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Obesity Hypoventilation Syndrome and Obstructive Sleep Apnea

An APCCMPD Ambulatory Care Curriculum Teaching Script

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Obesity Hypoventilation Syndrome and Obstructive Sleep Apnea

Scenario 1:

A 51-year-old morbidly obese man with mild persistent asthma presents for a yearly visit in the pulmonary clinic. He denies any recent wheezing, cough, or shortness of breath, but complains of fatigue that has been slowly worsening over the past year. He is embarrassed because he recently fell asleep during an important meeting at work. His wife made the trip with him today because she is concerned about him "zoning out" while driving.

Question 1: What is a differential diagnosis for the patient's sleepiness? What symptoms suggest a sleep disorder in this patient, and what other questions should be asked?

Scenario 1 (Continued):

This **51-year-old male** complains of sleepiness for at least a year. It is worst when he is sedentary, and he often falls asleep while reading or watching television. Recently, he has **fallen asleep several times at his job** as a software engineer, including during an important meeting. On days when he is especially tired, he uses his lunch break to take a quick nap in his car before returning to work. He has **difficulty staying awake while driving longer distances**, and recently, he dozed off momentarily and hit the rumble strips on the side of the highway. He usually gets into bed at 10:30 PM (though he often falls asleep earlier while watching TV) and falls asleep "instantly." He occasionally **awakens feeling as if he is choking or suffocating**. This sensation goes away quickly once he is awake. **He wakes up 3-4x per night to urinate.** He does not notice nocturnal wheezing and does not have nocturnal leg cramps. He wakes up in the morning at 6:30 AM using the alarm clock, but often hits the snooze until around 7 AM. He usually **feels in the morning as if he "barely slept."** He often has a **dry mouth and a headache** upon awakening. His wife notes that he **snores loudly.** Because of his snoring, she sometimes sleeps in the guest bedroom. On the weekends, he sleeps until 8:30 AM or 9 AM and tries not to nap, but often finds himself dozing off for up to an hour if he is not keeping active. No matter how long he sleeps, he rarely feels rested. **His Epworth Sleepiness Score is 13.** With regards to his asthma, he denies any exercise limitation ("it's my knees and my weight that keep me from walking more, doc"), recent episodes of wheezing or significant cough. He uses his rescue inhaler infrequently (less than once weekly). He does not smoke cigarettes.

Exam shows:
BP 145/85, HR 75, RR 18, **Weight 345 lbs., Height 5'10"**
General: morbidly obese middle-aged man, face somewhat flushed, in no distress

Neck circumference 19 inches, BMI 49.5 kg/m^2
HEENT: PERRL, **injected conjunctiva, large uvula, narrowing of the peritonsillar lateral walls and tongue ridging**, Mallampati III
Lungs: breath sounds clear to auscultation throughout, no rales, rhonchi, or wheezes
Cards: heart sounds regular, no murmurs, gallops, rubs
Abdomen: soft, non-tender, obese, bowel sounds present
Extremities: no clubbing or cyanosis, trace pedal edema
Neuro: non-focal exam, DTRs intact

Question 2: What is the most likely diagnosis for his sleepiness? What are his risk factors for each?

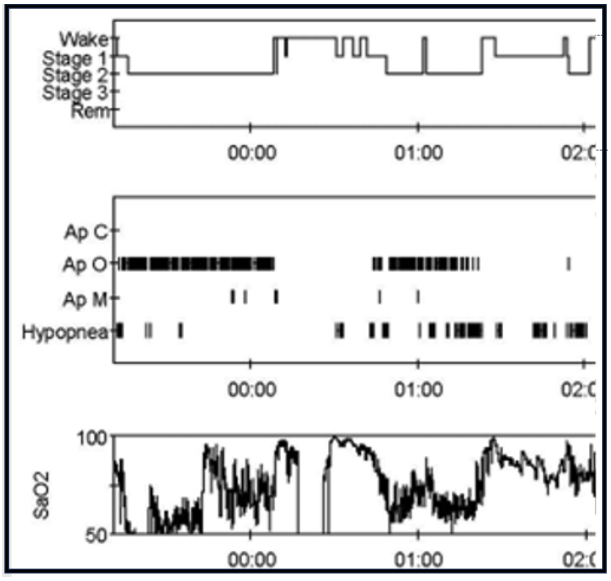
Question 3: What types of tests can be used to confirm a diagnosis of sleep apnea?

Scenario 1 (Continued):

The patient underwent a sleep study that demonstrated the following: his latency to sleep onset was <2 minutes; his sleep efficiency was 89%. He had fragmented sleep with 61 arousals/hour. His Apnea-Hypopnea Index (AHI) was 90 events/hour. There were no periodic limb movements.

His tracing is shown below:

Figure 2. Patient Hypnogram Report

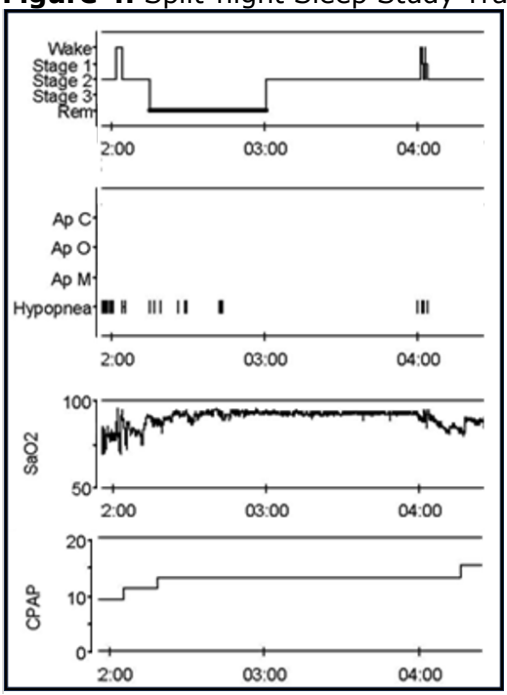


Question 4: What do the tracings demonstrate?

Question 5: What options exist for treating OSA?

Question 6: What does the tracing below demonstrate?

Figure 4. Split-night Sleep Study Tracing



Used with permission from W. Merwin, MD.

Question 7: What is the most likely explanation for this patient's persistent hypoxemia?

Question 8: What diagnostic tests can be used to confirm OHS?

Question 9: What treatment options exist for OHS?

Question 10: How would you approach starting this patient on PAP therapy?

Obesity Hypoventilation Syndrome and Obstructive Sleep Apnea

Educational Objectives:

1. Review the key differences in examination, laboratory values, and diagnostic testing between obesity hypoventilation syndrome (OHS) and obstructive sleep apnea (OSA)
2. Understand the different treatment options for OHS and OSA
3. Review different non-invasive ventilation techniques available

Scenario 1:
A 51-year-old morbidly obese man with mild persistent asthma presents for a yearly visit in the pulmonary clinic. He denies any recent wheezing, cough, or shortness of breath, but complains of fatigue that has been slowly worsening over the past year. He is embarrassed because he recently fell asleep during an important meeting at work. His wife made the trip with him today because she is concerned about him "zoning out" while driving.

Question 1: What is a differential diagnosis for the patient's sleepiness? What symptoms suggest a sleep disorder in this patient, and what other questions should be asked?

Sleep disordered breathing (SDB) refers to abnormalities of breathing during sleep, including obstructive sleep apnea (OSA), central sleep apnea (CSA), obesity hypoventilation syndrome (OHS), and sleep-related hypoventilation. The diagnosis of SDB can be very complex. Table 1 details pertinent patient history features and their associated sleep-related pathology, including diagnoses that span outside of SDB, as you may encounter them in the clinic. However, for this script, the discussion will focus on OSA and OHS as they are commonly evaluated and managed in the pulmonary clinic.

Table 1. Patient History Features and Sleep-Related Diagnosis

Symptom	Diagnosis & Key Features
Lack of sleep related to lifestyle, behavioral, or environmental factors	**Insufficient Sleep** — Common in young adults (residents/fellows). Sleep is essential for health. The AASM/SRS recommend ≥7 hours/night on a regular basis for adults.[1]
Snoring, nocturnal awakening due to gasping/choking, nocturnal hypoxia, apneic events	**Obstructive Sleep Apnea (OSA)** — Snoring occurs in 70–90% of patients. Persistent respiratory effort against an occluded airway. If suspicious for OSA, screen using STOP-BANG or DOISNORE50.[2-6]
Apneic events; snoring less prominent or absent	**Central Sleep Apnea (CSA)** — Characterized by lack of respiratory effort during apneas. Often occurs at high altitude (>2,000 m), in heart failure, or post-stroke patients.[6-8]
Normal sleep time but frequent interruptions	**Sleep Fragmentation** — A patient may sleep 8–9 hours yet have poor-quality rest if frequent awakenings disrupt N3 or REM sleep. Ask what causes awakenings (wheezing, nocturia, reflux, post-nasal drip, gasping). Sleep apnea and limb-movement disorders can also fragment sleep.[3,6,9]
Difficulty going to sleep, staying asleep, or early-morning awakenings >3 nights/week for >3 months	**Chronic Insomnia** — Multifactorial: can occur with psychiatric comorbidities (depression, anxiety), chronic pain, cardiopulmonary disease, medications, and environmental/situational stressors. Excessive screen time prior to sleep may worsen sleep quality. Insomnia without coughing/gasping may still coexist with sleep-disordered breathing in up to 50% of patients.[1,9]
Leg pain/movements during sleep	**PLMS / PLMD** — PLMS = repetitive movements of lower limbs during sleep, measured by polysomnography. PLMD = PLMS with sleep disturbance. **Restless Legs Syndrome (RLS)** — Clinical diagnosis (does NOT require polysomnogram): urge to move legs, often with unpleasant sensations; worsens with rest/inactivity, improves with movement, worsens in the evening or night.[10]
Morning headaches, orthopnea, elevated bicarbonate on CMP, signs of right heart disease	**Obesity Hypoventilation Syndrome (OHS)** — Defined by obesity (BMI > 30) and daytime hypercapnia ($PaCO_2$ > 45 mm Hg) not due to other causes (e.g., COPD, neuromuscular disease). Associated with sleep-disordered breathing in ~90% of patients.[11-14]
Sleep paralysis, cataplexy, hypnagogic hallucinations	**Narcolepsy** — Cataplexy (emotion-triggered weakness) is almost pathognomonic, though not present in all patients.[15]

**OSA, Obstructive Sleep Apnea; CSA, Central Sleep Apnea; PLMS, Periodic Limb Movements of Sleep; PLMD, Periodic Limb Movement Disorder; RLS, Restless Legs Syndrome; OHS, Obesity Hypoventilation Syndrome.*
Used with permission from A. Simon, MD.

When clinical suspicion for obstructive sleep apnea is high, as in patients with risk factors like morbid obesity and daytime somnolence, screening instruments such as STOP-BANG or DOISNORE50 can help quantify risk and guide further evaluation.

***The mnemonic for STOP-BANG.[4] One point is given for each feature, if present.**
S – Snoring Loudly (loud enough to be heard through a closed door)
T – Tiredness
O – Observed Apnea
P – Pressure (HTN)
B – BMI > 35 kg/m2
A – Age > 50 years
N – Neck circumference (>16 inch/40 cm)
G – Gender (male)

At scores from 0 to 2 the probability of moderate to severe OSA is 18%.
At scores from 7 to 8 the probability of moderate to severe OSA is 60%.

The sensitivity of STOP-Bang ≥ 3 for moderate OSA is 93% and 100% for severe OSA. **In general, a score < 3 predicts low-likelihood of OSA and score ≥3 predicts high-likelihood of OSA.**

While the STOP-BANG is commonly used, DOISNORE50 has been validated perioperatively to identify patients who may develop respiratory or neurological issues after surgery.[5]

***The mnemonic for DOISNORE50. One point is given for each feature, if present.**
D – Diseases (Stroke/afib/hypertension)
O – Observed apnea
I – Insomnia
S – Snoring
N – Neck circumference over 18 inches
O – Obesity
R – Are you male
E – Excessive daytime sleepiness
50 – Age ≥ 50

A cutoff of > 6 was associated with a sensitivity of 58% and 81% specificity for OSA.

The amount of sleepiness can be reported by patients using the Epworth Sleepiness Scale, as seen in Figure 1.[16] It can be helpful to quantify the degree of daytime sleepiness and monitor response to treatment. The score ranges from 0 to 24, with scores >10 consistent with pathologic sleepiness.

Figure 1. Epworth Sleepiness Scale Questionnaire

The Epworth sleepiness scale

THE EPWORTH SLEEPINESS SCALE

Name: _____
Today's date: _____ Your age (years): _____
Your sex (male = M; female = F): _____

How likely are you to doze off or fall asleep in the following situations, in contrast to feeling just tired? This refers to your usual way of life in recent times. Even if you have not done some of these things recently try to work out how they would have affected you. Use the following scale to choose the *most appropriate number* for each situation:

0 = would *never* doze
1 = *slight* chance of dozing
2 = *moderate* change of dozing
3 = *high* chance of dozing

Situation	Chance of dozing
Sitting and reading	_____
Watching TV	_____
Sitting, inactive in a public place (e.g. a theater or a meeting)	_____
As a passenger in a car for an hour without a break	_____
Lying down to rest in the afternoon when circumstances permit	_____
Sitting and talking to someone	_____
Sitting quietly after a lunch without alcohol	_____
In a car, while stopped for a few minutes in the traffic	_____

Adapted from Mokhlesi B, Masa JF, Brozek JL, et al. Evaluation *and management of obesity hypoventilation syndrome: an official American Thoracic Society clinical practice guideline.* Am J Respir Crit Care Med. *2019;200(3):e6-e24. doi:10.1164/rccm.201905-1071ST*

In addition to the risk factors discussed above, OSA is associated with:

- Age: Prevalence of OSA increases from adulthood through 6-7th decade of life[17]
- Male sex: It is 2-3x more common in men than women. This gender gap does narrow after menopause in women[18-19]
- Obesity: 10% increase in weight has shown to be associated with 6-fold increase in incidence of OSA.[20] In a population-based study, prevalence of moderate to severe OSA was demonstrated to be 63% with BMI > 30 kg/m[3, 21]
- Craniofacial and upper airway abnormalities
- Nasal congestion (in adults, doubles the risk for OSA compared to controls)[18]

Scenario 1 (Continued):

This **51-year-old male** complains of sleepiness for at least a year. It is worst when he is sedentary, and he often falls asleep while reading or watching television. Recently, he has **fallen asleep several times at his job** as a software engineer, including during an important meeting. On days when he is especially tired, he uses his lunch break to take a quick nap in his car before returning to work. He has **difficulty staying awake while driving longer distances**, and recently, he dozed off momentarily and hit the rumble strips on the side of the highway. He usually gets into bed at 10:30 PM (though he often falls asleep earlier while watching TV) and falls asleep "instantly." He occasionally **awakens feeling as if he is choking or suffocating**. This sensation goes away quickly once he is awake.

He wakes up 3-4x per night to urinate. He does not notice nocturnal wheezing and does not have nocturnal leg cramps. He wakes up in the morning at 6:30 AM using the alarm clock, but often hits the snooze until around 7 AM. He usually **feels in the morning as if he "barely slept."** He often has a **dry mouth and a headache** upon awakening. His wife notes that he **snores loudly.** Because of his snoring, she sometimes sleeps in the guest bedroom. On the weekends, he sleeps until 8:30 AM or 9 AM and tries not to nap, but often finds himself dozing off for up to an hour if he is not keeping active. No matter how long he sleeps, he rarely feels rested. **His Epworth Sleepiness Score is 13.** With regards to his asthma, he denies any exercise limitation ("it's my knees and my weight that keep me from walking more, doc"), recent episodes of wheezing or significant cough. He uses his rescue inhaler infrequently (less than once weekly). He does not smoke cigarettes.

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Extremities: no clubbing or cyanosis, trace pedal edema
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Question 2: What is the most likely diagnosis for his sleepiness? What are his risk factors for each?

Among causes of SDB, the patient likely has OSA. He gets approximately 8 hours of sleep on weekdays and 10 on weekends. The quantity of sleep is sufficient. He denies symptoms related to restless legs syndrome. Additionally, he does not have any REM-disturbance symptoms such as hypnagogic hallucination or sleep paralysis. Narcolepsy, though sometimes presenting in ages 40-50, is less likely – especially in this patient, given other symptoms (and also lack of cataplexy). Obesity hypoventilation syndrome, however, cannot be ruled out based on the information available.

This patient's risk factors for OSA include increased age, male gender, and obesity.

Symptoms that suggest OSA are: loud snoring, nocturnal choking/gasping for air, nocturnal polyuria (increased release of atrial natriuretic peptide (ANP) secondary to increased negative intrathoracic pressure due to inspiratory effort against a closed airway and in turn, increased venous return), dry mouth, morning headache, non-restorative sleep, and excessive daytime sleepiness.

Signs that suggest OSA are obesity, large neck, crowded upper airway, and hypertension.

This patient's STOP BANG Score = 7 and DOISNORE50 = 7.

Question 3: What types of tests can be used to confirm a diagnosis of sleep apnea?

Three different types of sleep studies could be ordered for this patient:

1. Diagnostic polysomnography (PSG): reports electroencephalogram (EEG), electrooculogram (EOG), electromyogram (EMG) for chin and legs, airflow signals, respiratory effort signals for chest and abdomen, oxygen saturation, body position, and electrocardiogram (ECG).

   PSG can be used to diagnose OSA, central sleep apnea (CSA), sleep-related hypoventilation disorders, REM behavior disorder, parasomnias, narcolepsy, and hypersomnia (in which case Multiple Sleep Latency Test would follow PSG), periodic limb movement disorder, bruxism, etc.

2. Split-night study: reports the same parameters as the diagnostic PSG; however, the study is split into a diagnostic first half, followed by a second treatment part initiated the same night. To qualify for PAP titration during the second half of the night, at least moderate OSA must be seen during at least 2 hours of sleep time, and 3 hours must remain left in the night for PAP titration. Split-night studies have adequate sensitivity to diagnose OSA and may enhance care efficiency by diagnosing OSA and establishing PAP treatment needs within a single night. Examples of risk factors for nondiagnostic studies include severe insomnia, claustrophobia, concern for non-breathing related sleep disorders, or concern for non-OSA types of SDB.[22]

3. Unattended, home sleep apnea test (HSAT): reports airflow signals, respiratory effort signals (single or dual), oxygen saturation, ECG, sleep/wake or monitoring time, and snoring. Per the American Academy of Sleep Medicine, HSAT is indicated to diagnose only OSA in uncomplicated adults presenting with signs and symptoms that indicate

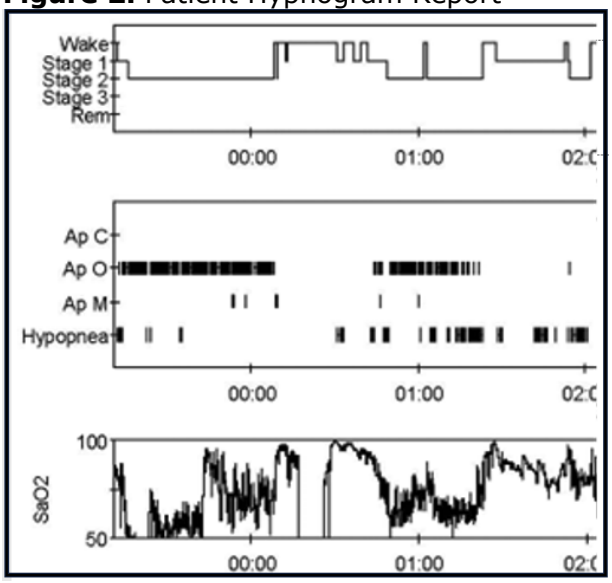
an increased risk of moderate to severe OSA.[23] It is essential to note the relative contraindications of HSAT, like central sleep apnea, HFrEF, severe pulmonary disease like COPD or ILD, patients on home oxygen, recent stroke, etc.

In our patient, HSAT is a good choice as he has high likelihood of OSA and absence of comorbid factors listed above. Importantly, if a HSAT is negative or inconclusive but suspicion for SDB remains, a diagnostic polysomnogram should be performed.

Scenario 1 (Continued):

The patient underwent a sleep study that demonstrated the following: his latency to sleep onset was <2 minutes; his sleep efficiency was 89%. He had fragmented sleep with 61 arousals/hour. His Apnea-Hypopnea Index (AHI) was 90 events/hour. There were no periodic limb movements. His tracing is shown below in Figure 2.

Figure 2. Patient Hypnogram Report



Question 4: What do the tracings demonstrate?

This type of tracing is known as a Hypnogram. The top panel shows different stages of sleep. Note that this patient never attains N3 or REM sleep. He also has multiple awakenings. The middle panel demonstrates that patient has multiple obstructive sleep apneas and hypopneas as soon as he falls asleep. The bottom panels show desaturation events throughout the study. During obstructive apneas and hypopneas, individuals have collapse of the upper airway, which leads to oxyhemoglobin desaturation and/or arousal from sleep. Thus, accompanying the many sleep-disordered breathing events observed in this patient (apnea graph), cyclical desaturations are recorded by the SaO2 graph. He has severe OSA based on AHI of 90/hour with predominantly obstructive apneas and hypopneas.

Table 2. Obstructive Sleep Apnea Severity Ratings in Adults

No OSA	Mild OSA	Moderate OSA	Severe OSA
AHI <5	AHI 5 to 15	AHI 15 to 30	AHI > 30

Question 5: What options exist for treating OSA?

Treatment options include:

1. **Continuous positive airway pressure (CPAP):** first line, most effective therapy, but not tolerated by all patients. CPAP increases intraluminal pressure above the pharyngeal transmural pressure, keeping the airway open.[6] A meta-analysis with 35 randomized control trials shows that CPAP was more effective compared to sham in reducing AHI and improving sleepiness based on ESS.[24]

2. **Behavior modification:**
    a. Weight loss and exercise should be recommended for all overweight patients with OSA. Weight loss rarely cures OSA unless there is a significant amount of weight loss, ie, from a bariatric procedure or injectable GLP-1 medication. Weight loss does reduce AHI.
        i. One of the GIP/GLP-1 drugs, tirzepatide (Zepbound), was approved for treatment of adults with moderate to severe sleep apnea and obesity (BMI >30) in December 2024 based on the results of the SURMOUNT-OSA trial.[28]
        ii. The trial was done in populations unable/unwilling to tolerate PAP and then in patients using adjunctive PAP, and had robust benefits for both groups, though in practice, it is difficult to get this covered by most insurance companies for patients not already compliant with PAP therapy.
    b. Positional therapy – if the patient has a strong positional component, i.e., a much higher AHI in the supine position, sleeping in a non-supine position can reduce the AHI. Several over-the-counter devices exist, such as Zzoma pillows, as seen in Figure 3.

    Figure 3. Zzoma Pillow

    Image from https://www.medicare.ie/product/zzoma-positional-device/

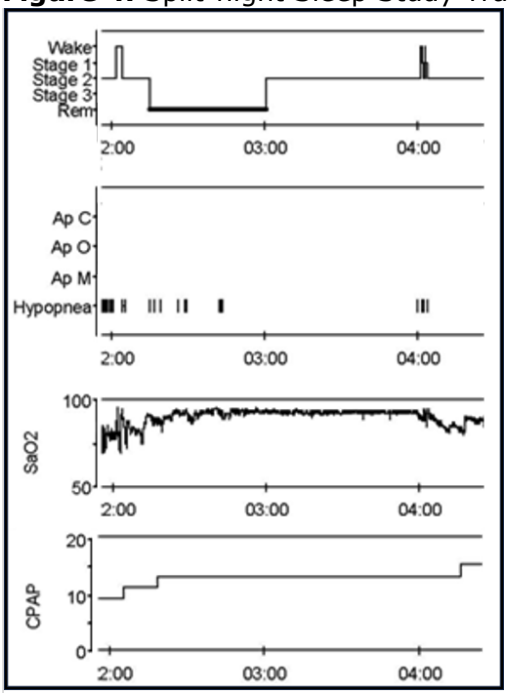
    c. Alcohol avoidance – alcohol can exacerbate OSA symptoms, cause sleep fragmentation, and depress the respiratory system.
    d. Sedative avoidance – any of the sedatives with respiratory depressant properties can also exacerbate the OSA symptoms.

3. **Oral appliance:** This is often used as second-line therapy in patients with moderate to severe OSA who cannot tolerate PAP therapy. It can be used as first-line therapy in patients with mild OSA and no other comorbidities. AASM recommends repeating the sleep study once the patient is fit with an oral mandibular device. Drawbacks to oral appliance therapy include jaw pain, malocclusion, and movement of teeth. Poor dentition is a contraindication.

4. **Upper airway surgery:** It is difficult to predict which patients would benefit from an upper airway surgery. Tonsillectomy and adenoidectomy are the mainstay of therapy in non-obese children with OSA, but these are rarely effective treatments in adults.
5. **Hypoglossal Nerve Stimulation:** Also used as second-line therapy for OSA. Patients will often need drug-induced sleep endoscopy to determine the pattern of airway collapse. This modality can be used in patients with moderate to severe OSA who have ineffective pharyngeal dilator muscles.
6. **Supplemental oxygen:** Can improve oxygen saturation but may prolong apneas and does not decrease the number of events; therefore, in general, oxygen alone is NOT considered to be effective therapy.

Question 6: What does the tracing below demonstrate (Figure 4)?

Figure 4. Split-night Sleep Study Tracing



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This tracing is from a split-night study. Around 2AM, the patient is started on CPAP 10, and titrated to 13 cm H20, due to persistent obstructive apneas and hypopneas. The increase in pressure resolves most of his sleep-related breathing events. Despite his AHI being reduced to less than 5 events/hour, his arousal index decreasing to 2, and his report of much more refreshing sleep, he remains hypoxemic, with O2 saturation below 88% for 15% of the night.

Question 7: What is the most likely explanation for this patient's persistent hypoxemia?

He likely has OHS. OHS is defined by BMI > 30 kg/m^2, a resting $PaCO_2$ of more than 45 mmHg, and absence of an alternative cause of alveolar hypoventilation. It is often associated with worsened nocturnal hypercapnia, nocturnal hypoxemia, and OSA.[7-8,11,25]

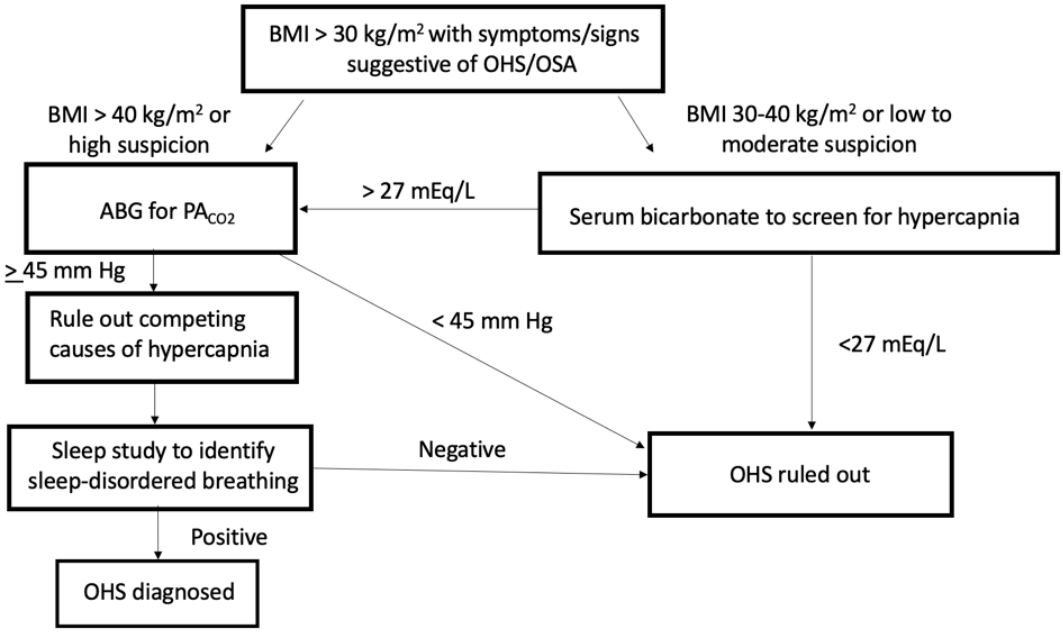
OHS is a diagnosis of exclusion, and other conditions commonly associated with hypercapnia should be ruled out. In 90% of patients with OHS, the sleep-disordered breathing is

obstructive sleep apnea. The other 10% may have sleep-related hypoventilation (an increase in $PaCO_2$ of >10 mm Hg above that during wakefulness or significant oxygen desaturations) that is not the result of obstructive apneas or hypopneas.

Question 8: What diagnostic tests can be used to confirm OHS?

You can check an arterial blood gas, which would likely show a chronic respiratory acidosis, with elevated PCO_2 and borderline PaO_2. You can also check serum chemistry, which would likely show an elevated HCO_3. Early diagnosis of OHS can improve outcomes and one way to work up an OHS patient is mentioned below.[12]

Figure 5. Evaluation Algorithm for OHS



Abbreviations: ABG, arterial blood gas; BMI, body mass index

Adapted from Mokhlesi B, Masa JF, Brozek JL, et al. *Evaluation and management of obesity hypoventilation syndrome: an official American Thoracic Society clinical practice guideline.* Am J Respir Crit Care Med. *2019;200(3):e6-e24. doi:10.1164/rccm.201905-1071ST*

Question 9: What treatment options exist for OHS?

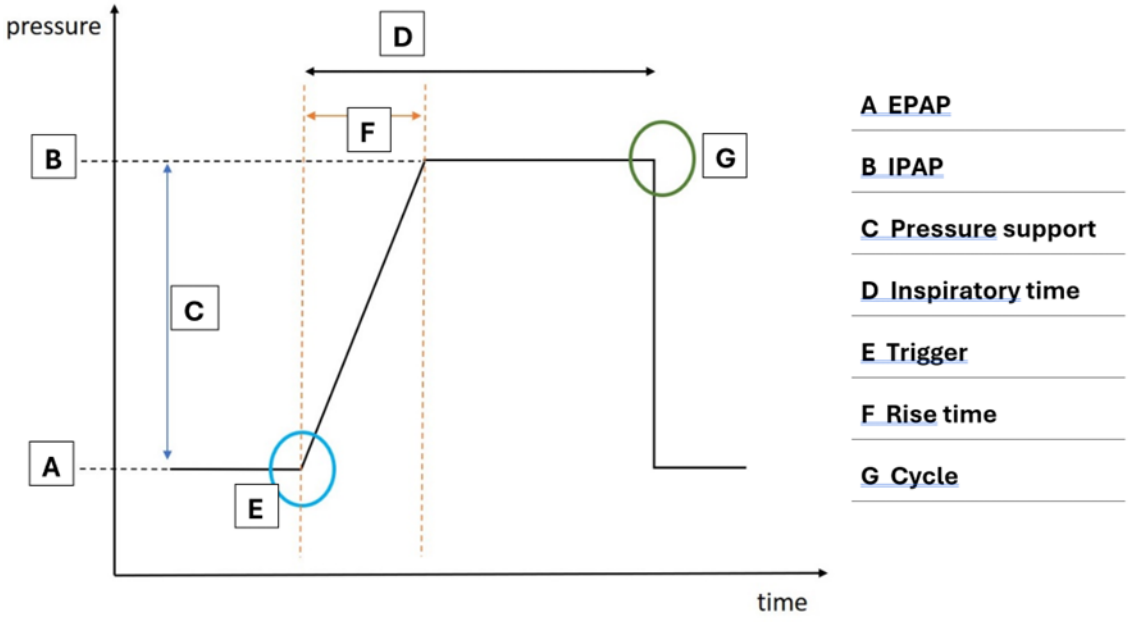
In many observational studies and RCTs, both CPAP and non-invasive ventilation (NIV) have shown similar improvement in awake respiratory failure, symptoms, and quality of life for patients with OHS.[23-31] Pathophysiologically, the primary mechanism by which CPAP treats OHS is enabling off-loading of carbon dioxide accumulated during airflow obstruction by stenting open the upper airway. In stable ambulatory patients (the majority of patients with OHS), CPAP is recommended over NIV for patients with OHS and concurrent severe OSA. There is less certainty of evidence for patients with OHS without concomitant OSA (30% of patients), and it is important to note that improvements in awake hypercapnia may be achieved more efficiently with bilevel PAP. In a prospective trial, 57% of ambulatory patients with severe OHS were able to be managed with CPAP alone. The remaining 43% of patients were persistently hypoxemic at therapeutic pressure and needed to be started on bilevel PAP therapy.[23]

Question 10: How would you approach starting this patient on PAP therapy?

The patient should first be started on CPAP, which, as above, maintains a continuous positive airway pressure above ambient pressure to stent open the upper airway. There are two "modes" of continuous positive airway pressure; fixed CPAP maintains one positive pressure throughout the respiratory cycle, while auto-titrating PAP (AutoCPAP, see table 3) adjusts pressure between a range set by the clinician to treat hypopneas and apneas.

If the patient is intolerant of high CPAP pressures, (>15 cmH2O) that are required to resolve apneas and hypopneas or if the hypoxemia persists despite elimination of apneas and hypopnas with adequate CPAP pressures bilevel positive airway pressure (BPAP) should be considered. During BPAP titration, IPAP should be 8-10 cmH2O above EPAP to increase ventilation. BPAP S/T can also be used. BPAP S/T adds a backup rate to bilevel positive airway pressure to help maintain ventilation. Adherence to PAP therapy has been shown to improve PaCO2 and PaO2 and to decrease the need for oxygen therapy.[14,26-27] See Figure 6 and Table 3.[13]

Figure 6. Understanding the Respiratory Cycle in Non-Invasive Ventilation



Used with permission from Jennifer Newitt, MD.

Table 3. Basic Modes of PAP Therapy for OSA and OHS

Type of Pressure	**Continuous**		**Bilevel**	
Machine Mode	**CPAP**[6, 12-13, 23]	**Auto CPAP**[6,22-23]	**BPAP S**[7-8,11-12,26]	**Auto Bilevel** [7-8,11-12,26]
Notes	Fixed, continuous pressure that stents open upper airway	Machine senses apneas and hypopneas and increases pressure within a range set by the clinician	Machine delivers fixed IPAP and fixed EPAP	Machine delivers a fixed pressure support; IPAP and EPAP are adjusted if hypopneas or apneas are sensed
Prescription	CPAP	PAP range	IPAP/EPAP (back up respiratory rate if S/T)	Minimum EPAP, Maximum IPAP, fixed pressure support

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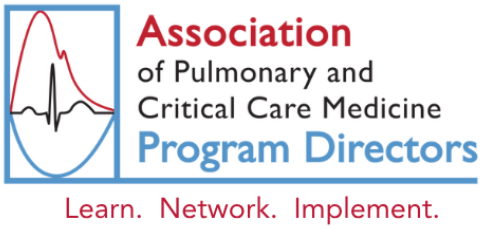

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