

Current reviews of allergy and clinical immunology

(Supported by a grant from GlaxoSmithKline, Research Triangle Park, NC)

Series editor: Harold S. Nelson, MD

Obstructive sleep apnea

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Obstructive sleep apnea is an increasingly well-recognized disease characterized by periodic collapse of the upper airway during sleep. This leads to either complete or partial obstruction of the airway, resulting in apneas, hypopneas, or both. This disorder causes daytime somnolence, neurocognitive defects, and depression. It affects almost every system in the body, resulting in an increased incidence of hypertension, cardiovascular disease, stroke, pulmonary hypertension, cardiac arrhythmias, and altered immune function. It also increases the risk of having an accident, presumably as a result of associated somnolence. The gold standard for the diagnosis of sleep apnea is an overnight polysomnogram. Split-night studies are becoming increasingly common and allow for quicker implementation of therapy at a reduced cost. Treatment options for sleep apnea include weight loss, positional therapy, oral devices, continuous positive airway pressure (CPAP), and upper airway surgery. CPAP is the most efficacious and widely used therapy. Its complications include nasal congestion or dryness, mask discomfort, and claustrophobia. Heated humidifiers, newer types of masks, and nasal steroids have improved tolerance of this therapy. Bilevel positive-pressure therapy can be considered for patients who find it difficult to exhale against the consistently increased pressure of CPAP. The disease requires aggressive treatment to improve quality of life and prevent its complications. (*J Allergy Clin Immunol* 2003;112:643-51.)

Key words: Obstructive sleep apnea, continuous positive airway pressure, hypertension, cardiovascular disease, rhinitis, immune function

Obstructive sleep apnea (OSA) is characterized by periodic complete or partial upper airway obstruction during sleep, causing intermittent cessations of breathing (apneas) or reductions in airflow (hypopneas) despite ongoing respiratory effort. This disorder has been described for decades, but its recognition has remained a problem. Prevalence studies of OSA with coexistent daytime somnolence (obstructive sleep apnea hypopnea syndrome) have reported an incidence of 2% in middle-aged

Abbreviations used

AHI: Apnea-hypopnea index
COPD: Chronic obstructive pulmonary disease
CPAP: Continuous positive airway pressure
OSA: Obstructive sleep apnea

women and 4% in middle-aged men.¹ The prevalence is higher (ie, 9% for women and 24% for men) if we use only an apnea-hypopnea index (AHI) of 5 or more as indicative of sleep-disordered breathing.¹ The prevalence in community-dwelling elderly (age ≥ 65 years) has been reported to be as high as 62%.² With the education of primary care physicians and heightened public awareness, the recognition of this disorder has substantially increased. In a national ambulatory care survey, Namen et al³ found a 12-fold increase in the annual diagnosis and reporting of OSA between 1990 and 1998.

The economic cost of untreated OSA is estimated to be in the range of billions of dollars.⁴ On average, patients with OSA use more health care resources before the diagnosis of OSA compared with matched control subjects without OSA.⁵ The long-term effects of OSA include hypertension,⁶⁻⁸ cardiovascular disease,^{9,10} stroke,^{11,12} and higher mortality.^{13,14} All of these issues make this disease an important societal concern.

DEFINITIONS

An apnea is defined in adults as cessation of airflow for 10 or more seconds. It usually indicates complete obstruction of the upper airway.¹⁵ A hypopnea is commonly defined as at least 30% reduction in airflow for 10 seconds associated with a 4% decrease in oxygen saturation.¹⁵ It connotes a transient reduction in inspiratory airflow caused by increased upper airway resistance. An apnea is considered obstructive if there is continued respiratory effort despite cessation of airflow. An apnea is considered to be of central origin if there is no concurrent respiratory effort.

OSA is defined by the presence of at least 5 obstructive apneas, hypopneas, or both per hour while the patient is sleeping.¹⁵ OSA is commonly divided into 3 levels of severity: mild (AHI = ≥ 5 but < 15 events per hour); moderate (AHI = 15-30 events per hour); and severe (AHI = > 30 events per hour).

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Received for publication August 14, 2003; accepted for publication August 19, 2003.

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0091-6749/2003 \$30.00 + 0

doi:10.1067/mai.2003.1813

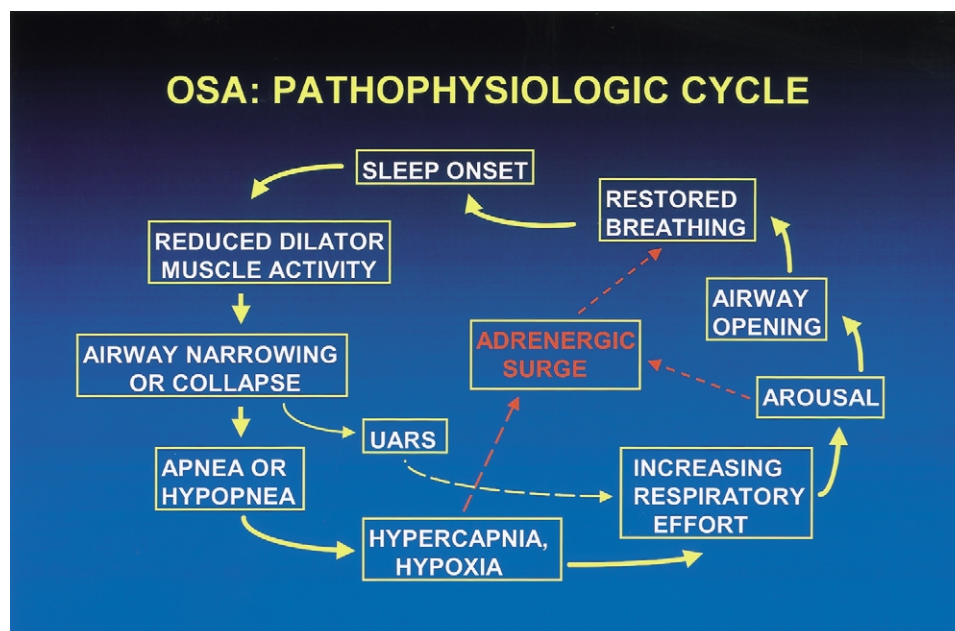


FIG 1. The recurrent pathophysiologic sequence typical of significant OSA. *UARS*, Upper airway resistance syndrome.

Recent clinical research has documented that significant clinical sequelae can occur with even mild OSA.

PATHOPHYSIOLOGY OF OSA

The site of upper airway obstruction typically lies in the pharynx, which is a surprisingly complex structure. The pharyngeal muscles assist in various functions, including swallowing, phonation, and maintenance of pharyngeal patency during inspiration. The function of each pharyngeal muscle varies depending on the contraction and position of other pharyngeal muscles. The pharyngeal luminal area during inspiration reflects a balance between collapsing intrapharyngeal negative suction pressure and dilating forces provided by the pharyngeal muscles.

The behavior of the relaxed pharynx is partly dependent on the transmural pressure. An increase in transmural pressure promotes pharyngeal patency, whereas a decrease in the pressure promotes pharyngeal collapse. The transmural pressure at which the pharyngeal area decreases to zero is called the closing pressure.¹⁶ The pharynx becomes increasingly susceptible to collapse as its luminal area decreases.¹⁷ Factors that tend to narrow the pharyngeal airway include surface adhesive forces, neck flexion, jaw opening,¹⁸ and gravity.¹⁹ Forces that tend to dilate the pharynx include tracheal tug caused by increased lung volume²⁰ and neck extension.¹⁸ Increased nasal resistance, the Bernoulli effect, and increased dynamic compliance of the pharynx are dynamic factors that can also promote pharyngeal narrowing.²¹

In healthy awake human subjects, pharyngeal patency is maintained by continual neuromuscular activation

of the pharyngeal muscles by the central nervous system. This activation is typically reduced during sleep, compromising the patency of the upper airway. The combination of reduced neural activation with anatomic abnormalities of the pharynx, such as excess posterior pharyngeal tissue, an enlarged tongue, or a low-hanging palate, sets the stage for obstructive hypopneas and apneas in patients with OSA. Pharyngeal muscle activation is also attenuated by alcohol, sleep deprivation, anesthesia, and sedative-hypnotics.²²⁻²⁵ Proprioceptive feedback from thoracic and upper airway receptors^{26,27} and chemical stimulation by hypercapnia and hypoxia²⁸ increase neural output to the pharyngeal muscles, resulting in increased activation. Sleep abolishes such proprioceptive feedback and suppresses chemical feedback.²⁹⁻³¹

Pharyngeal narrowing can be periodic or nonperiodic. Periodic obstruction produces apneas, hypopneas, or both interspersed with normal airflow. During nonperiodic obstruction, there is a sustained increase in airflow resistance with or without associated desaturation.¹⁵ This sustained increase in resistance can trigger sleep disruption and daytime somnolence, in which case it has been termed the upper airway resistance syndrome.³² Pharyngeal narrowing can occur at multiple sites in patients with OSA, including the velopharynx, oropharynx, and hypopharynx. The velopharynx is the primary site of occlusion in the majority of patients.^{16,33}

Total occlusion or critical narrowing of the upper airway either fully eliminates or substantially reduces ventilation. This in turn allows the development of hypercapnia and hypoxia. Respiratory effort progressively

TABLE I. Common symptoms of OSA

Common nocturnal symptoms
Snoring
Witnessed apneas
Nocturnal choking
Nocturnal snorting and gasping
Restlessness
Dyspnea
Diaphoresis
Nocturia
Dry mouth
Drooling
Gastroesophageal reflux
Common daytime symptoms
Daytime sleepiness and fatigue
Morning or nocturnal headaches
Impaired memory and concentration
Decreased dexterity
Personality changes
Irritability
Aggressiveness
Depression
Anxiety
Decreased libido
Impotence

increases in response to these chemostimuli and eventually triggers an arousal. This leads to a surge of pharyngeal dilator muscle activity and resolution of the upper airway obstruction. This process can repeat itself continuously during the night, allowing intermittent hypoxia and hypercarbia, fragmenting sleep, and triggering adrenergic surges with each cycle (Fig 1).

CLINICAL FEATURES OF OSA

The symptoms of OSA can be divided into daytime and nighttime symptoms (Table I). Daytime symptoms are usually the result of sleep disruption caused by apnea- or hypopnea-associated arousals and awakenings. Neck circumference is often increased in patients with OSA. This is believed to be indicative of central obesity, which is widely regarded as a significant risk factor for OSA. An increased body mass index can therefore also be associated with OSA. Other anatomic features that might be associated with the presence of OSA are shown in Table II. Upper airway anatomy should be assessed in the supine posture when possible because it is more representative of the anatomy of the sleeping patient. Predisposing factors, such as chronic sleep deprivation, sleeping in the supine position, and alcohol use, sedative use, or both, should be inquired about during patient evaluation. The presence of specific clinical features (Table III) can increase the probability of OSA but are usually not sufficient by themselves to establish the diagnosis of OSA. In a report by Hoffstein and Szali,³⁴ subjective impression on the basis of clinical features had a sensitivity of 60% and a specificity of 63%. The definitive diagnosis of OSA usually requires a sleep study.

TABLE II. Anatomic findings predictive of OSA

Obesity, large neck circumference, or both
Retrognathia
Dental overbite
Dental malocclusion
Dislocation of temporomandibular joint during mouth opening
Macroglossia
Edema, erythema, or both of the uvula
Elongated and low-hanging soft palate
Narrow mandible
Narrow maxilla
Narrow retroglossal area
Tonsillar hypertrophy
Adenoid hypertrophy
Nasal septal deviation
Nasal obstruction from any cause

TABLE III. Clinical predictors of OSA and its severity

Risk factors that enhance the probability of OSA
Obesity (BMI >30 kg/m ²)
Neck circumference >40 cm
Male sex
Positive family history of OSA
Race (African American, Pacific Islander, and Mexican American)
Down syndrome
Marfan disease
Pierre-Robin syndrome
Age >40 y
Postmenopausal status
Alcohol ingestion before bedtime
Respiratory allergies and nasal congestion
Underlying hypertension

BMI, Body mass index.

CLINICAL CONSEQUENCES OF OSA

Hypertension

Approximately 40% of patients with OSA have hypertension. Approximately 30% of middle-aged men with hypertension have OSA. Young et al³⁵ demonstrated a dose-response relationship between AHI and blood pressure, which was independent of confounding factors, such as age, sex, body mass index, and tobacco and alcohol use.³⁵ Three large clinical trials recently reported that the presence of OSA was associated with a 1.5- to 3-fold increase in the risk of hypertension development.³⁶⁻³⁸ The Wisconsin Sleep Cohort study also demonstrated a higher incidence of hypertension in patients with an AHI of 5 events per hour or more compared with that in healthy subjects and persons with only snoring.³⁹ In hypertensive patients the treatment of OSA often leads to a decrease in daytime systemic blood pressure.⁴⁰

Pulmonary disorders

Pulmonary hypertension. OSA can cause mild pulmonary hypertension, but it usually does not lead to clinical right-heart failure in the absence of daytime hypoxia or coexistent pulmonary disease.^{41,42} Weitzenblum et

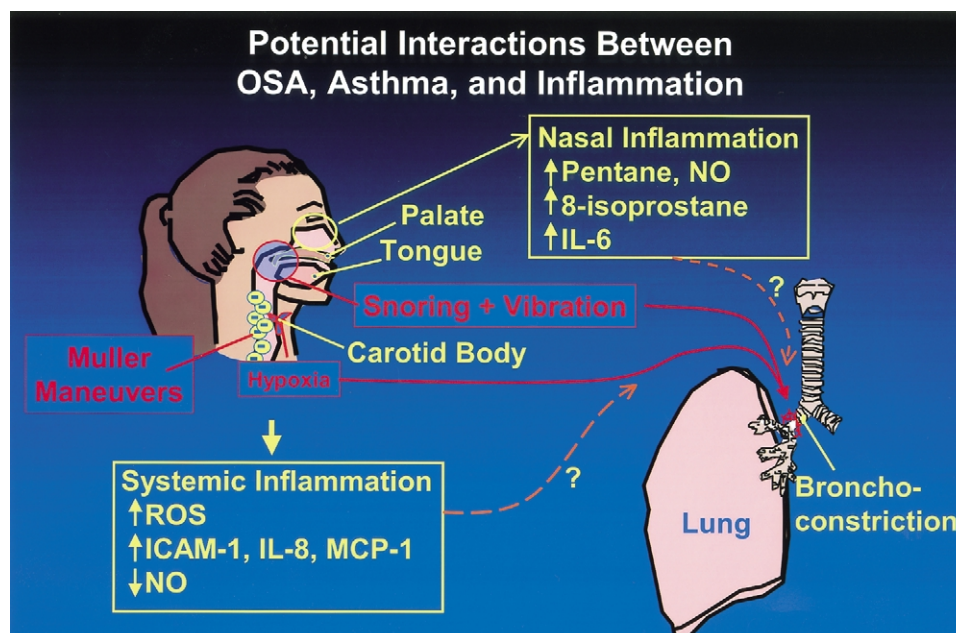


FIG 2. Potential interactions among OSA, nasal inflammation, systemic inflammation, and a possible link to asthma severity. *NO*, Nitric oxide; *ROS*, reactive oxygen species; *ICAM-1*, intercellular adhesion molecule 1; *MCP-1*, monocyte chemoattractant protein 1.

al⁴³ reported that about 15% of patients with OSA have resting increases of pulmonary artery pressure.

Asthma. Asthma often has a circadian pattern of severity, with exacerbations occurring more frequently in the early morning. Two studies have demonstrated increased asthma severity and frequent nocturnal exacerbations in asthmatic patients with concomitant snoring or OSA.^{44,45} These studies demonstrated improvement in asthma control and symptoms with effective treatment of OSA and snoring. The mechanism of this effect remains unknown, although it has been hypothesized to be neurogenically mediated from upper airway vibration, apnea-associated hypoxemia, or inspiratory efforts against an occluded pharynx (Muller Maneuver; Fig 2).

Chronic obstructive pulmonary disease. Chronic obstructive pulmonary disease (COPD) can also coexist with OSA. Approximately 10% of patients with OSA also have COPD.⁴⁶ However, the nocturnal oxygen desaturation commonly seen in COPD is more often caused by REM sleep-associated hypoventilation and less typically caused by apneas. Other causes of nocturnal desaturation can include sleep-associated ventilation-perfusion mismatch and decreased functional residual capacity. Hypercapnic patients with COPD have been observed to have a narrowed pharynx when awake, which could also predispose them to obstructive apneas during sleep. A recent Swedish study found that symptoms of lower respiratory tract disease and OSA coexist in a large number of patients with COPD.⁴⁷ Although Sanders et al⁴⁸ reported that there was no clear association between mild COPD and OSA, the degree of nocturnal desaturation was greater in patients who had a combination of the 2 diseases.

Cardiac disorders

Ischemic heart disease. Nocturnal hypoxemia and heightened adrenergic tone resulting from obstructive apneas-hypopneas might predispose to cardiac ischemia in patients with OSA and coronary artery disease. The risk of having a myocardial infarction is greatest at the oxygen saturation nadir, when the heart rate and blood pressure are increased. Reduced capacity for fibrinolysis has also been reported in patients with sleep apnea, possibly further increasing their risk for coronary occlusion. Population-based studies have demonstrated an increased risk of angina in adults with chronic snoring.⁴⁹ In a population of middle-aged adults referred to a sleep clinic for OSA evaluation, Peker et al⁵⁰ noted that 37% of patients with OSA initially reported cardiovascular disease, as opposed to only 7% in patients without OSA. Over 7 years of follow-up, new cardiovascular disease was identified in 57% of patients with inadequately treated OSA compared with only 7% of patients with adequately treated OSA. These findings strongly support a role of OSA in the development of coronary artery disease.

Arrhythmias. Patients with OSA are more prone to have cardiac arrhythmias. Hoffstein and Mateika⁵¹ detected arrhythmias in 58% of patients with OSA. Patients with arrhythmias usually had more severe OSA and nocturnal hypoxemia. Flemons et al⁵² reported that an AHI of at least 10 events per hour and oxygen desaturation to 60% or less are arrhythmogenic. The most common rhythm abnormalities seen with sleep apnea are bradycardia during the apneic phase, followed by tachycardia at the termination of the apnea. Other arrhythmias can include sinus arrest, second-degree heart block, atri-

al tachycardia, paroxysmal atrial fibrillation, atrial flutter, premature ventricular contractions, and nonsustained ventricular tachycardia.

Congestive heart failure. OSA elicits further increases in sympathetic activity in patients with congestive heart failure, possibly accelerating the cardiac disease process.⁵³ Continuous positive airway pressure (CPAP) treatment in patients with OSA and congestive heart failure increases baroreflex receptor sensitivity and resets the operating point of the baroreceptor to a lower blood pressure, thus improving neural control of heart rate and ultimately reducing heart rate.⁵⁴ CPAP treatment also resolves upper airway obstruction, allowing overnight reduction in mean intrathoracic pressure. By controlling OSA, CPAP also eliminates typical postapneic surges in blood pressure. These combined effects yield a reduction in cardiac afterload, which can ultimately improve cardiac function.

Cerebrovascular disease

Cerebral perfusion fluctuates during apneas, which might compromise blood flow to areas of the brain with already borderline or poor circulation. An odds ratio of 3.2 for the occurrence of stroke has been reported in patients with snoring, and an odds ratio of 8 for stroke has been reported in patients with witnessed apneas, sleepiness, and obesity with snoring.^{55,56} The incidence of OSA in patients who have had a previous stroke is also significantly increased.

Inflammatory changes

Evidence is also accumulating that OSA can be associated with upper airway inflammation. Olopade et al⁵⁷ measured exhaled pentane (an indicator of oxidative stress) and nitric oxide as indicators of inflammation in 20 patients with OSA and 8 healthy control subjects. They reported that exhaled nasal pentane and nitric oxide were increased after sleep only in patients with OSA. Carpagnano et al⁵⁸ more recently reported increased levels of both 8-isoprostane (another indicator of oxidative stress) and IL-6 in breath condensate from 18 patients with OSA.⁵⁸ These changes correlated with measured AHI. Such findings present convincing evidence of increased nasal–upper airway inflammation in patients with OSA.

Evidence is also accumulating that OSA is associated with changes in systemic indicators of inflammatory function. Schulz et al⁵⁹ reported, from a study 18 patients with OSA, that neutrophil superoxide generation was enhanced in comparison with that seen in control subjects, and this enhancement was immediately blunted by CPAP therapy. Dyugovskaya et al⁶⁰ demonstrated increased reactive oxygen species production from monocytes and granulocytes in 18 patients with OSA, an effect that was also blunted by subsequent CPAP therapy. These studies lend support to a role for cycling hypoxia-reoxygenation typical of OSA in promoting injury and inflammatory responses in patients with OSA. This concept is also supported by 2 separate reports that OSA is associated with reduced circulating nitric oxide and that

this reduction can be reversed by subsequent CPAP therapy.^{61,62} It has also been recently reported that OSA is associated with increased circulating levels of intercellular adhesion molecule 1, IL-8, and monocyte chemoattractant protein 1 and that these changes can again be reversed by effective CPAP therapy.⁶³ It is tempting to speculate that the apparently pro-inflammatory effect of OSA could account for the associations of this disorder with cardiovascular disease and asthma severity (Fig 2).

Neurocognitive function

Classic sleep apnea is well known to cause daytime somnolence, depression, and neurocognitive defects, presumably at least partly because of sleep deprivation. The Wisconsin Sleep Cohort study recently demonstrated reduced psychomotor efficiency in middle-aged adults with untreated sleep apnea. Kim et al⁶⁴ also reported that an AHI of 15 events per hour or greater impaired neurocognitive function in a manner similar to 5 years of additional age or equivalent to approximately 50% of the effect of administering a sedative-hypnotic.⁶⁴

Mortality and accidents

Increases in work-related and motor vehicle accidents are likely causes of increased mortality in patients with OSA. Masa et al⁶⁵ reported that habitually sleepy drivers as a group have a higher incidence of respiratory disturbance during sleep and significantly more automobile accidents if they have an AHI of greater than 15 events per hour. In a study conducted by Findley et al,⁶⁶ the accident rate of patients with OSA was 0.07 crashes per driver per year as opposed to 0.01 crashes per driver per year for all drivers in the state of Colorado. With the application of CPAP therapy, the accident rate decreased to zero in compliant patients with OSA.

DIAGNOSIS OF OSA

The gold standard for the diagnosis of OSA is the overnight polysomnogram. It includes electroencephalographic, electro-oculographic, electromyographic, oxygen saturation, oral and nasal airflow, respiratory effort, electrocardiographic, and leg movement recordings. The study is typically supervised by a technician and conducted in the sleep laboratory. A full-night polysomnogram usually provides an accurate picture of sleep characteristics and the severity of OSA.

Many sleep laboratories routinely conduct split-night studies, during which baseline sleep is assessed in the first portion of the study, followed by CPAP titration. Strollo et al⁶⁷ demonstrated comparable results between split-night polysomnograms and full-night diagnostic polysomnograms followed by full-night CPAP titration polysomnograms. Split-night polysomnograms typically allow for quicker diagnosis and treatment of OSA at reduced expense.

Home cardiorespiratory studies can also be used in the assessment of suspected OSA. These studies usually

monitor heart rate, respiratory effort, airflow, and oxygen saturation. Although useful for screening purposes, such tests usually do not provide information about sleep architecture and might be unable to distinguish sleep from wakefulness. The diagnosis of upper airway resistance syndrome and even mild sleep apnea might therefore be difficult. Overnight oximetry alone provides information about only nocturnal desaturation and is not a substitute for a polysomnogram.

TREATMENT OF OSA

Lifestyle modification

The approach to treating a patient with OSA is multifaceted. In addition to treating the site of obstruction itself, risk factors for OSA also should be modified. This includes encouraging patients to lose weight, avoid sleep deprivation, and refrain from tobacco, alcohol, and sedatives. The relationship between weight loss and apneas is apparently curvilinear. A critical amount of weight loss must often occur before a significant reduction in AHI is seen. Weight loss alone does not cure OSA in the majority of patients.

Positional therapy can be used in a selected group of patients or in combination with CPAP. This therapy usually consists of having patients sleep in the lateral position or with their heads propped up at 30° to 60°. Although positional therapy might not affect the length of apneas or apnea frequency during REM sleep, it typically reduces overall apnea frequency by stabilizing the pharynx. Patients more likely to respond to positional therapy include those with milder OSA and mild or no obesity. In addition, responsiveness to positional changes should be documented during a formal polysomnogram.

Electrical stimulation of the upper airway, nasal dilators, and drugs such as protriptyline and mirtazapine have been evaluated for the treatment of OSA, but all have typically been unsuccessful in adequately reducing apneas. Such therapies are therefore not currently recommended for widespread use.

CPAP therapy

Nasal CPAP therapy for OSA was first reported in 1981.⁶⁸ Since then, it has become the most effective and widely used treatment for OSA. It treats apneas-hypopneas by providing air under positive pressure through a nasal or facial mask, thus creating a pneumatic splint in the pharynx, which prevents collapse of the pharyngeal airway.

In one early study covert compliance monitoring revealed acceptable compliance rates to be as low as 46%.⁶⁹ Compliance rates of 65% to 90% have been reported in more recent literature. Compliance is likely higher if a patient has significant daytime somnolence before initiation of therapy and perceives subsequent improvement with CPAP therapy. Aggressive patient education and support from health care providers also improve compliance with treatment. Pepin et al⁷⁰ recently reported a compliance rate of 80% in response to augmented support for CPAP-treated patients with OSA.

Jenkinson et al⁷¹ substantiated the therapeutic efficacy of CPAP in a controlled parallel trial, in which therapeutic CPAP was compared with "sham CPAP." Patients with OSA undergoing therapeutic CPAP demonstrated clear-cut reductions in Epworth sleepiness scores and improvement in maintenance of wakefulness test results and energy-vitality scores on the SF-36.

Side effects of CPAP. Common side effects of CPAP include rhinorrhea, nasal congestion and dryness, mask discomfort, conjunctivitis from air leak, skin abrasions, claustrophobia, irritation from device noise, difficulty exhaling, aerophagy, chest discomfort, and bed-partner intolerance.

Nasal congestion and mask intolerance are the most common complaints that reduce compliance with CPAP. Many different kinds of mask interfaces are available. They range from full-face masks for preferential mouth breathers to nasal prongs, which insert into the nostrils and reduce claustrophobia. Gel and air cushion interfaces on many nasal masks prevent abrasions and skin breakdown.

Nasal symptoms reported by Pepin et al⁷² in association with CPAP therapy included nasal congestion (25%), rhinorrhea-sneezing (35%), and nasal dryness (65%). Similar symptoms were reported by Brander et al⁷³ and Hoffstein and Szali.³⁴ The addition of heated humidification to CPAP and the administration of nasal steroids are commonly used to combat these symptoms. Rakotonanahary et al⁷⁴ demonstrated that the addition of humidification to CPAP increased compliance and device use. Of 82 CPAP-treated patients enrolled in this study, 46 (56%) subsequently had nasal symptoms. A passive (cool) humidifier was added to their CPAP, despite which 23 patients had persistent nasal symptoms and subsequently required heated humidification. With heated humidification, CPAP use increased from 3.5 hours to 5.38 hours daily. Only one patient discontinued CPAP after addition of heated humidification.

Nasal steroids are also widely used to reduce nasal congestion. Craig et al⁷⁵ demonstrated that in patients with perennial allergic rhinitis but no diagnosed OSA, nasal steroids improved both nasal congestion and subjective sleep quality. In children with OSA, Brouillette et al⁷⁶ reported a decrease in baseline AHI from 10.7 to 5.8 events per hour after the initiation of nasal steroids. Desaturation events were also reduced.⁷⁶ Given previous observations by McNicholas et al⁷⁷ that snoring patients with seasonal rhinitis are more prone to have OSA during the pollen season, it appears that nasal steroid therapy might be helpful for some patients with OSA.

Finally, patients who cannot tolerate CPAP because of discomfort from exhaling against high pressure can be treated with the addition of a pressure "ramp" or the use of bilevel positive-pressure therapy. Autotitrating CPAP was also introduced in the last decade with the premise that by continuously adjusting pressure to meet the patient's variable needs, the overall mean airway pressure will be reduced. This would hopefully allow better tolerance and therefore better compliance. However, additional research is necessary to demonstrate cost benefits and better outcomes from this approach before its use can be widely recommended.

Oral devices

Most available oral devices are approved for snoring only. Only 14 devices have received clearance from the US Food and Drug Administration for treatment of OSA. Of these, only 7 are adjustable, allowing titration of the mandibular position, and only 3 have undergone controlled or randomized clinical trials. These devices work on the principle of mechanically displacing the tongue or the jaw and tongue forward to increase space in the posterior pharyngeal area. These devices should not be used for therapy of central sleep apnea or in patients with temporomandibular joint disease or problematic nasal obstruction. Patients need to have sufficiently healthy teeth to anchor the device. Such devices are usually not recommended in patients with severe OSA or with significant nocturnal oxygen desaturation. Two recent trials have shown that even in patients with mild-to-moderate OSA, CPAP therapy produced better results, with greater improvement in AHI and sleepiness than observed with oral devices.^{78,79}

Surgical treatment

Surgical approaches are likely more effective for patients with OSA with discrete craniofacial abnormalities than for simply obese patients. A wide variety of procedures are available, many of which are directed at the site of obstruction (Table IV). Surgery can be carried out in at least 2 separate phases. Phase I surgery can include combinations of all the listed procedures, with the exception of maxillomandibular advancement. The reported short-term response rate to these procedures in combination is approximately 60%. If the patient does not respond, Phase II surgery can be considered, which consists of maxillomandibular advancement. These surgeries are best conducted in tertiary centers by experienced surgeons. In selected patients the success rate from combined Phase I and II surgery can be as high as 90%. The success rate is likely lower if the patient is obese with severe OSA and oxygen desaturation to less than 70%.⁸⁰ Complications can include bleeding, infections, upper airway obstruction caused by surgical edema, hematomas, and facial anesthesia after maxillomandibular advancement. Patients should be closely monitored postoperatively in an intensive care unit setting by medical staff experienced in managing such cases.

If all treatment modalities fail, a tracheostomy can be considered as a final therapeutic option, especially in morbidly obese patients with severe OSA, significant oxygen desaturation, and/or associated cardiac disease.

SUMMARY

OSA is a disease manifested by intermittent closure or narrowing of the upper airway during sleep, which leads to apneas-hypopneas, arousals, and oxygen desaturations. It is being recognized with increasing frequency as physicians and patients become more aware of this disorder. OSA places a significant burden on society

TABLE IV. Surgical approaches to OSA

Phase I	Phase II
Nasal septoplasty	Maxillomandibular advancement
Turbinate reduction	
Tonsillectomy and adenoid resection	
Laser-assisted uvulopalatoplasty	
Uvulopalatopharyngoplasty	
Mandibular osteotomy with genioglossus advancement	
Hyoid myotomy-suspension	

because of its associated long-term morbidity and mortality. It adversely affects almost every system of the body. It is best diagnosed with an overnight polysomnogram. The most efficacious and widely used treatment for this disease is CPAP. CPAP therapy improves daytime function and reverses many adverse consequences of OSA. Other modalities, including surgery and oral devices, are best used in patients with mild-to-moderate disease who cannot use CPAP.

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