ACP
American College of Physicians

Principles and Practice of HOSPITAL MEDICINE

SECOND EDITION

SYLVIA McKEAN | DAN D. DRESSLER | JOHN J. ROSS | DANIELLE SCHEURER

Mc Graw Hill Education

Principles and Practice of Hospital Medicine

Second Edition

Editors

Sylvia C. McKean, MD, SFHM, FACP
Deputy Editor for Editorial Projects, UpToDate
Formerly:
Leave of absence:
Associate Professor of Medicine,
Harvard Medical School
Hospitalist
Brigham and Women's Hospital
Boston, Massachusetts

John J. Ross, MD, CM, FIDSA
Assistant Professor of Medicine
Harvard Medical School
Hospitalist Service
Brigham and Women's Hospital
Boston, Massachusetts

Daniel D. Dressler, MD, MSc, SFHM, FACP
Professor of Medicine
Director, Internal Medicine Teaching Services
Emory University Hospital
Associate Program Director
J. Willis Hurst Internal Medicine Residency Program
Co-Director, Semmelweis Society
Emory University School of Medicine
Atlanta, Georgia

Danielle B. Scheurer, MD, MSCR, SFHM
Chief Quality Officer and Hospitalist
Associate Professor of Medicine
Medical University of South Carolina
Charleston, South Carolina

Mc Graw Hill Education

New York Chicago San Francisco Athens London Madrid Mexico City
Milan New Delhi Singapore Sydney Toronto

Principles and Practice of Hospital Medicine, Second Edition

1 2 3 4 5 6 7 8 9 DOW 21 20 19 18 17 16

ISBN 978-0-07-184313-3
MHID 0-07-184313-2

This book was set in Myriad pro by Cenveo® Publisher Services.
The editors were Amanda Fielding and Kim J. Davis.
The production supervisor was Richard Ruzycka.
Project management was provided by Vastavikta Sharma, Cenveo Publisher Services.
The designer was Alan Barnett; the cover designer was Dreamit, Inc.

RR Donnelley was printer and binder.

Library of Congress Cataloging-in-Publication Data

Names: McKean, Sylvia C., editor. | Ross, John J. (John James), 1966-editor. | Dressler, Daniel D., editor. | Scheurer, Danielle, editor.
Title: Principles and practice of hospital medicine / editors, Sylvia C. McKean, John J. Ross, Daniel D. Dressler, Danielle B. Scheurer.
Description: Second edition. | New York : McGraw-Hill Education Medical, [2017] | Includes bibliographical references and index.
Identifiers: LCCN 2016022668 (print) | LCCN 2016023825 (ebook) | ISBN 9780071843133 (hardcover : alk. paper) | ISBN 0071843132 (hardcover : alk. paper) | ISBN 9780071843140 (ebook)
Subjects: | MESH: Hospital Medicine—methods | Hospitalization | Inpatients | Hospitalists Classification: LCC RA972 (print) | LCC RA972 (ebook) | NLM WX 21 | DDC 362.11—dc23
LC record available at https://lccn.loc.gov/2016022668

McGraw-Hill Education books are available at special quantity discounts to use as premiums and sales promotions, or for use in corporate training programs. To contact a representative, please visit the Contact Us pages at www.mhprofessional.com.

CHAPTER 235

Sleep Apnea and Obesity Hypoventilation Syndrome

Ji Yeon Lee, MD
David A. Schulman, MD, MPH, FCCP

1. What are the key distinguishing features between obstructive and central sleep apnea?
2. What are the consequences of untreated sleep apnea?
3. What are the indications for inpatient therapy for sleep apnea?
4. How should patients with suspected sleep apnea be managed at hospital discharge?
5. What is obesity hypoventilation syndrome and how is it best treated?

INTRODUCTION

Sleep apnea is defined by repeated transient cessations of respiration during sleep. The most common type of this disorder, obstructive sleep apnea (OSA), affects between 5% and 15% of middle-aged and older adults. The burden of disease in the hospitalized patient is likely to be even greater than that of the general population because inpatients carry many disorders that have been associated with OSA, including obesity, congestive heart failure, coronary artery disease, hypertension, stroke, and diabetes. Epidemiologic data suggest that the majority of patients with sleep apnea are undiagnosed; in 2004, national hospital discharge codes revealed fewer than 300,000 cases of sleep apnea among almost 35 million inpatient stays, yielding a prevalence of identified disease of less than 1%.

Although sleep apnea by itself is rarely a primary indication for hospitalization, recent evidence suggests that inpatient management of this disease needs to improve. Less than 6% of those identified as having sleep apnea in the 2004 National Hospital Discharge Survey received therapy with continuous positive airway pressure (CPAP) while in the hospital. Hospital Medicine physicians should identify and appropriately treat OSA patients. The key to identifying the possible presence of sleep apnea depends largely on an appreciation of risk factors and clinical features. In 2008, Goring and Collop showed that almost 80% of patients with suspected sleep apnea referred for a sleep study after an inpatient hospitalization were confirmed to have OSA. Therefore, inpatient identification and referral for evaluation can improve diagnosis rates and reduce the percentage of affected patients left untreated. This chapter will review the fundamentals of sleep apnea, address the management of patients with stable sleep apnea in the inpatient setting, delineate the necessity of preoperative screening in at-risk patients, and identify which patients should undergo further testing.

PATHOPHYSIOLOGY

Sleep-disordered breathing includes three related classes of diseases: OSA, central sleep apnea (CSA), and sleep-related hypoventilation—although as many as 90% of all cases are OSA. The distinction between OSA and the other categories is an important one as the treatment options and responses to therapy are quite different. Obesity hypoventilation syndrome (OHS) is a condition in which obese patients develop diurnal hypercapnia and hypoxemia in the absence of a causal pulmonary or neurologic disorder; up to 90% of these patients have OSA, though the causative association has yet to be proven.

■ OBSTRUCTIVE SLEEP APNEA

The upper airway is a compliant structure susceptible to collapse. Complex neurologic and musculoskeletal interactions cause a decrease in the cross-sectional area of the upper airway during sleep, which may result in a reduction or cessation of airflow in susceptible individuals. The imbalance between the forces that promote airway patency (the pharyngeal dilator muscles) and the negative inspiratory forces generated by the diaphragm (which promotes airway collapse) causes obstruction. The majority of patients with OSA demonstrate obstruction in the retropalatal or retrolingual areas as these locations carry the greatest risk of collapse when a negative pressure forms inside the airway during inspiration.

Even during wakefulness, the cross-sectional area of the upper airway is smaller in patients with OSA compared with disease-free controls, often due to anatomic contributors, such as enlarged

tonsils, a large tongue, a high-arched palate, and abnormal positioning of the maxilla and mandible. At sleep onset, a decrease in neural output to the pharyngeal dilator muscles increases the risk of collapse. In addition, supine positioning during sleep promotes collapse by bringing the weight of external tissue to bear upon the anteroposterior diameter of the pharynx, which is usually its shortest axis (and thus the one along which collapse is most likely). Snoring, caused by vibration of the soft tissues of the upper airway, may be the earliest sign of obstruction, although it is a very common clinical finding with a prevalence of up to 35% and has a positive predictive value as low as 20% for the presence of frank sleep apnea in an otherwise unselected population.

As obstruction worsens, periods of time during which airflow is inadequate (hypopnea) or absent (apnea) occur. During these episodes, there are transient increases in arterial carbon dioxide tension and decreases in arterial oxygen tension. Episodes of obstruction typically terminate with an arousal from sleep, precipitated by the changes in oxygen and carbon dioxide as well as marked pressure swings within the thoracic cavity, as the affected individual unconsciously attempts to inspire air past the obstruction, an effort that fails due to the negative pressure serving to further pull the airway walls inward, worsening collapse. The lack of restful sleep from repetitive arousals throughout the night is responsible for the symptom of excessive daytime sleepiness in affected patients. Notably, only about half of patients with sleep apnea report overt sleepiness; others may report symptoms of fatigue, lack of energy, or other related symptoms without frank sleepiness.

■ CENTRAL SLEEP APNEA

Patients with central events are marked by a transient absence of respiratory effort. Affected patients fall into two main groups, those with daytime hypercapnia and those without. All human beings have a slight decline in respiratory drive during sleep, with a typical increase in pCO_2 of about 5 mm of mercury; this change is normally achieved through a transient period of hypoventilation at sleep onset. Among patients with daytime hypoventilation (including those with chronic obstructive pulmonary disease and a number of neuromuscular disorders), an already impaired ventilatory drive is further suppressed, causing apnea. Hypercapnic CSA may also be related to a reduced central drive, as in the case of a brainstem stroke, use of CNS depressants, or an impaired respiratory motor, as seen in myopathy, neuromuscular junction disorders, and spinal lesions.

Patients with nonhypercapnic CSA tend to have an increased responsiveness to elevated $PaCO_2$ levels, leading to periods of apnea when carbon dioxide levels drop below the apneic threshold. The disorder is most often seen as Cheyne-Stokes respiration in patients with heart failure. When these patients lie in the supine position, rostral movement of fluid from the lower extremities into the lungs activates stretch receptors, stimulating ventilation and leading to hypocapnia. The $PaCO_2$ drops below the apneic threshold, leading to a cessation of respiration. Decreased cardiac output causes an increase in circulatory time, leading to delays in information feedback from peripheral receptors. In addition, decreased cerebrovascular reactivity impairs the respiratory control center's ability to appropriately target ventilatory responses to carbon dioxide. As a result, as the $PaCO_2$ rises again, there can be ventilatory overshoot, creating a cyclic crescendo-decrescendo respiratory pattern that characterizes Cheyne-Stokes respiration.

■ SLEEP-RELATED HYPOVENTILATION

The diagnosis of sleep-related hypoventilation requires a persistent elevation in carbon dioxide level. This class of disorders includes OHS, as well as hypoventilation due to medication use, congenital diseases (such as central alveolar hypoventilation syndrome) and medical disorders (most commonly pulmonary or neurologic diseases).

DIFFERENTIAL DIAGNOSIS

Snoring does not necessarily indicate the presence of sleep apnea. While a socially problematic condition, primary snoring has not clearly been associated with any long-term health repercussions. While history may be highly suggestive of OSA, several other disorders may mimic its symptoms (Table 235-1). Patients who awaken with paroxysmal dyspnea at night may suffer from congestive heart failure, chronic obstructive pulmonary disease, nocturnal gastroesophageal reflux or nocturnal panic attacks. A small percentage of patients with severe sleep apnea will demonstrate diurnal hypercapnia, although diagnoses of underlying pulmonary disease (either restrictive or obstructive) and neuromuscular disease affecting the muscles of respiration should also be entertained.

Central sleep apnea presents with witnessed breathing pauses at night and daytime fatigue, although the mechanism of disease and appropriate treatment is quite different. Other causes of frequent nocturnal arousals and daytime somnolence include periodic limb movement disorder, prostatism and environmental sleep disorder, in which an external stimulus causes recurrent awakenings from sleep. Patients with nocturnal pain and inadequately controlled mood disorders may also demonstrate frequent nocturnal arousals. Excessive daytime sleepiness may be a symptom of insuf cient sleep (the most common cause of daytime somnolence), depression, narcolepsy and idiopathic hypersomnia, as well as a number of endocrinologic, cardiac, pulmonary, renal, and hematologic disorders.

DIAGNOSIS

Patients with OSA present to the hospital with other medical problems and the clinician should make an effort to identify at-risk patients so that they undergo appropriate evaluation. Older age, obesity, and male sex are the most significant risk factors for OSA. Because the prevalence of these characteristics in the general population is too high for them to be useful as a screening tool, history and physical examination are critical in determining which patients are appropriate referrals for sleep testing (Table 235-2).

Taking a screening history for OSA includes an assessment of both nocturnal and diurnal symptoms; the accuracy of the history improves if the patient's bed partner is available for questioning, as

TABLE 235-1 Differential Diagnosis of Obstructive Sleep Apnea, Based on Symptom

Symptom	Differential Diagnosis
Frequent nocturnal arousal and daytime somnolence	Central sleep apnea
	Benign prostatic hypertrophy
	Periodic limb movement disorder
	Environmental sleep disorder
	Nocturnal pain
	Mood disorder (eg, depression) inadequately controlled
	Insufficient sleep (most common cause of daytime sleepiness)
	Narcolepsy
	Idiopathic hypersomnia
	Awakening from other endocrinologic, cardiac, pulmonary, renal, or hematologic disorders
Snoring	Primary snoring
Paroxysmal dyspnea	Congestive heart failure
	Chronic obstructive pulmonary disease
	Nocturnal gastroesophageal reflux
	Nocturnal panic attacks

TABLE 235-2 History and Physical Examination Findings Suggesting Sleep Apnea

Heroic snoring
Nocturia
Witnessed breathing pauses during sleep
Excessive daytime sleepiness
Tongue or tonsillar enlargement
Retrognathia
Large neck circumference (> 16 in in women, > 17 in in men)

that person can offer greater insight into events that occur when the patient is unconscious. Snoring is the most common symptom of obstructive sleep apnea, present in more than 90% of patients with the disease, suggesting that the absence of snoring (by history from bed partner or other witness) would have a good negative predictive value for OSA. The presence of witnessed apneas, seen in 75% of patients with OSA, has a positive predictive value of greater than 80% for the presence of sleep apnea, although data do not suggest that their presence is associated with the severity of the disease. However, the absence of witnessed apneas does not reliably exclude the diagnosis of OSA. Another suggestive clue in the history includes waking up with a sensation of choking or gasping for air. This symptom usually represents an arousal due to an apneic episode, with the sensation normally subsiding within seconds of waking. Rarely, patients may complain of insomnia (which probably represents disturbed sleep secondary to arousal), although the majority of patients report no difficulty initiating sleep. Nocturia is a frequent complaint among patients with apnea; it occurs, in part, because OSA patients have higher circulating levels of atrial natriuretic peptide, leading to a greater urine output. In addition, some patients who wake from an apnea have no recollection of the event (as the obstruction resolves with arousal), leading them to believe that they woke to urinate.

The presence of excessive daytime somnolence (EDS) is very common in OSA patients, although the complaint is nonspecific and its utility in discriminating affected patients from those without the disorder is poor. A validated questionnaire to assess sleepiness, such as the Epworth Sleepiness Scale (see http://epworthsleepinessscale.com/1997-version-ess/), may quantify fatigue, although the symptom remains nonspecific. Other daytime symptoms of OSA include irritability, memory disturbance, and morning headaches (secondary to cerebral vasodilation due to transient carbon-dioxide retention).

Other medical disorders have been associated with OSA (Table 235-3). Although most of these disorders do not have a good positive predictive value for sleep apnea, recent guidelines recommend considering the diagnosis of OSA in patients with refractory hypertension. Sleep apnea is also more common in patients with renal failure, hypothyroidism, and acromegaly. Diastolic dysfunction has been associated with sleep-disordered breathing, although a causal link has not been definitively proven. The presence of neuromuscular disease can increase the risk of both central and obstructive sleep apnea. In addition, the regular use of narcotics or benzodiazepines can cause or exacerbate OSA, as well as sleep-related hypoventilation. Any of these risk factors should trigger more detailed questioning about symptoms of OSA.

TABLE 235-3 Other Comorbid Conditions Associated with Obstructive Sleep Apnea

Systemic hypertension
Pulmonary hypertension
Myocardial infarction
Stroke
Diabetes mellitus and glucose intolerance
Atrial fibrillation
Congestive heart failure
Hypothyroidism
Neuromuscular disease

Physical examination may be helpful in identifying patients at higher risk of sleep apnea, but no exam findings can confirm or preclude the diagnosis with a significant degree of certainty. Obesity is associated with OSA, with a prevalence of OSA as high as 70% in morbidly obese patients. A large neck circumference (>17 in in men, > 16 in in women) confers an increased risk of OSA. Other anatomic factors conferring an increased risk include retrognathia, micrognathia, tonsillar hypertrophy, an enlarged uvula, macroglossia, and inferior displacement of the hyoid. Assessment of cross-sectional area of the pharynx using a validated tool such as the Mallampati scale can help to quantify airway crowding and OSA risk.

Several screening tools exist that attempt to integrate components of the history (and physical examination in some cases) to create a composite risk score. Both the STOP-BANG and the Berlin questionnaire (Figure 235-1) have a high sensitivity for significant sleep apnea (>90%) but only a modest specificity (approximately 50%), yielding a positive predictive value in the 10% to 20% range. As a result, a combination of such tools and clinical acumen can best determine which patients should undergo further evaluation.

■ OVERNIGHT PULSE OXIMETRY AND IN-HOSPITAL POLYSOMNOGRAPHY IN DIAGNOSIS OF OSA

Even when the diagnosis of OSA is highly suspected and can be confirmed by the presence of heroic snoring and witnessed apneas during hospitalization, formal testing is required before insurance will cover therapy with CPAP. The current reference standard test for the diagnosis for obstructive sleep apnea is overnight polysomnography (PSG), though this requires the patient to spend a night in the sleep laboratory and can be inconvenient and expensive, as well as impractical for an acutely ill, hospitalized patient.

As an alternative to PSG, home sleep apnea testing (HSAT) has been increasing in popularity due to its cost effectiveness and convenience. HSAT is an alternative to PSG, best in cases of high pretest probability of moderate-to-severe OSA. The test has not been validated for patients with significant comorbid conditions, specifically lung disease, congestive heart failure, morbid obesity, and neurologic disorders. Home testing is also not useful in the diagnosis of nonrespiratory sleep disorders.

Some have also proposed the use of overnight continuous pulse oximetry to diagnose OSA as it is comparatively inexpensive, simple to perform and may be performed in the patient's hospital room. However, the presence of nocturnal desaturation does not equate to the presence of obstructive sleep apnea and cannot reliably distinguish central from obstructive apnea as an explanation for any observed desaturation. In addition, the absence of desaturation does not exclude the diagnosis of OSA. In some cases, patients may not sleep for much of the night; the absence of significant desaturation could simply represent the absence of significant sleep. On the whole, overnight oximetry may be useful to exclude OSA in appropriately chosen patients who are at high risk, but patients would still require a full PSG prior to initiation of CPAP therapy if abnormalities are found.

In-hospital PSG for patients considered to be high-risk for sleep apnea may be considered during their inpatient stay, although this test is not usually reimbursed by insurers when performed during hospitalization. Because of its portability, there has been interest in using HSAT prior to hospital discharge in patients thought to have

sleep apnea. While this is a practical option, providers should be cautious about trying to diagnose sleep-disordered breathing in the hospital environment; false-negative tests may result from sleep disruption from environmental noise, nocturnal medication administration, or vital sign checks. In addition, sleep apnea may be exacerbated during hospitalizations as a result of physiologic derangements that will resolve as the patient recovers, leading inpatient HSATs to show disease that will diminish or disappear over time.

MANAGEMENT

■ MANAGEMENT OF STABLE OSA IN THE HOSPITALIZED PATIENT

Many hospitalized patients carry a prior diagnosis of OSA. Several studies have recently evaluated the prevalence and management of sleep apnea in the hospitalized patients, and found that management of OSA in the hospital setting is frequently overlooked. One large study showed that less than 6% of patients received this therapy during hospital stay, despite home adherence to CPAP; some smaller studies have fared better, though not markedly so. Several challenges impede optimal management of OSA patients during an acute hospitalization. The burden of disease is high and resources are limited to provide CPAP machines to all affected patients, as the setup often requires significant involvement by a respiratory therapist. Many patients are not able to report the correct home setting for their CPAP machine, and the mask interface with which the patient has become comfortable may not be available at the hospital.

- Patients already on treatment should be encouraged to bring in their home device for use during their hospital course. While this may not be feasible in all patients or in all facilities, it would tremendously decrease the burden on the hospital to provide devices for inpatient use. If patients are unable to retrieve their own device, contacting the patient's durable medical equipment provider often provides information about the machine's setting and the specific mask interface used by the patient at home, so that this can be replicated during hospitalization.

Berlin Questionnaire
Sleep Apnea

Height (m) _____ Weight (kg) _____ Age _____ Male/Female

Please choose the correct response to each question.

Category 1

1. Do you snore?
£ a. Yes
£ b. No
£ c. Don't know

If you answered 'Yes':

2. You snoring is:
£ a. Slightly louder than breathing
£ b. As loud as talking
£ c. Louder than talking
£ d. Very loud—can be heard in other rooms

3. How often do you snore?
£ a. Almost every day
£ b. 3-4 times per week
£ c. 1-2 times per week
£ d. 1-2 times per month
£ e. Rarely or never

4. Has your snoring ever bothered other people?
£ a. Yes
£ b. No
£ c. Don't know

5. Has anyone noticed that you stop breathing during your sleep?
£ a. Almost every day
£ b. 3-4 times per week
£ c. 1-2 times per week
£ d. 1-2 times per month
£ e. Rarely or never

Category 2

6. How often do you feel tired or fatigued after your sleep?
£ a. Almost every day
£ b. 3-4 times per week
£ c. 1-2 times per week
£ d. 1-2 times per month
£ e. Rarely or never

7. During your waking time, do you feel tired, fatigued or not up to par?
£ a. Almost every day
£ b. 3-4 times per week
£ c. 1-2 times per week
£ d. 1-2 times per month
£ e. Rarely or never

8. Have you ever nodded off or fallen asleep while driving a vehicle?
£ a. Yes
£ b. No

If you answered 'Yes':

9. How often does this occur?
£ a. Almost every day
£ b. 3-4 times per week
£ c. 1-2 times per week
£ d. 1-2 times per month
£ e. Rarely or never

Category 3

10. Do you have high blood pressure?
£ a. Yes
£ b. No
£ c. Don't know

Figure 235-1 The Berlin Questionnaire. (Data from the Netzer NC, Stoohs RA, Netzer CM, Clark K, Strohl KP. Using the Berlin Questionnaire to identify patients at risk for the sleep apnea syndrome. Ann Intern Med. 1999;131(17):485-491.)

Berlin Questionnaire
Sleep Apnea

Scoring Berlin Questionnaire

The questionnaire consists of 3 categories related to the risk of having sleep apnea. Patients can be classified into High Risk or Low Risk based on their responses to the individual items and their overall scores in the symptom categories.

Categories and Scoring:

Category 1: items 1, 2, 3, 4, and 5;

Item 1: if **'Yes'**, assign **1 point**
Item 2: if '**c**' or '**d**' is the response, assign **1 point**
Item 3: if '**a**' or '**b**' is the response, assign **1 point**
Item 4: if '**a**' is the response, assign **1 point**
Item 5: if '**a**' or '**b**' is the response, assign **2 points**

Add points. Category 1 is positive if the total score is 2 or more points.

Category 2: items 6, 7, 8 (item 9 should be noted separately).

Item 6: if '**a**' or '**b**' is the response, assign **1 point**
Item 7: if '**a**' or '**b**' is the response, assign **1 point**
Item 8: if '**a**' is the response, assign **1 point**

Add points. Category 2 is positive if the total score is 2 or more points.

Category 3 is positive if the answer to item 10 is '**Yes**' or if the BMI of the patient is > 30 kg/m^2.

(BMI is defined as weight (kg) divided by height (m) squared, ie, kg/m^2).

High Risk: if there are 2 or more categories where the score is positive.

Low Risk: if there is only 1 or no categories where the score is positive.

Additional Question: item 9 should be noted separately.

Figure 235-1 (Continued)

■ MANAGEMENT OF HOSPITALIZED PATIENTS WITH UNTREATED OSA

While CPAP yields significant benefit in many patients with respiratory failure, little evidence supports urgent implementation of CPAP strictly for management of OSA during hospitalization.

In one study looking at cardiac patients with suspected OSA, inpatient portable monitoring was performed to diagnose sleep apnea; patients were trialed on CPAP for OSA during hospitalization and discharged home with therapy. Those who were adherent to therapy after discharge were less likely to be readmitted to the hospital within 30 days of discharge. The only other related diagnosis for which acute therapy with positive airway pressure has been shown to be beneficial is acutely decompensated obesity hypoventilation, for which bilevel pressure (not CPAP) has been shown to be associated with a decreased risk of need for intubation and improved survival.

On the negative side, implementation of positive airway pressure for the first time to an acutely ill patient may lead to significant discomfort and noncompliance absent close supervision by a qualified respiratory therapist. In addition, inpatient respiratory therapy departments may not have resources to initiate therapy in all patients. When CPAP therapy is used during hospitalization in patients that are suspected to have OSA, the patient will still require formal diagnostic testing prior to insurance authorizing coverage for home therapy.

- There is insuf cient evidence to support initiation of positive airway pressure for the majority of therapy-naïve patients during their hospitalization, with the possible exceptions

of acute decompensations of congestive heart failure and obesity hypoventilation. Even if therapy is used during hospitalization in patients with suspected OSA, hospitalists should refer patients for inpatient or outpatient PSG or portable monitoring in order to have insurance coverage for outpatient therapy with positive airway pressure.

■ SCREENING FOR OSA IN THE SURGICAL POPULATION

Patients with sleep apnea bear a high risk of perioperative complications, including postoperative ICU admission, prolonged length of hospital stay, encephalopathy, cardiac arrhythmias, pulmonary embolisms, acute MI, infection, and the need for reintubation, particularly if they receive general anesthesia or opioids. Morbidly obese patients often desaturate rapidly during periods of apnea due to low resting lung volumes; care during intubation should focus on preventing significant desaturation during the induction of anesthesia. Failure to recognize OSA patients prior to surgery may leave providers unable to anticipate possible complications and implement preventative measures. One recent study screened all patients presenting for elective surgery using the Berlin Questionnaire and performed PSG in those that were found to be high risk; the prevalence of sleep apnea in this surgical population was more than 20%, but over 70% of these cases were undiagnosed.

Recent American Society of Anesthesiologists guidelines recommend evaluating patients for OSA risk factors, as perioperative pulmonary complications increase with increasing severity of sleep apnea. Depending on clinical suspicion based on record review, focused history, and physical exam, one should consider sleep testing and preoperative initiation of CPAP, particularly if there is severe OSA. Patients with sleep apnea may also be optimized with the use of oral appliances or weight loss. Even if testing is not feasible preoperatively, identification of high-risk patients should generate a lower threshold to reduce pulmonary complications with active management when necessary. High clinical suspicion or a prior diagnosis of sleep apnea may affect the decision to admit the patient postoperatively, may play a role in deciding the type of anesthesia used, (eg, using general anesthesia with a secure airway instead of deep sedation), and should be managed as a dif cult airway. Although there have been no large studies evaluating the benefit of using CPAP postoperatively in OSA patients, postop CPAP use in abdominal surgery patients may reduce the risk of pneumonia, atelectasis, and postop respiratory complications.

■ TREATMENT OF OSA IN THE CPAP INTOLERANT

While continuous positive airway pressure serves as the most effective therapy for obstructive sleep apnea, its use is associated with significant noncompliance (> 50% in some studies). Efforts to improve compliance with CPAP may include the use of different facial interfaces (Figure 235-2) to improve patient comfort, desensitization therapy, the addition of heated humidification or a switch to bilevel positive airway pressure therapy. No single intervention improves satisfaction in all patients requiring CPAP therapy, although a qualified respiratory therapist may help determine which to try first.

Other therapeutic options include oral appliance therapy (OAT), surgical treatments, and supplemental oxygen. Oral appliances work either by repositioning the lower jaw (mandibular advancement) or by holding the tongue forward. Although mandibular advancement devices (Figure 235-3) have been better studied, both treatments are effective in patients with less-severe disease and among those who are not morbidly obese. Surgical treatments have also shown benefit in these groups as well as those with specific anatomic defects on physical examination.

Oxygen therapy had been suggested as a possible therapy for OSA patients who have significant desaturation at night. While supplemental oxygen does not have a marked impact on the frequency of obstructive events, it attenuates nocturnal hypoxemia, which seems to be a greater predictor of cardiovascular risk than the frequency of apneas or hypopneas. However, a recent study has shown that CPAP may help reduce blood pressure, while nocturnal oxygen therapy provided no significant benefit. In OSA patients who use medications that may increase apnea severity or frequency

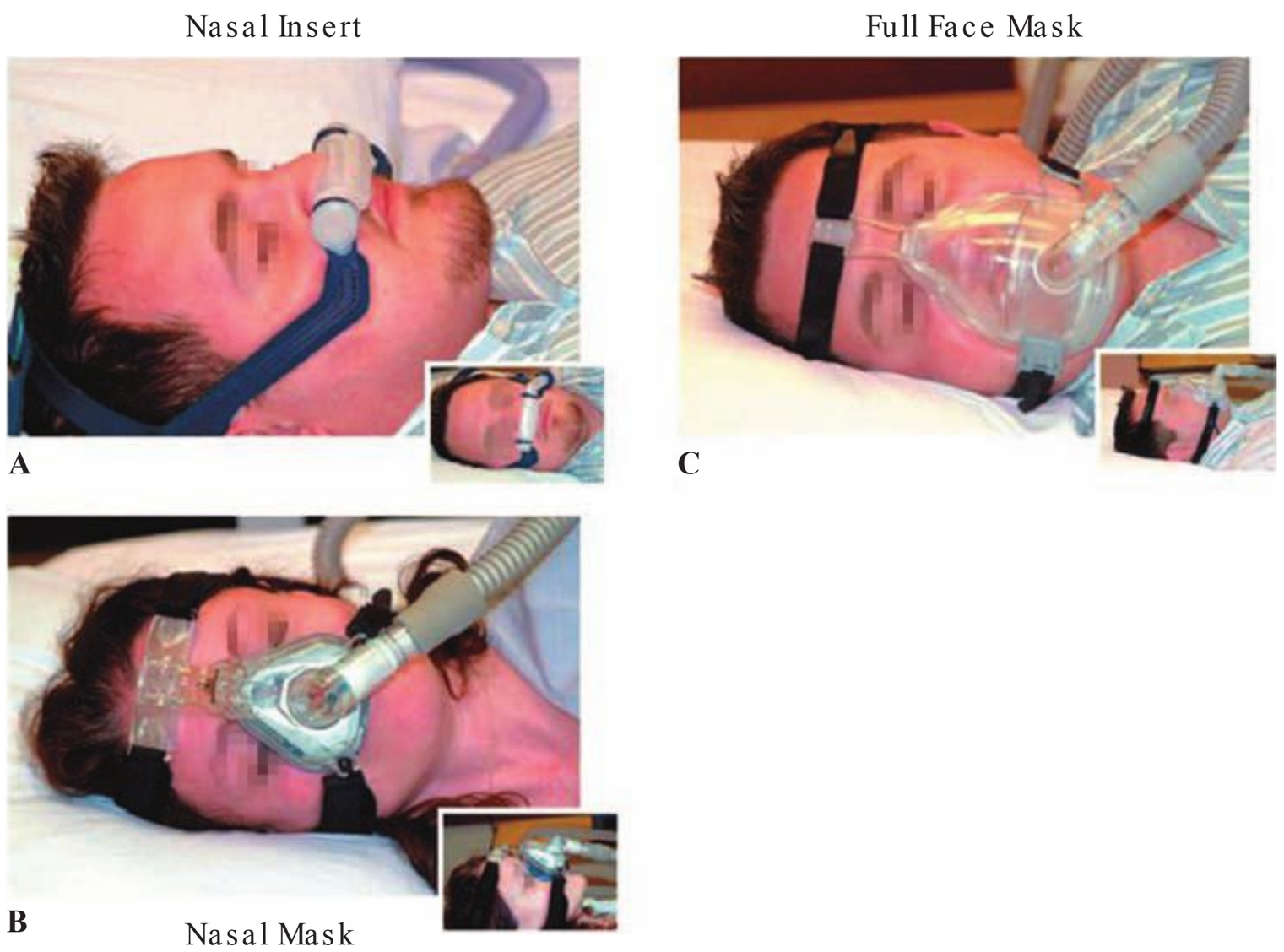

Figure 235-2 Pictures of different continuous positive airway pressure interfaces. (A) Nasal Insert, (B) Nasal Mask, (C) Full Face Mask. (Reproduced, with permission, from Pack A. Fishman's Pulmonary Diseases and Disorders, 4th ed. New York: McGraw-Hill; 2008. Fig. 97-16.)

Figure 235-3 Mandibular advancement devices. (Reproduced, with permission, from Pack A. Fishman's Pulmonary Diseases and Disorders, 4th ed. New York: McGraw-Hill; 2008. Fig. 97-22.)

(such as opiates and benzodiazepines), dose reduction or discontinuation should be considered, when possible.

■ CONSULTATIONS

Respiratory therapy consultation for all patients for whom CPAP is prescribed during the hospitalization will allow the patient selection of interface (mask type) and appropriate fitting to minimize nocturnal mask leak and optimize patient comfort. Many respiratory therapists are also trained in desensitizing techniques for patients who complain of intolerance.

Consultation from a pulmonologist or sleep specialist (when available) should be strongly considered when CPAP nonadherence cannot be adequately addressed by a respiratory therapist. In addition, patients who require more aggressive outpatient therapy may benefit from specialist evaluation during their hospital stay. Symptoms suggesting the need for such a consultation include persistent sleepiness despite compliance with CPAP therapy, persistent snoring on treatment, the development or worsening of cor pulmonale, or the presence of obesity hypoventilation. Finally, consultation should be considered in all patients with predominantly central sleep apnea due to the complexity of optimizing treatment in patients with this disease.

COMPLICATIONS AND PROGNOSIS

OSA confers an increased risk of incident hypertension, coronary artery disease, atrial fibrillation, complex ventricular ectopy, cerebrovascular disease, a worsened prognosis in heart failure, and increased cardiovascular mortality. In one study evaluating outcomes in hospitalized patients with pneumonia, OSA was associated with an increased risk of mechanical ventilation and clinical deterioration. While few data support an increased rate of complications during hospitalization for medical illness attributable to OSA, multiple trials of postoperative complications demonstrate a higher rate of pulmonary, cardiac, gastrointestinal, and bleeding complications in patients with sleep apnea. As a result, many anesthesiologists screen patients for sleep-disordered breathing during preoperative evaluation. However, evidence has not yet proven any attenuation of surgical risk in OSA patients with CPAP therapy.

■ OBESITY HYPOVENTILATION SYNDROME

Obesity hypoventilation syndrome (OHS) is a disorder related to sleep apnea, defined by the presence of waking alveolar hypoventilation ($PaCO_2$ > 45 mm Hg) in the setting of obesity (BMI > 30) with no other clear predisposing risk factors. It occurs in approximately 11% of OSA patients, while approximately 90% of patients with OHS have comorbid sleep-disordered breathing.

The pathogenesis of OHS is not completely understood, but is likely related to physiologic derangements and consequences of obesity, including respiratory muscle dysfunction, diminished central ventilatory drive, and increased resistance to certain hormonal modulators, such as leptin. Obesity reduces respiratory system compliance, increases airways resistance, and creates a state of relative respiratory muscle weakness—possibly related to fatigue from the increased work of breathing. Other as-yet-unidentified contributors must also exist, as the majority of patients with obesity do not develop OHS. In most cases, correction of obesity improves the elevated daytime $PaCO_2$ and hypoventilation. CPAP therapy, the mainstay of therapy if weight loss does not occur, has been shown to yield significant increases in PaO_2 and decreases in $PaCO_2$. Of note, bilevel pressure has not been shown to be superior to CPAP, though it may be helpful in cases when there is failure of CPAP therapy or inability to tolerate high CPAP pressures.

Patients with OHS may come to the attention in the inpatient setting after admission with severe hypercapnia or admission for other acute medical illnesses such as pneumonia. Despite obesity, these patients will not have other obvious contributors to hypercapnia (sedative use, chronic obstructive pulmonary disease, or neuromuscular disease). Although they are often admitted to the intensive care unit for rapid correction of their hypercapnia, transfer to the medical floor allows optimization of care prior to discharge. Unlike patients with pure OSA, these patients do not typically require additional diagnostic testing to justify the use of home positive pressure therapy, as hypoventilation is an approved indication for this treatment. Most providers implement bilevel pressure acutely to assist with the probable underlying OSA as well as the hypoventilation with empiric choices of pressure levels based upon patient tolerance and the need to eliminate carbon dioxide. Commonly used starting pressures include inspiratory positive airway pressure of 10 cm H_2O and expiratory positive airway pressure of 5 cm H_2O. Hospitalists should refer these patients for a formal CPAP or bilevel titration after discharge, as the optimal pressure levels will depend upon the severity of the underlying obstructive physiology.

Patients with OHS use significantly more resources and have far greater medical expenses when compared with morbidly obese eucapnic patients and sleep-disordered breathing patients without hypercapnia. Additionally, OHS patients have a greater morbidity and mortality than obese subjects without OHS. These patients have higher rates of comorbid congestive heart failure, angina, and cor pulmonale, as well as a greater rate of hospitalization, need for intensive care, and need for mechanical ventilation.

DISCHARGE PLANNING

Patients with sleep apnea who were on therapy prior to their hospitalization and who do not demonstrate any signs of inadequate treatment have no specific discharge needs. Masks or tubing are required. Even if disease is stable and well controlled, annual follow-up with a sleep specialist should be considered to ensure continued compliance with therapy and monitoring for complications of undertreated disease.

For patients without previously documented disease, ensuring rapid confirmation and implementation of therapy by arranging an outpatient polysomnogram or home sleep apnea test is strongly recommended, more so if the patient was admitted with a disease that might have resulted from inadequately treated apnea, such as

TABLE 235-4 Evidence-based Medicine Key References

Study	Methodology	Results	Limitations	Bottom Line
Goring K, Collop N. J Clin Sleep Med. 2008;4(2):105-110.	Retrospective chart review of 100 polysomnographies ordered after hospitalization	High frequency of sleep disordered breathing (77%) in sample, especially with underlying cardiopulmonary disease	Retrospective; cannot determine impact of sleep-disordered breathing on acute exacerbations of cardiopulmonary disease	Patients with sleep-disordered breathing can be successfully identified during hospitalization
Finkel KJ, et al. Sleep Med. 2009;10(7): 753-758.	Prospective observational study of surgical patients screened for OSA	23.7% of patients screened high risk for OSA of which 81% did not have prior diagnosis of OSA; among patients tested 82% had OSA	Did not look at outcomes of patients with OSA; home studies used to make diagnosis of OSA	Undiagnosed OSA is prevalent in adult surgery population, and universal screening can help identify these patients
Hwang D, et al. Chest. 2008;133: 1128-1134.	Preoperative patients were screened for sleep-disordered breathing and tested with home oximetry; postoperative complications were assessed	57% of patients had evidence of sleep-disordered breathing by nocturnal oximetry; postoperative complications were much higher with greater degrees of nocturnal desaturation	Nocturnal desaturation used to identify patients with sleep-disordered breathing	Postoperative complications are much greater in patients with sleep-disordered breathing, highlighting importance of preoperative screening and testing
Khayat RN, et al. Chest. 2009;136(4): 991-997.	Randomized clinical trial of patients admitted with decompensated heart failure who underwent sleep study and randomized to treatment with CPAP vs usual care for heart failure	Left ventricular ejection fraction was significantly superior in intervention arm	Small study of 46 patients; unable to look at other outcomes	Early identification of OSA in patients with acute decompensated heart failure is important as treatment with CPAP can improve systolic function in this setting

CPAP, continuous positive airway pressure; OSA, obstructive sleep apnea.

myocardial infarction, congestive heart failure, or cerebrovascular accident. The hospitalist may need to arrange nocturnal oxygen supplementation for severely affected patients if polysomnography cannot be performed soon after discharge. However, not all such patients will qualify for therapy, due to lack of definitive evidence of benefits of this treatment in patients with sleep apnea. Arranging an outpatient appointment with a sleep specialist for these patients is critical, as close follow-up after implementation of CPAP therapy is one of the best predictors of long-term compliance with treatment (Table 235-4).

SUGGESTED READINGS

American Society of Anesthesiologists: practice guidelines for the perioperative management of patients with obstructive sleep apnea: An updated report by the American Society of Anesthesiologists Task Force on Perioperative Management of Patients with Obstructive Sleep Apnea. Anesthesiology. 2014;120:268-286.

American Academy of Sleep Medicine. International Classification of Sleep Disorders, 3rd ed. Darien, IL: AASM; 2014.

Basner RC. Continuous positive airway pressure for obstructive sleep apnea. N Engl J Med. 2007;356(17):1751-1758.

Costanzo MR, Khayat R, Ponikowski P, et al. Mechanisms and clinical consequences of untreated central sleep apnea in heart failure. J Am Coll Cardiol. 2015;65(1):72-84.

Pérez de Llano LA, Golpe R, Ortiz Piquer M, et al. Short-term and long-term effects of nasal intermittent positive pressure ventilation in patients with obesity-hypoventilation syndrome. Chest. 2005;128(2): 587-594.

Finkel KJ, Searleman AC, Tymkew H, et al. Prevalence of undiagnosed obstructive sleep apnea among adult surgical patients in an academic medical center. Sleep Med. 2009;10(7):753-758.

Flemons WW. Clinical practice. Obstructive sleep apnea. N Engl J Med. 2002;347(7):498-504.

Goring K, Collop N. Sleep disordered breathing in hospitalized patients. J Clin Sleep Med. 2008;4(2):105-110.

Hwang D, Shakir N, Limann B, et al. Association of sleep-disordered breathing with postoperative complications. Chest. 2008;133: 1128-1134.

Kauta, SR, Keenen, BT, Goldberg, L, Schwab, RJ. Diagnosis and treatment of sleep disordered breathing in hospitalized cardiac patients: a reduction in 30-day hospital readmission rates. J Clin Sleep Med. 2014;10(10):1051-1059.

Khayat RN, Abraham WT, Patt B, Pu M, Jarjoura D. In-hospital treatment of obstructive sleep apnea during decompensation of heart failure. Chest. 2009;136(4):991-997.

Lindenauer PK, Stefan MS, Johnson KG, Priya A, Pekow PS, Rothberg MB. Prevalence, treatment, and outcomes associated with OSA among patients hospitalized with pneumonia. Chest. 2014;145(5):1032-1038.

McNicholas WT. Diagnosis of obstructive sleep apnea in adults. Proc Am Thorac Soc. 2008;5(2):154-160.

Mokhlesi B, Kryger MH, Grunstein RR. Assessment and management of patients with obesity hypoventilation syndrome. Proc Am Thorac Soc. 2008;5(2):218-225.

Myers, KA, Mrkobrada M, Simel DL, Does This Patient Have Obstructive Sleep Apnea? The Rational Clinical Examination Systematic Review. JAMA. 2013;310(7):731-741.

Netzer N, Eliasson AH, Netzer C, Kristo DA. Overnight pulse oximetry for sleep-disordered breathing in adults: a review. Chest. 2001;120(2): 625-633.

Spurr KF, Graven MA, Gilbert RW. Prevalence of unspecified sleep apnea and the use of continuous positive airway pressure in hospitalized patients, 2004 National Hospital Discharge Survey. Sleep Breath. 2008;12(3):229-234.

Young T, Palta M, Dempsey J, Peppard PE, Nieto FJ, Mae Hla K. Burden of Sleep Apnea: Rationale, Design, and Major Findings of the Wisconsin Sleep Cohort Study. WMJ. 2009;108(5): 246-249.