Assessment and Management of Patients with Obesity Hypoventilation Syndrome

Babak Mokhlesi[1], Meir H. Kryger[2], and Ronald R. Grunstein[3]

[1]Section of Pulmonary and Critical Care Medicine, and Sleep Disorders Center, University of Chicago Pritzker School of Medicine, Chicago, Illinois; [2]Sleep Medicine, Gaylord Hospital, Wallingford, Connecticut; and [3]Sleep and Circadian Research Group and Centre for Respiratory and Sleep Medicine, Woolcock Institute of Medical Research, University of Sydney, Camperdown, Sydney, Australia

Obesity hypoventilation syndrome (OHS) is characterized by obesity, daytime hypercapnia, and sleep-disordered breathing in the absence of significant lung or respiratory muscle disease. Compared with eucapnic morbidly obese patients and eucapnic patients with sleep-disordered breathing, patients with OHS have increased health care expenses and are at higher risk of developing serious cardiovascular disease leading to early mortality. Despite the significant morbidity and mortality associated with this syndrome, diagnosis and institution of effective treatment occur late in the course of the syndrome. Given that the prevalence of extreme obesity has increased considerably, it is likely that clinicians will encounter patients with OHS in their clinical practice. Therefore maintaining a high index of suspicion can lead to early recognition and treatment reducing the high burden of morbidity and mortality and related health care expenditure associated with undiagnosed and untreated OHS. In this review we define the clinical characteristics of the syndrome and review the pathophysiology, morbidity, and mortality associated with it. Last, we discuss currently available treatment modalities.

Keywords: Pickwickian syndrome; hypercapnia; sleep apnea; continuous positive airway pressure; noninvasive positive-pressure ventilation

In the United States, the prevalence of extreme obesity (body mass index [BMI] $\geq$ 40 kg/m^2) is increasing rapidly. From 1986 to 2000, the prevalence of BMI of at least 40 kg/m^2 quadrupled and that of BMI of at least 50 kg/m^2 increased by fivefold (1, 2). Unfortunately, the obesity epidemic is a global phenomenon affecting not just adults, but also children and adolescents (3–6). With such a global epidemic of extreme obesity the prevalence of obesity hypoventilation syndrome (OHS) is likely to increase and therefore clinicians need to maintain a high index of suspicion, particularly given that early recognition and treatment reduce the high burden of morbidity and mortality associated with this syndrome.

DEFINITION

Auchincloss and coworkers, in 1955, described in detail a patient with OHS (7) and the following year, Burwell and colleagues compared patients with OHS with an obese, somnolent Charles Dickens character and popularized the description "Pickwickian syndrome" (8). The central features of OHS, as currently accepted, include obesity (BMI $\geq$ 30 kg/m^2), chronic alveolar hypoventilation leading to daytime hypercapnia and hypoxia ($Pa_{CO_2} \geq$ 45 mm Hg and $Pa_{O_2} <$ 70 mm Hg), and sleep-disordered breathing (9–11). Essential to the diagnosis is exclusion of other causes of alveolar hypoventilation such as severe obstructive or restrictive pulmonary disease, significant kyphoscoliosis, severe hypothyroidism, neuromuscular diseases, or other central hypoventilation syndromes. Although OHS can exist autonomously, it is frequently associated with obstructive sleep apnea (OSA), which is characterized by recurrent upper airway obstruction resulting in apneas, hypopneas, oxygen desaturation, and arousals from sleep. In approximately 90% of patients with OHS the sleep-disordered breathing consists of OSA. The remaining 10% of patients with OHS have an apnea–hypopnea index less than 5 (10, 12, 13). The sleep-disordered breathing in this subset of patients has been labeled as sleep hypoventilation and is defined as an increase in Pa_{CO_2} during sleep by 10 mm Hg above wakefulness or significant oxygen desaturation that is not explained by obstructive apneas or hypopneas.

EPIDEMIOLOGY AND CLINICAL PRESENTATION

The precise prevalence of OHS in the general population remains uncertain because no general population-based studies have been performed to examine this issue. However, the prevalence of OHS among patients with OSA has been estimated as between 10 and 20% (10, 12, 14–18) and is higher in the subgroup of patients with extreme obesity (Table 1 and Figure 1) (10, 15, 19).

Although most patients with OHS have had prior hospitalizations, in the majority of these patients the formal diagnosis of OHS is established late, in the fifth or sixth decade of life, after consultation with a pulmonary and critical care specialist (12, 20, 21). The vast majority of patients have the classic symptoms of OSA including loud snoring, nocturnal choking episodes with witnessed apneas, excessive daytime sleepiness, and morning headaches. In contrast to eucapnic OSA, patients with stable OHS frequently complain of dyspnea and may have signs of cor pulmonale. Physical examination findings can include a plethoric obese patient with an enlarged neck circumference, crowded oropharynx, a prominent P2 on cardiac auscultation (this is often difficult to hear because of obesity), and lower extremity edema. Table 2 summarizes the clinical features of 757 patients with OHS reported in the literature (10, 12–19, 21–27).

Several laboratory findings are supportive of OHS, yet the definitive test for alveolar hypoventilation is an arterial blood gas performed on room air. Elevated serum bicarbonate level due to metabolic compensation of respiratory acidosis is common in patients with OHS and points toward the chronic nature of hypercapnia (13, 21, 28), and could be used as a sensitive test to screen for hypercapnia (10). In addition, hypoxemia detected on room air pulse oximetry during wakefulness in patients with sleep-disordered breathing should prompt clinicians to exclude hypercapnia (10, 29). If hypercapnia is present, pulmonary function testing and chest imaging should be performed to exclude other causes of hypercapnia.

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Correspondence and requests for reprints should be addressed to Babak Mokhlesi, M.D., M.Sc., Section of Pulmonary and Critical Care Medicine, University of Chicago Pritzker School of Medicine, 5841 S. Maryland Avenue, MC 0999/Room L11B, Chicago, IL 60637. E-mail: bmokhles@medicine.bsd.uchicago.edu

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TABLE 1. PREVALENCE OF OBESITY HYPOVENTILATION SYNDROME IN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA

Authors	n	Design	Country	Age (*yr*)	BMI	AHI	OHS (%)
Verin and colleagues (17)	218	Retrospective	France	55	34	51	10
Laaban and Chailleux (15)	1,141	Retrospective	France	56	34	55	11
Kessler and colleagues (12)	254	Prospective	France	54	33	76	13
Resta and colleagues (16)	219	Prospective	Italy	51	40	42	17
Golpe and colleagues (18)	175	Retrospective	Spain	NA	32	42	14
Akashiba and colleagues (14)	611	Retrospective	Japan	48	29	52	9
Mokhlesi and colleagues. (10)	359	Prospective	USA	48	43	62	20

Definition of abbreviations: AHI = apnea–hypopnea index; BMI = body mass index; NA = not available; OHS = obesity hypoventilation syndrome; OSA = obstructive sleep apnea.

Age, BMI, and AHI values represent means of all patients (OSA and OHS) and were calculated from data provided by the authors of the articles.

Pulmonary function tests can be normal but typically reveal a mild to moderate restrictive defect due to body habitus without significant reduction in FEV_1/FVC accompanied by a significant reduction in the expiratory reserve volume. Patients with OHS may also have mild reductions in maximal expiratory and inspiratory pressures related to the combination of abnormal respiratory mechanics and weak respiratory muscles (30). Other laboratory testing should include a complete blood count to rule out secondary erythrocytosis and severe hypothyroidism.

PATHOPHYSIOLOGY

The mechanism by which morbid obesity leads to hypoventilation is complex and not fully understood. Several mechanisms have been proposed in the pathogenesis of OHS, including abnormal respiratory system mechanics due to obesity, impaired central responses to hypercapnia and hypoxia, sleep-disordered breathing, and neurohormonal abnormalities such as leptin resistance (Figure 2).

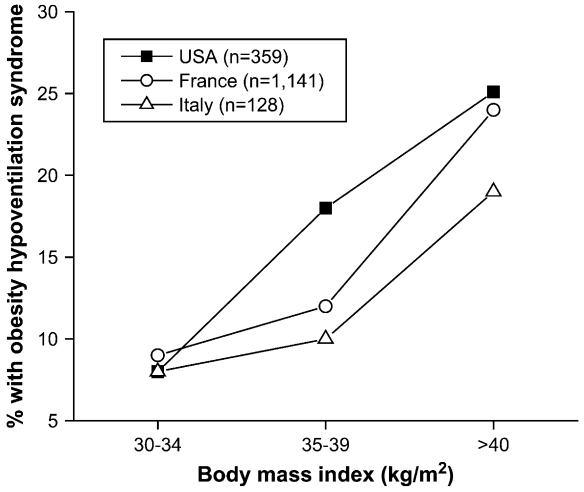

Figure 1. Prevalence of obesity hypoventilation syndrome (OHS) in patients with obstructive sleep apnea (OSA) by categories of body mass index (BMI) in the United States (10), France (15), and Italy. The data from Italy were provided by O. Resta (University of Bari, Bari, Italy). In the study from the United States the mean BMI was 43 kg/m^2 and 60% of the subjects had a BMI above 40 kg/m^2. In contrast, the mean BMI in the French study was 34 kg/m^2 and 15% of the subjects had a BMI above 40 kg/m^2. Consequently, OHS may be more prevalent in the United States compared with other nations because of its more exuberant obesity epidemic. Reprinted by permission from Reference 11.

Obesity imposes a significant mechanical load leading to a reduction in total respiratory system compliance (16, 19, 23, 31, 32), increased lung resistance (33, 34), and a relative state of respiratory muscle weakness leading to increased work of breathing (30, 34–36). However, it does not appear that obesity is the only determinant of hypoventilation as less than one-third of morbidly obese patients develop chronic hypercapnia (10, 15). Other determinants of hypoventilation include a blunted central responsiveness to hypercapnia and hypoxia (25, 28, 35, 37–39), a state of leptin resistance (a satiety protein that increases ventilation) (40–43), and sleep-disordered breathing. The role of sleep-disordered breathing in the pathogenesis of hypoventilation has been well established by the resolution of hypercapnia in the majority of patients with OHS with either positive airway pressure therapy or tracheostomy without any concomitant change in body mass (13, 22, 25, 26, 28, 44–46), CO_2 production, or the volume of dead space (28).

A model that combines sleep-disordered breathing, central respiratory drive, and renal buffering has been proposed to explain the pathophysiology of OHS (47–49). In patients with OSA, the minute ventilation during sleep does not decrease, due to the large increase in the minute ventilation between the obstructive respiratory events. Obstructive respiratory events can, however, lead to acute hypercapnia if the duration of the interevent hyperventilation is inadequate to eliminate the accumulated CO_2 (50). This acute hypercapnia causes a small increase in serum bicarbonate level that is not corrected before the next sleep period if the time constant of bicarbonate excretion is longer

TABLE 2. CLINICAL FEATURES OF PATIENTS WITH OBESITY HYPOVENTILATION SYNDROME

Variable	Mean (*range*)
Age, yr	52 (42–61)
Men, %	60 (49–90)
Body mass index, kg/m^2	44 (35–56)
Neck circumference, cm	46.5 (45–47)
pH	7.38 (7.34–7.40)
Pa_{CO_2}, mm Hg	53 (47–61)
Pa_{O_2}, mm Hg	56 (46–74)
Serum bicarbonate, mEq/L	32 (31–33)
Hemoglobin, g/dl	15
Apnea–hypopnea index	66 (20–100)
Oxygen nadir during sleep, %	65 (59–76)
Percent time Sa_{O_2} less than 90%, %	50 (46–56)
FVC, %pred	68 (57–102)
FEV_1, %pred	64 (53–92)
FEV_1/FVC	77 (74–88)
Medical Research Council dyspnea class 3 and 4, %	69
Epworth Sleepiness Scale, score	14 (12–16)

Data are presented as means (range) of the 16 studies (10, 12–19, 21–27) and include a total of 757 patients with obesity hypoventilation syndrome.

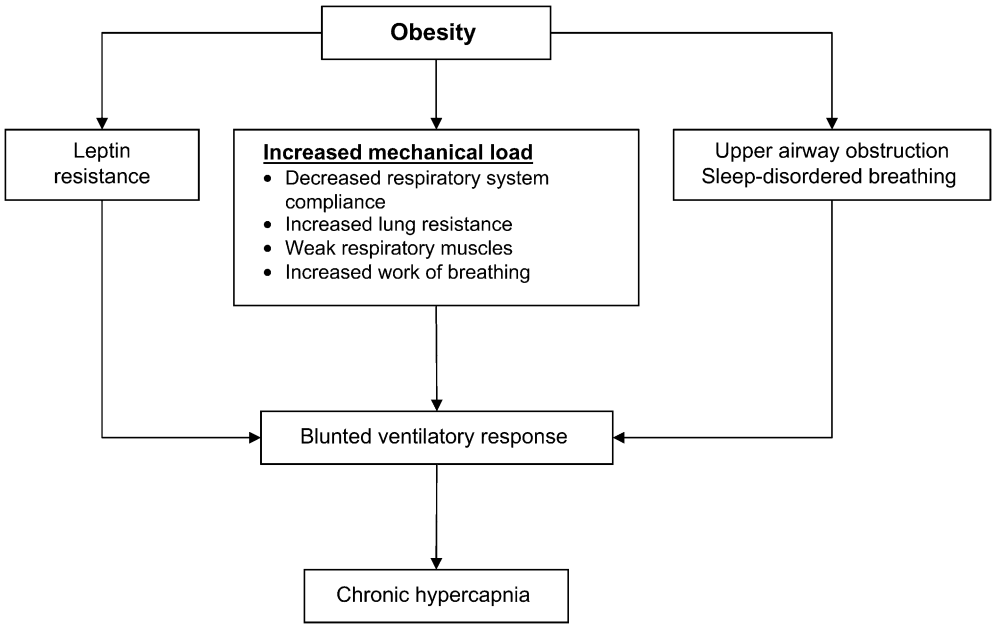

Figure 2. Mechanisms by which obesity can lead to chronic daytime hypercapnia.

than that of CO_2 (48). The elevated bicarbonate level blunts the ventilatory response to CO_2 from its initial value by reducing the change in hydrogen ions for a given change in CO_2 and would ultimately result in a higher awake CO_2 level (47, 51–53).

MORBIDITY AND MORTALITY

Because the defining pathogenic characteristics in OHS are obesity and respiratory failure, not surprisingly the morbidity documented clinically will be most often related to these two factors. Berg and colleagues showed that patients with OHS use much more health care than do obese patients without hypoventilation or general population control subjects in the 5 years before OHS is actually confirmed (24). By comparing health care use in cases versus obese and general population control subjects, this group was able to tease apart the comorbidities related to the obesity per se and those related to hypoventilation.

Morbidity Related to Hypoventilation

Compared with obese control subjects, patients with OHS were statistically much more likely to have been diagnosed with congestive heart failure (odds ratio [OR], 9; 95% confidence interval [95% 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) (24). Patients with OHS were more likely to be hospitalized and, compared with patients with a similar degree of obesity but without hypoventilation, had higher rates of admission to the intensive care unit and need for invasive mechanical ventilation (21, 24).

Morbidity Related to Obesity

Obesity is associated with many medical problems and is a component of the metabolic syndrome. Consequently, patients with OHS are at increased risk of morbidities that span several organ systems in addition to those related to hypoventilation. Specifically, they are much more likely to be diagnosed with arterial hypertension (OR, 3.8; 95% CI, 1.5–9.8), diabetes mellitus (OR, 17.2; 95% CI, 7.3–40.7), hypothyroidism (OR, 6.5; 95% CI, 2.4–17.5), and osteoarthritis (OR, 3.3; 95% CI, 1.1–10.3) (24). Also probably related to obesity, there is an increased risk of hepatic dysfunction and hyperlipidemia (14). Others have reported that patients with OHS have a higher rate of pulmonary hypertension compared with eucapnic patients with OSA (12, 54). Up to one-quarter of patients with OHS also carry a diagnosis of asthma (13, 26). It is not surprising, given the large number of comorbidities, that patients with OHS have impaired quality of life compared with eucapnic patients with OSA matched for age, BMI, and lung function (55).

Mortality

Although older series had reported a high mortality rate among hospitalized patients with OHS (56, 57), two prospective studies reported no in-hospital deaths among a total of 64 consecutive hospitalized patients with OHS (21, 58). Of course, respiratory failure, if untreated, places these patients at markedly increased risk of death. A retrospective study reported that 7 of 15 patients with OHS who refused long-term noninvasive positive airway pressure (NPPV) therapy died during an average follow-up period of 50 months (13). Similarly, a prospective study monitored 47 untreated patients with OHS for 18 months after hospital discharge. The mortality of patients with OHS was 23 versus 9% in patients with a similar degree of obesity but without hypoventilation (hazards ratio of 4.0 after adjusting for age, sex, BMI, and renal function) and most deaths occurred in the first 3 months after hospital discharge (21). In contrast, one retrospective study of 126 patients with OHS who were adherent with NPPV therapy reported an 18-month mortality of 3% (Figure 3), and the 2- and 5-year mortality rates were 8 and 30%, respectively (27). Moreover, current evidence also suggests that adherence with positive airway pressure therapy reduces health care expenses and hospital readmission rates among patients with OHS (13, 24, 45).

Collectively, the foregoing evidence would suggest that identifying patients with OHS in a timely manner is important and treatment should be initiated without delay to avoid adverse outcomes such as readmission to the hospital, acute-on-chronic respiratory failure requiring intensive care monitoring, and death.

TREATMENT

There are no established guidelines on treatment of OHS. In effect, treatment modalities are each based on different perspectives concerning the underlying pathophysiology of the condition. First, upper airway obstruction is an important factor in the pathogenesis of OHS and there is evidence that strategies

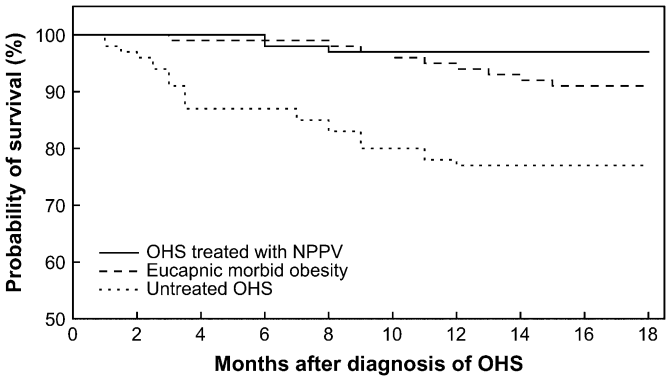

Figure 3. Survival curves for patients with untreated obesity hypoventilation syndrome (OHS) (n = 47; mean age, 55 ± 14 yr; mean body mass index [BMI], 45 ± 9 kg/m^2; mean Pa_{CO_2}, 52 ± 7 mm Hg) and eucapnic, morbidly obese patients (n = 103; mean age, 53 ± 13 yr; mean BMI, 42 ± 8 kg/m^2) as reported by Nowbar and colleagues (21) compared with patients with OHS treated with noninvasive positive airway pressure (NPPV) therapy (n = 126; mean age, 55.6 ± 10.6 yr; mean BMI, 44.6 ± 7.8 kg/m^2; mean baseline Pa_{CO_2}, 55.5 ± 7.7 mm Hg; mean adherence with NPPV, 6.5 ± 2.3 h/d). Data for patients with OHS treated with NPPV were provided by S. Budweiser and colleagues (University of Regensburg, Regensburg, Germany) (27). Modified by permission from Reference 21.

for reversing upper airway obstruction, such as tracheostomy and nasal continuous positive airway pressure (CPAP), are effective. Second, failure of normal mechanisms that prevent hypoventilation during sleep are implicated in OHS and therefore noninvasive or even invasive ventilation to support breathing and reverse hypoventilation has been advocated. Alternatively, pharmacologic methods to stimulate breathing have been used. Finally, by definition, OHS does not occur in the absence of obesity. Therefore, methods that result in major weight reduction will effectively reverse OHS.

Reversing Upper Airway Obstruction

Kuhlo and colleagues first described the use of tracheostomy to reverse Pickwickian syndrome in 1969 (59). It was observed that this therapy could not only reverse sleep-related respiratory failure but also awake hypoventilation in most cases (28, 60, 61). There was also a report of a mechanical device (a supportive collar designed to hold the mandible forward) reversing upper airway obstruction during sleep and improving awake respiratory failure (62). After the advent of nasal CPAP, this therapy was used in patients with OHS, resulting in remission of awake respiratory failure (63, 64). Subsequent reports have supported the initial observation of the efficacy of nasal CPAP alone as a treatment of OHS (26, 55, 65–67). Patients who were successfully treated with CPAP typically required pressures of 12–14 cm H_2O (13, 26, 65). However, there were also early reports of partial success or failure of nasal CPAP to reverse some cases of OHS (68) and confirmed by subsequent studies (15, 26, 65, 69, 70).

Ventilation and OHS

Ventilatory support via tracheostomy for obesity-related respiratory failure has been used since the 1960s. Although effective, this was obviously not an ideal technique given the difficulties of maintaining a tracheostomy, especially in patients with markedly excessive fat in the neck region. NPPV using a face or, later, nasal mask was regularly used in OHS from the late 1980s, based on effective use of this approach in patients with other forms of chest wall disease (71). Studies using bilevel positive airway pressure (PAP)—the most common mode of NPPV—or volume-cycled ventilation showed efficacy in reversing diurnal respiratory failure in patients who had failed nasal CPAP for OHS (45, 72, 73). Subsequent research has confirmed the efficacy of NPPV in OHS (13, 27, 74).

Oxygen Therapy

Approximately half of patients with OHS require supplemental nocturnal oxygen in addition to some form of PAP therapy (13, 26, 45, 75). The need for nocturnal and daytime oxygen therapy decreases significantly in patients adherent with PAP therapy (13, 26, 45). Supplemental oxygen without PAP therapy, however, is inadequate and does not improve hypoventilation (76).

Pharmacological Respiratory Stimulation

Given the potential role of impaired respiratory control in the pathogenesis of OHS, using pharmacologic agents to stimulate breathing would be an attractive option. However, there are few data on this approach. Reports of initial positive results with either progesterone (77), almitrine (78), or acetazolamide (28) have never resulted in ongoing randomized controlled trials. Furthermore, medroxyprogesterone can increase the risk of venous thromboembolism (79, 80).

Research using a putative animal model of OHS, the leptin-deficient *ob/ob* mouse, has demonstrated improvement in awake respiratory failure with leptin replacement (81). This has never been verified in humans, where leptin resistance, rather than leptin deficiency, is present (41).

Weight Loss

Starting with the original report by Burwell and colleagues (8), a number of subsequent studies have identified that weight loss results in improvement in sleep-disordered breathing, reduction in awake respiratory failure, and improvement in lung function in patients with OHS. Rapid weight reduction can be achieved by a range of surgical methods, although most data concerning OHS are available from surgical procedures such as gastric bypass or gastric banding (54, 82, 83). The significant weight loss associated with bariatric surgery can improve ventilation during sleep, which can eventually lead to improvement in diurnal ventilation (84).

Patients with OHS are at increased risk of death related to gastric bypass surgery, in part because of the increased risk of postoperative respiratory failure and the development of pulmonary embolism (85). Appropriate management in such patients undergoing surgery should include perioperative treatment with PAP therapy until weight loss results in enough improvement of disordered breathing during sleep that withdrawal of therapy is allowed. Therefore, we believe that patients with OHS should be treated with CPAP or bilevel PAP preoperatively and immediately after extubation to avoid postoperative respiratory failure (86–88). Furthermore, there is no evidence that PAP therapy used during the immediate postoperative period leads to increased risk of anastomotic disruption or intestinal leakage (89). In the long term, weight reduction provides the most effective solution to OHS. Evidence is accumulating that in patients with OHS bariatric surgery may be the best option in treating the multitude of comorbidities that are related to extreme obesity such as hypertension, hyperlipidemia, and type 2 diabetes (90). However, weight gain and significant increase in the apnea–hypopnea index can occur between 3 and 7 years after gastric bypass surgery (91).

Overall Treatment Approach

Clearly treatment of OHS will depend on the state of the patient at presentation. In extreme cases patients with OHS may present with decompensated respiratory failure. Although there are reports that nasal CPAP may be effective in these situations (67), clinical consensus suggests such patients should receive NPPV (13, 45, 72, 73). Figure 4 provides a general approach to the management of patients with OHS hospitalized because of acute-on-chronic hypercapnic respiratory failure (92).

More commonly, however, patients with OHS will present to ambulatory settings such as sleep disorders clinics or in a semi-elective consultation setting in hospital with stable awake respiratory failure without impaired consciousness. Most studies indicate that patients with stable OHS should undergo an initial titration with nasal CPAP and, when there is persistent moderate hypoxemia despite adequate resolution of upper airway obstruction with CPAP, bilevel PAP should then be considered (26, 65). There is no standard protocol for bilevel PAP titration in stable patients with OHS. However, the titration protocol followed in most studies consisted of increasing the expiratory PAP (EPAP) to abolish apneas, hypopneas, and any evidence of flow limitation and, if the oxygen saturation remained persistently below 90%, then inspiratory PAP (IPAP) was added to the final EPAP to improve ventilation. To achieve long-term improvement in daytime hypercapnia and hypoxia with bilevel PAP the IPAP needs to be at least 8 to 10 cm H_2O above EPAP and most patients with OHS require inspiratory pressures of 16–20 cm H_2O and expiratory pressures of 6–10 cm H_2O (11, 13, 22, 46, 93). In the subset of patients with OHS who do not have obstructive sleep apnea EPAP can be set at 5 cm

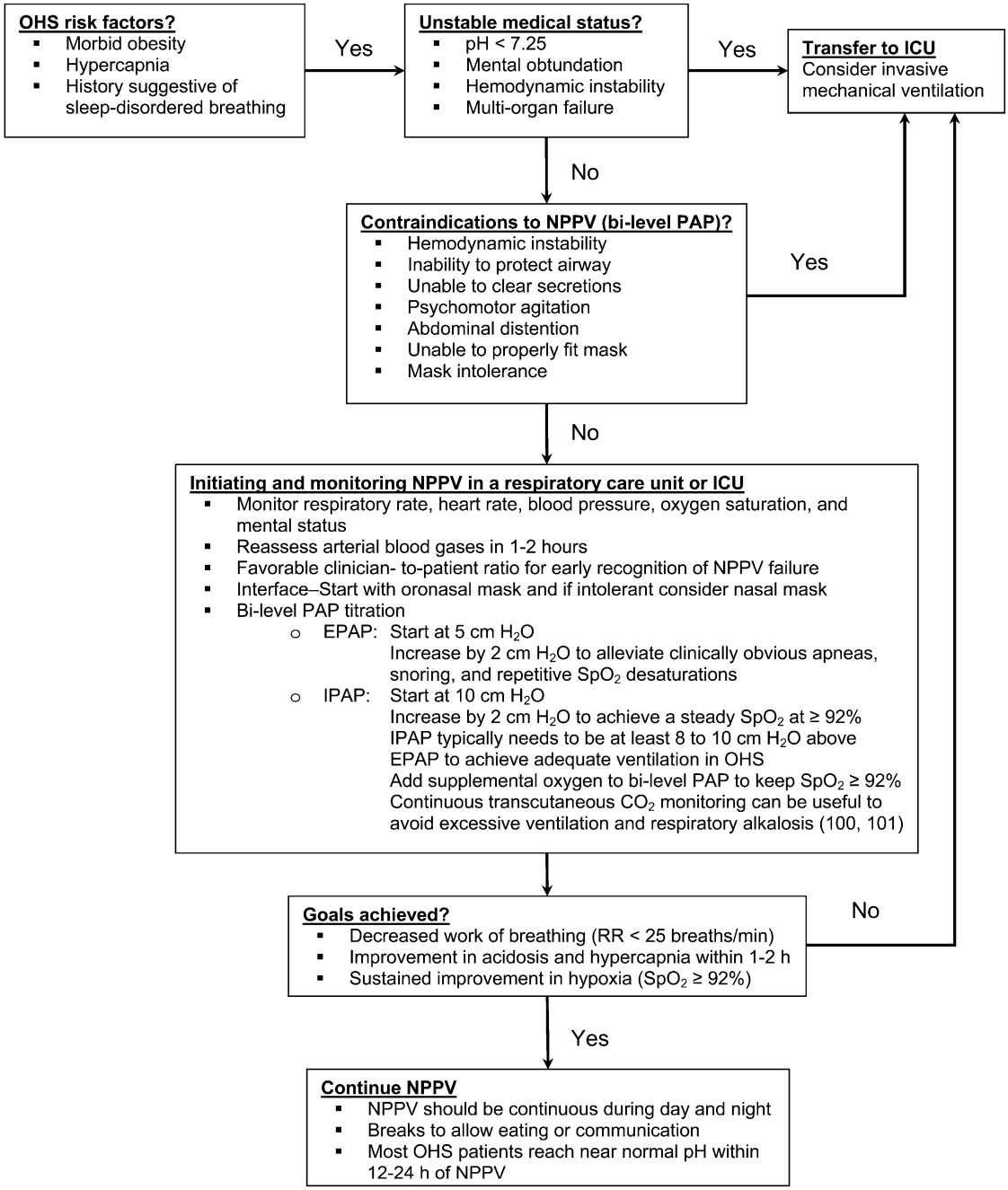

Figure 4. Management of patients with OHS requiring hospitalization because of acute-on-chronic hypercapnic respiratory failure. EPAP = expiratory positive airway pressure; ICU = intensive care unit; IPAP = inspiratory positive airway pressure; RR = respiratory rate; Sp_{O_2} = oxygen saturation by pulse oximetry.

H_2O and IPAP can be titrated to improve ventilation (46, 93). Whether the persistent moderate hypoxemia improves over time with CPAP remains to be elucidated (69). Some have also advocated adding low-flow oxygen to CPAP in these situations, similar to patients with overlap syndrome (OSA plus chronic obstructive pulmonary disease), but this approach has not been investigated in any detail and has not been compared to NPPV with bilevel PAP without supplemental oxygen (69). CPAP is also a less costly therapy than bilevel PAP and there is evidence that a majority of patients with OHS will have immediate improvement with CPAP alone (26, 65). There are no published large-scale randomized controlled trials that inform on best management practice in these patients (studies comparing initial treatment with CPAP or NPPV). If patients with CPAP failure due to persisting marked hypoxemia while undergoing this therapy are excluded, then preliminary data suggest that the remaining patients will have similar outcomes with CPAP at 3 months as with bilevel PAP (94). Perez de Llano and coworkers reported that up to one-third of patients with OHS who were initially treated with a few months of NPPV could be successfully switched to CPAP therapy as long as a repeat polysomnogram did not reveal significant and persistent oxygen desaturation (arbitrarily defined as oxygen saturation below 90% for at least 15% of the sleep period) despite adequate CPAP titration (13). In fact, more recently the same group of investigators reported long-term success in patients who were switched to CPAP therapy (95). Predictors of CPAP failure include greater degrees of obesity, significant restrictive chest physiology, severity of hypoxemia during polysomnography, and higher Pa_{CO_2} levels during wakefulness (65, 70, 95).

Taken together, there is compelling evidence that CPAP is effective in the majority of patients with stable OHS, particularly in the subgroup with severe OSA. Bilevel PAP should be strongly considered in patients who fail CPAP, patients with OHS who experience acute-on-chronic respiratory failure (13, 58), and patients who have OHS without OSA (46, 93). Treatment of OHS with positive airway pressure improves blood gases, morning headaches, excessive daytime sleepiness and vigilance, dyspnea, pulmonary hypertension, leg edema, and secondary erythrocytosis (13, 45, 75, 96). Improvement in symptoms and blood gases is directly related to adherence with therapy and maximal improvement in blood gases can be achieved as early as 2 to 4 weeks (26). Therefore, early follow-up is imperative and should include repeat measurement of arterial blood gases and objective assessment of adherence with positive airway pressure as patients frequently overestimate adherence (97–99). Changes in serum bicarbonate level and pulse oximetry could be used as a less invasive measure of ventilation. After a few months of treatment with bilevel PAP a subgroup of patients may no longer need nighttime or daytime supplemental oxygen therapy. In these cases simplifying the treatment regimen by discontinuing oxygen therapy can lead to significant cost savings and decrease the amount of equipment in the patient's bedroom. Although it is possible to successfully switch this subgroup of patients with OHS from bilevel PAP to CPAP therapy, it may not add to the cost savings because many patients may have completed the initial rental period on the bilevel PAP device.

In summary, the prevalence of OHS is likely to increase because of the global obesity epidemic. A high index of suspicion can lead to early recognition of the syndrome and initiation of appropriate therapy. The treatment options other than positive airway pressure have been poorly studied and further research is needed to better understand the long-term treatment outcomes of patients with OHS. In the meantime, clinicians should encourage adherence with positive airway pressure therapy to prevent the serious adverse outcomes of untreated OHS. If positive airway pressure fails to achieve the desired results, weight reduction surgery or tracheostomy with or without pharmacotherapy with respiratory stimulants should be considered.

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