Published in final edited form as:
Sleep Med Clin. 2013 March ; 8(1): 135–147.

Obesity Hypoventilation Syndrome and Anesthesia

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Keywords

Obesity hypoventilation syndrome; Perioperative management; Positive airway pressure

INTRODUCTION

Obesity is a global health concern. One of the complications associated with morbid obesity is obesity hypoventilation syndrome (OHS). OHS is defined by the triad of obesity (body mass index [BMI] ≥30 kg/m^2), daytime hypoventilation with hypercapnia (partial pressure of arterial carbon dioxide [Pa_cO_2] ≥45 mm Hg at sea level), and hypoxemia (partial pressure of arterial oxygen [Pa_{O2}] less than 70 mm Hg at sea level), and sleep-disordered breathing.[1] OHS is diagnosed after excluding other known causes of hypoventilation, such as severe obstructive or restrictive parenchymal lung disease, kyphoscoliosis, severe hypothyroidism, neuromuscular disease, and congenital central hypoventilation syndrome. In 90% of cases of OHS, the sleep-disordered breathing present is obstructive sleep apnea (OSA).[2] The prevalence of OHS in the general adult population is estimated to be 0.15% to 0.3%.[2] In patients undergoing bariatric surgery, approximately 8% present with OHS.[3]

Patients with OHS have a higher burden of comorbidities and increased risk for perioperative morbidity and mortality.[4-6] Therefore, a thorough plan of evaluation and management is essential for patients with OHS who undergo surgery. Currently, information on the perioperative management of OHS is extremely limited in the literature. As the prevalence of OHS is likely to increase as a result of the current global obesity epidemic, it is crucial for physicians to recognize and manage patients with this syndrome. This review examines the current data on OHS and discusses its optimal perioperative management.

PATHOPHYSIOLOGY

Daytime hypercapnia is a distinguishing feature in OHS and is entirely due to hypoventilation; a short course of noninvasive positive airway pressure (PAP) therapy (<2 weeks) improves hypercapnia without any significant changes in body weight, carbon dioxide (CO_2) production, or dead space volume.[7] There are currently 3 main hypotheses regarding the development of OHS: obesity-induced impairment in respiratory mechanics, leptin resistance, and impaired compensation for acute hypercapnia in OSA.[2,8]

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Obesity-Induced Impairment in Respiratory Mechanics

Obesity induces hypoventilation by increasing the mechanical load on the respiratory system, resulting in fatigue and weakness of the respiratory muscles.[9-11] In several studies, patients with OHS were shown to have higher BMI than eucapnic obese individuals.[12-15] However, because less than one third of morbidly obese individuals develop hypercapnia, other mechanisms may contribute to hypoventilation.[14-16]

Leptin Resistance

Leptin is a protein secreted specifically by adipocytes to regulate appetite and energy expenditure.[17-19] Leptin crosses the blood-brain barrier and exerts its effect by binding to leptin receptors in various areas of the brain.[18] In obese individuals, a higher level of leptin is found to be associated with an increase in ventilation to compensate for the increased CO_2 production by excess body mass.[17,20,21] Patients with OHS have an even higher serum leptin level than eucapnic individuals matched for BMI.[22,23] Although the precise relationship between leptin and OHS remains to be determined, it is speculated that leptin resistance may lead to central hypoventilation in OHS.

Impaired Compensation of Acute Hypercapnia in OSA

Hypoventilation during sleep secondary to obstructive apneas and hypopneas results in transient episodes of acute hypercapnia and serum bicarbonate (HCO_3^-) retention. Eucapnic patients with OSA present several compensatory mechanisms to maintain acid-base homeostasis. During sleep, they hyperventilate between periods of apnea.[24] In addition, during wakefulness in daytime, acute hypercapnia is corrected and the excess HCO_3^- is excreted. On the other hand, patients with OHS have a reduced duration of ventilation between periods of apnea while sleeping.[25] The resulting acute hypercapnia persists during wakefulness and HCO_3^- retention occurs, causing gradual adaptation by chemoreceptors and further blunting of ventilatory CO_2 responsiveness. In a computer model, when both CO_2 response and the rate of renal HCO_3^- excretion was abnormally low, an increase in awake Pa_cO_2 and HCO_3^- developed over multiple days.[26]

DISTINGUISHING CLINICAL FEATURES

Compared with eucapnic obese individuals with and without OSA, patients with OHS demonstrate more severe upper airway obstruction, restrictive pulmonary physiology, blunted central respiratory drive, and increased incidence of pulmonary hypertension. Patients with OHS display increased upper airway resistance in both the sitting and supine position in comparison with obese individuals with eucapnia.[27] In perioperative settings, patients with OHS are at increased risk of life-threatening apneic events because sedatives and narcotics increase the collapsibility of the upper airway and attenuate respiratory drive.[28,29]

Spirometric values from morbidly obese patients typically reveal a restrictive pattern with a reduction in forced expiratory volume in the first second (FEV_1) and forced vital capacity (FVC) but normal FEV_1/FVC ratio. This restrictive pulmonary physiology is further impaired in OHS.[3] Chest wall compliance is reduced and respiratory resistance is increased, likely secondary to the reduction in functional residual capacity and expiratory reserve volume. As a result, the work of breathing in OHS patients is twice that of obese eucapnic individuals[30,31] and increases further when these patients are positioned supine from sitting as a result of the cephaled shift of abdominal contents.[30,32]

In contrast to obese eucapnic individuals who possess a substantially increased central respiratory drive,[32] patients with OHS have a blunted central respiratory drive to both

hypercapnia and hypoxia. They do not hyperventilate to the same extent as obese eucapnic individuals when forced to rebreathe CO_2[33-35] or breathe a hypoxic gas mixture.[35]

The prevalence of pulmonary hypertension in patients with OHS is high, ranging from 30% to 88%.[4,36-38] Seventy-seven percent of patients with OHS with respiratory failure in the intensive care unit have moderate to severe pulmonary hypertension (pulmonary systolic pressure >45 mm Hg).[37] The cause of pulmonary hypertension is likely secondary to chronic alveolar hypoxia and hypercapnia. In morbidly obese patients, left-heart failure is not uncommon and may increase pulmonary arterial pressure.[39]

MORBIDITY AND MORTALITY

Obesity and OSA are associated with a spectrum of comorbidities such as coronary artery disease, heart failure, stroke, and metabolic syndrome, which result in increased morbidity and mortality.[40-44] Furthermore, patients with OSA are at increased risk of developing postoperative complications including arrhythmias and hypoxemia.[45-47] An increased risk of transfer to the intensive care unit and increased length of hospital stay were also observed among patients with OSA who underwent noncardiac surgery.[46]

Several studies have shown that patients with OHS may experience higher morbidity and mortality than patients who are similarly obese and have OSA. Compared with obese individuals with eucapnia, patients with OHS were more likely to develop heart failure (odds ratio [OR] 9, 95% confidence interval [CI] 2.3–35), angina pectoris (OR 9, 95% CI 1.4–57.1), and cor pulmonale (OR 9, 95% CI 1.4–57.1).[4] They also received higher rates of long-term care at discharge (19% vs 2%, $P = .01$), and invasive mechanical ventilation (6% vs 0%, P 5 .01).[48]

Hospitalized patients with untreated OHS had a high mortality rate of 46% during a 50-month follow-up period after discharge.[49] In addition, their mortality rate is higher compared with obese eucapnic patients after hospital discharge at 18 months (23% vs 9%).[48] In patients undergoing open bariatric surgery, those with either OHS or OSA suffered a surgical mortality rate of 4%, significantly higher than those without the disease (0.2%, $P<.01$).[50] In patients with OHS with additional risk factors (previous history of venous thromboembolism, BMI ≥50 kg/m^2, male sex, hypertension and age ≥45 years) undergoing bariatric surgery, mortality ranges between 2% and 8%.[5,6,51]

In summary, patients with OHS experience higher morbidity and mortality than those with eucapnia who are obese. Previous history of venous thromboembolism, morbid obesity, male sex, hypertension, increasing age, and noncompliance with PAP treatment may further increase mortality risk. The surgical mortality rate in high-risk patients with OHS undergoing bariatric surgery is between 2% and 8%.

TREATMENT

Therapeutic interventions for OHS include 4 main components: PAP therapy, supplemental oxygen, bariatric surgery, and pharmacologic respiratory stimulants.

PAP Therapy

The 2 main forms of PAP therapy currently being used are continuous positive airway pressure (CPAP) and bilevel PAP. The overall short-term and long-term benefits were summarized in a recent systematic review.[3]

Short-term benefits of PAP include an improvement in gas exchange and sleep-disordered breathing. A short course (≤3 weeks) of PAP results in a significant decrease in Pa_cO_2 and

an increase in Pa_{O_2}.[49,52-54] Furthermore, sleep study parameters, including the apnea-hypopnea index (AHI) and oxygen saturation during sleep, were reported to be significantly improved.[53-56]

Long-term benefits of PAP include an improvement in pulmonary function and central respiratory drive to CO_2. A course of PAP therapy for 24 to 48 weeks significantly increased FEV_1 and FVC.[57-59] The effect of PAP on central respiratory drive, measured as the change in minute ventilation per unit change in end-tidal CO_2, was reported in several studies to be favorable.[52,58,60]

PAP may also reduce mortality in OHS. Two retrospective studies have reported a mortality rate of 13% to 19% in patients with OHS on PAP throughout a mean period of 4 years.[57,61] Through indirect comparison, this mortality rate is lower than the 23% mortality rate reported in patients with untreated OHS at 18 months of follow-up.[48]

CPAP failure, defined by a residual AHI of 5 or more or a mean nocturnal pulse oximeter oxygen saturation (SpO_2) less than 90%, has been reported.[62] A recent prospective randomized study compared the long-term efficacy of bilevel PAP versus CPAP.[63] Two groups of 18 patients with OHS who underwent successful CPAP titration were randomized to either bilevel PAP or CPAP for 3 months. Both groups experienced a similar degree of improvement in Pa_CO_2 and daytime sleepiness. Overall, bilevel PAP was not considerably superior to CPAP if CPAP titration was successful. However, if CPAP titration is unsuccessful, bilevel PAP should be strongly considered and treatment should be individualized to each patient.[2] Bilevel PAP should be instituted if the patient is intolerant of higher CPAP pressure (>15 cm H_2O) or if hypoxemia persists despite adequate resolution of obstructive respiratory events.[64] A therapeutic algorithm for CPAP titration in OHS patients is shown in Fig. 1.

A new treatment modality in patients with OHS is average volume-assured pressure-support (AVAPS) ventilation. This mode of PAP therapy ensures the delivery of a preset tidal volume during bilevel PAP mode. The expiratory tidal volume and leak are estimated based on pneumotacographic inspiratory and expiratory flows. Target tidal volume is typically set at 8 to 10 mL/kg of ideal body weight. The expiratory PAP is set to resolve upper airway obstruction and the inspiratory PAP is automatically adjusted to achieve the targeted tidal volume. This mode of PAP also provides a backup rate to alleviate central apneas that may emerge during PAP therapy.[65] In 1 clinical trial, AVAPS was more effective in improving nocturnal hypoventilation compared with CPAP and bilevel PAP in ST mode (activated backup rate). However, the degree of improvement in daytime Pa_CO_2 did not reach statistical significance.[66] In a recent randomized controlled trial of patients with OHS, there was no significant difference between AVAPS and bilevel PAP ST mode in the degree of improvement in daytime and nocturnal gas exchange.[67] However, in this study, those randomized to bilevel PAP ST mode underwent aggressive bilevel PAP titration focusing on achieving adequate tidal volumes (mean inspiratory PAP of 23 cm H_2O and mean expiratory PAP of 10 cm H_2O), as recommended by the American Academy of Sleep Medicine guidelines on noninvasive ventilation.[68]

In summary, PAP therapy improves gas exchange, sleep-disordered breathing, lung function, and central respiratory drive to CO_2. Long-term PAP therapy also lowers the mortality rate in patients with OHS. Because of its noninvasiveness and effectiveness, PAP is the first-line therapy for OHS. CPAP is usually the initials modality of choice because of its relative simplicity and low cost. However, if CPAP titration fails, bilevel PAP or AVAPS should be applied. A need for a backup rate should be strongly considered because central apneas occur commonly in patients with OHS undergoing PAP therapy.

Supplemental Oxygen

Approximately 20% to 30% of patients with OHS continue to desaturate to SpO_2 less than 90% during sleep while on adequate CPAP or bilevel PAP settings, thereby requiring supplemental oxygen.[55] Administration of high-concentration supplemental oxygen without PAP therapy may induce hypoventilation and worsen hyercapnia.[69,70] In a recent study, a significant decrease in minute ventilation, resulting in an increase in transcutaneous CO_2 tension by 5 mm Hg, was found in patients with OHS while breathing 100% oxygen compared with those breathing room air.[71] Therefore, clinicians should administer the lowest concentration of oxygen to patients with OHS to avoid worsening of hypercapnia while maintaining optimized oxygenation, particularly in patients with OHS experiencing an exacerbation or recovering from sedatives/narcotics or general anesthesia.[72]

Bariatric Surgery

Bariatric surgery is a mainstay treatment of obesity, especially for morbidly obese patients in whom more conservative approaches have failed or who have developed comorbidities. Bariatric surgery improves gas exchange and lung function in OHS. At 1 year after surgery, Pa_{O_2}, Pa_CO_2, FEV_1, and FVC all improved significantly.[50,73] To better understand the effect of surgical weight loss on OSA, Greenburg and colleagues[74] performed a meta-analysis of 12 studies including a total of 342 patients in whom polysomnography before and after maximum weight loss were available. They found that bariatric surgery led to significant weight loss with a mean reduction in BMI from 55.3 kg/m^2 to 37.7 kg/m^2. This robust weight loss was accompanied by a reduction in the AHI from baseline values of 55 events/h (95% CI 49–60) to 16 events/h (95% CI 13–19). However, many of these patients (62%) had persistent OSA of moderate severity (AHI ≥15 events/h). Thus, although improvements should be anticipated, OSA does not resolve in all patients after surgically achieved weight loss. CPAP therapy could still benefit patients with residual OSA after maximum weight loss. Similarly, 14% of patients with OHS still require PAP therapy after weight loss.[73] Therefore, patients with OHS should undergo reevaluation after bariatric surgery before discontinuing PAP therapy. As patients age and/or regain some weight over the years, the severity of OSA can increase.

Bariatric surgery is associated with significant risk. The overall perioperative mortality ranges between 0.5% and 1.5%.[75,76] The presence of OSA and extreme preoperative weight are independent risk factors associated with perioperative death and adverse events including venous thromboembolism, surgical reintervention, and prolonged hospital stay.[76,77] Ideally, PAP should be initiated in all patients with OHS and bariatric surgery should be considered as a second-line intervention.

Pharmacotherapy

Medications that increase respiratory drive have been investigated for the treatment of OHS. Limited evidence is available for 2 respiratory stimulants: medroxyprogesterone acetate and acetazolamide.

Medroxyprogesterone acetate stimulates respiration at the hypothalamic level.[78] Its role in OHS is uncertain. An early study reported an increase in Pa_{O_2} and a decrease in Pa_CO_2 in patients with OHS treated with medroxyprogesterone acetate.[79] However, a later study did not demonstrate the same benefits.[7] Because medroxyprogesterone acetate increases the risk of venous thromboembolism,[80] administration to patients with OHS whose mobility is limited may be unsafe.

Acetazolamide is a carbonic anhydrase inhibitor that increases minute ventilation by inducing metabolic acidosis through increased excretion of bicarbonate by the kidneys.

Acetazolamide has been shown to improve AHI, increase Pa_{O_2}, and reduce Pa_cO_2 in patients with OSA.[81,82] More recently, in mechanically ventilated patients with OHS, acetazolamide reduced plasma HCO_3^- and increased hypercapnic drive response.[83] Given the limited data on pharmacotherapy and because it is not widely used, the authors do not recommend it as a mainstay therapy but rather an adjunctive therapy in patients with OHS who remain hypercapnic despite adequate adherence to optimally titrated PAP therapy. Specifically, OHS patients requiring high doses of loop diuretics, which can lead to further HCO_3^- retention, may be ideal candidates for acetazolamide. Caution should be exercised in prescribing a respiratory stimulant in patients with ventilatory limitation because it can lead to exacerbation of acidosis and worsening of dyspnea. Acetazolamide should not be prescribed as a respiratory stimulant if a patient cannot normalize or near normalize their Pa_cO_2 (or endtidal CO_2) levels with 1 to 2 minutes of voluntary hyperventilation.

PREOPERATIVE ASSESSMENT OF PATIENTS WITH OHS

The 3 main challenges in OHS are OSA, obesity, and hypoventilation (hypercapnia and hypoxemia). For a patient with suspected OHS presenting for elective surgery, the preoperative assessment should begin with the history and physical examination directed to identify comorbidities in OHS, including coronary artery disease, congestive heart failure, pulmonary hypertension, and diabetes mellitus. A detailed examination of the airway and sites for venous access should also be performed. Further laboratory and imaging investigations should be focused on screening for sleep-disordered breathing and stratification of surgical risk. An algorithm for the perioperative evaluation and management of OHS is given in Fig. 2.

Preoperative Screening for OHS

OHS is often undiagnosed and may increase perioperative risk. Appropriate screening facilitates the identification of patients at risk for OHS, referral to sleep medicine for PAP therapy, and modifications in the surgical approach, anesthetic technique, and postoperative monitoring.

The definitive test for alveolar hypoventilation is an arterial blood gas performed on room air during wakefulness. As this is a relatively invasive procedure, several screening tools have been proposed. Mokhlesi and colleagues[15] suggested 3 clinical predictors of OHS: serum HCO_3^-, AHI, and lowest oxygen saturation during sleep. Increased serum HCO_3^- level caused by metabolic compensation of chronic respiratory acidosis is common in patients with OHS. In a cohort of obese patients with OSA referred to the sleep laboratory for suspicion of OSA, a serum HCO_3^- threshold of 27 mEq/L demonstrated a 92% sensitivity in predicting hypercapnia on arterial blood gas.[15] To complement the highly sensitive serum HCO_3^-, a highly specific (95%) AHI threshold of 100 was identified. A 2-step screening process was proposed, with serum HCO_3^- as the initial test to exclude patients without OHS and then AHI asthe second test to improve specificity. In addition, hypoxemia (Sp_{O_2} <90%, corresponding to Pa_{O_2} <60 mm Hg)[84] during wakefulness should lead clinicians to suspect OHS in patients with OSA. In a recent meta-analysis, patients with OSA and higher BMI, higher AHI, and more restrictive chest wall mechanics were more likely to develop OHS.[85] In these patients with OHS, the mean BMI, AHI, percent predicted FEV_1, and percent predicted FVC were 39 kg/m^2, 64 events/h, 71%, and 85%, respectively.

In summary, patients presenting with a high BMI and AHI should alert the physician to screen for OHS. The serum HCO_3^- is an easy initial screening test. If it is increased or hypoxemia by room air Sp_{O_2} during wakefulness is present, a measurement of arterial blood gases, or end-tidal CO_2, is recommended. Once hypercapnia is confirmed, referral to sleep medicine and further testing, such as pulmonary function testing, chest imaging,

measurement of thyroid-stimulating hormone level, and clinical assessment of neuromuscular strength, should be considered to rule out other important causes of hypoventilation.

Preoperative Screening for OSA

OSA screening in patients suspected to have OHS provides valuable information that may modify perioperative management. Approximately 90% of patients with OHS present with OSA, therefore a positive screen increases the index of suspicion for OHS. Multiple screening tools have been developed to evaluate patients at risk for OSA. The STOP-Bang questionnaire was used in preoperative patients.[86,87] It is a scoring model combining the STOP (snoring, tiredness, observed apneas, and increased blood pressure) questionnaire and Bang (BMI ≥35 kg/m^2, age >50 years, neck circumference >40 cm, and male gender). A systematic review has suggested using the STOP-Bang questionnaire in the surgical population due to its high methodological quality and easy-to-use features.[88] A positive screen (≥3 questions answered yes) is highly sensitive for moderate to severe OSA and is useful to exclude patients with the disease. On the other hand, patients with an STOP-Bang score of 5 to 8 have been shown to be at higher risk for moderate to severe OSA.[89] If these patients present with concurrent morbid obesity, they are at high risk for OHS and should be referred to sleep medicine for further evaluation.

In summary, the STOP-Bang questionnaire is a validated and easy tool to screen for OSA as part of the preoperative evaluation for patients with suspected OHS. A high STOP-Bang score with coexisting morbid obesity indicates an increased risk for OHS.

Preoperative Risk Stratification and Cardiopulmonary Testing

The Lee revised cardiac risk index represents a valuable tool to predict cardiac risk for elective major noncardiac surgery in the general population.[90] However, other risk factors specifically related to OHS, such as pulmonary hypertension and a history of venous thromboembolism, should be considered when evaluating perioperative risk. The Obesity Surgery Mortality Risk Score was developed for patients undergoing gastric bypass, and includes 5 risk factors: hypertension, BMI of 50 kg/m^2 or greater, male sex, age 45 years or more, and known risk factors for pulmonary embolism (OHS, previous thromboembolism, preoperative vena cava filter, pulmonary hypertension).[5,6,51] This risk score stratifies mortality risk into low (0 or 1 comorbidity), intermediate (2 to 3 comorbidities) and high (4 to 5 comorbidities). Mortality rates were 0.2%, 1.2%, and 2.4% for low-risk, intermediate-risk, and high-risk classes, respectively.[6] The most common causes of death were pulmonary embolism (30%), cardiac causes (27%), and gastrointestinal leak (21%).

A 12-lead electrocardiogram and chest radiograph should be obtained in patients suspected to have OHS to evaluate for coronary artery disease, congestive heart failure, and pulmonary hypertension. Indications for further cardiovascular testing should be based on the patient's cardiovascular risk factors and the invasiveness of surgery according to current American Heart Association guidelines.[90,91] The assessment of functional capacity is of particular importance in obese individuals because cardiorespiratory fitness levels and the postoperative complication rate are inversely related to BMI.[92,93] If these patients are undergoing major surgery and present with multiple cardiac risk factors, stress testing and transthoracic echocardiogram may be considered if management may be changed.[91]

Studies evaluating postoperative pulmonary complications have generally found no increased risk attributable to obesity.[94] However, patients with OSA were found to have a higher risk of pulmonary complications than patients without OSA in a recent retrospective study.[95] Routine pulmonary function tests may not translate into an effective risk prediction

for postoperative pulmonary complications in noncardiothoracic surgery.[96] However, if coexisting chronic obstructive pulmonary disease is suspected in the patient with OHS, spirometry may be considered for diagnosis and subsequent optimization.

INTRAOPERATIVE MANAGEMENT OF OHS

Key considerations specific to the intraoperative management of OHS include airway management and emergence from anesthesia.

Airway Management

OSA is a risk factor for both difficult mask ventilation and tracheal intubation.[97] In addition, patients with severe OSA (AHI ≥40 events/h) showed a significantly higher prevalence of difficult intubation than patients with lower AHI.[98]

Obesity results in a threefold increase in difficulty with mask ventilation.[99] Whether obesity increases the difficulty of tracheal intubation is more controversial. A retrospective study of 18,500 surgical patients reported that obesity is a risk factor for difficult intubation.[100] However, other studies have not found an association between BMI and intubation difficulties.[101,102] More recently, Kheterpal and colleagues[103] identified 5 risk factors (limited mandibular protrusion, thick/obese neck anatomy, OSA, snoring, and BMI more than 30 kg/m^2) as independent predictors of difficult mask ventilation and intubation during anesthesia induction, which suggests that patients with OHS with limited mandibular protrusion are in the highest risk group for airway complications.

During induction of anesthesia, patients with OHS should be placed in the ramp position with tilting of the torso and head by 25°. This position has been shown to improve the glottic view during intubation and reduce atelectasis.[104] Preoxygenation for more than 3 minutes with a tightly fitted mask can increase apnea tolerance time. A variety of airway adjuncts and skilled anesthesiology assistance should be made available in advance. Awake fiber optic intubation should be considered in patients with OHS with markers for difficult mask ventilation and intubation. In situations during which a patient with OHS is hypoxemic, concomitant use of PAP during fiber optic intubation prevents further deterioration of oxygen saturation.[105,106] In addition, PAP splints the airway open and thus facilitates the visualization of anatomic landmarks.[107]

Emergence from Anesthesia

Patients with OHS are sensitive to the respiratory depressant effects of anesthetic agents due to the propensity for airway collapse, sleep deprivation, and blunting of physiologic response to hypercapnia and hypoxemia. A semi-upright or lateral position is recommended at the end of surgery for better oxygenation and airway maintenance.[108] Rapid emergence from anesthesia is preferred because tracheal extubation should be performed only after the patient is fully conscious. A systematic analysis of the literature comparing postoperative recovery after propofol, isoflurane, desflurane, and sevoflurane-based anesthesia in adults demonstrated that early recovery was faster in the desflurane and sevoflurane groups.[109] Another strategy to accelerate emergence is to decrease volatile anesthetic requirement and minimize washout time from fat/muscle by using other short-acting anesthetic adjuvants, such as remifentanil, or a combined general regional anesthetic.[29]

POSTOPERATIVE MANAGEMENT OF OHS

Key considerations specific to the postoperative management of OHS include monitoring for opioid-induced ventilatory impairment (OIVI) and prompt use of PAP therapy. The dual

roles of postoperative PAP therapy are to prevent and treat respiratory failure secondary to sleep-disordered breathing.

OIVI

OIVI induces central respiratory depression, decreased level of consciousness, and upper airway obstruction, ultimately resulting in alveolar ventilation. The incidence of OIVI after major surgery varies with the different routes of opioid administration. The incidence of decreased respiratory rate was 0.8%, 1.2%, and 1.1% for intramuscular, intravenous patient-controlled analgesia, and epidural analgesia, respectively.[110] The incidence of oxygen desaturation was 37%, 11.5%, and 15.1% for intramuscular, intravenous patient-controlled analgesia, and epidural analgesia, respectively.[110] Patients with OHS could be at significant risk for OIVI because of their susceptibility to upper airway obstruction, depressed central respiratory drive, and impaired pulmonary mechanics. An opioid-sparing analgesic regimen, including local anesthetic–infused nerve block catheters and nonopioid adjuncts (acetaminophen, nonsteroidal antiinflammatory drugs), should be considered in these patients.

Improved postoperative monitoring is key in reducing the risk of OIVI. Patient-specific, anesthetic, and surgical factors determine the requirements for postoperative monitoring. Patients with OHS undergoing major surgery who require high doses of postoperative opioid should be monitored with continuous oximetry. Recurrent respiratory events in the postanesthesia care unit, including apnea for 10 seconds or more, bradypnea of less than 8 breaths/min, pain-sedation mismatch, or desaturations to less than 90%, can be used to identify patients at high risk of postoperative respiratory complications.[111] Recently, Macintyre and colleagues[112] proposed that sedation level is a more reliable sign of OIVI than respiratory rate because multiple reports suggest that OIVI is not always accompanied by a decrease in respiratory rate.[112-114] Thus, sedation scoring systems should be used postoperatively to recognize OIVI so that appropriate interventions are triggered. In patients with OHS requiring high doses of postoperative opioids, sedation monitoring should be considered every 1 to 2 hours for the first 24 hours.[115]

Postoperative PAP Therapy: Prevention of Respiratory Failure

There is limited evidence demonstrating a reduction in postoperative complications with PAP in patients with OHS. However, a case series of 14 patients with OSA suggested that the use of CPAP continuously for 24 to 48 hours after extubation may reduce the risk of postoperative complications.[116] In addition, PAP was found to decrease respiratory failure after extubation in severely obese patients admitted to the intensive care unit (absolute risk reduction of 16%).[117] Subgroup analysis of patients with hypercapnia showed reduced hospital mortality in the PAP group compared with the control group. Other potential benefits of postoperative PAP include reduced hemodynamic fluctuations and arrhythmia related to hypoxemia.

In summary, patients with OHS who were previously on PAP should resume therapy as soon as possible postoperatively. In patients suspected to have OHS experiencing postoperative ventilatory impairment, PAP should be considered. Based on the available literature, patients with OHS typically require an inspiratory PAP and the expiratory PAP of 16 to 22 cm H_2O and 9 to 10 cm H_2O, respectively, to achieve adequate resolution of upper airway obstruction and to improve ventilation. Bilevel PAP can be empirically set at these pressures in patients suspected of having OHS.

Postoperative PAP Therapy: Treatment of Respiratory Failure

Although the incidence of postoperative respiratory failure in patients with OHS is unknown, these patients are particularly susceptible to cardiopulmonary complications secondary to increased respiratory load, blunted central drive, pulmonary hypertension, and impaired ventricular function. In the postoperative period, these patients may decompensate acutely due to multiple factors, including sedation, sleep deprivation, and deconditioning.[118] Of concern, misdiagnosis is common if the physician is not aware of the potential for sleep-disordered breathing causing acute cardiopulmonary failure.[37,118] It was reported that 77% of patients with OHS admitted to the intensive care unit for hypercapnic respiratory failure were erroneously diagnosed and treated for chronic obstructive pulmonary disease/asthma.[37]

Four presentations of acute cardiopulmonary failure may be encountered postoperatively: hypercapnic respiratory failure, acute congestive heart failure, acute cor pulmonale, and sudden death, an extreme manifestation. The mechanisms leading to the development of such complications were described by Carr and colleagues[118] in detail. A high index of suspicion and early initiation of PAP therapy are key in managing patients with suspected OHS who develop respiratory failure postoperatively. Adjunctive interventions include judicious sedation/analgesia, minimal sleep disruption at night, and close follow-up with a sleep specialist.

SUMMARY

OHS is an important disease entity that requires the anesthesiologist's thorough understanding. The prevalence of OHS is estimated to be 0.15% to 0.3% in the general population and 8% in patients undergoing bariatric surgery.[2,3] Patients with OHS present with severe upper airway obstruction, restrictive pulmonary physiology, blunted central respiratory drive, and pulmonary hypertension. The primary therapy for OHS is PAP.

Perioperative management begins with a high index of suspicion for OHS in the morbidly obese patient. Screening questionnaires such as the validated STOP-Bang questionnaire can identify patients at high risk of OSA. This screening tool can be further complemented by the presence of low Sp_{O_2}, increased end-tidal CO_2 or Pa_cO_2, and serum HCO_3^- level to identify patients at high risk of OHS. Before major elective surgery, these patients should be referred to sleep medicine for polysomnography and PAP titration. An echocardiogram should be considered to assess right ventricular function and pulmonary hypertension. Perioperative precautions for OHS include prudent airway management, rapid emergence, monitoring for ventilatory impairment, and early resumption of PAP therapy. Future research should focus on the perioperative strategies of screening, monitoring, and treatment of OHS and associated complications.

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KEY POINTS

- Obesity hypoventilation syndrome (OHS) is an important disease entity that requires the anesthesiologist's thorough understanding.
- Patients with OHS present with severe upper airway obstruction, restrictive pulmonary physiology, blunted central respiratory drive, and pulmonary hypertension. The primary therapy for OHS is positive airway pressure (PAP) therapy.
- Screening questionnaires such as the validated STOP-Bang questionnaire can identify patients at high risk of obstructive sleep apnea.
- Before major elective surgery, these patients should be referred to sleep medicine for polysomnography and PAP titration.
- Future research should focus on the perioperative strategies of screening, monitoring, and treatment of OHS and associated complications.

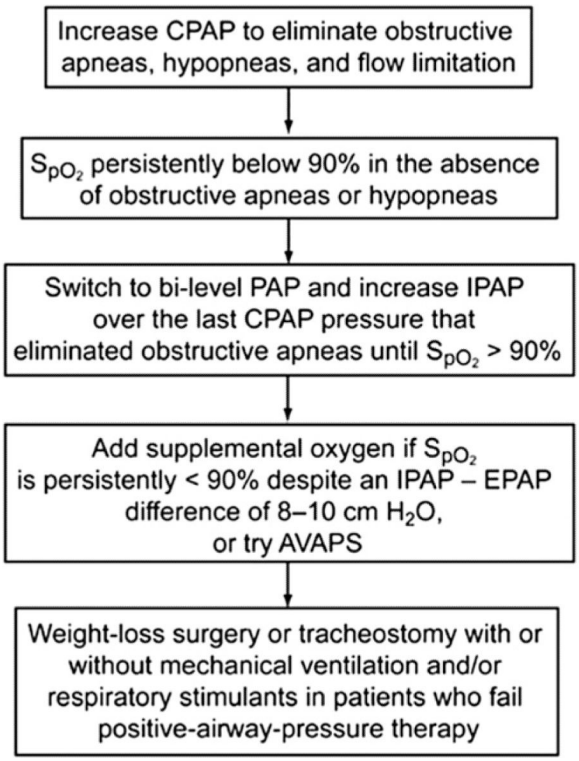

Fig. 1.

Algorithm for continuous PAP titration in patients with OHS. AVAPS, average volume-assured pressure-support ventilation; CPAP, continuous positive airway pressure; EPAP, expiratory positive airway pressure; IPAP, inspiratory positive airway pressure.

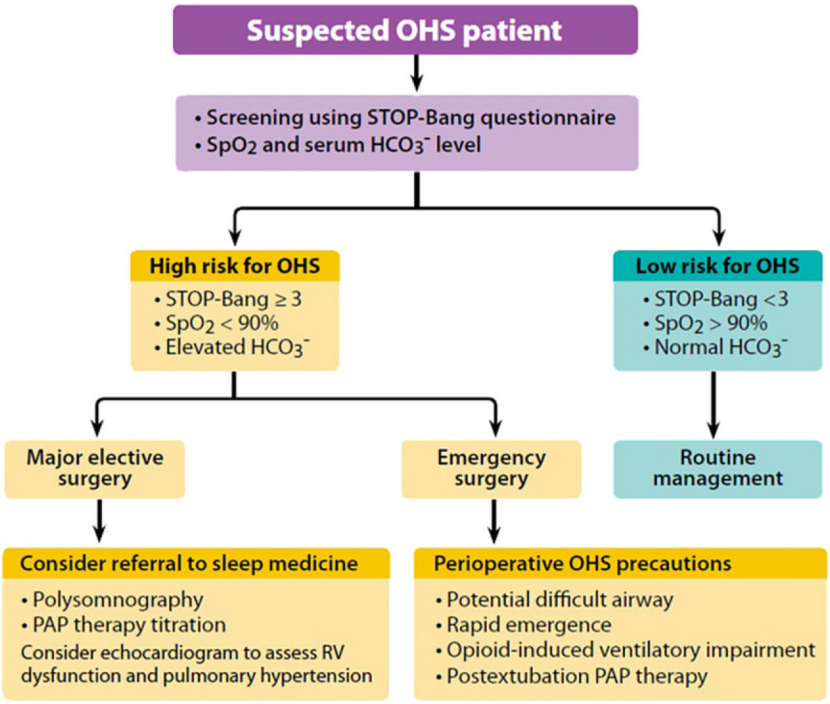

Fig. 2.

Perioperative management of the patient suspected to have OHS. HCO_3^-, serum bicarbonate; RV, right ventricular. (*Adapted from* Mokhlesi B. Obesity hypoventilation syndrome: a state-of-the-art review. Respir Care 2010;55:1347–62; with permission.)