



Obesity hypoventilation syndrome: a current review

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INTRODUCTION

Obesity hypoventilation syndrome (OHS) is defined as the presence of obesity and daytime hypoventilation ($\text{PaCO}_2 \geq 45$ mmHg) in patients without central, pulmonary, neuromuscular, metabolic, or chest wall disease that explains the hypercapnia.⁽¹⁾ Therefore, OHS is a diagnosis of exclusion, and other causes of hypercapnia should be investigated. Obesity is the hallmark of the disease, there being a correlation between body mass index (BMI) and disease prevalence.⁽¹⁻⁵⁾ The identification of OHS is important because of the possibility of clinical exacerbation leading to respiratory failure and the high mortality rate in untreated patients. OHS is accompanied by obstructive sleep apnea (OSA) in more than 90% of cases, and both share the same major risk factor, that is, obesity; however, the presence of OSA is not necessary for the diagnosis of OHS. This explains why polysomnography is not necessary for this diagnosis. Signs of right heart failure can be present in OHS and are secondary to chronic hypoxemia and pulmonary hypertension, both of which can accompany the clinical picture. In addition, arterial hypertension and insulin resistance are more prevalent in patients with OHS than in obese individuals without OHS.⁽¹⁻³⁾

Since OHS is associated with high morbidity and mortality,⁽⁵⁻⁷⁾ the objective of the present study was to conduct a current review of the epidemiology, pathophysiology, and treatment of OHS.

ABSTRACT

Obesity hypoventilation syndrome (OHS) is defined as the presence of obesity (body mass index ≥ 30 kg/m²) and daytime arterial hypercapnia ($\text{PaCO}_2 \geq 45$ mmHg) in the absence of other causes of hypoventilation. OHS is often overlooked and confused with other conditions associated with hypoventilation, particularly COPD. The recognition of OHS is important because of its high prevalence and the fact that, if left untreated, it is associated with high morbidity and mortality. In the present review, we address recent advances in the pathophysiology and management of OHS, the usefulness of determination of venous bicarbonate in screening for OHS, and diagnostic criteria for OHS that eliminate the need for polysomnography. In addition, we review advances in the treatment of OHS, including behavioral measures, and recent studies comparing the efficacy of continuous positive airway pressure with that of noninvasive ventilation.

Keywords: Obesity; Obesity hypoventilation syndrome; Noninvasive ventilation.

HISTORY

Obesity-related sleepiness was described in 1889, even prior to the recognition of OSA.⁽⁸⁾ Bickelmann et al. published a case report in 1956⁽³⁾ and popularized the term "Pickwickian syndrome" (an eponym that has fallen into disuse) in a reference to the character Fat Boy Joe from Charles Dickens's "The Posthumous Papers of the Pickwick Club," who was always sleepy and hungry and would often fall asleep on the job any time during the day.⁽⁹⁾ The patient reported by Bickelmann et al.⁽³⁾ had daytime hypoventilation, chronic hypoxemia, polycythemia, and pulmonary hypertension, with evidence of *cor pulmonale*. Several studies have since characterized the epidemiology, clinical picture, and pathophysiology of OHS.^(1,2,4,7) Since 1999, the American Academy of Sleep Medicine has defined the diagnostic criteria for OHS.^(10,11)

EPIDEMIOLOGY

The prevalence of OHS is unknown because of the lack of population-based studies. The prevalence of OHS is estimated to be 10-20% in patients with OSA^(7,12-16) and is estimated to be even higher in extremely obese patients.^(7,14) Mokhlesi et al.⁽⁷⁾ evaluated a population in the USA referred to a sleep medicine center for suspicion of OSA—180 patients were retrospectively selected, and 410 patients were prospectively selected. Of the patients diagnosed with OSA in the retrospective and prospective samples, 30% and 20%, respectively, met

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the criteria for OHS, and those percentages increased with increasing BMI. Laaban et al.⁽¹⁴⁾ retrospectively evaluated patients receiving home treatment for OSA in France. The sample included 1,114 adults, of whom approximately 10% met the diagnostic criteria for OHS, and a positive association was also found with increasing BMI.⁽¹⁴⁾ Akashiba et al.⁽¹²⁾ evaluated 611 patients in Japan referred to sleep medicine centers for OSA and diagnosed OHS in 9% of the patients. The patients with OHS were younger, were more obese, and had more severe OSA when compared with those without OHS. In a different approach, Kessler et al.⁽¹⁷⁾ evaluated patients with OHS and detected OSA in most of the patients (90%); in addition, OHS patients with OSA were found to have poorer gas exchange and poorer pulmonary hemodynamics than did those without OSA.

Seeking to determine the prevalence and, consequently, the degree of underdiagnosis of OHS, Nowbar et al.⁽⁵⁾ conducted a study involving obese patients admitted to internal medicine services for any cause. Of 29 obese inpatients with a BMI > 50 kg/m², 14 (48%) were diagnosed with OHS. In the same study, 31% of 150 obese inpatients did not have a previous diagnosis of OHS, although they met the criteria for this diagnosis.⁽⁵⁾

Because of the lack of studies on the prevalence of OHS in the general population, an exercise on epidemiological correlations has been repeatedly cited. Mokhlesi⁽¹⁸⁾ infers that if approximately 3% of the general population in the USA are severely obese (BMI > 40 kg/m²), half of those individuals would have OSA. Considering, therefore, the estimate that 10-20% of severely obese patients with OSA would have OHS, a conservative estimate indicates a prevalence of OHS of 0.15-0.30% in the general population in the USA (ranging approximately from 1:300 to 1:600 adults).⁽¹⁸⁾

MORBIDITY AND MORTALITY

Patients with OHS use more health care resources in the period prior to the diagnosis than do obese individuals without OHS or the general population.⁽¹⁹⁾ Obesity *per se* leads to a greater likelihood of diseases

such as systemic arterial hypertension, diabetes, dyslipidemia, and hypothyroidism. Comorbidities such as heart failure, coronary artery disease, and *cor pulmonale* are more common in patients with OHS, and the likelihood that such patients will require invasive mechanical ventilation or ICU admission is also increased.^(5,20) In addition, pulmonary hypertension is more common (50% vs. 15%) and more severe in patients with OHS than in patients with OSA.^(16,21,22)

Berg et al.⁽¹⁹⁾ conducted a study involving 20 patients with OHS, who were matched to control subjects by age, gender, and zip code (to try to equate socioeconomic factors). A comparison with controls revealed that the most common morbidities in patients with OHS were cardiovascular diseases: congestive heart failure (OR = 9.0; 95% CI: 2.3-35.0); angina pectoris (OR = 9.0; 95% CI: 1.4-57.1); and *cor pulmonale* (OR = 9.0; 95% CI: 1.4-57.1). In a retrospective study conducted by Basoglu & Tasbakan, having a BMI > 40 kg/m² and obesity-related complications showed a strong association with an increased risk of premature death in hospitalized patients.⁽²⁾ Nowbar et al.⁽⁵⁾ reported that, at 18 months following hospital discharge, mortality was 23% in patients with obesity-related hypoventilation, which was almost twice as high as that among obese patients without hypoventilation.

CLINICAL PRESENTATION AND DIAGNOSIS

OHS occurs within a triad: obesity; daytime gas exchange abnormalities (hypercapnia); and the absence of other causes for the findings (Chart 1).⁽²³⁾ The American Academy of Sleep Medicine defines OHS as follows: the presence of awake daytime alveolar hypoventilation (PaCO₂ > 45 mmHg as measured at sea level) in patients with a BMI ≥ 30 kg/m² in the absence of other causes of hypoventilation.⁽¹¹⁾

The vast majority of patients with OHS have symptoms of OSA, including snoring, nighttime choking, witnessed apneas, nonrestorative sleep, excessive daytime sleepiness, and fatigue. In contrast to patients with OSA alone, patients with OHS complain of dyspnea, are often hypoxemic, and can have signs of *cor*

Chart 1. Diagnosis of obesity hypoventilation syndrome.

Diagnostic criteria
<ul style="list-style-type: none"> • Presence of awake daytime alveolar hypoventilation (PaCO₂ > 45 mmHg as measured at sea level) • BMI ≥ 30 kg/m² • Absence of other causes of hypoventilation
Diagnoses of exclusion
<ul style="list-style-type: none"> • COPD or other severe obstructive lung diseases • Severe interstitial lung disease • Mechanical respiratory limitation such as in severe chest wall disorders (such as kyphoscoliosis) • Neuropathic and myopathic conditions (such as amyotrophic lateral sclerosis, Duchenne muscular dystrophy, myasthenia gravis, myositis, and diaphragmatic paralysis) • Electrolyte disturbances (such as hypophosphatemia, hypomagnesemia, hypermagnesemia, hypokalemia, and hypocalcemia) • Central causes (such as cerebrovascular disease and untreated hypothyroidism) • Congenital alveolar hypoventilation syndrome (Ondine's syndrome) • Use of sedatives, hypnotics, opiates, or alcohol

pulmonale. Plethoric obese patients with hypoxemia, an increased neck circumference, a decreased airway area, a prominent P2 (a loud second heart sound) on cardiac auscultation, and leg edema, as determined by physical examination, are at risk of having OHS.⁽¹⁾

OHS is a diagnosis of exclusion. Other causes of hypoventilation, such as COPD; severe interstitial lung disease; mechanical respiratory limitation (for example, chest wall disorders such as kyphoscoliosis); myopathies (such as myasthenia gravis); neurological diseases; central causes (such as cerebrovascular disease and untreated hypothyroidism); and congenital causes (such as Ondine's syndrome; Chart 1), should be ruled out.

Patients suspected of having OHS can initially be screened by pulse oximetry and by determination of serum levels of venous bicarbonate. Borderline oximetry values are common findings. Patients with OHS undergoing arterial blood gas analysis rarely have PaO₂ values > 70 mmHg. Consequently, SpO₂ values < 93% on pulse oximetry would be suggestive of hypoventilation. However, higher values are not exclusionary, which explains why this is not a necessary criterion to establish the diagnosis, although it helps in screening. Nocturnal oximetry showing sustained hypoxemia and no associated apneas strengthens the suspicion for hypoventilation. A serum bicarbonate level ≥ 27 mEq/L had a sensitivity of 92% and a specificity of 50%, justifying its use in screening.^(7,24,25) After such screening, arterial blood gas analysis is mandatory. For excluding other causes of hypoventilation (Chart 1), pulmonary function testing and assessment of respiratory muscle strength (MIP and MEP), chest X-ray, electrocardiography, and thyroid function testing

should be performed. In addition, the use of drugs and medications, such as sedatives, hypnotics, opiates, and alcohol (alcohol abuse), should be investigated. Polysomnography is not necessary for the diagnosis of OHS.⁽¹¹⁾ However, since it has been observed that individuals with OHS have obstructive events, as well as lower saturation in REM sleep (Figure 1), polysomnography is requested with a view to treating comorbid sleep apnea and to justifying possible treatments.⁽⁶⁾

Unfortunately, despite being simple in concept, the diagnosis of OHS is delayed in most cases, occurring during acute events of respiratory failure or cardiac decompensation.^(5,26)

PATHOPHYSIOLOGY

Several mechanisms are related to the pathogenesis of OHS (Figure 2), including an abnormal organic response of the respiratory system in certain obese individuals, as well as an inappropriate central response to hypercapnia and hypoxemia, in addition to neurohumoral changes. In comparison with other obese individuals, patients with OHS have decreased lung compliance, important reductions in functional residual capacity and chest wall compliance, and increased pulmonary resistance.^(23,27)

Changes in pulmonary function

Obesity and the resulting greater chest wall thickness cause an excessive increase in the work of breathing. Breathing smaller volumes affects respiratory mechanics, reducing respiratory system compliance and increasing its resistance (which, in

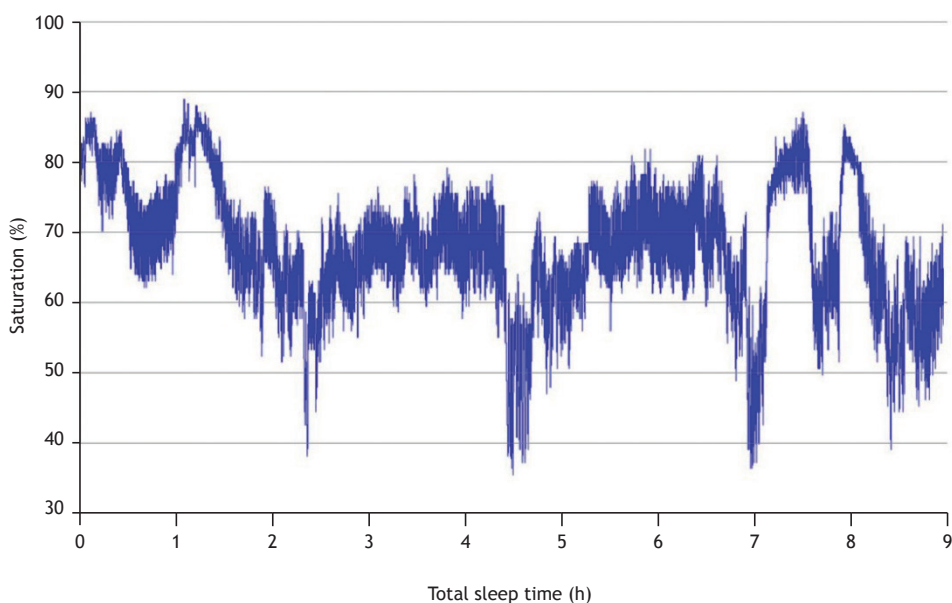


Figure 1. Female patient with a body mass index of 45 kg/m², PaCO₂ = 55.6 mmHg, obstructive sleep apnea, and obesity hypoventilation syndrome presenting with persistent hypoxemia and frequent desaturations, which were more pronounced at three time points (at between 2 and 3 h of sleep, at between 4 and 5 h of sleep, and at 7 h of sleep), suggestive of occurring during REM sleep.

individuals with OHS, is approximately 20% higher than in other obese individuals and 60% higher than in normal-weight individuals).^(23,27) Gas exchange is also affected, worsening the ventilation/perfusion ratio. Individuals with OHS tend to have lower tidal volume and higher RR, which increases the dead space effect. Consequently, hypoxemia is a common finding, which leads to an equally common outcome of pulmonary hypertension secondary to hypoxia.^(16,17) In addition, abdominal fat deposition compromises the diaphragm's influence on ventilation, compromising muscle function. Furthermore, there is thinning of the diaphragm and increased oxidative stress.⁽²⁸⁾

Ventilatory control

Patients with OHS have arterial CO₂ retention. A reduction in CO₂ chemosensitivity was initially believed to be the possible cause of this finding, which was proven untrue.⁽²⁹⁻³¹⁾ Unlike what occurs in chronic hypoxia, low daytime and nighttime saturation can be the cause of decreased ventilatory response.⁽³²⁾ Chemosensitivity

is progressively impaired by increased CO₂ levels. Chronic hypercapnia is also believed to result from the inability to eliminate CO₂, which accumulates at night during apnea and hypopnea episodes, during the day (Figure 3).⁽³⁰⁾ A secondary mechanism that also impairs chemosensitivity is elevated serum and cerebrospinal fluid levels of bicarbonate.

Role of leptin

Leptin is a cytokine produced by adipocytes and may explain a causal relationship among obesity, ventilatory control, and chronic hypercapnia. Most data come from studies of mice. When obese, these animals, like humans, develop daytime hypercapnia and reduced ventilatory response to CO₂. In mice, there is deficiency of leptin. Leptin replacement reverses hypoventilation in mice with leptin deficiency.⁽³³⁾

Unlike in the animal model, there is no deficiency but rather an increase in leptin levels in obese humans. Leptin is believed to initially have a protective effect, stimulating the ventilatory response. The persistence

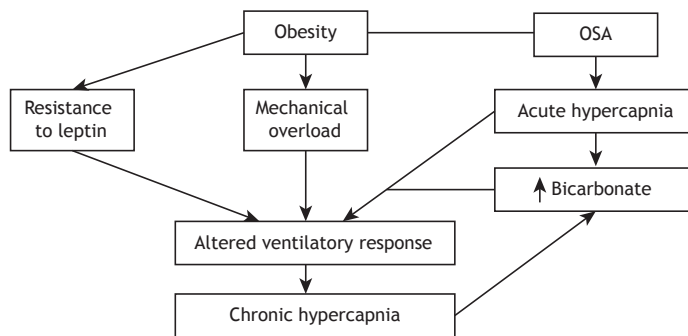


Figure 2. Pathophysiology of obesity hypoventilation syndrome. OSA: obstructive sleep apnea. Adapted from Mokhlesi.⁽¹⁸⁾

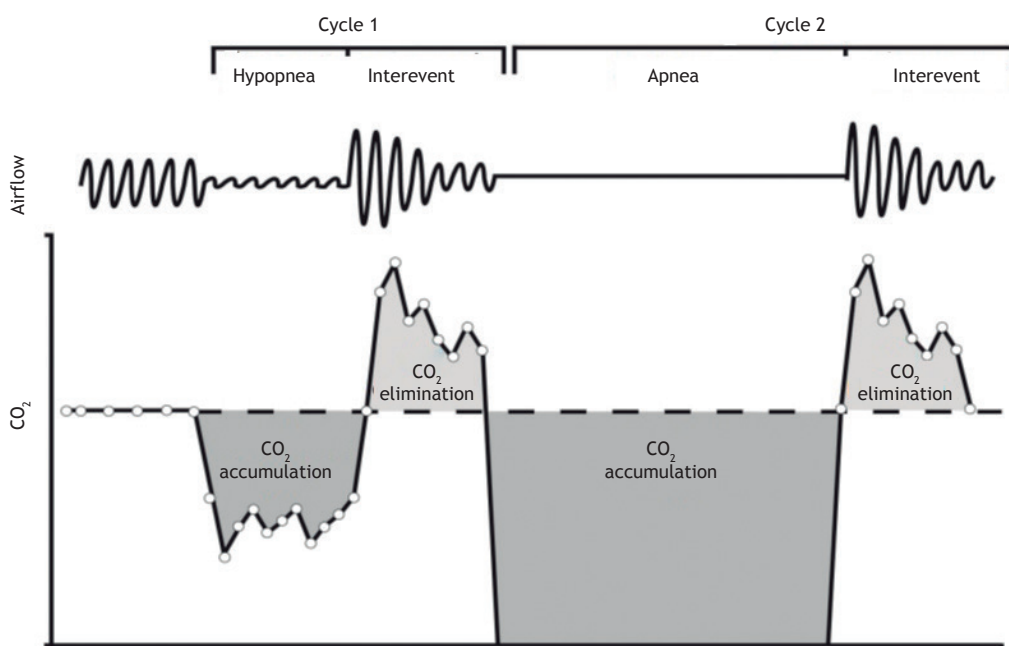


Figure 3. Influence of obstructive sleep events on hypercapnia. Adapted from Berger et al.⁽³¹⁾

of obesity would lead to leptin resistance (which is conceptually similar to insulin resistance), and thus, a consequent decrease in the ventilatory response to CO₂.^(25,32,34)

GENERAL TREATMENT MEASURES

Weight loss

Significant weight loss promotes improvement in ventilatory parameters.^(3,32) Bariatric surgery is the intervention resulting in the best outcome.⁽³⁵⁾ However, low-calorie diets may have satisfactory results. Bariatric surgery is the treatment of choice in the management of morbidly obese patients, but not every patient is a candidate for the procedure, given that the number of comorbidities that increase surgical risk is high. In fact, in some cases, the procedure will be contraindicated because of such comorbidities.

Although treatment improves ventilatory variables, it does not always resolve the problem. In a study conducted by Dixon et al.⁽³⁶⁾ involving 60 obese patients with a diagnosis of OSA who were divided into two groups—those undergoing calorie restriction and those undergoing bariatric surgery—weight loss was greater in the bariatric surgery group, but there was no statistically significant difference regarding the apnea-hypopnea index. Greenburg et al.⁽³⁷⁾ published a meta-analysis that included 12 studies involving 342 patients who underwent polysomnography before bariatric surgery and after maximal weight loss. There was a 71% reduction in the apnea-hypopnea index, from 55 events/h (95% IC: 49-60 events/h) to 16 events/h (95% CI: 13-19 events/h). It is known that 7% to 20% of such patients are unable to maintain a BMI loss of at least 20% after 5-10 years,^(38,39) which requires continued surveillance even after the procedure. Only one study evaluated the impact of bariatric surgery in patients with OHS. Sugerma et al.⁽⁴⁰⁾ evaluated 61 patients with OHS undergoing bariatric surgery. In 31 patients, there was improvement in PaO₂ (from 53 mmHg to 73 mmHg) and in PaCO₂ (from 53 mmHg to 44 mmHg) at 1 year. At 5 years, only 12 patients underwent arterial blood gas analysis, which revealed marked worsening (mean PaO₂ = 68 mmHg and mean PaCO₂ = 47 mmHg); in addition, the mean BMI was found to have increased (from 38 kg/m² to 40 kg/m²), having been high since the first postoperative year.

Oxygen therapy alone

Oxygen therapy alone is not appropriate, even in acute events, because it increases nocturnal CO₂ retention (Haldane effect or “dead space” ventilation effect), which worsens sleep quality, and is considered a common error in the management of patients with OHS (this subject will be discussed below).⁽⁴¹⁾

Phlebotomy

There are no studies that examine the indications for phlebotomy in patients with OHS. Our group uses the

indications for phlebotomy for heart disease patients and lung disease patients (hematocrit > 56% or symptoms of hyperviscosity).⁽⁴²⁾

Tracheostomy

Tracheostomy was the first treatment instituted for OHS; however, today, tracheostomy is reserved only for patients who are refractory to noninvasive ventilation (NIV), because of risk and complications inherent in the procedure and in obese patients.⁽³⁴⁾

Pharmacotherapy

Several medications (such as medroxyprogesterone and acetazolamide) have been tried to increase ventilatory response, without success, and are not recommended for the treatment of OHS.^(25,32,34,43)

Positive pressure

Continuous positive airway pressure (CPAP) is the treatment of choice for stable OHS. CPAP improves alveolar ventilation by decreasing upper airway resistance, relieving the respiratory muscle load, and/or increasing central respiratory activity.^(6,19,24,41,44-52) Patients with OHS should be initially treated with CPAP if they are clinically stable and if PaCO₂ is not severely altered (< 55 mmHg). If either of these conditions is not met, NIV should be used. In OHS patients without OSA, NIV should also be used. CPAP therapy is typically administered via a nasal mask. Some studies have shown that oronasal masks are less efficient and are associated with poorer adherence and greater side effects than are nasal masks in patients with OSA.⁽⁵³⁾ Therefore, for long-term use, nasal masks are recommended. In critically ill patients with respiratory failure, oronasal masks are preferred.

In a randomized multicenter study involving 221 patients conducted in Spain, NIV, CPAP, and lifestyle change were compared. NIV and CPAP were more effective than lifestyle change in improving clinical symptoms and polysomnographic parameters. However, there were no significant differences between NIV and CPAP, although NIV resulted in slightly improved pulmonary function values.⁽⁵⁴⁾ Howard et al.⁽⁵⁵⁾ conducted a double-blind, randomized trial of CPAP versus NIV in 57 patients with OHS admitted to either the emergency room or an outpatient clinic. There were no differences in treatment failure between CPAP and NIV, and there were similarities in ventilatory parameters, quality of life, and cardiovascular risk markers at 3 months, regardless of OHS severity. Although there was a trend toward early improvement in the group treated with NIV, use of CPAP was safe even in patients who were more severely ill, provided that it occurred in the emergency room after stabilization with NIV and that patients were monitored for treatment failure (PaCO₂ > 60 mmHg at 3 months of treatment or a 10-mmHg increase in PaCO₂ at any given time point).⁽⁵⁵⁾ However, further long-term comparative studies are needed to compare NIV versus CPAP in terms of variables such as length of hospital stay, cardiovascular events, and

mortality. In patients with refractory hypoventilation ($\text{PaCO}_2 > 45$ mmHg despite proven adherence to treatment and use of PAP determined by titration and despite the elimination of obstructive events) or with persistent desaturation ($\text{SpO}_2 < 90\%$ despite proven adherence to treatment and use of PAP determined by titration and despite the elimination of obstructive events), NIV should be used.^(43,46,47,49,52,56,57)

Treatment objectives

The objective of therapy in OHS is to reverse the major abnormalities that give rise to the disease, that is, to normalize ventilation during sleep and to reduce body weight. The therapeutic goals for patients with OHS include normalization of PaCO_2 during wakefulness and sleep; prevention of desaturations during sleep and wakefulness; control of erythrocytosis, pulmonary hypertension, and *cor pulmonale*; and relief of hypersomnia. Poor adherence to PAP is associated with incomplete clinical improvement. Adherence can be assessed by reviewing the memory card of NIV and CPAP devices.

Management in the emergency room: common errors in caring for patients with OHS

Overuse of supplemental oxygen

Hypercapnia can be aggravated by hyperoxia via several mechanisms: an increase in FiO_2 can result in a decrease in minute volume and, consequently, a decrease in tidal volume due to the activity of peripheral chemoreceptors; oxygenation of hypoxic areas causes vasodilation that changes blood flow to previously poorly ventilated areas, causing an increase in dead space; and the Haldane effect causes a reduction in hemoglobin affinity for CO_2 and decreases correction of hypoxia, causing increased release of CO_2 in plasma, which increases hypercapnia.^(29,41,58) Therefore, oxygen therapy alone is best indicated in hemodynamically stable patients with no excessive work of breathing ($\text{RR} \leq 30$ breaths/min without use of accessory muscles or with other signs of risk of ventilatory failure), under clinical surveillance, with an SpO_2 target of 89-92%.⁽⁴¹⁾

Overuse of loop diuretics

Patients with OHS are commonly affected by conditions that cause edema due to *cor pulmonale*. Since decompensation of *cor pulmonale* can be the cause for seeking medical care, a loop diuretic (furosemide) usually is used for the initial treatment of these patients in order to achieve a euvolemic state. However, overuse of diuretics can lead to acute prerenal renal failure. Contraction alkalosis secondary to the use of diuretics can worsen CO_2 retention. In addition, overuse of furosemide can cause hypokalemia. Cautious use of diuretics is indicated in OHS, at the lowest dose possible to achieve a favorable clinical response and minimize the electrolytic and acid-metabolic impact.⁽⁴¹⁾ The use of spironolactone for the prevention of hypokalemia is plausible.

Overuse of psychotropic drugs

The use of sedative/hypnotic drugs not only increases airway collapsibility but also decreases ventilatory response, which is harmful to patients with OHS.

Diagnostic confusion with COPD

Patients with chronic CO_2 retention, such as patients with OHS, are commonly diagnosed with COPD, despite the absence of documented obstructive ventilatory disorders. A retrospective study by Marik & Desai⁽⁵⁹⁾ showed that, of the morbidly obese patients admitted to the ICU for respiratory failure secondary to OHS, 75% had been erroneously treated for COPD and 86% had been treated for congestive heart failure (Chart 2).

PERIOPERATIVE PERIOD IN PATIENTS WITH OHS

Patients with OHS commonly have a consultation with a pulmonologist in the preoperative period. In addition to comorbidity care and the required cardiovascular evaluation in obese patients or in those who are known to have or are highly suspected of having OSA, specific perioperative care is required for these patients whatever the procedure. In addition to the already suggested screening with pulse oximetry and determination of serum bicarbonate, other measures are required. If screening is positive and OHS is confirmed by arterial blood gas analysis, treatment should be started immediately, even a few days or weeks after the procedure; there is significant evidence of improved gas exchange and improved ventilatory control, either with one-level positive pressure or with two-level positive pressure.⁽⁶⁰⁾

Obesity is a risk factor for difficult mask ventilation.⁽⁶¹⁾ A retrospective study by Rose & Cohen, involving 18,500 patients, showed that obesity is also an independent risk factor for difficult intubation.⁽⁶²⁾ Khetarpal et al.⁽⁶³⁾ evaluated 22,660 procedures and identified five risk factors (limited mandibular protrusion, increased neck circumference, OSA, snoring alone, and $\text{BMI} \geq 30$ kg/m^2) as independent predictors of difficult mask ventilation and difficult intubation during anesthesia induction. This suggests that patients with OHS are among those at highest risk for airway complications.⁽⁶⁴⁾ During anesthesia induction, patients with OHS should be placed in the ramp position with elevation of the torso and head (preferably at a 25° tilt). This has been shown to improve ventilation and the glottic view,⁽⁶⁵⁾ as well as oxygenation.⁽⁶⁶⁾

Patients with OHS are more sensitive to the respiratory depressant effects of anesthetic agents and opioids because they are prone to airway collapse and

Chart 2. Common errors in the emergency care of patients with obesity hypoventilation syndrome.

- Overuse of supplemental oxygen
- Overuse of loop diuretics
- Overuse of psychotropic drugs
- Diagnostic confusion with COPD

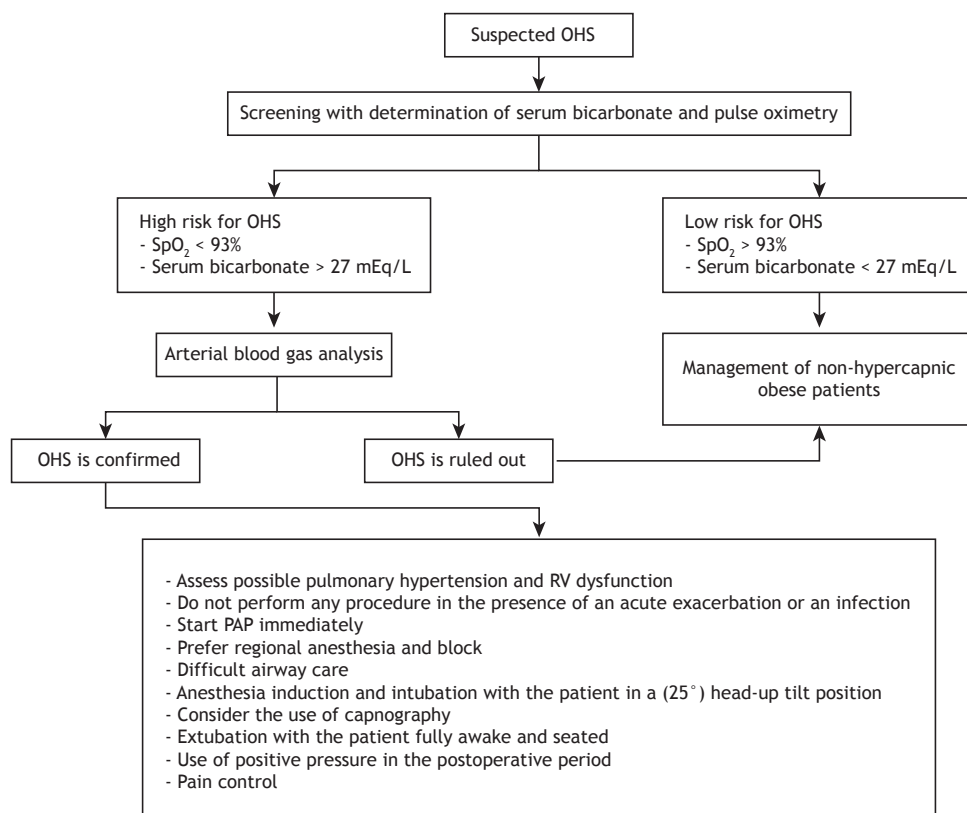


Figure 4. Suggested algorithm for the screening and perioperative management of patients with suspected or confirmed obesity hypoventilation syndrome (OHS). PAP: positive airway pressure; and RV: right ventricle. Adapted from Chau et al.⁽⁶⁴⁾

inappropriate physiological response to hypercapnia and hypoxemia. Regional block should be chosen, when possible. In addition, during the procedure if possible, patients with OHS should be monitored with a capnograph. At the end of the procedure, it is recommended that patients be placed in the ramp position or in the lateral decubitus position for improved oxygenation and maintenance of the airways, and tracheal extubation should be performed only after the patient is fully conscious.⁽⁶⁴⁾

With regard to postoperative care, the use of CPAP for 24-48 h after extubation can reduce the risk of postoperative complications and extubation failure in severely obese patients admitted to the ICU (an absolute risk reduction of 16%), with a reduction in mortality in patients with hypercapnia.^(67,68) In addition, pain

control has an impact on ventilatory status. Therefore, optimal analgesia is also required.

Figure 4 outlines a suggested algorithm for the screening and perioperative management of patients with suspected or confirmed OHS.

FINAL CONSIDERATIONS

OHS is still a poorly recognized entity in Brazil. Delayed diagnosis of OHS is associated with an increase in morbidity, mortality, and costs of care of patients who are more severely ill. However, breaking free from myths and paradigms regarding diagnosis, such as that related to polysomnography, which is unnecessary, the possibility of screening for OHS with determination of venous bicarbonate, and the possibility of treatment with CPAP enable the diagnosis and treatment of OHS in a larger number of patients.

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