ANESTHETIC CARE OF THE PATIENT WITH OBSTRUCTIVE SLEEP APNEA

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Introduction

Obstructive sleep apnea (OSA) is an insidious, progressive disease[1] that is significantly under diagnosed in the general population. It carries increased risk of difficult intubation preoperatively[2] and increased risk of postoperative respiratory depression and airway collapse leading to hypoxia and possibly asphyxia[3]. In light of the estimated prevalence of symptomatic OSA in 5%[4] of the general populace, and the fact that 80% of these patients remain undiagnosed[5], it is crucial for anesthesia personnel to screen every patient undergoing anesthesia for this disorder quickly and effectively, and likewise, to have a strategy for perioperative care.

Definitions

Several commonly used terms include:

1. **Obstructive Apnea:** an absence of airflow during sleep for greater than 10 seconds[6,7].
2. **Obstructive Hypopnea:** The latest manual of the American Academy of Sleep Medicine (AASM) provides two definitions of hypopnea. The recommended definition is a drop in airflow ≥30% from baseline lasting for at least 10 seconds and associated with a ≥4% oxygen desaturation.
3. **Obstructive Sleep Apnea:** recurrent apneic and/or hypopneic episodes despite continuing ventilatory effort, usually associated with a decrease of ≥4% in oxygen saturation[6,7].
4. **Obstructive Sleep Apnea Hypopnea Syndrome** (OSAHS): symptomatic OSA. The main symptom is daytime sleepiness, but symptoms can manifest as choking or gasping during sleep, recurrent awakening during sleep, unrefreshing sleep, and impaired concentration[6,7].
5. **Apnea-Hypopnea Index** (AHI): a tool used to diagnose and measure the severity of OSA by measuring apneic-hypopneic events *per hour* during sleep. An AHI greater than 5 but less than 15 is the criteria for mild OSA. Moderate OSA is defined by an AHI greater than 15 but less than 30, and severe OSA is an AHI greater than 30[6]. Respiratory Disturbance Index (RDI) is the same measurement as AHI[8].

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6. **Sleep Disordered Breathing** (SDB): a spectrum of disorders based on irregular breathing during sleep. It includes *obstructive sleep apnea-hypopnea syndrome*, *central sleep apnea syndrome* (recurrent apneic-hypopneic events during sleep without upper airway obstruction), *Cheyne-Stokes breathing syndrome* (the waxing and waning breathing patterns of patients with cardiac dysfunction or intracranial disease), and *sleep hypoventilation syndrome* (hypoxia for greater than 50% of sleep without apneic-hypopneic events)[6].
7. **Chronic Intermittent Hypoxia:** the physiologic terminology for chronic repetitive episodes of oxygen desaturation and resaturation unique to SDB disorders.

Epidemiology

Eighty percent of mild to severe OSA is undiagnosed in the United States[5]. It is estimated that 20 million Americans have OSA[9]. Mild OSA is present in 24% of men and 9% of women, or 20% of the total population. OSA syndrome (OSAHS), or symptomatic OSA, occurs in 5% of the population, 4% of men and 2% of women. The male to female ratio of OSA is 3:1[4]. Race does not correlate significantly with OSA prevalence.

As a person matures through middle age, the risk for OSA increases and then plateaus at age 65. Correspondingly, OSA is three times as prevalent in the elderly as in middle age individuals, independent of obesity or snoring[10]. Mechanisms proposed for the age-related increase in prevalence include increased deposition of fat in the parapharyngeal area, lengthening of the soft palate, and changes in body structures surrounding the pharynx[11,12].

Fig. 1[1]
Estimated Percent Change in AHI for Selected Decrements and Increments of Percent Body Weight

Weight Change (%)	Change in AHI (%)
-20	-48
-10	-26
-5	-14
5	15
10	32
20	70

Obesity is the most common modifiable risk factor for OSA. 90% of OSA patients have a BMI ≥28[3]. Lifestyle modifications aimed at losing weight are the best way to decrease the number of nighttime apneas/hypopneas. Changes in obesity have been shown to directly correlate with disease severity (Figure 1)[1].

Pathogenesis of Upper Airway Collapse

OSA is a progressive disorder of the obese[4]. Fat deposition in the upper airway (UA) is most common at the lateral pharyngeal walls, decreasing pharyngeal caliber and adding external compression forces on the pharynx[13]. The decrease in pharyngeal size leads to increases in both UA resistance and negative intrathoracic pressure[14,15]. A combination of anatomical narrowing, a more collapsible airway, and increasingly negative intrathoracic pressure, predisposes OSA patients to UA collapse during sleep and anesthesia[16,17].

Repeat trauma induced by snoring[18] can lead to inflammation in mucosal and muscular layers of the soft palate along with denervation of the upper airway mucosa, resulting in decreased sensory and motor activity in the upper airway in OSA patients[19].

In the awake state, OSA patients with obstruction at the genioglossus have increased genioglossal tone (compared to non-OSA subjects) in order to maintain pharyngeal patency. When continuous positive airway pressure (CPAP) is applied, there is inhibited genioglossal activity in OSA patients but little effect in control subjects. This decrement in muscle activity (with CPAP) in apnea (OSA) patients likely represents inhibition of a neuromuscular reflex designed to preserve airway patency[20].

Sensory and motor denervation found in OSA patients could lead to decreased activity of neuromuscular reflexes designed to maintain patency. Patients with OSA show evidence of UA tissue damage, edema, and reduced UA mucosal sensation. These changes may be brought on by the mechanical events associated with nights/years of snoring and repetitive nighttime obstructions. Such mechanical events may cause peripheral nerve or muscle damage and ultimately impair the ability of the UA to defend itself from suction collapse[21].

Collapse of the UA is determined by two processes: a) increased airway resistance with an associated increase in negative intrathoracic pressure, and b) the loss of neuromuscular control of the pharynx. Collapse most often occurs at multiple sites within the pharynx with the most common site overall being the nasopharynx where the soft palate contacts the posterior pharyngeal wall[22,23].

With UA collapse, the extra work of the diaphragm causes increasingly negative intrathroracic pressure along with concomitant hypoxia and hypercapnea. Apnea-hypopnea episodes increase sympathetic output up to 125% of normal levels, increasing muscle tone, blood pressure, and other processes which cause arousal. With arousal comes restoration of pharyngeal dilator muscle tone, reoxygenation, and an abrupt decrease in sympathetic response[24].

Sequelae

Chronic intermittent hypoxia (CIH) is a newly recognized physiologic phenomenon and shares similarities with chronic hypoxia and ischemic reperfusion injury[25]. Much about CIH and its long and short terms effects are unknown. Known sequelae of CIH are increased sympathetic output and oxidative damage, sleep fragmentation, and the mechanical effects of negative intrathoracic pressure.

Effects of increased sympathetic output

Sympathetic output during apneas or hypopneas occurs in bursts of sympathetic activity, 125% above normal. Repeated sympathetic burst activity increases blood pressure upon termination of apnea. Over time, nocturnal dysregulation of sympathetic responses eventually leads to a tonic increase in sympathetic output after arousal and elevation in diurnal blood pressure[24].

OSA is an independent risk factor for hypertension. In a study by Logan et al., 34 of 41 patients (83%) in a drug-resistant hypertensive population, were found to be undiagnosed OSAHS patients[26]. CPAP decreases nocturnal hypertension in OSAS patients with refractory hypertension[27]. In severe OSA (OSAHS) but not in mild cases, CPAP has been proven to lower blood pressure[28,29]. Eleven to 37% of CHF patients have been found to have OSAHS in prevalence studies. In patients with CHF and OSAHS, treatment with CPAP has been associated with increased ejection fraction, lower systolic blood pressure, and decreased heart rate[30].

Consequences of increased oxidative damage

The main atherogenic sequelae of OSA are thought to occur through oxidative damage. CIH brings about a significant increase in inflammatory mediators, adhesion molecules, and free radicals, all thought to contribute to endothelial dysfunction[31].

OSAHS patients are at higher risk for stroke, myocardial infarction (MI), unstable angina, coronary artery disease, ischemic heart disease, and an increased 10-year risk for cardiovascular events. These risks can be decreased with CPAP therapy[31,32].

Stroke and transient ischemic attack (TIA) populations have an increased prevalence of OSA at 43 to 72%. Patients who suffer a stroke and have OSA have increased functional impairment and longer hospital stays. Treatment of OSA may lead to better functional outcomes and increased rehabilitation of patients with stroke[30,32].

Another pathology postulated to be caused by increased inflammatory mediators is insulin resistance. Insulin resistance has been linked to increased levels of tumor necrosis factor-α and interleukin-6, and both of these inflammatory mediators are increased with cyclical hypoxia as found in OSA and CIH. OSA has been proven as an independent risk factor for both glucose intolerance and insulin resistance independent of BMI, visceral obesity, and waist-to-hip ratio[33]. Also, CPAP has been shown to improve glycemic control in Type 2 diabetic OSA patients[34]. CPAP can help improve insulin sensitivity in OSA patients; however, obesity is the primary determinant of insulin sensitivity. Harsch and colleagues found that in OSA patients using CPAP therapy, "patients with a BMI of less than 30 kg/m^2 have a seven-fold higher chance of experiencing an improvement in insulin sensitivity".

Sleep fragmentation

Repetitive episodes of microarousal produce fragmented sleep[15,24]; and with chronicity, wakefulness is impaired with increased daytime sleepiness and decreased quality of life[35]. Not only is quality of life affected, but patient safety is compromised. OSA patients are at 2.5 times increased risk for traffic accidents versus non-OSA patients[36].

Mechanical effects of increasingly negative intrathoracic pressure

Preload to the right heart increases as intrathoracic pressure becomes more negative.

In hypertensive OSA patients, afterload is increased, and the myocardium consumes more oxygen to maintain cardiac output. Decreases in oxygenation and increased myocardial oxygen demand during events can produce subendocardial ischemia. Roughly 30% of OSAHS patients show increased nocturnal cardiovascular events such as ST depressions, the clinical significance of which has not been investigated thoroughly[37].

When coupled with all mechanisms of CIH, the final pathological outcome of OSA has been associated with congestive heart failure, systolic and diastolic dysfunction, cerebral ischemia, cardiac arrhythmias, pulmonary hypertension, cor pulmonale, and pulmonary edema[32,38].

Risk factors (Figure 2)[22]

The most prevalent risk factor for development of OSA is obesity[4]. In the absence of neck masses, such as goiters, increasing neck circumference is the best correlate of disease progression and severity of OSA[3,39]. Increasing age is another risk factor. Alcohol has acute adverse effects of worsening AHI but has unknown long term effects. Smoking worsens OSA but its effects are reversible upon cessation[10]. But, because former smokers do not manifest the increased risk for obstructive sleep apnea, airway inflammation and damage due to direct contact with cigarette smoke could alter the mechanical and neural properties of upper airway and increase collapsibility during sleep[40].

Fig. 2[22]
Risk factors and symptoms of OSA in adults

Demographical risk factors

1	Male
2	40-70 years of age
3	Familial aggregation

Suspected risk factors

1	Smoking
2	Genetics
3	Menopausal
4	Alcohol before sleep
5	Evening nasal congestion

Established risk factors

1	Obese or overweight
2	Central body fat distribution
3	Large neck circumference Craniofacial/airway
4	Abnormalities

OSA Symptoms

1	Habitus
2	Snoring
3	Nocturnal periods of apnea
4	Choking
5	Gasping
6	Daytime sleepiness

Postmenopausal women have 4 times the risk of OSA versus premenopausal women[41], perhaps secondary to decreased progesterone levels. Ventilatory drive is increased during the luteal phase of the menstrual cycle when progesterone levels are highest[42,43]. Polycystic ovarian syndrome (PCOS) also predisposes an increased risk for OSA, independent of obesity. Increased circulating androgens secondary to PCOS can increase soft tissue deposition in the pharynx, affect function of pharyngeal dilator muscles, and alter ventilatory control mechanisms[44]. Other risk factors for OSA are: craniofacial disorder; retroposed mandible/maxillae; adenotonsillar hypertrophy; nasal pathologies: deviated septum, allergic rhinitis; hyperthyroidism, acromegaly, and Down syndrome[30].

Genetic factors also play a role. A first-degree relative with OSA increases a person's risk of having OSA by 1.5 to 2 times independent of the genetics for obesity[45-47].

Anatomical narrowing of the UA is a risk factor for apneic events during sleep or anesthesia[17]. Factors that decrease skeletal confines of the tongue and

decrease pharyngeal dimensions predispose individuals to OSA.

A patient with a difficult airway is also at risk. Therefore, patients who are difficult intubations or whose airway closes intraoperatively may benefit from postoperative screening for OSA[48]. Screening positive for OSA should trigger diagnostic procedures and therapy.

Pre-operative Assessment

Screening for OSA

Due to its high prevalence and the high number of undiagnosed OSA patients, all patients should be screened for OSA[49]. Simple screening factors include obesity, snoring or apneic episodes in sleep, daytime sleepiness, and hypertension (Table1)[50].

Table 1[50]
Univariate Obstructive Sleep Apnea Predictors (using an RDI cutoff value of 10 hours[1])

Variable	Odds ratio	P value	95% confidence interval
Age	1.10	0.001	1.03,1.16
Epworth Sleepiness Scale	1.03	0.558	0.93,1.13
Snoring history	12.5	0.023	1.42,110.6
Choking episodes	2.02	0.169	0.74,5.49
Witnessed apneas	3.37	0.016	1.25,0.06
Hypertension	10.3	0.029	1.27,83.9
Pharyngeal grade, I-IV	1.52	0.046	1.01,2.30
Sampsoon-Young class, I-IV	1.77	0.018	1.1,2.86
Palatal Elevation	1.41	0.303	0.73,2.71
Overbite	2.19	0.044	1.02,4.70

Proven physical exam findings that are risk factors for OSA are BMI ≥28, neck circumference >17 inches for men or >16 inches for women[48], cricomental distance ≤1.5 cm, pharyngeal grade greater than III or IV, and presence of an overbite. Proven findings that do not accurately predict the presence of OSA are retrognathia, large tonsil size, enlargement of the uvula, and a change in palatal elevation with phonation[3,14,50].

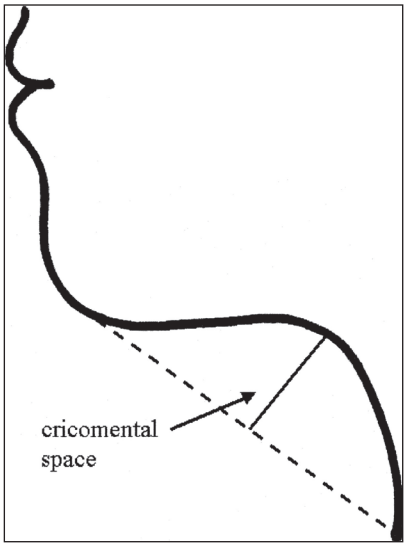
Risk factors for OSA also correlate with risk factors for difficult intubation. Mallampati score, anterior mandibular depth, lower mandibular angle, cervical angle, and the occlusal-geniohyoid angle, correlate with both difficult intubation and increased risk of OSA.

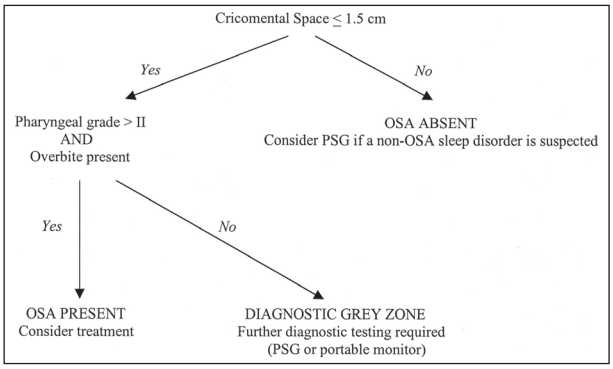
Predicting undiagnosed OSA

There are many prediction models of OSA in previously undiagnosed patients. In general, OSA prediction models are highly sensitive with poor specificity[51]. Two prediction models are as follows:

1. Designed by Tsai et al., this model uses the cricomental distance ≥1.5 cm as its basis (Fig. 3). When the cricomental distance is ≥1.5 cm, OSA can be excluded in a patient with a negative predictive value of 100% (CI 95%). If cricomental distance is <1.5 cm, the pharyngeal grade and overbite are assessed.

Fig. 3[50]





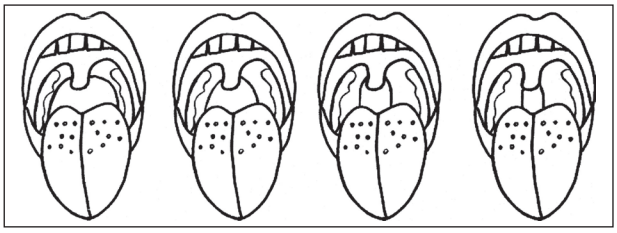
If pharyngeal grade is a III or IV (Fig. 4)[50] and overbite is present, OSA is assumed in a patient with a sensitivity of 40% and a specificity of 96%. In the study population, 60% of patients did not fall into either category, leaving them in the "gray zone" of

diagnosis[50].

2. Another prediction model is the "modified neck circumference" described by Flemons. The patient is screened for the following criteria: 1) snoring, 2) daytime sleepiness or drowsiness while driving, and 3) obesity or hypertension. Patients who meet at least 2 of the criteria are then measured for neck circumference; the circumference is then 'modified' for potential sequelae of OSA. If the patient has a history of hypertension, 4 cm is added, a habitual snorer has 3 cm added; and a patient reported to choke or gasp most nights has 3 cm added. A score of 43 cm or less indicates low risk for OSA, 43-48 cm is intermediate risk (4-8 times risk), and more than 48 cm indicates a high risk for OSA (20x's risk)[52].

Fig. 4[50]

Pharyngeal grading system. *Class I = palatopharyngeal arch intersects at the edge of the tongue. Class II = palatopharyngeal arch intersects at 25% or more of the tongue diameter. Class III = palatopharyngeal arch intersects at 50% or more of the tongue diameter. Class IV = palatopharyngeal arch intersects at 75% or more of the tongue diameter*



If a patient is suspected of having OSA and is not yet diagnosed, adequate perioperative preparation should be made.

Pre-operative sedation

Pre-operative sedation and medications should be carefully chosen and administered in a setting that is well supervised and includes O_2 saturation monitoring[21]. The presence of snoring and apneic events during sleep may be unmasked by pre-operative sedation. Due to sedatives increasing respiratory depression and apnea, some authors have warned against the use of preoperative sedation in OSA patients[53].

Outpatient versus Inpatient

ASA guidelines for outpatient care in OSA patients interprets many factors including, "(1) sleep apnea status, (2) anatomical and physiologic abnormalities, (3) status of coexisting diseases, (4) nature of surgery, (5) type of anesthesia, (6) need for postoperative opioids, (7) patient age, (8) adequacy of postdischarge observation, and (9) capabilities of the outpatient facility." Current ASA guidelines for outpatient strategies are outlined in Table 2[49].

Table 2[49]

Consult opinion on outpatient surgical procedures in patients with OSA

Consult agrees procedure is safe

1	superficial surgery/local or regional anesthesia
2	minor orthopedic/local or regional anesthesia
3	Lithotripsy

Consult is equivocal on procedure safety

1	superficial surgery/general anesthesia
2	tonsillectomy in age >3
3	minor orthopedic/general anesthesia
4	gynecologic laparoscopy

Consult is disapproves of safety

1	airway surgery
2	tonsillectomy in age <3
3	laparoscopic surgery/upper abdomen

Use of regional anesthesia

Regional anesthesia should be strongly considered as the method of choice for OSA patients[49]. Under regional anesthesia, patients do not lose conscious reflexes that protect the airway. Use of regional anesthesia may be technically difficult due to patient body habitus and is limited by procedure type. Potential poor outcomes of regional anesthesia are associated with unintentional side effects if the regional block spreads further than intended, and if better control of the airway is needed mid-procedure due to heavier than expected sedation. There is no evidence that regional anesthesia is associated with better outcome for OSA patients[22].

Intra-operative Management

Pre-oxygenation

Pre-oxygenation may be technically difficult in OSA patients. Due to obesity, functional residual capacity is greatly reduced leading to faster oxygen desaturation compared to non-obese patients[54] because of unopposed increases in intra-abdominal and intra-

thoracic pressures that decrease lung compliance and impair PaO_2[55-57]. A recent study of preoxygenation in the severely obese suggests that preoxygenation with the head at 25 degrees elevation versus supine can increase the time before arterial desaturation[58].

Airway management requires a blend of proper positioning, artificial airways, and may require CPAP[22]. The importance of proper positioning and the difficulty of repositioning in the obese patient should not be underestimated[59].

Preinduction positive end expiratory pressure (PEEP) can increase time before oxygen desaturation upon intubation. Preoxygenation with 100% FiO_2 and 10 cm PEEP for 5 minutes before induction followed by 10 cm PEEP with mask ventilation after intubation, decreases postintubation atelectasis[60]. In a study by Pelosi, et al, the use of 10 cm of water PEEP in morbidly obese patients during the maintenance phase of general anesthesia was shown to increase PaO_2 throughout surgery, whereas in normal patients PEEP had no significant effect on PaO_2. Patients in the study were taken to the ICU post-operatively and weaned off PEEP gradually[61].

Intubation of the OSA patient

OSA is a risk factor for difficult intubation[2]. The intubation failure rate for OSA patients using direct laryngoscopy is 5%, roughly 100 times the failure rate of the general population – necessitating either cancellation of cases or intubation via alternative methods[3]. Laryngeal mask airways are useful adjuncts. Further difficult airway management should be performed according to *ASA Difficult Airway Guidelines 2003*.

Extubation

As with all patients, complete reversal of neuromuscular blockade must be ensured before extubation, and extubation must be attempted in Stage I, the analgesia stage, when the patient is conscious and able to follow commands. Extubation is particularly dangerous following OSA corrective surgeries as 5% of patients have life threatening post-extubation airway obstruction[3]. Airway collapse as a result of early extubation can lead to rapid development of severe negative pressure pulmonary edema from spontaneous ventilation against an obstructed airway[3].

For morbidly obese OSA patients undergoing non-nasal or non-oral surgery, the decision to extubate while awake or to maintain postoperative mechanical ventilation should be considered. Factors in this decision include ease of mask ventilation, difficulty of tracheal intubation at the beginning of the case, existing cardiopulmonary disease, length and type of surgery, BMI, and severity of OSA (Fig. 5).

Criteria that should be fulfilled before extubation are as follows:

1. The patient should be fully awake before extubation - rational and responsive to commands. Mindless reflex movement is not purposeful movement.
2. Full recovery from neuromuscular blockade must be proven by both use of a neuromuscular blockade monitor and a sustained head lift >5 s. In the ICU, an adequate vital capacity (>15 cc/kg) and negative inspiratory force (NIF) <-25cm H_2O should be present.
3. Concentration of serum narcotic should be titrated to a spontaneous respiratory rate >12-14/min.

Fig. 5
Major determining factors when deciding awake vs. asleep extubation during uvulopalatopharyngeoplasty or nasal surgery

Individual factors	**Factor status and extubation plan**
BMI AHI Ease of mask ventilation Ease of tracheal intubation Experience at start of case Associated cardiopulmonary disease	If all factors good → patient may be asleep If one factor severe → awake extubation Factors in between extremes → requires clinical judgement

After extubation, the patient should be positioned in reverse Trendelenburg at 30 degrees or upright as these positions minimize compression of abdominal contents against the diaphragm.

Post-operative Management

Continuous positive airway pressure (CPAP)

If patients use CPAP machines preoperatively, their machines should be available immediately after surgery to decrease apneic events[3]. In the postoperative setting, sleep is disturbed and patients are at increased risk of apneic events during sleep for 1 week following surgery[62]. For the first 3 days after surgery, the deeper stages of sleep, non-rapid eye movement (NREM) stages 3 & 4 and rapid eye movement (REM), are suppressed. In post-operative days 4 through 6, REM rebounds and natural deep sleep apnea increase[63,64].

Monitoring

Due to the increased risk of apnea, the monitoring needs for these patients are more intense, and strategies for dealing with them postoperatively vary from an overnight stay in the ICU to a PACU environment with a nursing ratio of 2 or 3:1[65]. Deutscher and colleagues provide a strategy to try to guide the level of care needed for OSA patients based on the risk of surgery and severity of OSA displayed in Table 3[66].

Approximately 60% of postoperative hypertension occurs in patients who have a history of hypertension preoperatively[67]. OSA patients are at risk for postoperative hypertension and blood pressure should be closely monitored and controlled so as to minimize the risk of surgical site bleeding.

Current ASA recommendations for monitoring are that OSA patients, "should not be discharged from the recovery area to an unmonitored setting (*i.e.*, home or unmonitored hospital bed) until they are no longer at risk for postoperative respiratory depression. Adequacy of postoperative respiratory function may be documented by observing patients in an unstimulated environment, preferably while they seem to be asleep, to establish that they are able to maintain their baseline oxygen saturation while breathing room air"[49].

Pain management

OSA carries an increased risk of postoperative complications, with most complications occurring within 2 hours[68,69]. Judicious use of opioids and other respiratory depressants used for pain management is essential in the post-operative period, as OSA patients are at increased risk for upper airway obstruction when sedated[70,71]. Regional nerve blocks may be useful for postoperative pain management. The judicious use of post-operative narcotics and sedatives in patients with OSA should be emphasized to healthcare professionals involved in the patient's care[49,72].

Conclusion

Obstructive Sleep Apnea (OSA) is an often undiagnosed condition which presents many challenges to the anesthesiologist. The prevalence of obesity in world wide suggests that an increasing number of patients with sleep apnea will present for surgery. OSA frequently coexists with other disease processes such as hypertension, congestive heart failure, and pulmonary hypertension. An understanding of the pathophysiology behind OSA is necessary to assure proper perioperative management. Recent emphasis has been placed by the American Society of Anesthesiologists on this condition and guidelines for management have been published.

Table 3[66]

Sleep Apnea Scale for Monitoring Needs Postoperatively					M = monitored area W = standard ward H = home * = case by case basis
		OSA Severity			
		Mild	Moderate	Severe	
Surgical	Low	W/H*	W/H	W	
Risk	Intermediate	W	M/W*	M	
	High	M/W*	M	M	

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