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In the Clinic®

Obstructive Sleep Apnea

Screening and Prevention

Diagnosis

Treatment

Practice Improvement

Obstructive sleep apnea (OSA) is very common but is frequently undiagnosed. Symptoms include loud snoring, nocturnal awakening, and daytime sleepiness. Motor vehicle accidents due to drowsy driving are a particular concern. Evaluation and treatment should focus on symptomatic patients, both to alleviate symptoms and to potentially decrease cardiovascular risk. In recent years, a strategy of home sleep apnea testing followed by initiation of autotitrating continuous positive airway pressure therapy in the home has greatly reduced barriers to diagnosis and treatment and has also facilitated routine management of OSA by primary care providers.

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Obstructive sleep apnea (OSA) is characterized by repeated episodes of upper airway closure during sleep that result in recurrent oxyhemoglobin desaturation and sleep fragmentation. OSA syndrome is defined by the combination of OSA and resulting symptoms (typically daytime sleepiness). In the general adult population, OSA syndrome occurs in 14% of men and 5% of women (1). The prevalence of OSA is increasing in conjunction with increasing rates of obesity (1), with 5-year incidence of 7%–11% in middle-aged adults (2). Despite this, only about 1 in 50 patients with symptoms suggestive of OSA syndrome is evaluated and treated for the disease (3).

The most common symptoms associated with OSA include snoring that is bothersome to others, nocturnal awakening, nocturia, unrefreshing sleep, and daytime sleepiness resulting in reduced quality of life. It also impairs the sleep quality of bedpartners. If left untreated, OSA can have long-term consequences, such as increased risk for motor vehicle and occupational accidents (4). In addition, the physiologic stresses from repetitive upper airway obstruction can lead to increased blood pressure (5). OSA is associated with increased risk for atrial fibrillation, heart failure, and stroke as well as type 2 diabetes and Alzheimer disease. However, whether treatment can prevent or reverse these conditions is unclear.

Screening and Prevention

Who should be screened for OSA?

Because most patients with OSA symptoms do not report them to their primary care provider (6), there is reason to believe that screening could be beneficial. The U.S. Preventive Services Task Force has highlighted the lack of high-quality research to justify routine screening for OSA (7). Nevertheless, the American Academy of Sleep Medicine (AASM) recommends asking all adults whether they are dissatisfied with their sleep or have daytime sleepiness as part of a routine health maintenance evaluation (8). This can be achieved by including sleep as part of a review of systems. Those with positive responses should be screened for OSA by using further clinical history or screening instruments.

Patients with risk factors should also be screened (see the **Box**: Risk Factors for Obstructive Sleep Apnea). Because obesity is a major risk factor for OSA, all obese patients should be screened. Excess weight is responsible for 41% of all cases and 58% of moderate to severe cases (9), and risk for OSA increases as obesity increases. Of note, patients of East Asian heritage are at risk for OSA at lower levels of obesity than other racial groups because of differences in facial bone structure.

The AASM also recommends screening patients with a family history of OSA and those who have retrognathia. Patients who have high-risk driving occupa-

Risk Factors for Obstructive Sleep Apnea

- Obesity, especially with body mass index >35 kg/m^2
- Family history of obstructive sleep apnea
- Retrognathia
- Treatment-resistant hypertension
- Congestive heart failure
- Atrial fibrillation
- Stroke
- Type 2 diabetes
- Polycystic ovary syndrome
- Acromegaly
- Down syndrome

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tions, such as commercial truck drivers and public transit operators, should be screened for OSA because of the potential public health effect, and any patient with a history of a recent motor vehicle crash or near miss attributable to sleepiness should be screened (10).

Screening should also be done in patient populations with diseases that commonly co-occur with OSA. For example, in those with treatment-resistant hypertension, atrial fibrillation, heart failure, stroke, and type 2 diabetes, the prevalence of OSA is high, ranging from 35%–85%.

What screening tools can be used?

Several screening questionnaires have been developed to identify high-risk patients (7, 11), but none is as accurate as formal sleep testing. The Berlin Questionnaire and the STOP-BANG (see the **Box**) screening test are 2 widely used, well-validated instruments.

The Berlin Questionnaire (**Supplement**, available at Annals.org) was developed for a primary care population and consists of 10 questions focused on the severity of snoring, witnessed apnea, the significance of daytime sleepiness, and the presence of obesity and hypertension. When the questionnaire was evaluated in a primary care setting, more than 1 in 3 respondents was found to be at high risk for OSA and sensitivity was 86% for predicting OSA (12).

The STOP-BANG screening test was developed to assess patients in the preoperative setting. It is an 8-item tool with 1 point for each item. A score of 3 or higher among preoperative patients had a sensitivity of 84% for predicting any OSA, and a score of 5 or higher was more predictive of clinically relevant, moderate to severe OSA (13).

STOP–BANG Screening Test*

STOP

S Do you snore loudly (louder than talking or loud enough to be heard through closed doors)?

T Do you often feel tired, fatigued, or sleepy during the day?

O Has anyone observed you stop breathing during sleep?

P Do you have or are you being treated for high blood pressure?

BANG

B Body mass index >35 kg/m^2?

A Age >50 years?

N Neck circumference >40 cm?

G Gender male?

*In a perioperative setting, answering "yes" to ≥3 questions indicates high risk for obstructive sleep apnea. Answering "yes" to ≥5 questions indicates high risk for moderate to severe obstructive sleep apnea.

Can OSA be prevented?

Weight gain over time is associated with OSA incidence: A 10% increase in weight predicts a 6-fold increase in the likelihood of developing clinically significant OSA (14). As such, avoiding weight gain reduces OSA risk. Furthermore, weight loss of 10% among patients followed over 10 years predicted a 26% decrease in OSA severity. Among patients with mild OSA, nearly 9 in 10 who lost an estimated 15% of body weight through diet and lifestyle modification achieved remission (15).

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Screening and Prevention... Although there is no strong evidence to justify routine screening, it is reasonable to consider asking about sleep problems as part of a review of systems, particularly in high-risk populations. Patients with sleep symptoms should be screened with further clinical history or validated questionnaires for OSA. Prevention should focus on achieving and maintaining an ideal body weight.

CLINICAL BOTTOM LINE

Diagnosis

What symptoms should prompt consideration of OSA?

Symptoms of OSA are shown in the **Box**. Snoring has the highest sensitivity for OSA but is nonspecific (16). To distinguish simple snoring from that suggestive of OSA, patients should be asked for details about the snoring. Patients with OSA are more likely than simple snorers to report loud, nightly snoring that is bothersome to others (12).

Excessive daytime sleepiness is also a nonspecific finding but is critical to elicit in determining therapy options and following the response to therapy. The Epworth Sleepiness Scale (**Figure 1**) is an 8-item scale that quantifies the propensity for dozing off during everyday activities (17). Although correlation with objective measurements of sleepiness is inconsistent and correlation with OSA severity is poor, it can help standardize the evaluation of a patient's subjective perception. It can also be used to follow response to therapy. The Epworth Sleepiness Scale has been adopted by many insurance plans as a required part of the sleep history before payment for a sleep study is authorized. A history of drowsiness or falling asleep while driving should be explicitly explored. Patients should also be questioned about consumption of caffeine or other stimulants because it may indicate attempts to self-treat sleepiness.

Symptoms of Obstructive Sleep Apnea

- Loud, frequent, bothersome snoring
- Witnessed episodes of apnea
- Choking/gasping during sleep
- Excessive daytime sleepiness
- Drowsy driving (recent motor vehicle accident or near miss associated with sleepiness)
- Unrefreshing sleep
- Frequent nocturnal awakening
- Sleep-maintenance insomnia (prolonged wake after sleep onset)
- Nocturia
- Morning headaches
- Decreased concentration ("brain fog")
- Depressed mood
- Irritability
- Decreased libido

Although relatively insensitive, choking or gasping during sleep is highly specific for moderate to severe OSA, as is the presence of morning headaches (16). Obtaining a history from a bedpartner or cohabitant can be particularly helpful because many of these symptoms may not be apparent to the patient. Manifestations of untreated OSA may also include depressive symptoms, decreased libido, and frequent nocturnal awakening. Patients with OSA can describe being in a "brain fog" or having difficulty concentrating as opposed to sleepiness.

Of note, OSA can sometimes present in an atypical fashion, with insomnia and fatigue as the predominant symptoms, particularly in women. Sleep-maintenance insomnia (difficulty with falling back to

sleep after nocturnal awakening) is more likely related to OSA than sleep-onset insomnia (18). Despite population-based studies that found a 2:1 male–female ratio of prevalence, utilization data indicate that the male–female ratio for referrals is 9:1, suggesting that clinicians do not adequately consider OSA in women (19).

In the absence of symptoms, what other diseases should prompt evaluation?

No high-level evidence supports routine testing for OSA in asymptomatic patients. However, diagnostic testing in asymptomatic, morbidly obese patients scheduled for bariatric surgery may be reasonable given the high prevalence in this population, the association of OSA with adverse perioperative outcomes (20), and low-level evidence that perioperative treatment improves outcomes (21).

Evaluation and treatment of OSA may also benefit asymptomatic patients with hypertension that is refractory to 5 or more medications. Prevalence of OSA is extremely high in this patient population, and treatment produces a clinically important reduction in blood pressure (22).

What other conditions should be considered when evaluating patients with possible OSA?

OSA frequently coexists with obesity hypoventilation syndrome (OHS), a condition defined by daytime hypercapnia among obese patients without other causes of hypoventilation. This syndrome is present in up to 10%–20% of morbidly obese patients with OSA, and patients with OHS have higher rates of cardiovascular complications, such as cor pulmonale.

Figure 1. **Epworth Sleepiness Scale.**

Consider how you have felt over the past week or two. How likely are you to doze off or fall asleep in the following situations?

0 = None
1 = Slight
2 = Moderate
3 = High

Situation	Score
Sitting and reading	
Watching television	
Sitting inactive in a public place (e.g., theater or meeting)	
As a passenger in a car for an hour without a break	
Lying down in the afternoon when able	
Sitting and talking to someone	
Sitting quietly after lunch without alcohol	
In a car while stopped for a few minutes in traffic	
Add above for total score	

Although imperfect, this scale can be a useful guide to quantifying the subjective concept of "sleepiness." Scores >10 are consistent with excessive daytime sleepiness and should prompt further clinical evaluation.

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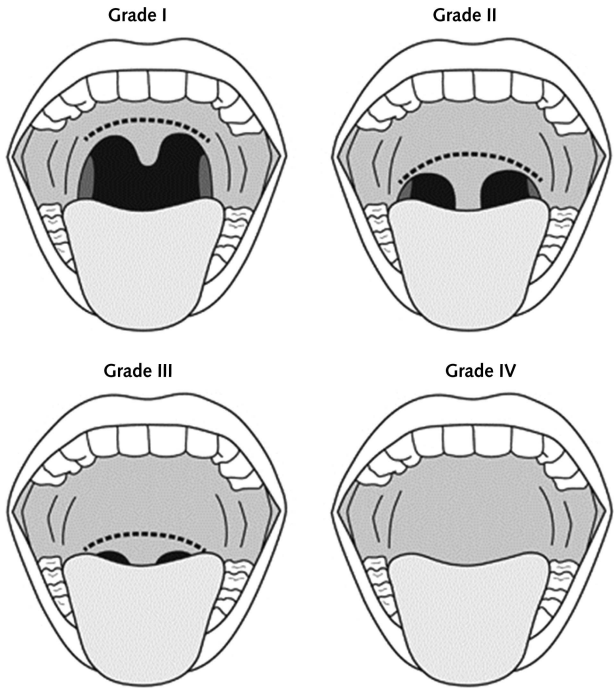
Figure 2. **Modified Mallampati classification.**



The mouth is evaluated while the patient is sitting with the tongue protruded. A higher classification is associated with higher risk for obstructive sleep apnea. Grade I = soft palate, uvula, tonsillar fauces, and pillars visible; grade II = soft palate, uvula, and tonsillar fauces visible; grade III = only soft palate and base of uvula visible; grade IV = only hard palate visible.

Observed episodes of apnea and nocturnal gasping may indicate a central sleep apnea syndrome rather than OSA. Patients with congestive heart failure are at risk for Cheyne–Stokes respiration, and those receiving long-term opioid therapy are at risk for opioid-induced central sleep apnea.

Finally, it is important to note that many symptoms of OSA, such as sleepiness, poor concentration, and difficulty staying asleep, are nonspecific. Other common sleep disorders, including insomnia, chronic sleep deprivation, and circadian rhythm disorders (such as shift work sleep disorder), may cause these symptoms.

What physical examination findings are important?

The physical examination should include the respiratory, cardiovascular, and neurologic systems. The presence and degree of obesity should be noted. Particular attention should be paid to the following signs of upper airway narrowing: enlarged neck circumference (>16 inches in women and >17 inches in men), a modified Mallampati score of 3 or 4 (**Figure 2**), macroglossia, tonsillar hypertrophy, an enlarged or elongated uvula, a high or arched palate, signs of nasal obstruction (polyps, septal deviation, turbinate hypertrophy, significant congestion), and retrognathia.

What type of sleep study should be ordered?

A diagnosis of OSA requires formal testing to document obstructive respiratory events during sleep. Historically, such testing was performed in a sleep laboratory with a technician present, with determination of continuous positive airway pressure (CPAP) treatment pressures requiring similar intensive monitoring. However, several randomized

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trials have found that a home-based diagnosis and treatment strategy incorporating home sleep apnea testing (HSAT) followed by initiation of treatment in the home leads to equivalent outcomes for patients with uncomplicated OSA (23–26). HSAT does not include electroencephalography (EEG) and so does not measure sleep per se. Rather, it only records respiratory channels, such as oximetry, airflow, and chest movement, and these monitors are relatively easy to self-apply. A home-based strategy is cheaper, leads to more rapid initiation of treatment, can broaden access for patients in remote or underserved areas (23, 24), and is preferred by patients (25). Therefore, for most patients being evaluated for OSA, HSAT is the preferred diagnostic test. However, some insurance plans, such as many Medicaid programs, do not cover HSAT despite the lower cost and patient preference.

Of note, use of overnight oximetry to diagnose OSA leads to poorer outcomes than either HSAT or in-laboratory testing (27). There is also no evidence to support use of overnight oximetry to screen patients before formal testing, particularly because HSAT adds little additional burden over oximetry alone. Commonly used terms to quantify and classify OSA severity obtained from both HSAT and in-laboratory studies are presented in the **Box**.

What is the role of in-laboratory sleep studies?

An in-laboratory sleep study or polysomnography involves overnight recording of multiple physiologic channels (**Figure 3**) and is useful to evaluate for OSA in patients who are unable to complete HSAT. Reasons for this could include unstable housing or cognitive or physical disabilities that preclude the patient from self-applying a sleep monitor. Causes of oxyhemoglobin desaturation other than OSA, such as chronic lung disease, congestive heart failure, or high likelihood of central sleep apnea, are all relative contraindications for HSAT because the accuracy of HSAT in these settings has not been evaluated. An in-laboratory sleep study should be considered when such alternative causes of sleep-disordered breathing are in the differential diagnosis. However, in some cases, in-laboratory sleep studies are impractical, so HSAT can still

Sleep Study Terminology and OSA Definitions

Terminology

- Apnea: Breathing cessation for ≥10 seconds
- Hypopnea: Breathing flow reduction for ≥10 seconds, accompanied by a ≥3% or ≥4% oxyhemoglobin desaturation or arousal from sleep
- AHI: Number of apnea and hypopnea episodes per hour of sleep
- REI: Number of apnea and hypopnea episodes per hour of recording
- ODI: Number of ≥3% or ≥4% oxyhemoglobin desaturation episodes per hour of sleep
- Time below SpO_2 90%: Sleep or recording time spent with oxyhemoglobin saturation <90%

Definitions*

- Mild OSA: AHI or REI ≥5 but <15 events/h
- Moderate OSA: AHI or REI ≥15 but <30 events/h
- Severe OSA: AHI or REI ≥30 events/h
- OSA syndrome: AHI or REI ≥5 events/h with daytime sleepiness

AHI = apnea–hypopnea index; ODI = oxygen desaturation index; OSA = obstructive sleep apnea; REI = respiratory event index.

*All definitions presume that most cases of apnea and hypopnea have an obstructive (rather than central) cause.

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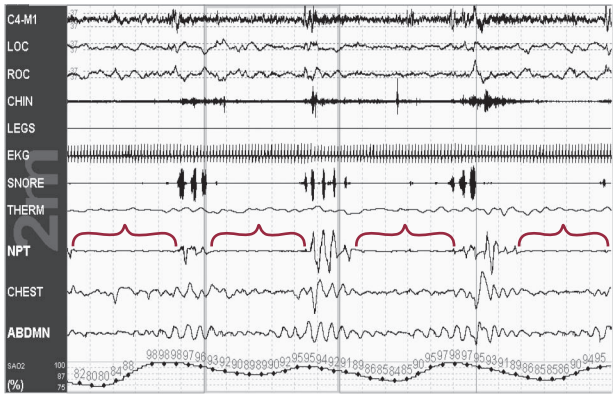
Figure 3. **Sample recording from overnight polysomnography demonstrating obstructive sleep apnea.**



This 2-minute window demonstrates repeated episodes of obstructive apnea (denoted by curly brackets), characterized by airflow cessation lasting ≥10 seconds despite respiratory effort. Here, apnea is associated with oxyhemoglobin desaturation and is terminated by arousal from sleep. ABDMN = respiratory inductance plethysmography bands placed around the abdomen; C4-M1 = electroencephalogram; CHEST = respiratory inductance plethysmography bands placed around the thorax; CHIN = chin electromyogram; EKG = electrocardiogram; LEGS = leg electromyogram; LOC = left electro-oculogram; NPT = airflow monitoring by nasal pressure transducer; ROC = right electro-oculogram; SaO_2 = arterial oxyhemoglobin saturation; SNORE = vibratory snore; THERM = airflow monitoring by thermal air sensor.

be useful in these situations. In-laboratory sleep studies are preferred over HSAT to evaluate for nonrespiratory sleep disorders (for example, narcolepsy) because HSAT only records respiratory parameters. A diagnostic in-laboratory sleep study can be ordered as either a "full-night" to allow for a full night of recording without intervention or a "split-night," in which an initial diagnostic recording of at least 2 hours that documents OSA is immediately followed by CPAP titration. Use of split-night studies allows for determination of CPAP requirements on the same night as diagnosis but requires laboratory staff capable of accurately diagnosing OSA in real time and a patient who is well prepared and accepting of the possibility of being awakened to start CPAP therapy.

What variables are reported on a sleep study report, and what do they mean?

The key metric used to stratify OSA severity is the apnea–hypopnea index (AHI), defined as the number of episodes of apnea (cessation of airflow for ≥10 seconds) and hypopnea (airflow reduction for ≥10 seconds, accompanied by a ≥3% or ≥4% oxyhemoglobin desaturation or arousal from sleep) per hour of sleep. With HSAT where sleep is not measured, the number of respiratory events is divided by the total recording time to compute the respiratory event index (REI). When a patient spends a large portion of the night awake, the REI can substantially underestimate the AHI. Of note, varying definitions of hypopnea (for example, 3% vs. 4% desaturation with or without arousal) can lead to markedly different AHI or REI calculations and can limit comparisons of polysomnography reports from different sleep laboratories. The AHI based on a 4% desaturation criterion for hypopnea has been more closely associated with cardiovascular risk (28) and is preferred by the Centers for Medicare & Medicaid Services (CMS). However, this definition is more restrictive and may preclude diagnosis in some patients with classic symptoms

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but without significant oxyhemoglobin desaturations who would benefit from therapy.

Other measures of sleep-disordered breathing provided by both in-laboratory and home studies include the number of central versus obstructive respiratory events, the frequency of oxyhemoglobin desaturation events, time spent with an oxyhemoglobin saturation below 90%, and nadir oxyhemoglobin saturation. In-laboratory sleep studies also provide nonrespiratory information about a patient's sleep, such as total sleep time, measures of sleep architecture and fragmentation (sleep latency, sleep efficiency, wake time after sleep onset, time in each sleep stage, and arousal index), assessment of any EEG epileptiform activity, nocturnal arrhythmia, limb movements, and video or audio recording of sleep-related behaviors.

Do patients need to be seen by a sleep specialist before a sleep study is ordered?

Consultation with a sleep specialist is not necessary before evaluation of uncomplicated OSA. However, clinicians should adequately counsel patients on the rationale for diagnostic testing, OSA-related health risks, and what to expect with a sleep study and should address concerns about subsequent management. Without this information, no-show and cancellation rates for sleep studies are often high. An important practical consideration is that CMS requires documentation of the symptoms leading to consideration of an OSA diagnosis as part of a face-to-face clinical encounter before diagnostic testing in order to cover OSA treatment. Thus, clinicians need to adequately document the rationale for OSA testing as part of a face-to-face (not telephone or e-mail) encounter.

Evaluation by a sleep specialist before a sleep study is ordered is recommended when complex forms of sleep-disordered breathing (for example, suspected nocturnal hypoventilation due to chronic lung or neuromuscular disease, OHS, central sleep apnea) or nonrespiratory sleep disorders (for example, parasomnia, narcolepsy) are in the differential diagnosis so that the most appropriate test and monitoring during the test can be planned.

Diagnosis... Patients who snore loudly, have significant daytime sleepiness, or have a history of drowsy driving should be evaluated for OSA, particularly if they also have nocturnal choking or gasping or witnessed episodes of apnea. Although in-laboratory sleep studies are considered the gold standard, HSAT is now the preferred diagnostic method for most patients with clinical suspicion for OSA and low suspicion for other sleep disorders. Primary care providers can order HSAT without the need for sleep specialists for uncomplicated patients.

CLINICAL BOTTOM LINE

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Treatment

Which patients with OSA require treatment?

High-level evidence indicates that patients with daytime sleepiness, regardless of severity, should be offered therapy (29). In particular, those who have recently had a motor vehicle accident or near miss attributable to sleepiness should be aggressively treated. Treatment should also be offered to patients with other symptoms that are attributable to OSA, and shared decision making can be used to balance the benefits of symptom abatement against the inconvenience of therapy. A trial of therapy can often be helpful to allow the patient to make an informed decision based on the magnitude of benefit and the inconvenience of treatment. It is important to include the bedpartner in treatment decisions because partner support is an important predictor of treatment acceptance and adherence and because treatment improves sleep quality in the bedpartner (30).

Randomized trials of CPAP therapy in nonsleepy patients with moderate to severe OSA have found no reduction in risk for cardiovascular events (31–33). As such, CPAP therapy should not be routinely recommended to asymptomatic patients with moderate to severe OSA. There is no evidence that asymptomatic patients with mild OSA are at elevated cardiovascular risk and would benefit from treatment (34).

What is the role of weight loss and exercise?

Randomized trials have shown that behavioral weight loss interventions reduce OSA severity and symptoms (35). Furthermore, initiating CPAP therapy and behavioral weight loss interventions together does not lead to a reduction in adherence to either treatment, and the achieved benefits are additive (36). Therefore, comprehensive weight loss interventions that combine diet, exercise, and behavior modification should be recommended to all overweight or obese patients with OSA. Bariatric surgery can also substantially reduce the severity of OSA in morbidly obese patients. Although the likelihood of normalizing the AHI with bariatric surgery is low, improvements in OSA symptoms are much greater than improvements in AHI.

Can OSA be effectively managed by alterations in sleep position?

In many patients, gravitational forces on the tongue lead to worsening of upper airway narrowing and OSA severity when the patient is supine. Position-dependent OSA is found in up to one third of patients with mild or moderate OSA. Positional therapy involves wearing a tennis ball, backpack, or foam device strapped to the back or, alternatively, a monitor that sounds a gentle alarm when the patient is supine to minimize the time spent sleeping on the back. Short-term data suggest that these treatments can reduce OSA severity and sleepiness(37).

How should CPAP therapy be initiated?

CPAP provides pneumatic splinting of the upper airway and should be considered the first-line treatment option for moderate to severe OSA and for any OSA in high-risk drivers. CPAP settings have traditionally been determined via an overnight titration study in a sleep laboratory. However, for patients with uncomplicated OSA, empirical prescription of autotitrating CPAP is an effective alternative to in-laboratory overnight titration. Autotitrating CPAP devices detect flow limitation as a surrogate marker for upper airway narrowing and automatically adjust

pressure to remain therapeutic. Autotitrating CPAP performs as well as fixed-pressure CPAP in terms of patient adherence and reduction in sleepiness (29). It is contraindicated in patients with Cheyne–Stokes breathing and has not been well studied in patients with comorbid pulmonary disease. An advantage of autotitrating CPAP is that pressures self-adjust as therapy requirements change, such as with weight gain over time. A prescription for CPAP should include specifications for the pressure setting, mask type, and associated device supplies (tubing, filters, mask straps). Autotitrating CPAP is typically prescribed with a wide pressure range, such as 5–20 cm H_2O.

When therapy is initiated, patients should be educated about function, care, and maintenance of the equipment; the benefits of therapy; and potential problems, such as mask or pressure intolerance, nasal congestion, excessive dryness or moisture, or air leak around the mask. Masks have traditionally been selected in the laboratory at the time of overnight titration. With home-based treatment using autotitrating CPAP, the mask must be selected at the time of CPAP delivery. It is important to allow the patient to try several different mask styles and sizes to find the most comfortable fit.

What amount of CPAP use constitutes sufficient adherence?

There is no threshold of use at which optimal benefit from CPAP therapy is derived. Strategies to maximize adherence are shown in the **Appendix Figure** (available at Annals.org). Studies show a linear relationship between hours of use and improvements in sleepiness, quality of life, and blood pressure (38, 39); therefore, patients should use CPAP whenever they sleep. However, CMS has defined "adequate" adherence as 4 or more hours of use per night on 70% of nights. CMS and most insurance plans require documentation of this level of adherence during a consecutive 30-day period within the first 90 days of CPAP therapy to continue payment. Nationally, 75% of patients achieve this threshold (40).

What factors can optimize patient adherence to CPAP therapy?

Adherence patterns are determined within the first week after CPAP initiation, and a patient's early perceived benefit of therapy is a strong predictor of long-term use. Accordingly, optimal management of patients with OSA should include early follow-up (for example, 1–2 weeks after therapy initiation) to identify and address any problems. Patients should be instructed to bring their CPAP devices and masks to the clinic to review proper use. Adherence data can be obtained directly from the device or via remote transmission and are important to review because patients tend to overestimate use.

Early on, poor mask fit, nasal congestion, and airway drying are the most common adverse effects that can impair adherence. Changing masks and/or adjusting fit, using nasal steroids, and increasing humidification settings can readily ameliorate these problems. Otherwise, the most important barriers to adherence are those related to behavior change, such as denial, knowledge deficits, low self-efficacy, lack of perceived improvement, and lack of social support. Education about the adverse effects of OSA and the benefits of treatment, troubleshooting, and behavioral interventions that enhance motivation have all been shown to increase adherence (30). Daily feedback to patients on CPAP use as well as telemonitoring of use with automated messaging to provide encouragement or positive feedback have both been shown to increase adherence (41, 42). All CPAP manufacturers now provide free apps that combine daily feedback with education and automated feedback. Use of these apps should be encouraged.

Claustrophobia can occasionally affect one's ability to tolerate a CPAP mask. This can be addressed through desensitization, whereby the patient slowly increases the time spent wearing the mask while engaging in distracting activities, such as watching television during the daytime (without pressure at first). Pressure intolerance is a rare reason for low adherence, and interventions that decrease pressure, such as bi-level PAP or expiratory pressure relief, do not improve outcomes compared with CPAP when used routinely. Nevertheless, for selected patients who report difficulty exhaling or have other pressure-related adverse effects (such as aerophagia), these options should be considered.

How should a CPAP mask be chosen?

Masks should be chosen to maximize patient comfort. Involving patients in mask selection can facilitate acclimatization. Nasal masks or nasal pillows (which sit under the nose and fit in the nares) may be better tolerated in patients with claustrophobia. Nasal pillows may be more effective in patients with unusual nasal bridge anatomy, facial hair, or absent dentition leading to a lack of infranasal support.

For patients whose mouths open during sleep, the airflow delivered by a nasal mask may leak out of the mouth, preventing effective treatment. In many patients, keeping the mouth closed can be learned over a few nights or can be accomplished with use of a chin strap. Treatment of nasal congestion with saline irrigation, nasal steroids, or antihista-

mines can also enhance nasal breathing. In patients who remain mouth breathers, an oronasal ("full-face") mask can be used to deliver pressure through the nose and mouth. However, these masks are bulkier, leading to greater problems with mask leak and psychological distress. Further, they can worsen OSA severity by pushing the mandible back. Overall, clinical outcomes are worse with oronasal masks versus nasal interfaces, so they should generally be used only when other options have failed (43).

Poor fit or intolerance often does not become apparent until the mask has been used for a few nights. Patients should be encouraged to report problems early because masks can typically be replaced without cost in the first 30 days.

What is the role of mandibular advancement devices?

Custom-made mandibular advancement devices (MADs) are oral appliances that treat OSA by holding the mandible in a forward position, thus decreasing airway collapsibility and enlarging the upper airway. These devices are less effective than CPAP at normalizing the AHI and are therefore not recommended as initial therapy for severe OSA (44). However, MADs are a reasonable initial therapy for patients with mild or moderate OSA because despite reduced efficacy, they tend to be more acceptable to patients and are therefore associated with greater adherence. Among patients with mild to moderate OSA, MADs have real-world effectiveness similar to that of CPAP with regard to improvements in sleepiness and quality of life (45). For patients with severe OSA who do not tolerate CPAP therapy despite attempts to address barriers, MADs are a secondary option. Although insurance will typically cover only 1 treatment, use of both devices can be considered in patients who can afford it. In this situation, the CPAP is often used at home, and the MAD, which is more portable and inconspicuous, is used when traveling.

Use of MADs requires adequate dentition and may exacerbate temporomandibular joint disease. Patients who need these devices should be referred to a dentist with expertise in OSA management, preferably with sleep dentistry accreditation. In addition, if a MAD is being used as primary therapy, a follow-up sleep study to document adequacy in reducing OSA is recommended.

What is the role of surgical intervention?

For patients with difficulty tolerating CPAP because of anatomical abnormalities, nasal procedures, such as nasal septoplasty or turbinate reduction, can increase tolerability. Most surgeries to decrease upper airway collapsibility do not significantly reduce OSA severity or symptoms. Uvulopalatopharyngoplasty is perhaps the best-known procedure, but improvement in symptoms is generally small, and fewer than half of patients have significant reduction in OSA severity over the long term. Maxillomandibular advancement is an invasive procedure with prolonged postoperative recovery but has an OSA cure rate above 90%, particularly in nonobese patients with retrognathia. Tracheostomy also cures OSA and can be used in life-threatening situations. These 2 surgeries may be preferable to a lifetime of CPAP therapy for selected patients, but their intensity and associated morbidity preclude routine application. Hypoglossal nerve stimulation has recently emerged as a relatively minor surgical procedure with durable response rates in appropriately selected patients (46). The procedure should be limited to those with a body mass index less than 32 kg/m^2 in whom drug-induced sleep endoscopy demonstrates collapse in an anteroposterior direction.

How should treatment be monitored?

All CPAP devices store and can report data on use, and many provide information about therapy effectiveness and mask leak (47). Current devices have a built-in modem, which transmits these data to a cloud server that can be accessed by the clinician via the Internet. These data should be reviewed at follow-up visits to confirm patient reports of use and to troubleshoot problems. Clinicians should periodically ensure that CPAP supplies, such as masks and filters, are replaced regularly by the device supplier. Because adherence often wanes over time, it is important to provide regular motivational enhancement to continue nightly CPAP use at follow-up visits as part of chronic disease management.

Therapy monitoring should focus on ensuring CPAP use during all sleep sessions; assessing for symptom resolution; monitoring for adverse effects; and assessing for comorbid conditions that are commonly associated with OSA, such as obesity and hypertension. Patients with OSA remission due to weight loss or surgery should be monitored for a return of symptoms. Those with a history of drowsy driving or sleepiness-related motor vehicle or occupational accidents should be closely monitored for continued remission of sleepiness.

Relapse should prompt the clinician to investigate for the following possibilities in a stepwise fashion: decreased adherence, problems with CPAP delivery (mask leak, device malfunction), change in pressure requirements, and non-OSA sleep fac-

tors (insufficient sleep duration, medication effect, other sleep disorders). If sleepiness persists, a trial of modafinil or armodafinil should be considered as adjunctive treatment. Solriamfetol was also recently approved for this indication.

No evidence supports routine follow-up sleep studies in patients whose symptoms do not recur. Even in those with recurrent symptoms, CPAP-recorded data should be carefully reviewed before additional sleep testing is considered.

How should OSA be treated when a patient is admitted to the hospital?

Little evidence supports any particular management strategy for patients with OSA admitted to a medical service. Nevertheless, they should be encouraged to use their CPAP or MAD while hospitalized, just as they would at home. Hospital policies often do not allow patients to use their own CPAP machine. In such cases, home settings on a hospital-owned machine should be used along with the patient's own mask. Sedative and opioid medications can worsen OSA and should be used with caution in inpatients with OSA.

In surgical patients, untreated OSA in the perioperative setting is associated with a higher rate of cardiopulmonary complications and intensive care unit transfers (48). Accordingly, the American Society of Anesthesiologists recommends that if moderate sedation is used, ventilation should be monitored by continuous oximetry and continuous capnography if feasible, and CPAP administration during sedation should be considered (49).

When should a sleep specialist be consulted?

For uncomplicated OSA, studies suggest that primary care clinicians who are educated about disease management can produce treatment outcomes similar to those of sleep specialists (50). In complicated situations, such as a CPAP-intolerant patient or one with persistent symptoms despite treatment, a sleep specialist consultation may aid in further clinical evaluation and management. In addition, for patients with multiple sleep disorders or complex sleep-disordered breathing (for example, OSA with comorbid hypoventilation syndromes or central sleep apnea), consultation with a sleep specialist may facilitate use of more advanced PAP devices where necessary.

What should patients know about the effects of medications and supplemental oxygen?

Benzodiazepine and opioid medications can worsen OSA and should be used with caution. Low-level evidence suggests that exogenous testosterone administration can exacerbate or induce OSA, so patients treated with androgen therapy should be screened and followed for symptoms. In patients with hypothyroidism, appropriate treatment with thyroid hormone reduces OSA severity and symptoms. However, routine screening for hypothyroidism in patients with OSA is not cost-effective.

Although supplemental oxygen is effective in treating oxyhemoglobin desaturation associated with OSA, there is little evidence that it reduces symptoms, blood pressure, or cardiovascular risk (51). Therefore, supplemental oxygen should not be used to treat OSA.

Can treatment prevent or modify outcomes in other diseases?

CPAP therapy has been found to reduce depression symptoms and incidence (52). However, the role of OSA screening and treatment in patients with severe depression has not been studied.

Among patients with OSA and hypertension, high-level evidence indicates that both CPAP and MAD therapy can lead to modest reductions in blood pressure and that the degree of adherence correlates with the blood pressure response (39). In those with treatment-refractory hypertension, CPAP has a much larger, clinically important effect (22). Of note, the effect of CPAP therapy is additive to other antihypertensive therapies (36, 53).

The effect of OSA therapy on cardiovascular outcomes is unclear. Although clinical trials in nonsleepy patients with moderate to severe OSA have shown no cardiovascular risk reduction with treatment, the elevated cardiovascular risk attributable to OSA seems to be restricted to patients with OSA who have daytime sleepiness (54). Among patients clinically referred for OSA treatment (presumably because of symptoms), those treated with CPAP have reduced risk for cardiovascular events compared with those who decline treatment (55). This suggests a cardiovascular benefit of treatment in symptomatic patients, but these findings may be confounded in that patients who adhere to CPAP are more likely to adhere to other cardioprotective measures.

Other diseases may also be modified by OSA therapy, but only low-level evidence exists. For example, among patients with OSA and preexisting heart failure with reduced ejection fraction, CPAP therapy may modestly increase ejection fraction. Among patients with OSA and atrial fibrillation who undergo cardioversion, therapy may be associated with a reduced likelihood of recurrence of atrial fibrillation.

Treatment... All patients with OSA should be encouraged to pursue conservative measures, such as weight control and avoidance of alcohol and sedatives before bedtime. Symptomatic patients, particularly those with drowsy driving, should be treated. CPAP is considered the primary therapy for OSA, and therapy initiation requires close follow-up. Many factors facilitate patient acceptance of and adherence to CPAP therapy, including education, participation in mask selection, early troubleshooting of problems, and behavioral strategies to increase motivation and self-efficacy. For patients who are unable to tolerate CPAP therapy, alternate options include custom-fitted oral appliances, positional therapy devices, or surgical therapies. Maximizing adherence to therapy is important in order to maximize symptom resolution and blood pressure reduction. The role of OSA treatment in improving outcomes related to atrial fibrillation, heart failure, stroke, and other common comorbid conditions remains an active area of research.

CLINICAL BOTTOM LINE

Practice Improvement

What do professional organizations recommend with regard to the care of patients with OSA?

The American College of Physicians (ACP), the AASM, and the American Thoracic Society have issued guidelines for the diagnosis and management of OSA that reflect the themes of this article (11, 29, 35, 44, 56, 57).

Of note, ACP guidelines endorse in-laboratory sleep studies for OSA diagnosis, with HSAT as an alternative when in-laboratory testing is unavailable. However, the strong evidence base for equivalent outcomes with a home-based strategy (much of which has emerged since the ACP statement), patient preferences, and lower costs all suggest that HSAT should be the preferred diagnostic method in uncomplicated patients. Similarly, although ACP guidelines recommend focusing treatment on patients with daytime sleepiness, evidence exists that other OSA symptoms, such as bothersome snoring, nocturnal awakening, and depressive feelings, also improve with treatment.

What is the role of patient education in management?

Patient education about OSA and involvement in deciding on appropriate therapy is accepted by consensus as the standard of care. Educational interventions lead to improved adherence with treatment (29).

What measures do stakeholders use to evaluate the quality of care?

The AASM and the National Committee for Quality Assurance developed performance measures for individual clinicians to assess quality of care in OSA management (58). Clinicians should assess relevant symptoms initially and longitudinally with treatment, determine the severity of disease using objective sleep testing, offer CPAP therapy, and assess treatment adherence objectively. The AASM subsequently endorsed additional performance measures focusing on cardiovascular risk reduction by measuring and addressing comorbid obesity and hypertension (59).

In the Clinic
Tool Kit

Obstructive Sleep Apnea

Patient Information

https://medlineplus.gov/sleepapnea.html
https://medlineplus.gov/languages/sleepapnea.html
Information on living with sleep apnea in English and other languages from the National Institutes of Health's MedlinePlus.

www.nhlbi.nih.gov/health/health-topics/topics/sleepapnea
www.nhlbi.nih.gov/health-topics/espanol/apnea-del-sueno
Information for patients on sleep apnea in English and Spanish from the National Heart, Lung, and Blood Institute.

www.thoracic.org/patients/patient-resources/resources/obstructive-sleep-apnea-in-adults.pdf
Patient education handout on obstructive sleep apnea in adults from the American Thoracic Society.

Information for Health Professionals

https://annals.org/aim/fullarticle/1742606/management-obstructive-sleep-apnea-adults-clinical-practice-guideline-from-american
2013 clinical practice guideline on management of obstructive sleep apnea in adults from the American College of Physicians.

https://aasm.org/resources/clinicalguidelines/diagnostic-testing-osa.pdf
2017 clinical practice guideline on diagnostic testing for obstructive sleep apnea in adults from the American Academy of Sleep Medicine.

http://jcsm.aasm.org/ViewAbstract.aspx?pid=31513
2019 clinical practice guideline on treatment of obstructive sleep apnea with positive airway pressure in adults from the American Academy of Sleep Medicine.

http://jcsm.aasm.org/ViewAbstract.aspx?pid=30098
Clinical practice guideline, updated in 2015, on treatment of obstructive sleep apnea and snoring with oral appliance therapy from the American Academy of Sleep Medicine.

www.aafp.org/afp/2016/0901/p355.html
Information on diagnosis and treatment of obstructive sleep apnea in adults from the American Academy of Family Physicians.

WHAT YOU SHOULD KNOW ABOUT OBSTRUCTIVE SLEEP APNEA

What Is Obstructive Sleep Apnea?

Obstructive sleep apnea (OSA) is a common problem that disrupts breathing during sleep. People with OSA temporarily stop breathing or have shallow breathing while sleeping. Pauses in breathing during sleep lower blood oxygen levels and trigger a person to wake up during the night. Lack of sleep can lead to an increase in accidents and reduce quality of life due to constant sleepiness. Untreated OSA can have serious health consequences, such as high blood pressure, heart disease, and stroke. It occurs more often in men than in women and in people who are overweight or obese.

What Are the Signs and Symptoms?

People with OSA may have symptoms during sleep and while awake. Common signs and symptoms include:

- Loud snoring that bothers others
- Daytime sleepiness
- Feeling drowsy while driving
- Waking often at night
- Frequent need to urinate at night
- Choking or gasping during sleep (usually observed by a sleep partner)
- Morning headaches
- Feeling depressed and irritable due to sleepiness and fatigue
- Elevated blood pressure that is difficult to control

Can I Prevent It?

If you are overweight or obese, even mild weight loss (10%) through diet and exercise may prevent you from developing OSA.

How Is It Diagnosed?

- Your doctor will ask about your medical history, the quality of your sleep, and how rested you feel. Any symptoms observed by a sleep partner, such as snoring and gasping during sleep, are important to discuss.
- You will have a physical examination. Your doctor will listen to your heart and lungs, measure your neck size, and look down your throat to check your airway for anything that may disrupt your breathing.
- If your provider suspects OSA, you will need a sleep study. The study measures your breathing, heart rate, and oxygen levels during sleep. Most sleep studies can be done at home, but some need to take place in a special laboratory.

How Is It Treated?

Your doctor can talk to you about available treatment. If you have a sleeping partner, he or she should be included in the discussion. There are different options based on how severe your OSA is. Your doctor may suggest lifestyle changes, such as losing weight, cutting back on the use of alcohol or sedatives, or using strategies to change the position you sleep in (such as wearing a tennis ball on your back to encourage sleeping on your side).

Most patients with OSA have significant improvement in their symptoms from continuous positive airway pressure (CPAP) therapy. This involves wearing a mask over the nose while you sleep. The mask connects to a machine that pushes air through your airway to prevent blockages. It is important that you understand how the machine works and how to maintain it. Potential problems with the device may include a poorly fitting or uncomfortable mask, nasal congestion, and dry airways. It is easy to fix most of these, so talk to your doctor about any concerns. You should use the machine whenever you sleep. All machines have guides that provide additional education and feedback on use.

Other options for treating OSA are available, such as oral devices or surgery, based on your particular situation. Talk with your doctor about the best treatment plan.

Questions for My Doctor

- What should I expect from a sleep study?
- May I have my sleep study at home?
- What alternatives do I have to CPAP for treatment of OSA?
- How much weight should I lose to improve my OSA symptoms?
- How can I make my CPAP treatment more comfortable?
- How can I tell if CPAP treatment is working?
- Do I need to see a sleep specialist?

For More Information

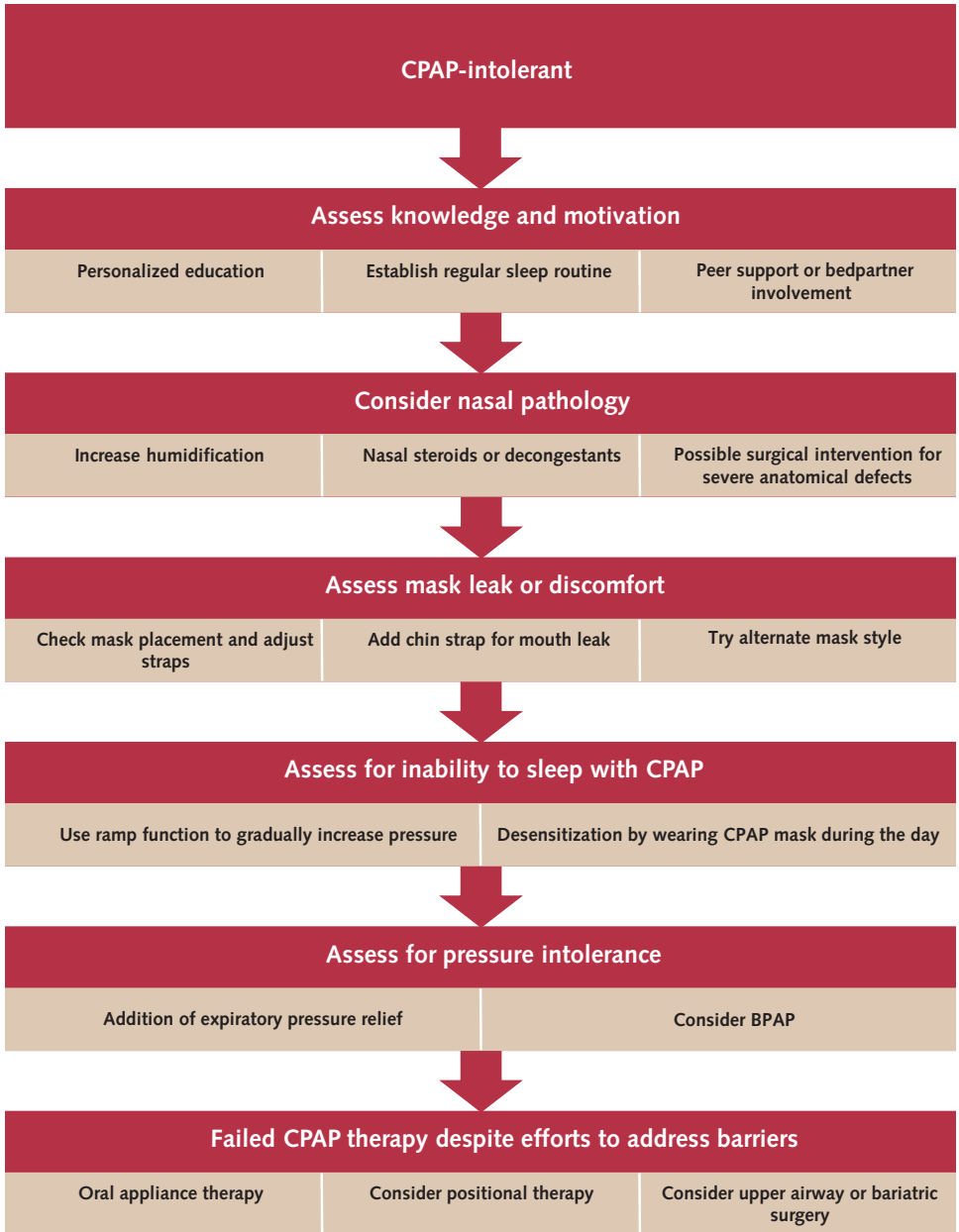
ACP℠
American College of Physicians
Leading Internal Medicine, Improving Lives

Medline Plus
www.nlm.nih.gov/medlineplus/ency/article/000811.htm

American Sleep Apnea Association
www.sleepapnea.org

Patient Information

Appendix Figure. **Strategies to maximize adherence to CPAP therapy.**



BPAP = bi-level positive airway pressure; CPAP = continuous positive airway pressure.