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Obstructive sleep apnea in the adult obese patient: implications for airway management

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Obstructive sleep apnea in the adult obese patient may be due, in part, to an increased amount of pharyngeal tissue; therefore, there is an increased risk of intubation and extubation difficulties, and pain management can be expected to be complicated by narcotic/sedative-induced pharyngeal collapse.

The number of adult obese patients with obstructive sleep apnea (OSA) is very large. It has been estimated, that among the middle-aged, 4% of men and 2% of women have clinically significant symptomatic OSA [1,2]. Prevalence rates of OSA and snoring increase with age [3,4] and the data in Table 1 is considered to be representative of this relationship [5]. As another very important independent factor, 60% to 90% of persons with OSA are obese (defined as a body mass index [BMI] > 29 kg/m^2) [5,6] 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 [1,2,7]. Indeed, in 1993 the National Commission on Sleep Disorders Research estimated that 18 million Americans have OSA [8].

At present, 80% to 90% [9] to 95% [8] of persons with OSA are undiagnosed; obviously, this includes those who require anesthesia and surgery now. At the same time, general physician recognition of the problem is rapidly growing [10] resulting in an increase in polysomography testing of approximately 124% every three years [11]. Thus, the incidence of adult obese patients presenting for anesthesia and surgery with either a presumptive clinical and/or a sleep study diagnosis of OSA can be expected to increase five- to ten-fold in the next decade. Finally, on the basis of the facts that the mean age and weight of the USA population is increasing steadily [7], and that most excess body fat accrues after 20 years of age [7], we can expect the incidence of adult obese patients with OSA presenting for anesthesia and surgery, both with and without a diagnosis of OSA, to increase for many years.

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Table 1
Distribution by age of categorical levels of AHI* (AHI = Apneas + Hyponeas/Hour of sleep)

Age (y)	Habitual snoring (%)	AHI > 5 (%)	AHI > 10 (%)	AHI > 15 (%)
< 25	14	10	2	0
26–50	41	26	15	0
>50	46	61	50	36

AHI = Apnea Hyponea Index.

* *From* Strohl KP, Redline S. Recognition of sleep apnea. Am J Respir Crit Care Med 1996;154: 279–86.

This review is limited to considering OSA in adult obese patients for two reasons. First, although other causes of OSA may coexist, an important mechanism of the obstruction in obese patients, namely, pharyngeal airway tissue enlargement, is relatively specific to the obese patient. To the contrary, the etiology of OSA in nonobese patients appears to be different and consists of predominantly of craniofacial and orofacial bony abnormalities [12]. However, excess neck fat may be present in nonobese snorers [13] and superimposition of obesity on any pre-existing bony abnormality may increase the severity of OSA. Similarly, nasal pathology and tonsillar hypertrophy are well-defined obvious causes of obstructed breathing in patients of any weight and are not considered here. Because OSA in pediatric patients is usually related to bony, nasal, and tonsillar pathology [14], pediatric patients are also not considered in this review. Second, obesity is by far the single most important physical characteristic associated with OSA in the adult population (60%–90% incidence) [5,6] and therefore the large majority of the adult OSA population is covered by this review.

Definition of OSA terms

OSA is defined as cessation of airflow for more than 10 seconds despite continuing ventilatory effort, five or more times per hour of sleep, and is usually associated with a decrease in arterial oxygen saturation (SaO_2) of more than 4% (Table 2) [15]. 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 2). Both OSA and OSH repeatedly disrupt sleep due to increased ventilatory effort-induced arousal which, in turn, causes daytime sleepiness and altered cardiopulmonary function (Table 2) [15].

Table 2
Major characteristics of obstructive sleep apnea (OSA) and obstructive sleep hypopnea (OSH)*

Obstruction	↓in Airflow >10 s	Times/h	↓in O_2 saturation	Disrupted sleep	Daytime sleepiness
OSA	100%	>5	≥ 4%	Yes	Yes
OSH	>50%	>15	≥ 4%	Yes	Yes

* *Data from* Strollo PJ, Rogers RM. Obstructive sleep apnea. Current concepts. N Engl J Med 1996;334:99–104.

Pathophysiology of OSA in the adult obese patient

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 collapsible 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) (Fig. 1). These pharyngeal segments are collapsible because the anterior and lateral walls lack bony support. The adult human is the only mammal to have an oropharynx (in all other mammals the tip of the uvula touches the tip of the epiglottis) and to suffer from OSA [16].

Patency of the collapsible segments of the pharynx depends on the inspiratory function of the pharyngeal dilator muscles which act to stiffen or distend the collapsible pharyngeal airway during inspiration [17]. The larger the negative inspiratory pressure, the larger the force of contraction of the pharyngeal dilator muscles required to keep the airway open. This negative pressure reflex response seems to be driven by pressure-sensitive airway receptors since pharyngeal anesthesia diminishes or abolishes it [18,19]. Activity of these muscles, at least during wakefulness, is thus precisely controlled both to maintain upper airway patency and to allow hyperventilation during hypoxia or hypercapnia. 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 (Fig. 1) [18,20].

The tensor palatini retracts the soft palate away from the posterior pharyngeal wall, thereby maintaining retropalatal pharyngeal patency during nasal breathing. The action of the tensor palatini is predominantly tonic [20]. Tonic activity of the tensor palatini decreases during sleep and correlates well with increased upper airway resistance (UAR) during sleep [17,20].

The genioglossus moves the tongue anteriorly to open the retroglossal air space and is considered to be the most important muscle in keeping the upper airway patent. The point of insertion of the genioglossus on the mandible determines the efficiency of genioglossus muscle contraction; anterior insertion is advantageous. The action of the genioglossus is phasic with inspiration [17]; this activity decreases with sleep in normal subjects and almost ceases during deep rapid eye movement (REM) sleep and is abolished in patients with OSA at the onset of an apnea and increases at the termination of an obstruction [17].

The muscles that cause forward movement of the hyoid bone (geniohyoid, sternohyoid, thyrohyoid) are thought to enlarge and stabilize the retroepiglottic laryngopharynx by tensing the hyoepiglottic ligament. The activity of these muscles are phasic with inspiration and the activity of these muscles correlates inversely with UAR [17,20]. Factors that influence hyoid position, such as neck

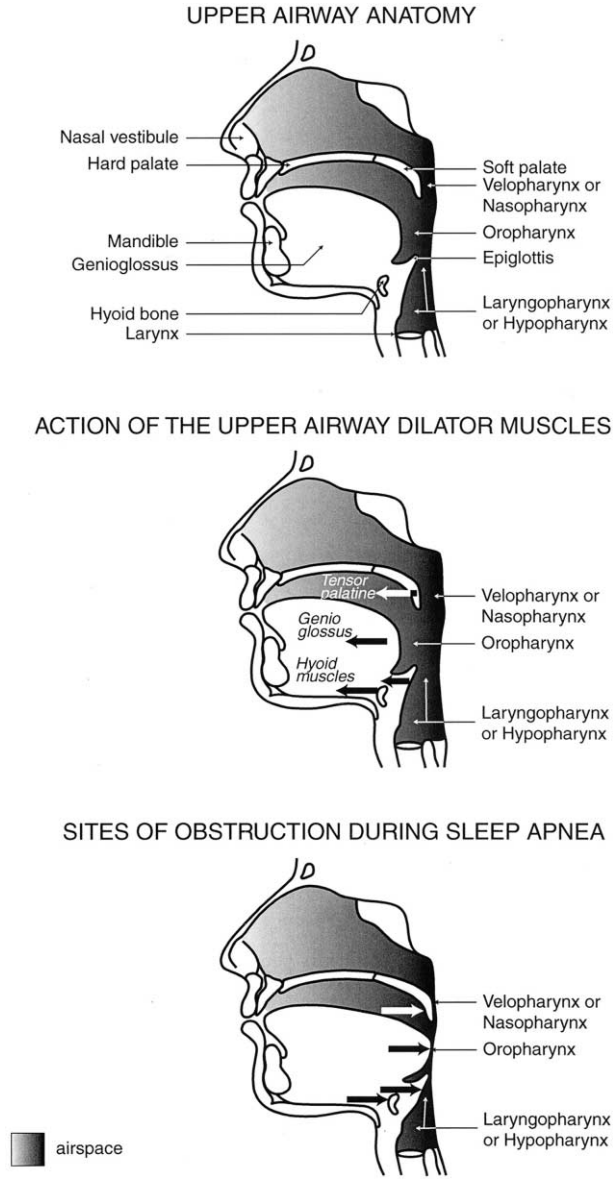

Fig. 1. The upper panel schematic shows the important upper airway anatomy. The nasopharynx ends at the tip of the uvula; the oropharynx extends from the tip of the uvula to the epiglottis; the laryngopharynx extends from the tip of the epiglottis to the posterior cricoid cartilage. The middle panel shows the action of the most important dilator muscles of the upper airway. The tensor palatine, genioglossus, and hyoid muscles enlarge the nasopharynx, oropharynx, and the laryngopharynx, respectively. The bottom panel shows collapse of the nasopharynx at the palatal level, the oropharynx at the glottic level, and the laryngopharynx at the epiglottic level. (*From* Benumof JL. Obstructive sleep apnea in the adult obese patient: implications for airway management. J Clin Anesth 2001;13(2):144–56, with permission.)

flexion or mandibular abnormalities, can adversely affect the function of these muscles, leading to narrowing of the laryngopharynx.

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 four to six cycles of non-rapid eye movement (NREM) sleep followed by REM sleep. There are four stages of NREM sleep which represent progressively deeper sleep with progressive slowing of the electroencephalogram (EEG) waves. Stages 3 and 4 of NREM sleep differ only in the relative amount of slow waves and together Stage 3 and 4 are called slow wave or deep sleep and is a restorative period of sleep. REM sleep is also very deep sleep and is almost the exclusive domain of dreaming [18]. REM sleep is characterized by a generalized loss of muscle tone as evidenced by electromyography (EMG). However, the eye muscles are not paralyzed, intermittent conjugate REMs still occur, and can be monitored by electro-oculography (EOG).

During NREM sleep, the rhythmic activity of the upper airway muscles decreases, UAR increases significantly and can be twice that during the awake state [21,22]. 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 [23]. As pharyngeal pressure becomes more negative, pharyngeal collapse increases. 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 [24].

Fig. 2 shows this sequence of events over the course of four breaths in a sleeping 39-year-old male with a BMI of 30.5 kg/m^2 [23]. Over these four breaths the esophageal pressure became increasingly negative, airflow progressively decreased to zero, and the fiberoptic images show near total occlusion on the third breath and total occlusion on the fourth. If the occlusion is partial, snoring is likely to occur and is due to a fluttering of the pharyngeal walls and soft palate. The sound intensity of the snoring correlates well with the severity of OSH and OSA [26]. If airway occlusion is total, then apnea results and snoring is absent.

Obesity and OSA: pharyngeal pathology and incidence

There are two reasons why obesity per se may cause OSA and OSH. First, there is an inverse relationship between obesity and pharyngeal area [27,28]. Magnetic resonance imaging shows that the decreased pharyngeal area in obesity results from deposition of adipose tissue into pharyngeal tissues. The pharyngeal structures that may increase in size with deposition of adipose tissue, from the nasopharynx down to the laryngopharynx, are the uvula, the tonsils, the tonsillar pillars, the tongue, aryepiglottic folds, and the lateral pharyngeal

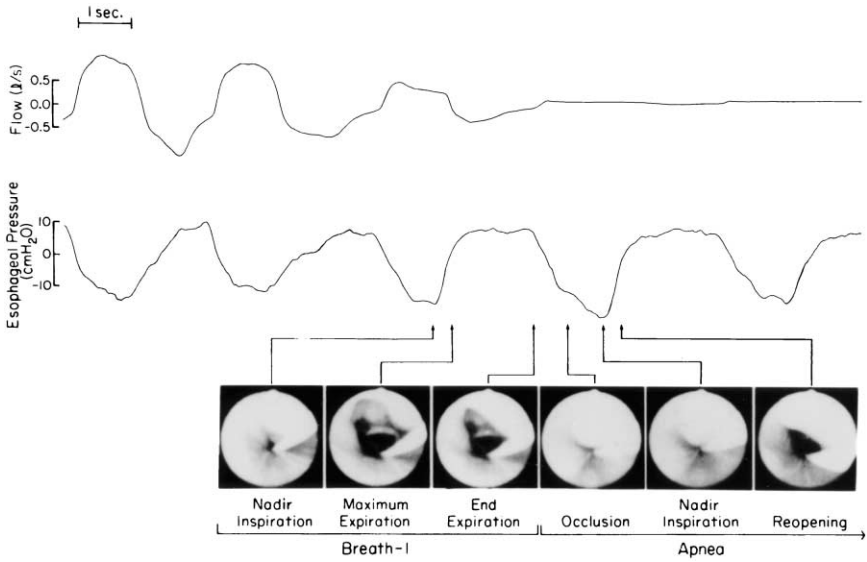

Fig. 2. An original recording (Patient 6, BMI = 30.5 kg/m^2 of Reference [23]) of airflow (flow; inspiration positive) and esophageal pressure. Tracings show three breaths leading to an obstructive apnea. Fiberoptic images are shown for the breath immediately preceding the apnea (Breath-1) and the apneic period. During Breath-1, the images selected correspond to the smallest cross-sectional area that occurred during inspiration (Nadir Inspiration), the largest cross-sectional area during expiration (Maximum Expiraton), and the cross-sectional area at the end-expiration (End Expiration). During the apnea, the images correspond to the time at which airway occlusion occurred (Occlusion), the time at which the maximum esophageal pressure was generated (Nadir Inspiration), and the time at which the airway reopened (Reopening). Within each image the dark area is the airway lumen, the lighter horseshoe shape in the middle of the image is the epiglottis, and the narrow white triangular/cylindrical shape on the right is the esophageal pressure catheter. (*From* Morrell MJ, Arabi Y, Zahn B, Badr MS. Progressive retropalatal narrowing preceding obstructive apnea. AM J Respir Crit Care Med 1998;158:1974–81, with permission.)

walls. 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 pharynx between the uvula and epiglottis [4,15,16].

The deposition of fat in the pharynx of OSA patients appears to be predominantly into the lateral walls of the pharynx [13,29–33] and the volume of fat in the lateral pharyngeal walls correlates well with the severity of OSA [31,32]. The converse is also true; ie, weight loss improves the pharyngeal and glottic function of OSA patients [34]. The deposition of fat into the lateral walls of the pharynx not only narrows the airway but also changes the shape of the pharynx in obese patients from being an ellipse with a long transverse (lateral) and a short anterior-posterior axis to an ellipse with a short transverse and a long anterior-posterior axis [20,30]. The external expression of very large internal lateral parapharyngeal fat pads is submandibular (lateral) jowls.

It has been theorized that the change in long axis from transverse to anterior-posterior is functionally significant [35]. Since the muscles that increase upper airway size are all located along the anterior border of the pharynx (tensor palatini, genioglossus, hyoid muscles), they pull the anterior wall of the pharynx forward increasing the anterior-posterior axis (Fig. 1). Fig. 3 shows that this action of the anterior pharyngeal dilator airway muscles is less efficient in a pharynx with a long anterior-posterior elliptical axis than in one with a long transverse elliptical axis [35].

Second, the patency of the pharynx (which is a collapsible tube) 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 [36,37]; ie, the upper airway is compressed externally. This external mechanism of increasing UAR is easily demonstrated in animals by experimentally increasing the amount of anterior cervical neck fat [36]. Therefore, it is not surprising that the neck is significantly fatter in obese OSA patients compared to equally obese non-OSA patients [38] and that the incidence and severity of OSA correlates better with increased neck circumference than with general obesity [39–41].

Snoring occurs in 30% to 40% and 15% to 25% of obese (upper 25th percentile of the BMI) and nonobese men, respectively, and in 15% to 25% and 5% to 10% of obese and nonobese women, respectively [25]. Approximately half of individuals that snore have some degree of OSA and virtually all patients with OSH and OSA snore to some extent [4,15]. In two small series of persons with BMI > 45.3 and > 50.2 (N = 250 and 27) 40% to 77% of men, but only 3% to 7% of women, had significant OSA [42,43].

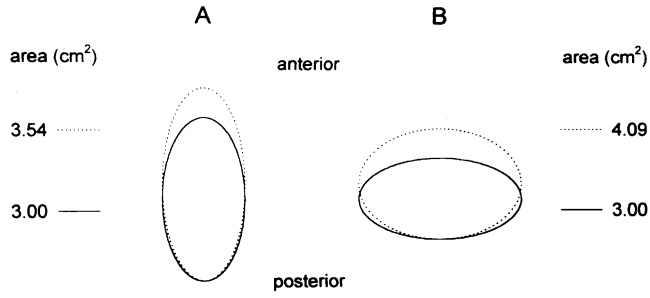

Fig. 3. The effect of a 5-mm change in the anteroposterior (AP) diameter of the airway on airway cross-sectional area is shown for two equally elliptical airways with different lateral/AP ratios. (A) The lateral/AP ratio = 0.5. (B) The lateral/AP ratio = 2. The lateral dimension of each ellipse was held constant. The solid line represents the starting area (3 cm^2 in both ellipses), and the dotted line represents the area after a 5-mm increase in the AP diameter. The area change is greater in the ellipse with a more lateral orientation (B). (*From* Leiter JC. Upper airway shape. Is it important in the pathogenesis of obstructive sleep apnea? Am J Respir Crit Care Med 1996;153:894–8, with permission).

Arousal

Over the course of an apnea a number of important respiratory events occur. First, arterial oxygen tension (PaO_2) decreases as a function of the initial PaO_2, functional residual capacity (FRC), and the duration of apnea. Arousal may occur, in part, because decreased PaO_2 augments carotid body output [44,45]. Second, arterial carbon dioxide tension ($PaCO_2$) increases as a function of duration of apnea. Arousal may, in part, be caused by central nervous system receptors sensitive to increasing $PaCO_2$ [46]. Third, ventilatory effort progressively increases as the apnea proceeds as a function of both the decreasing PaO_2 and increasing $PaCO_2$ [47,48]. Finally, as a function of the increased ventilatory effort, intra-airway pressure becomes progressively more negative. Arousal may, in part, be mediated by pressure-sensitive receptors in the upper airway [49].

Any or all of these four mechanisms could increase neural traffic in the reticular activating system and arouse the individual. Arousal often consists of just increased neural activity in the brain but sometimes has a visible external expression that ranges from simple one extremity twitching and movement to multiple extremity flailing and gasping for air. Once arousal occurs, the muscles of the upper airway reactivate, thereby opening the pharyngeal airway. The opening of the pharyngeal airway may cause a snorting noise. Ventilation then resumes and the hypoxia and hypercapnia are corrected [18]. The individual then returns to sleep and the cycle begins again [18].

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.

Systemic pathophysiology of OSA

Fig. 4 outlines the systemic effects of the sleep → arousal → sleep cycles that occur many times during every sleep. Decreases in PaO_2 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 [50]. When SaO_2 decreases below 60%, the severity of the bradycardia and the onset of ventricular ectopy increases markedly [51]. The high incidence of arrthymias in OSA patients may explain the higher incidence of nocturnal angina and myocardial infarction in these patients [5]. Proper treatment of OSA decreases the incidence of these arrthymias [52] and presumably decreases the incidence of myocardial ischemia.

Three mechanisms may account for pulmonary hypertension (ppa) in OSA. First, decreases in PaO_2 may directly cause hypoxic pulmonary vasoconstriction. Second, transmural ppa increases during each obstructive apnea because intrathoracic pressure becomes more negative with increasing ventilatory effort against the obstruction [53]. Third, increase in both pulmonary and systemic arterial pressures (Ppa and Psa) correlate directly with decreases in PaO_2, with the maximal increase in both pressures occurring during the nadir in PaO_2 during REM sleep [54]. Diurnal Ppa and Psa hypertension in OSA patients is likely

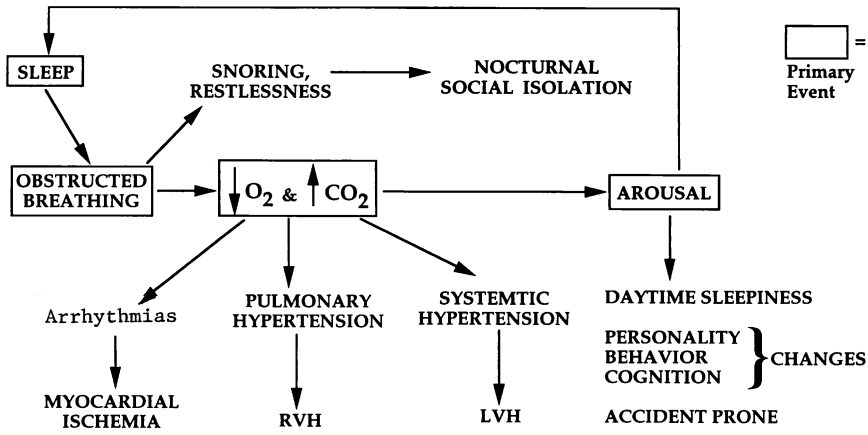

Fig. 4. The systemic pathophysiology of repeated sleep arousal sleep cycles in obstructive sleep apnea. RVH = ventricular hypertrophy; LVH = left ventricular hypertrophy. (*From* Benumof JL. Obstructive sleep apnea in the adult obese patient: implications for airway management. J Clin Anesth 2001;13(2):144–56, with permission.)

caused by the innumerable repetitive increases in sympathetic tone that occurs with each hypoxemic-hypercapnic arousal event [15,55]. Diurnal Psa hypertension is present in 50% of OSA patients [56] and is independent of obesity, age. and sex [4,6,15,52]; proper treatment of OSA results in a decrease in psa hypertension [52].

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 [15,52,57]. In view of the dual circulation hypertension, biventricular hypertrophy, increased incidence of arrthymias, myocardial infarction and stroke, it is not surprising that the cumulative eight-year mortality in patients with moderate untreated OSA is 37% compared to 4% in patients with mild OSA [15,58–60].

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 [15]. 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 [61,62] and independent of any other possible confounding factor [61,63].

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, and daytime sleepiness or fatigue.

Increased neck circumference is associated with OSA [39–41]; specifically, neck circumference of OSA patients = 41.1 ± 3.5 cm versus neck circumference in patients without OSA = 38.0 ± 3.5 cm, $P < 0.001$ [64].

Obesity is best expressed quantitatively as BMI:

$$\text{BMI} = \text{mass/height}^2 = \text{kg/m}^2 \text{ or } 703 \times \text{lb/in}^2$$

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 [65]. Ninety percent of OSA patients may have a BMI > 28 kg/m^2 [5]. Whereas the principal limitation of use of the BMI is that it does not distinguish between fat and muscle, in the general population it is much more likely that an individual with a BMI > 30 kg/m^2 is obese rather than muscular.

The definitive diagnosis of OSA and OSH, however, must be made by some form of sleep study. A complete comprehensive sleep study exam is done by a technologist in a formal soundproof infrared video monitored sleep study laboratory. However, such studies can be logistically complex and expensive to perform and may be relatively inaccessible some areas. Under these circumstances some simpler derivation (eg, portable abbreviated monitoring/screening system) is often performed and may be cost-effective [15,52].

A full sleep study consists of monitoring the EEG (for stage of sleep and arousal), the EOG (for NREM versus REM sleep), oral and nasal airflow sensors and capnography (for actual movement of air), noise (for snoring and snorting),

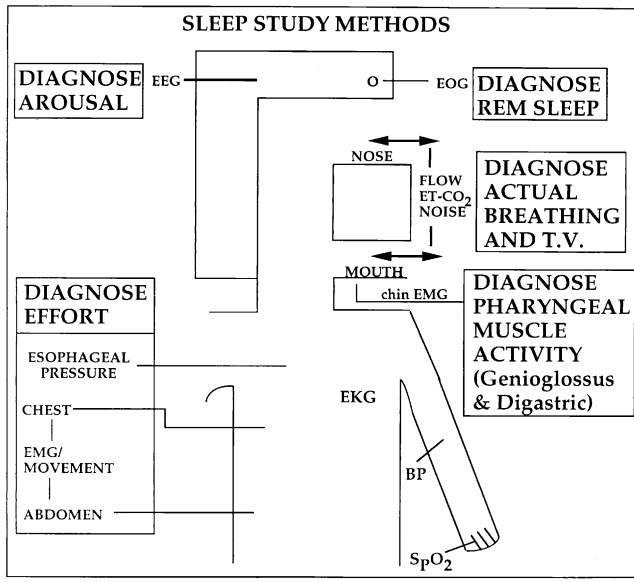

Fig. 5. Schematic illustrating the methodology of polysomography (sleep study). (*From* Benumof JL. Obstructive sleep apnea in the adult obese patient: implications for airway management. J Clin Anesth 2001;13(2):144–56, with permission.)

esophageal pressure and chest and abdominal movements (for breathing effort), submental and extremity EMG (for pharyngeal [genioglossus] muscle activity and extremity movement, respectively), oximetry (pulse, ear, transcutaneous) (for SpO_2 and noninvasive blood pressure), and ECG (for cardiovascular function) (Fig. 5). Rarely, direct systemic and pulmonary artery pressure monitoring is performed for more precise determination of cardiopulmonary function.

Apnea is defined as the cessation of airflow for >10 seconds and is considered obstructive if there is respiratory effort during the apnea. Hypopnea is defined as greater than a 50% reduction in airflow for >10 seconds. Fig. 6 shows simple examples of OSA (effort with no flow), central sleep apnea (no effort, no flow), and mixed obstructive and central sleep apnea [66]. Fig. 7 illustrates an example of obstructed breathing during sleep leading to apnea (panel A) and obstructed breathing during sleep leading to hypopnea (panel B) [15].

The results of a sleep study are reported 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. 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% [42].

Oxygen data are reported in several ways. Most laboratories report the number of SaO_2 desaturations greater than 4%, the lowest SaO_2, and time spent at a certain

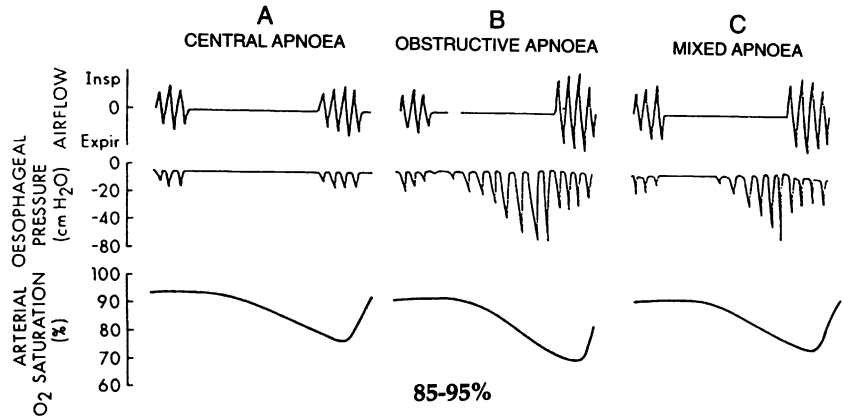

Fig. 6. Simple classical examples of central sleep apnea. Panel A = no effort, no flow; Panel B = obstructive apnea (effort, no flow); and Panel C = mixed apnea (initially no effort, then effort, but no flow). Effort is indicated by downward, negative deflections in esophageal pressure. As illustrated, all three types of apnea result in large decreases in arterial oxygen saturation. In the typical patient with obstructive sleep apnea, 85% to 95% of the apneas are purely obstructive, some are mixed, and a few are central. (*Adapted from* Boushra NN. Anaesthetic management of patients with sleep apnea syndrome. Can J Anaesth 1996;43:599–616, with permission.)

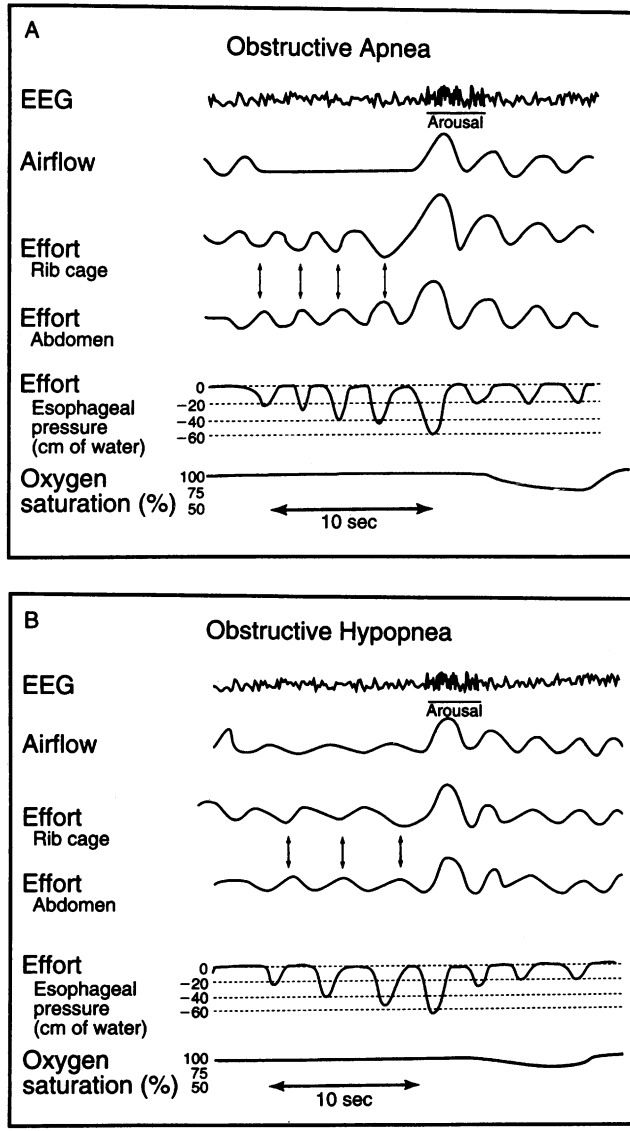

Fig. 7. Simple examples of obstructed breathing during sleep leading to apnea (A) and hypopnea (B). Increasing effort to inspire (esophageal pressure) against the obstructed upper airway results in paradoxical chest and abdominal movement, which are indicated by the small vertical arrows. (*From* Strollo PJ, Rogers RM. Obstructive sleep apnea. Current concepts. N Engl J Med 1996;334:99–104, with permission.)

range of SaO_2 (eg, 89%–80%, 79%–70%, etc.). The cardiovascular manifestations of SaO2 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 ECG changes that are consistent with myocardial ischemia. If N-CPAP was used for part of the sleep period, then all of the above data will be reported with and without N-CPAP.

Effect of anesthetic drugs on airway patency in the adult obese OSA patient

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 [67–73]. The commonly-used anesthetic drugs that have been demonstrated to cause pharyngeal collapse are propofol [74], thiopental [75,76], narcotics [70,77,78], benzodiazepines [70,79–81], small doses of neuromuscular blockers [82–84], and nitrous oxide [85]. 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" [86]. Furthermore, if opioids cause airway obstruction, then the opioids may also cause a poor ventilatory response to the ensuing hypoxemia and hypercapnia [87].

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 [88]. High levels of pain result 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 [88,89]. 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 [90,91].

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 SpO_2 during sleep postoperatively than normal patients [92]. This consideration is highlighted by numerous reports of the need for rescue airway management in postoperative OSA patients [93–100].

Implications for airway management

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 nor sleep study

diagnosis 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 snorting and/or apnea during sleep and daytime sleepiness. The severity of these historical items correlates with the severity of sleep study–proven OSA [6,7,15,26,101]. 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/snorting/apnea and diurnal sleepiness [91,92,102]. Prediction of OSA is increased if there is a history of hypertension [64] or a neck circumference >40–42 cm [39,40,64]. 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 (see fig. 4).

If the anesthesiologist is the first care-giver to diagnose OSA, then it may sometimes 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. Alternatively, if general anesthesia is required, the patient could be treated as though severe OSA existed (see remainder of article). 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 may obviate the need for sedative and narcotic drugs both intra- and postoperatively.

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 [103–105]. 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 [68,106]. Second, a short thick neck is significantly related to difficult intubation [103,107]. Third, obesity [1,2,5–7] and a short thick neck [36–41,64] are significantly related to OSA and to each other [103]. Fourth, since excess pharyngeal tissue is deposited in the lateral walls of the pharynx of obese OSA patients [13,29–33], 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 had been found to be significantly related [108]. In fact, the strength of the relationship was such that the author felt that "all patients who have a trachea that is difficult to intubate should be regarded as having OSA until excluded by clinical features and, where doubt exist, sleep studies." Indeed, in one large series of patients undergoing surgery for OSA, the incidence of failed intubation was 5% [93]. 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 [109–111].

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 [68,71]. The danger of premedication in these patients is well illustrated by descriptions of several cases of complete airway obstruction [112–115]. Thus, proper preparation should depend on thorough topical and nerve block anesthesia of the upper airway [106,110]. Use of a flexible fiberscope through a rigid oropharyngeal conduit technique of intubation permits visualization of structures in an atraumatic manner [111,116].

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

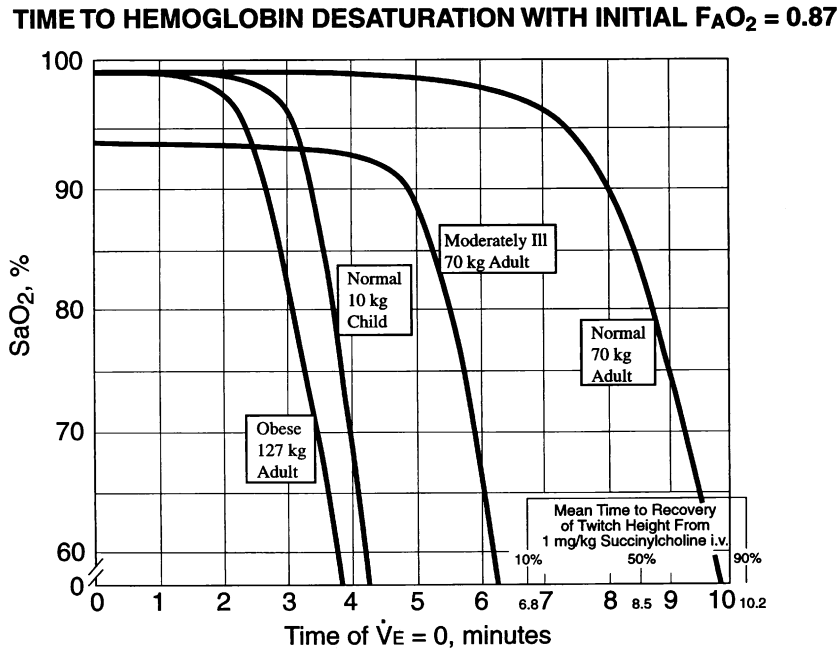

Fig. 8. SaO_2 versus time of apnea for various types of patients. The SaO_2 curves were produced by the computer apnea model of Reference [118]. An FaO_2 = 0.87 corresponds to an FIO_2 = 1.0. (*From* Benumof JL, Dagg R, Benumof R. Critical hemoglobin desaturation will occur before return to an unparalyzed state following 1 mg/kg intravenous succinylcholine. Anesthesiology 1997;87:979–82, with permission.)

(small oxygen reservoir) and high oxygen consumption desaturates much more rapidly during obstructive apnea compared with a normal patient [117,118] (Fig. 8).Maximal total body preoxygenation (filling of the alveolar, arterial, venous, and tissue spaces) requires that the patient breathe F_IO_2 = 1.0 for $\geq$ 3 minutes in a well sealed system [119]. Fig. 9 shows how decreasing initial FaO_2 (eg, 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 [117,118]. 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 [119,120]. 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 [121,122]. 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 [109–111].

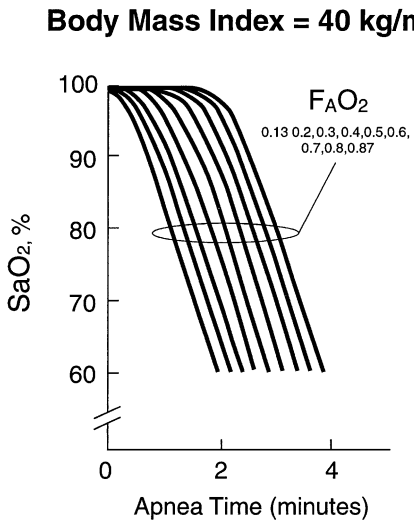

Fig. 9. SaO_2 versus time of apnea for a patient with a BMI = 40 kg/m^2 for various initial preapnea FaO_2. The family of curves was produced by the apnea model of Reference [118]. An FaO_2 = 0.87 (most right-hand curve) corresponds to an F_IO_2 = 1.0 and an $Fa0_2$ = 0.13 (most left-hand curve) corresponds to an F_IO_2 = 0.21. The FaO_2 = 0.87 desaturation curve (most right-hand curve) is the same as the desaturation curve for the 127-kg patient in Fig. 8. (*From* Benumof JL. Obstructive sleep apnea in the adult obese patient: implications for airway management. J Clin Anesth 2001;13(2): 144–56, with permission.)

Extubation: awake versus leaving the tube in

The risk of airway obstruction following extubation is increased in OSA patients [123]. The risk is further increased in OSA patients who have had nasal packing following nasal surgery [124]; therefore, packing around a nasopharyngeal airway (creating a central conduit for gas exchange) should be considered [69,91]. 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 [93]. Aside from the threat of death from airway obstruction, another great danger of spontaneous ventilation against an obstructed airway is rapid development of severe negative pressure pulmonary edema [93,125]. The treatment of negative pressure pulmonary edema in this setting usually requires re-intubation [125].

Depending on the mask ventilation and tracheal intubation experience at the beginning of the case, the length and type of surgery, and the severity of the OSA, one should consider leaving the patient intubated for a period of postoperative mechanical ventilation. 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. 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. 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. The patient should not have a high blood level of narcotic as indicated by a respiratory rate $< 12-14$ breaths/minute while the endotracheal tube is in situ. It is helpful for regional analgesia to be operative at the time of extubation. Extubation in the reverse Trendelenburg or semi-upright position minimizes compression of the diaphragm by abdominal contents [126].

When extubating, an oropharyngeal and/or a long nasopharyngeal airway (ie, 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. 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 [127]. If the patient does well initially, one should consider pneumatically splinting the oropharynx by applying N-CPAP, first with oxygen and then later with air [123,128]. Beyond the initial immediate application of N-CPAP, the F_IO_2 should only be increased if the SpO_2 is significantly decreased [94].

Opioid pain management: location of patient

Obese OSA patients have an increased risk of opioid-induced upper airway obstruction (even epidural and patient controlled analgesia may be problematic) [96,97] and is a reason why these patients may need a monitored care environment (ie, continuous electronic and frequent visual monitoring [respiratory rate,

sedation level, snoring]) [95]. Factors to be considered in this risk benefit analysis are the BMI of the patient, the AHI (ie, the severity of the OSA), the degree of associated cardiopulmonary disease, and the postoperative narcotic requirement. When all of these factors are mild, then the patient may reasonably go to a relatively unmonitored environment, and when any of these factors are severe, the patient should go to an ICU. The large gray zone in between these extremes requires careful judgment. Putting aside the one negative report involving epidural opioids in a OSA patient [96], the risk of opioid-induced airway obstruction may be avoidable by using alternative techniques, for example, regional block with local anesthesia to provide postoperative analgesia [129].

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 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 judgment and is a function of many anesthesia, medical, and surgical considerations.

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