snorting), and daytime sleepiness. The severity of these historical items correlates with the severity of sleep study-proven OSA **(4,5,10,87,88)**. Since a firm diagnosis of OSA will likely impact on anesthetic management, it is reasonable to suggest that all adult obese patients (or those who observe them while asleep) be routinely asked about nocturnal snoring/apnea, arousals and diurnal sleepiness**(77,78,89)**. Prediction of OSA is increased if there is a history of hypertension**(50)** or a neck circumference > 40-42 cm **(27,28,50)**. Other signs and symptoms consistent with a clinical presumptive diagnosis of OSA are nocturnal diaphoresis and enuresis, frequent nocturia, morning headaches, abnormal cardiovascular and neuropsychiatric function.

If the anesthesiologist is the first care-giver to diagnose OSA, then it may be prudent to postpone the surgery and refer the patient to an appropriate physician and perhaps a formal sleep study obtained to quantify the severity of OSA. It is important that anesthesiologists petition their OR committees to recognize that a presumptive clinical diagnosis of OSA is just as legitimate a reason to delay surgery in order to get a proper workup as it is to a get a cardiology workup for newly discovered cardiovascular disease. Alternatively, if general anesthesia is required, the patient could be treated as though severe OSA existed (see C, D, E and F below). Finally, regional anesthesia is worth considering if it can be technically performed, the awake patient can tolerate the surgical position and the respiratory effects of the regional anesthetic, access to the airway is adequate, and the surgery can be quickly terminated. Regional anesthesia should largely obviate the need for sedative and narcotic drugs both intra- and immediately postoperatively.

**Table 3. Reasons why Surgery on Typical Patient with OSA Should be Done as an Inpatient**

| FACTORS | GENERAL REASON | | SPECIFIC REASON |
|---|---|---|---|
| **OSA Patient Factors** | ↑sensitivity to narcotics and sedatives | | 1) ↓ $\dot{V}_E$; 2) Relaxation of pharyngeal muscles; 3) ↓ arousal |
| | 60-90% are obese | Respiration | 1) ↑ airway resistance; 2) ↓ FRC; 3) ↑ $\dot{V}O_2$ |
| | | Cardiovascular | 1)↑ BP; 2) biventricular hypertrophy |
| **Hospital Factors: ↑ Need For Rx & Equipment** | 1) Specialized airway equipment (e.g., FOB); 2) Respiratory Rx and equipment (e.g., CPAP, ventilators); 3) Monitoring equipment (e.g., $SpO_2$, CXR, ABGs, 12-lead EKGs, Invasive Vascular; 4) Availability of skilled personnel (for consults, ACLS) | | |

### B. INPATIENT VERSUS OUTPATIENT SURGERY

The typical patient with OSA should be done as an inpatient (see ***TABLE*** 3). Once again, it is important for anesthesiologists to petition their OR committees to formally consider whether patients with OSA are appropriate candidates for outpatient surgery (***TABLE*** 3 argues outpatient surgery is inappropriate for patient with OSA). Possible outpatient surgery exceptions for OSA

patients include those who have mild disease, have local or regional anesthesia with no or minimal sedation, have a several hour to 23 hour postoperative PACU observation period and are on oral medication at the time of discharge.

**C. TRACHEAL INTUBATION**

Several lines of evidence in the literature indicate that obese OSA patients are, in general, more difficult to intubate than normal controls. First, obesity is significantly related to difficult intubation **(90-92)**. Indeed, in two series of morbidly obese patients undergoing upper abdominal surgery, the incidence of difficult of intubation under general anesthesia was 13% and 24% and the incidence of patients requiring awake intubation was 8% in both studies **(54,93)**. Second, a short thick neck is significantly related to difficult intubation **(90,94)**. Third, obesity **(1-5)** and a short thick neck **(24-29,50)** are significantly related to OSA and to each other **(90)**. Fourth, since excess pharyngeal tissue is deposited in the lateral walls of the pharynx of obese OSA patients **(6,18-22)**, the excess tissue may not be visualized during routine oropharyngeal classification. Finally, based on the above, it is not surprising that difficult intubation and OSA have been found to be significantly related **(95**, A&A 2002; 95:1098). Indeed, in one large series of patients undergoing surgery for OSA, the incidence of failed intubation was 5% **(79)**, which is approximately 100 times the incidence in the general population. Thus, given the above literature, and the fact that excess pharyngeal tissue may not be revealed by routine examination, it is reasonable that the practitioner have an increased index of suspicion regarding intubation difficulty.

Several more tracheal intubation points are especially relevant to obese OSA patients. Within the context of an increased index of suspicion of intubation difficulty, the decision as to whether to do tracheal intubation with the patient awake or under general anesthesia must be individualized on the basis of a complete preoperative airway evaluation. If difficulty with either mask ventilation or tracheal intubation is expected, then according to the ASA Difficult Airway Algorithm, intubation and extubation should be performed while the patient is awake **(96-98)**.

If tracheal intubation is to be done while the patient is awake it is essential that the patient be properly prepared. One component of proper preparation is judicious administration of sedative and analgesic medication **(54,57)**. The danger of premedication in these patients is well illustrated by descriptions of several cases of complete airway obstruction **(99-102)**. Thus, proper preparation should depend on thorough topical and nerve block anesthesia of the upper airway **(93,97)**. Use of a flexible fiberscope through a rigid oropharyngeal conduit technique of intubation permits visualization of structures in an atraumatic manner **(98,103)**.

If intubation is to be done with the patient asleep, it is important to fully preoxygenate the patient because the obese patient with a relatively small FRC (small oxygen reservoir) and high oxygen consumption (***TABLE 3***) desaturates much more rapidly during obstructive apnea compared to a normal patient **(104,105)**. Maximal total body preoxygenation (filling of the alveolar, arterial, venous and tissue spaces) requires that the patient breathe $F_IO_2$ = 1.0 for ≥ 3 minutes in a well sealed system **(106)**. ***FIGURE 4*** shows how decreasing initial $FAO_2$ (e.g., resulting from a poorly sealed preoxygenation system) results in progressively more rapid oxyghemoglobin desaturation during obstructive apnea in a BMI=40 kg/m$^2$ patient **(104,105)**. Oxygen insufflation into the pharynx via a small nasopharyngeal catheter during laryngoscopy of the obese patient may further delay the onset on arterial oxygen desaturation **(107)**. Laryngoscopy must be performed in an optimal manner, which means that the patient is in the optimal 'sniff" position before induction of general anesthesia (may require building a ramp under the patient from the scapula to the head) and optimal external laryngeal manipulation should be used on the first attempt if the view of the larynx is poor **(108,109)**. Mask ventilation must be performed optimally, which may require two anesthesia providers using two- or three-handed bilateral jaw thrust and mask seal, with oropharyngeal and/or nasopharyngeal airways in-situ, and the airway pressure relief valve and mask seal set so that CPAP (5-15 cm$H_2O$) is delivered to the pharynx. "Cannot ventilate, cannot intubate" options must be immediately available at the anesthetizing location **(96-98)**.

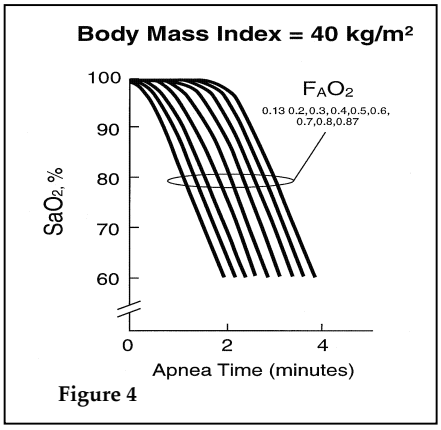


**Figure 4**

**D. EXTUBATION: AWAKE VERSUS LEAVING THE TUBE IN**

The risk of airway obstruction following extubation is increased in OSA patients **(110)**. The risk is further increased in OSA patients who have had nasal packing following nasal surgery **(111)**; therefore packing around a nasopharyngeal airway (creating a central conduit for gas exchange) should be considered **(55,77)**. In a retrospective review of 135 patients undergoing surgery for the treatment of OSA the incidence of life-threatening postextubation obstruction was 5%; those patients who obstructed were extubated in the operating room **(79)**. Aside from the threat of death from airway obstruction, another great danger of spontaneous ventilation against an obstructed airway is the rapid development of severe negative pressure pulmonary edema **(79,112)**. The treatment of negative pressure pulmonary edema in this setting usually requires re-intubation **(112)**.

Following non-nasal and/or non-oral surgery for morbidly obese OSA patients, the extubation choice is usually between awake versus leaving the patient intubated for a period of postoperative mechanical ventilation. The determinants one should consider as to whether to leave the patient intubated for a period of postoperative mechanical ventilation are the ease of the mask ventilation and tracheal intubation experience at the beginning of the case, associated cardiopulmonary disease and length and type of surgery, the BMI of the patient and the severity of the OSA. It is highly desirable/necessary for several conditions to be present at the time of extubation. First***, whenever the patient is to be extubated (either in the operating room or later in the PACU or ICU) the patient should be fully awake; this means the patient should be rational, oriented and responding to commands in a clear, crisp and unambiguous manner***. A dangerous mistake is to interpret mindless movement, such as reflex reaching for an endotracheal tube, or suddenly trying to sit up, for purposeful movement. Second, full recovery from neuromuscular blockade should

be proven by a neuromuscular blockade monitor, sustained head lift for > 5 seconds and, in the ICU, with an adequate vital capacity and peak inspiratory pressure. Third, the patient should not have a high blood level of narcotic as indicated by a respiratory rate < 12-14 breath/min while the endotracheal tube is in-situ. It is helpful for regional analgesia to be operative at the time of extubation. Fourth, extubation in the reverse Trendelenburg or semi-upright position minimizes compression of the diaphragm by abdominal contents **(113)**. Fifth, an oropharyngeal and/or a long nasopharyngeal airway (i.e., it is desirable for the distal end to be retroglossal) should be in-situ if possible and two-person mask ventilation should be ready to be applied. Sixth, if doubt exists about the ability of the patient to breathe adequately and the practitioner to re-intubate if the patient does breathe inadequately, then the tracheal tube should be removed over an airway exchange catheter or fiberscope**(114)**. Finally, **IF THE PATIENT WAS ON CPAP** (nasal or full face mask) **PREOPERATIVELY, THEN THE PATIENT SHOULD BE ON CPAP POSTOPERATIVELY (SEE E BELOW)**. The CPAP protects the patient against airway obstruction during sleep by pneumatically splinting the oropharynx. CPAP can be by applied first with oxygen and then later with air **(110,115)**. Beyond the initial immediate application of CPAP, the $F_IO_2$ should only be increased if the $SpO_2$ is significantly decreased **(80)**. Automatically adjusting CPAP devices are now available (e.g., Resmed) and make great therapeutic sense.

The determinants of awake versus asleep extubation in uvulopalatopharyngeoplasty (UPPP) and nasal surgery patients are shown in ***TABLE*** 4. If significant doubt exists as to the best extubation decision (awake versus asleep), it is usually prudent to extubate with patient fully awake ("□ awake" in ***TABLE*** 4).

**Table 4. Major Determinants of Awake Vs. Asleep (DEEP) Extubation in OSA Patients Having UPPP and/or Nasal Surgery**

| Individual Factors | Status Of Factors | Extubation Decision |
|---|---|---|
| Ease of Mask Ventilation } Experience at the Ease of Tracheal Intubation } Beginning of the Case<br>BMI } Severity<br>AHI }<br>Associated Cardiopulmonary | All factors good | Can be asleep if really indicated |
| | One factor severe | Awake |
| | In between the above extremes | Requires judgment → awake |

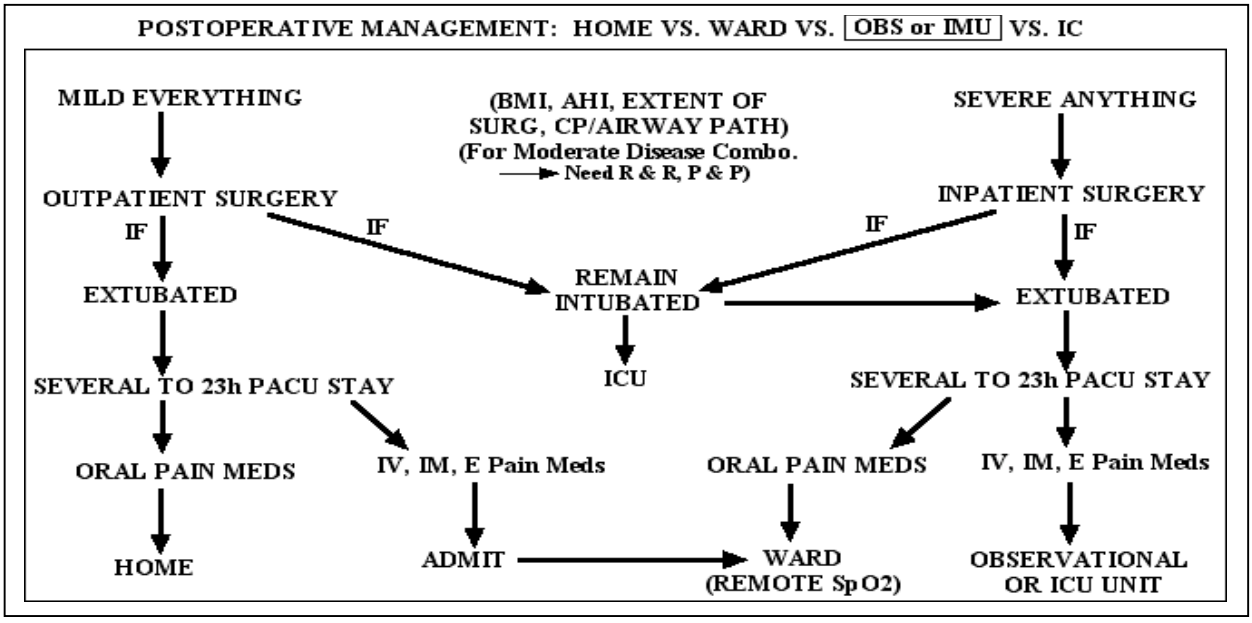
Table 5. Reasons Why Immediate Post-GA CPAP May Be Ill Advised

1. Impair access for suction, deal with N&V
2. Impair communication (speech)
3. Impair monitoring facial color and LOC
4. Fit of mask may change post-op (e.g. edema)
5. CPAP is Rx for natural sleep/not drug induced sleep; requirements may change greatly

**E. TIMING OF CPAP (N-CPAP OR FULL FACE CPAP)**

CPAP may be applied in the PACU, but ***TABLE*** 5 indicates why it should not be immediately upon, or within the first few minutes of arrival in the PACU. **WHEN ACCESS, COMMUNICATION, MONITORING ISSUES ABATE, CPAP SHOULD BE APPLIED AT ALL REST TIMES IN THE EARLY POST-GA PERIOD (TIMING OF SLEEP IS UNPREDICTABLE).**

**F. POSTOPERATIVE MANAGEMENT: WHERE? (See Figure 5 Immediately Below)**



The most straightforward of decisions to make regarding location of postoperative management involves those patients at the extremes of pathology (see "Mild Everything" and "Severe Anything" in Figure 5). The most difficult decisions regarding postoperative patient location involve patients with moderate plus or severe minus disease; it is these patients that the ASA should promulgate guidelines and individual institutional OR Committees should promulgate rules and regulations/policies and procedures **now** for postoperative management location. For all extubated patients with OSA, a several to 23 hour PACU stay is the logical management choice now.

## VII. SUMMARY

Adult obese patients with suspected or sleep test confirmed OSA present a formidable challenge throughout the perioperative period. Life-threatening problems can arise with respect to tracheal intubation, tracheal extubation and providing satisfactory and safe postoperative analgesia. Tracheal intubation and extubation decisions in obese patients with either a presumptive and/or sleep study diagnosis of OSA must be made within the context that there may be excess pharyngeal tissue that cannot be visualized by routine examination and the literature indicates an increased risk of intubation difficulty. Regional anesthesia for postoperative pain control is desirable (although such management is not necessary or possible for many of these patients). If opioids are used for the extubated postoperative patient, then one must keep in mind an increased risk of pharyngeal collapse and consider the need for continuous visual and electronic monitoring. The exact management of each sleep apnea patient with regard to intubation, extubation, and pain control requires judgement and is a function of many anesthesia, medical and surgical considerations. **Nevertheless, because of an apparent current high rate of major negative outcomes, it is my opinion that it is imperative for anesthesiologists to petition their operating committees NOW to recognize a presumptive clinical diagnosis of OSA as a legitimate reason to delay surgery to get a proper workup, to define appropriate outpatient versus inpatient boundaries for OSA patients, and to create appropriate monitored care environments for OSA patients.**

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