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Obesity Hypoventilation Syndrome: Understanding, Diagnosing, and Treating

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OBESITY HYPOVENTILATION SYNDROME:

UNDERSTANDING, DIAGNOSING, AND TREATING

by

Chad W. Padovich

An Evidence Based Scholarly Paper submitted to the faculty of

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ABSTRACT

OBESITY HYPOVENTILATION SYNDROME:

UNDERSTANDING, DIAGNOSING, AND TREATING

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Master of Science

Purpose: The effects of obesity are multifaceted and lead to poor quality of life, increased risk of cardiovascular disease, stroke, and death. Obesity hypoventilation syndrome (OHS) is a widely misunderstood and under diagnosed disease process, which carries specific diagnostic criteria. The purpose of this work is to: (1) provide practitioners with a better understanding of OHS and how it differs from other obesity related breathing disorders (such as Obstructive Sleep Apnea, OSA), (2) provide diagnostic criteria of OHS, (3) provide work up recommendations, and (4) provide current recommended treatment.

Data Sources: An electronic search of the literature was conducted to identify studies from 2008 to 2014 in the following databases: CINAHL, National Library of Medicine PubMed®/MEDLINE®, EBSCO, SciVerse®, Springer Link®, and the Cochrane library.

Conclusions: The effects of obesity are multifaceted and lead to poor quality of life, increased risk of cardiovascular disease, stroke, and death. Obese individuals are more prone to respiratory complications, such as obstructive sleep apnea (OSA) and obesity hypoventilations syndrome (OHS). OHS is commonly diagnosed as OSA, as symptomology is similar. Widely misunderstood and undertreated, OHS is a distinct disease, with specific diagnostic criteria. The

only proven method to reverse and cure OHS is bariatric surgery. These individuals require an interdisciplinary team approach to manage them.

Results/Implications for Practice: Nurse practitioners often see obese and overweight, patients who may be at risk for OHS. While, OSA is commonly recognized in the medical community, many providers are unaware of OHS and its serious complications. OHS is often misdiagnosed, undertreated and thought of as severe OSA, as both carry a similar patient symptomology. It is important nurse practitioners recognize the difference between OSA and OHS. This includes understanding the diagnostic criteria; appropriate tests to order, and treatment plan options.

Keywords: Obesity hypoventilation syndrome, OHS, obesity, treatment, Pickwickian Syndrome

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OBESITY HYPOVENTILATION SYNDROME:

UNDERSTANDING, DIAGNOSING, AND TREATING

INTRODUCTION

A recent study from the U.S. Department of Health and Human Services (2014) found more than two-thirds (68.8%) of Americans, ages 20 years and older, are considered overweight (Body Mass Index or BMI >25 kg/m^2). It is projected by 2030, 42% of U.S. citizens will be considered obese (BMI >30 kg/m^2) and 11% extremely obese (BMI >40 kg/m^2). In addition, Finkelstein and colleagues (2012) estimate that over the next 20 years there will be a 33% increase in obesity and a 130% increase in extreme obesity. Obese individuals are more prone to respiratory complications, even without a history of underlying lung disease. Demand on the lungs in these patients' leads to deteriorating respiratory muscle function. However, the exact link between increased respiratory complications and obesity is not completely understood (Borel et al., 2012).

These numbers are concerning because obese adults are at a greater risk for comorbidities such as: arrhythmia, asthma, chronic kidney disease, chronic obstructive pulmonary disease, diabetes, gastroesophageal reflux disease, hyperlipidemia, hypertension, hypothyroidism, and trans-ischemic attack or stroke (Akinnusi, Saliba, Porhomayon, & El-Solh, 2012; Macavei, Spurling, Loft, & Makker, 2013). In addition, Vucenik and Stains (2012) suggest "colon, endometrium, postmenopausal breast, kidney, esophagus, pancreas, gallbladder, liver, and hematological malignancy" (p.38) are common cancers often associated with obesity.

Most research related to obesity and respiratory disorders focus on Obstructive Sleep Apnea (OSA), with its associated risk of cardiovascular disease, stroke, and death. In fact, OSA

is the most commonly diagnosed respiratory complication of obesity. Currently OSA is diagnosed in 70-95% of obese patients (Riad & Chung, 2013).

With the prevalence of OSA and increasing obesity rates, a lesser known disease, Obesity Hypoventilation Syndrome (OHS), is widely misunderstood and often misdiagnosed as OSA (Borel et al., 2012). However, OHS occurs when obesity (BMI > 30 kg/m^2) is seen along with daytime hypercapnia (PaCO$_2$ >45 mm Hg) and various sleep-disordered breathing problems, with no underlying lung disease. OHS carries a greater mortality risk than OSA, and is fatal in nearly one in four patients (Budweiser, Riedl, Jorres, Heinemann, & Pfeifer, 2007). However, the diagnosis of OHS is often missed due to the low number of case studies; more importantly, diagnosis is delayed due to unfamiliarity with the disease process and diagnostic criteria (Borel et al., 2012). While improper diagnosis precludes an accurate count of patients who suffer from OHS, the prevalence of the disease is estimated to be 10-20% in the obese patient population and 0.15-0.3% in the general population (Chau, Lam, Wong, Mokhlesi, & Chung, 2012). This estimate translates to several hundred thousand people with OHS, most of whom have never been diagnosed (Piper, 2011).

With a growing obese population, it is important for a nurse practitioner to accurately diagnose OHS. Therefore, this paper will initially present a brief overview and associated diagnostic criteria of OSA since it is often considered before OHS is diagnosed, and then provide information on the history and misconceptions associated with OHS, patients at risk, diagnostic criteria, and potential treatment.

OBSTRUCTIVE SLEEP APNEA -- OSA

In 1965, the identification of OSA was considered to be the most important discovery in sleep medicine to date (Bahammam, 2011). As the most common sleep related breathing disorder, OSA occurs in a wide variety of patients, although a majority--roughly 70% according to some studies, and as high as 95% in others--are obese (Akinnusi et al., 2012; Rakel, 2009; Riad & Chung, 2013). It is defined as a recurrent collapse of the pharyngeal airway during sleep, leading to reduced or complete occlusion of airflow (Strohl, 2014). Patients often present with nocturnal symptoms: loud snoring; witnessed apneas, which end with a snort; sudden arousal from sleep with a choking, or gasping sensation; and insomnia (Downey III, Gold, Rowley, & Wickramasinghe, 2014). Daytime symptoms may include irritability, depression, morning headaches, awakening with a dry mouth or a sore throat, and neurocognitive impairments, such as sleepiness, forgetfulness, and impairment in memory, attention, vigilance, and executive function (Jackson, Howard, & Barnes, 2011; Kline, 2013). However, these symptoms do not need to be present for a diagnosis of OSA.

If a patient presents with the above named symptoms, the nurse practitioner should begin by conducting a thorough patient history and physical assessment. The assessment should include a detailed discussion of sleep habits and patterns. Physical exam is often normal in these patients, other than the presence of obesity, a large circumferential neck, and hypertension. The oral assessment should include a Mallampati score, since it is the most commonly used screening tool of an obstructive airway, and it is used for anesthesia airway evaluation and tonsillar hypertrophy grading in OSA evaluation (Moses, 2012). Scoring criteria includes assessment of the soft palate, fauces, uvula and tonsillar pillars (Mahmoodpoor et al., 2013)

If OSA is suspected a home sleep study or overnight polysomnogram must be conducted to confirm diagnosis. The polysomnogram will provide objective data regarding labored, obstructive, or apneic sleep related breathing (McNicholas, 2008).

OBESITY HYPOVENTILATION SYNDROME -- OHS

OHS is often thought to be and commonly misdiagnosed as OSA, as it is thought to predate and be a prerequisite for developing OHS; and, in fact, is seen in roughly 90% of patients diagnosed with OHS (Fayyaz & Lessnau, 2013). However, OHS has been documented since the early 1850's, and in the past was referred to as Pickwickian Syndrome. For example, in his 1856 *The Posthumous Papers of the Pickwick Club*, Charles Dickens described the character "Joe" as an overweight young man constantly falling asleep, no matter what he was doing (Mokhlesi, 2010). Burwell, Robin, Whaley, and Bickelmann (1956) coined the medical term "Pickwickian Syndrome" in 1956 to reflect this description of "Joe". It was based on one of Burwell's medical cases in which the patient's physical description resembled "Joe," and that he fell asleep holding a full house in a game of poker (Morgan & Zwillich, 1978).

OHS patients often complain of the same symptomology seen with OSA. However, only 4-20% of OSA patients have OHS. Furthermore, the respiratory disorders and complications associated with OHS differ than those seen in OSA (Martin, 2012).

Specifically, OHS is clinically defined as an obese patient, with chronic daytime alveolar hypoventilation, and no underlying lung disease. Chau et al. (2012) suggest "Daytime hypercapnia is the distinguishing feature of OHS that separates it from simple obesity and OSA" (p. 190) and is directly related to hypoventilation. Furthermore, sleep hypoventilation alone does not classify a patient as having OHS (Martin, 2012; Piper & Grunstein, 2011).

Daytime alveolar hypoventilation causes a prolonged, chronic hypoxia and often leads to a triad of polycythemia, pulmonary hypertension, and right-sided heart failure (cor pulmonale) (Naim & Wallace, 2010). Furthermore, OHS patients more frequently suffer from acute respiratory distress, congestive heart failure, pulmonary hypertension; increased psychiatric disturbances (such as increased paranoia, agitated depression, and hostility); worsening neurocognitive impairment, and diabetes than those diagnosed with OSA (Borel et al., 2012; Morgan & Zwillich, 1978).

OHS patients also exhibit obesity related impairments including a diminished respiratory drive; hypoxia, sleep disturbed breathing, and three types of respiratory abnormalities (Martin, 2012). Piper (2011) classifies OHS respiratory abnormalities as: pulmonary function, ventilatory control, and sleep disordered breathing.

Alterations in pulmonary function occur, as fat builds up around the abdomen and chest. This leads to decreased tidal volumes; total lung volume, expiratory reserve, and residual capacity. Low lung volumes reduce chest wall and lung compliance, increasing airway resistance. Therefore, OHS patients work harder to breathe at rest than normal weight or obese patients with OSA. In a supine position, OHS patients experience further difficulties and restrictions in breathing. As these impairments in respiratory function occur, breathing patterns change and consist of smaller tidal volumes combined with a higher respiratory rate (Piper, 2011).

In addition, Piper (2011) notes impaired ventilatory control and breathing rates increase as body weight increases; correspondingly, the same amount of weight on a healthy person's chest increases their respiratory drive. In OSA, patient's ventilatory control is augmented due to upper airway obstruction, resulting in hypoxemia. In OHS, patients fail to compensate for the

added excess weight allowing for a rise in CO_2. OHS patients also differ from OSA patients in that they become hypoxemic with or without obstructive events. Frequent arousal occurs in OHS patients as they experience severely fragmented sleep due to repetitive respiratory events (Piper, 2011).

The final respiratory abnormality, according to Piper (2011), is sleep-disordered breathing. Sleep-disordered breathing refers to any abnormal respiratory pattern including hypopnea (decrease of at least 50% in depth and rate of breathing), change in respiratory effort due to arousal, or hypoventilatory events, which occur during sleep (Al Dabal & Bahammam, 2009; Farre, Rigau, Montserrat, Ballester, & Navajas, 2001). Patients may suffer hypoxemia due to sleep-disordered breathing, or exhibit signs of disturbed sleep, such as those described with OSA (Rinaldi, Casale, Faiella, Pappancena, & Salvinelli, 2013; Strohl, 2014).

CLINICAL WORKUP

Nurse practitioners routinely see overweight and obese patients who may be at risk for OSA and OHS. Due to high mortality rates associated with OHS, nurse practitioners should become familiar with proper OHS diagnostic criteria as these present with symptoms and physical findings similar to OSA, making it indistinguishable from OSA (Piper & Yee, 2014). Al Dabal and Bahammam (2009) suggest a nurse practitioner will see a classic presentation: a middle-aged, obese patient, more commonly male than female, with excessive daytime sleepiness, neurocognitive impairment, and complaints of OSA related symptoms. Later symptoms include signs of pulmonary hypertension, such as exertional dyspnea and lower extremity edema. For definitive OHS diagnosis the criteria are specific and must include an obese patient and daytime hypercapnia, with no underlying lung disease (Surrat, 2013).

Therefore, further diagnostic testing is needed to exclude other causes of daytime hypercapnia and hypoventilation (Piper & Yee, 2014).

OHS patients should be evaluated for potential differential diagnoses, also causing hypoventilation such as: primary pulmonary disease (chronic obstructive pulmonary disease, interstitial lung disease, or tracheal stenosis); chest wall disorders (kyphoscoliosis, or thoracoplasty); neuromuscular disorders (muscular dystrophies, Guillain-Barret, amyotrophic lateral sclerosis, myasthenia gravis, or cervical spine injury); primary CNS disorders (primary central hypoventilation syndromes, or brain stem infarction or tumor); myxedema; drugs; or metabolic abnormalities (hypokalemia, hypomagnesaemia, metabolic acidosis) (Naim & Wallace, 2010).

After a complete history and physical, diagnostic tools are available, which can further help differentiate between OSA and OHS. These include screening questionnaires, laboratory testing, polysomnography, pulmonary function testing, and imaging. A discussion of these measures follows.

Screening

OSA is seen in 90% of OHS patients. Therefore, an effective way to screen for potential OSA and OHS is use of the Stop-Bang questionnaire and the Four-variable Tool (Chung et al., 2012; El-Sayed, 2012). The STOP-Bang questionnaire is the most sensitive instrument available for identifying patients with moderately severe and severe OSA; while the Four-variable Tool is the most effective measure in ruling out OSA (Silva, Vana, Goodwin, Sherrill, & Quan, 2011). While there is not a known or specific OHS screening instrument, clinical findings combined with a positive OSA questionnaire may suggest a diagnosis of OHS. Clinical findings in an OHS

patient may include a BMI >30 kg/m^2 with a positive OSA questionnaire, signs of pulmonary hypertension, increasing neurocognitive impairment, or psychiatric disturbances (Morgan & Zwillich, 1978).

Laboratory testing

Laboratory testing to rule out other causes of daytime hypercapnia and hypoventilation should be completed. This includes a chemistry panel, complete blood count, arterial blood gas, and thyroid function panel. A discussion of each follows.

A chemistry panel provides information about metabolic imbalances and electrolyte abnormalities. Specifically, oxygen consumption increases with weight gain and an obese habitus, causing an increase in carbon dioxide (CO_2), which is the hallmark of OHS (Powers, 2010). Therefore, a total CO_2 level can be drawn; however, a chemistry panel is also helpful in ruling out other causes of hypoventilation. The total CO_2 content includes the serum bicarbonate, carbonic acid, and dissolved CO_2. Serum bicarbonate comprises roughly 95% of the total CO_2 content making it an excellent reflection of the serum bicarbonate (HCO_3) level (Centor, 1990). Due to the chronic respiratory acidosis, a compensated metabolic alkalosis is seen in OHS. A subtle increase in the chemistry serum CO_2 may be an early sign warranting further investigation into the cause (Dugdale, 2013; Olson & Zwillich, 2005). Major electrolyte imbalances, for example, hypomagnesaemia, hypocalcaemia, or hypophosphatemia, can lead to neuromuscular weakness causing hypercapnia (Lee & Mokhlesi, 2008; Surrat, 2013).

A complete blood count (CBC) allows for assessment of hypoventilation causes, such as anemia and polycythemia. OHS patients may present with secondary polycythemia related to chronic hypoxia. Laboratory findings will include an elevated hematocrit, (>45 in women or >52

in men), or hemoglobin, (>16.5 in women or >18.5 in men) (Tefferi, 2014), which is a response

to chronic cellular hypoxemia and is reliant on transcription hypoxia-inducible factor (HIF)-1.

HIF-1 regulates cellular oxygen hemostasis; and in combination with chronic cellular hypoxia

allows adaptive genes, such as erythropoietin and vascular endothelial growth to generate. HIF-1

causes an increase in gene expression leading to an abnormal response, such as polycythemia

(Kent, Mitchell, & McNicholas, 2011).

Arterial blood gases (ABG) are the gold standard in assessing pulmonary status and

alveolar ventilation (Al Dabal & Bahammam, 2009; Martin, 2014). An ABG in a hypercapnic

OHS patient will show an elevated $PaCO_2$ ($PaCO_2 > 45$ mmHg) reflecting chronic respiratory

acidosis. Compensating metabolic alkalosis will be reflected in an elevated HCO_3 ($HCO_3 > 26$).

The OHS patient will often have associated hypoxemia with a low a low PaO_2 ($PaO_2 < 70$)

(Mokhlesi, Kryger, & Grunstein, 2008).

Another contributing factor, which may affect OHS patients is hypothyroidism, (TSH >

4-5 mU/l) (Ross, 2013). Hypothyroidism in OHS leads to decreased chemo-responsiveness,

stimulation of chemical receptors, causing OSA; due to macroglossia, upper airway muscle

dysfunction, and myopathy or neuropathy of respiratory muscles. Therefore, all OHS patients

should be screened for hypothyroidism; as OSA complications associated with hypothyroidism

in OHS may be improved with thyroid replacement (Martin, 2012).

Polysomnography

Obese patients with difficulty breathing, nocturnal symptoms, or clinician suspicion of

OSA or OHS, need to undergo an overnight polysomnography. Polysomnography analyzes,

monitors, and records physiological data of the patient's sleep and wakefulness patterns during

the study (AAST, n.d.). During a polysomnogram OSA is classified as: mild OSA (asymptomatic with five to 15 respiratory events; apneic, hypopneic, or other sleep disturbed events; per hour of sleep), moderate OSA (symptomatic with 15 to 30 events per hour of sleep), or severe OSA (symptomatic with greater than 30 events per hour of sleep) (Kline, 2013). During sleep OSA and OHS patients experience obstructive hypoventilation, and periods of hypoxia, and severe hypoxia. Patients with OHS have an abnormal number of apneic and hypopneic events each hour and a more significant drop in oxyhemaglobin, (oxygenated arterial blood) (American Heritage Dictionary, 2007; Surrat, 2013). Of importance, in OHS patients, these symptoms continue after treatment of obstructions, either via surgery or non-invasive positive airway pressure ventilation (Naim & Wallace, 2010).

Pulmonary Function Testing

Pulmonary function testing (PFT) provides information on the severity of obstructive lung disease. The ratio of forced expiratory volume in one second (FEV-1) to forced vital capacity (FVC) is reduced in airflow obstruction. Therefore, lung volume measurements provide information of functional volume residual, forced lung capacity, and residual volume. Increases in pressures may suggest obstructive pulmonary disease (Fayyaz & Lessnau, 2013).

Imaging

Chest radiography, electrocardiograms, and echocardiograms may be helpful in diagnosing chronic hypercapnia. The location and shape of the diaphragm may assist in evaluation of other disease processes. Lung hyperinflation and flattened diaphragms can suggest a diagnosis of COPD. Bilateral elevated hemidiaphragms, due to an obese abdomen, and an

enlarged heart, due to right ventricular hypertrophy, is commonly seen. An asymmetrical diaphragm suggests diaphragmatic paralysis, which may cause hypoventilation. Hyperinflation and bullous disease, an air space in the lung, which when distended measure larger than one centimeter in diameter, may indicate hypercapnia due to pulmonary disease and not OHS (Martinez, 2013; Surrat, 2013).

Chronic hypoxia may lead to pulmonary vascular remodeling, vascular resistance, and pulmonary congestion, causing pulmonary hypertension, right ventricular and atrial hypertrophy. Electrocardiograms and echocardiograms are useful in the diagnosis of cardiac hypertrophy. While, cardiac catheterization may also help diagnose the extent of pulmonary hypertension (Weitzenblum, 2003). With a suspected central cause of hypoventilation, a brain CT and MRI are indicated.

TREATMENT

Several treatment modalities are available for OHS patients. These include non-invasive positive airway pressure ventilation (NIPPV), tracheostomy, and/or weight loss. In addition, Mokhlesi et al. (2008) and Al Dabal and Bahammam (2009) suggest pharmacotherapy, such as medroxyprogesterone or acetazolamide, may help. Treatment implementations should be based on the patient's clinical presentation, while improving a specific impairment, dysfunction, or handicap (Borel et al., 2012; Mokhlesi et al., 2008). A discussion of these treatment modalities follows.

Non-invasive Positive Pressure Ventilation (NIPPV)

First line treatment of OHS is non-invasive positive pressure ventilation (NIPPV), such as Continuous Positive Airway Pressure (CPAP) or Bi-level Positive Airway Pressure (BiPAP). These devices eliminate airway obstructions by increasing the caliber of the retropalatal and retroglossal regions of the upper airway (Borel et al., 2012). Furthermore, airflow increases the lateral dimensions of the upper airway and thins the lateral pharyngeal walls, which are thicker in obese patients with sleep disordered breathing (Downey III et al., 2014). Improvement occurs in daytime hypercapnia and hypoxia; in some instances as quickly as one month (Naim & Wallace, 2010). In OHS patients, lung compliance and chest wall expansion are impaired, due to the patient's body habitus and weight on their lungs, while in a recumbent position.

NIPPV treatment improves lung compliance, chest wall expansion, and central ventilatory drive (the body's response to the rise and fall of CO_2) during sleep (Al Dabal & Bahammam, 2009; Dellborg, Olofson, Hamnegard, Skoogh, & Bake, 2000). This is done in two ways; first by decreasing stress on ventilatory muscles, which in turn, allows patients to breathe easier. This results in less daytime fatigue (Martin, 2014). Second, NIPPV can augment ventilatory drive by alleviating sleep fragmentation, or repetitive, short, sleep interruptions; and nocturnal asphyxia (Smurra, Dury, Aubert, Rodenstein, & Liistro, 2001).

OHS patients must be carefully monitored for compliance with NIPPV therapy. With each office visit, the provider should assess for factors that may interrupt treatment. It is essential to educate the patient on the health benefits associated with NIPPV therapy. The patient may view the therapy as intrusive and it may take several trials to obtain a comfortable nasal or facial mask fit. Second, an inadequate NIPPV titration or pressure settings will not properly ventilate or oxygenate the patient; consequently, negating the benefit of therapy. Finally, lack of

improvement after NIPPV use, may be due to undiagnosed hypothyroidism, neuromuscular diseases, or metabolic acidosis (Mokhlesi & Tulaimat, 2007).

Tracheostomy

A small group of patients may benefit from a temporary tracheostomy, specifically, those intolerant to NIPPV therapy or in life threatening situations. A tracheostomy bypasses upper airway obstructions and was thought to lower CO_2 levels. However, due to a high potential of external blockage, secondary to increased neck adipose tissue, tracheostomy placement in an obese patient carries a higher risk of life threatening complications (Piper, 2011). Prior to 1980, tracheostomy was thought as the most effective and advanced treatment of OSA and OHS (Bahammam, 2011). Current studies agree a tracheostomy should be placed as last resort in OHS patients and not considered a viable long-term option in any situation (Lee & Mokhlesi, 2008; Martin, 2012; Piper, 2011; Piper & Grunstein, 2011).

Pharmacotherapy

The third method of treating OHS is pharmacotherapy. Two medications, medroxyprogesterone and acetazolamide, used in conjunction with NIPPV, may increase ventilatory response, augment the body's pH, or stimulate the respiratory system (Al Dabal & Bahammam, 2009; Mokhlesi et al., 2008; Mokhlesi & Tulaimat, 2007).

Medroxyprogesterone, a synthetic progesterone derivative, improves breathing by increasing the ventilatory response to hypercapnia. This in turn, drops the $PaCO_2$ level, increases the PaO_2, and provides significant improvement in ventilation and oxygenation (Al Dabal & Bahammam, 2009; Saaresranta, Irjala, & Polo, 2002).

Acetazolamide, a carbonic anhydrase inhibitor, is a mild diuretic. In OHS, patients have a chronic respiratory acidosis with compensatory metabolic alkalosis. The primary effect of acetazolamide drops the serum pH, by 0.05-0.1, and serum bicarbonate (HCO_3), by 4-6 mEq/L, via excretion of bicarbonate in the urine, within 24 hours (Mokhlesi & Tulaimat, 2007). A secondary effect of acetazolamide, is improvement of minute ventilation rates, by roughly 15%, which reduces PaCO2 by 5-6 mm Hg. Finally, acetazolamide appears to reduce frequency of obstructive events during sleep (Mokhlesi & Tulaimat).

There is limited research on the effects of medroxyprogesterone and acetazolamide in the long-term treatment of OHS. Currently no strong recommended use of these medications can be made; however, use should only occur in conjunction with NIPPV therapy, and only if other OHS treatment modalities have failed (Al Dabal & Bahammam, 2009). These medications may temporarily improve ventilation; but, they are not effective as a standalone therapy, nor considered first line therapy (Lee & Mokhlesi, 2008).

Weight loss

A final treatment modality is weight loss. Obesity associated with OHS places strain on the lungs and increases work of breathing, due to decreased lung compliance and respiratory muscle impairment. As work of breathing increases, lung compliance decreases due to respiratory muscle fatigue. In this state, OHS patient are unable to maintain or sustain the effort required to move the rib cage and diaphragm, leading to hypoventilation (Surrat, 2012).

Weight loss provides many benefits for obese patients suffering from sleep disordered breathing. For example, a weight gain or loss of 10% significantly increases or decreases breathing dysfunction (Borel et al., 2012). In addition, a 10 kg weight loss improves pulmonary

function by increasing forced vital capacity and expiratory volume (Al Dabal & Bahammam, 2009). Weight loss improves lung compliance, respiratory muscle impairment, and pulmonary function. Of note, calorie counting, exercise, and lifestyle changes are all viable options for weight loss, although adherence to these measures does not always occur and often weight is regained (Martin, 2012)

OHS patients require a multi-disciplinary team approach in order to achieve successful and sustained weight loss and modified lifestyle changes (Surrat, 2013). Weight loss, in conjunction with increased physical activity, improve the OHS patients overall health, metabolic profile, body muscle and respiratory function, and decreases comorbidities (Borel et al., 2012).

The most effective and successful long-term weight management for OHS patients is bariatric surgery. Bariatric surgery includes the following procedures: gastric bypass, gastric sleeve resection, adjustable gastric banding, biliopancreatic diversion, duodenal switch, intragrastric balloon, and jejunoileal bypass (Martin, 2012; Piper, 2011). However, the OHS patient is at greater risk for developing peri- and post- operative complications. These complications include: surgical injury to bowel, liver, and spleen; respiratory failure, pulmonary emboli, pneumonia, myocardial infarction, wound infection, and urinary tract infections (Bamgbade, Rutter, Nafiu, & Dorje, 2007).

With these associated risks and complications, the mortality rate with bariatric surgery is less than 2% and dependent on current comorbidities (Martin, 2012). Therefore, the benefits of bariatric surgery, including: successful weight loss, improved quality of life, longevity, reduction of cancer mortality by 60% and diabetes mortality by 90%, outweigh the risks (ASMBS, 2014).

After surgery, significant weight loss, and lifestyle changes, bariatric surgery patients with OHS, are revaluated after one- to two- years (Martin, 2012). At this point, many patients

have lost weight, see improvement in respiratory function, and experience fewer comorbidities (Mokhlesi, 2010). Currently, follow-up visits show significantly improved oxygenation (SpO2), forced expiratory volume (FEV), and forced expiratory volume in one second (FEV1) (Lumachi et al., 2010). Other studies show increased mean PaO2, decreased mean PaCO2, and reduced pulmonary hypertension, related to weight loss. However, long-term outcome and success rates, in OHS patients after bariatric surgery, are limited (Marik, 2012; Naim & Wallace, 2010; Piper & Grunstein, 2011).

CONCLUSION

Nurse practitioners often see obese and overweight, patients who may be at risk for OSA and OHS. While, OSA is commonly recognized in the medical community, many providers are unaware of OHS and its serious complications. OHS is often misdiagnosed, undertreated and thought of as severe OSA, as both carry a similar patient symptomology (Borel et al., 2012). While OSA is extremely prevalent among the obese population, the mortality rate of OHS is one in four patients. Furthermore, 90% of OHS patients have an underlying OSA; while less than 20% of OSA, patients have OHS. Classic presentation of OHS includes: a middle aged, obese patient, more commonly male than female; with excessive daytime sleepiness, neurocognitive impairment, and complaints of OSA symptoms. Additional symptoms include: daytime hypercapnia, various sleep-disordered breathing problems, obstructive hypoventilation, and severe hypoxia during sleep. These symptoms are present with no underlying lung disease and continue to occur after treatment (Chau et al., 2012; Martin, 2012).

It is important nurse practitioners recognize the difference between OSA and OHS. This includes understanding the diagnostic criteria; appropriate tests to order, and treatment plan

options. Treatment modalities for patients with OHS include: NIPPV therapy; pharmacotherapy in conjunction with NIPPV treatment; temporary tracheostomy, and weight loss through bariatric surgery (Al Dabal & Bahammam, 2009; Piper, 2011; Piper & Grunstein, 2011). Current treatment recommendations should include NIPPV as first line therapy and bariatric surgery to decrease associated comorbidities. These treatments have shown an 89% decrease in overall mortality, with a decrease in cancer mortality, by 60%; mortality associated with diabetes, by 90%; and mortality from heart disease by 50%; in obese patients (ASMBS, 2014).

These complex patients will require an interdisciplinary team approach. The interdisciplinary team should include the nurse practitioner, pulmonologist, respiratory therapist, physical therapist, dietician, and bariatric surgeon. Further research of OHS treatment is needed, as there are few long-term studies of patient outcomes.

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