

Obstructive Sleep Apnea

a report by

Nirav P Patel, MD^{1,2} and Richard J Schwab, MD^{1,3}

1. Pulmonary, Allergy, and Critical Care Division; 2. Center for Sleep and Respiratory Neurobiology; 3. Sleep Center, University of Pennsylvania

Obstructive sleep apnea (OSA) is a highly prevalent disease that represents the most frequent form of sleep-disordered breathing (SDB) encountered, and is associated with serious medical, public health, and economic consequences. The first definitive description of OSA was in 1966 by Gastaut,¹ although descriptions of SDB date back to the 19th century.²

OSA is characterized by repetitive episodes of partial or complete obstruction of the upper airway during sleep, disrupting airflow into the respiratory tract in spite of continued thoracic efforts to breathe. The disorder is categorized in two ways:

- hypopnea—manifests as transient decrements causing a reduction in airflow by 25–50%, and is associated with a decrease in oxygen saturation by 3–4% and/or an arousal or fragmentation of sleep; or
- apnea: a cessation of airflow for at least 10 seconds.

Occlusion of the airway institutes several immediate physiological abnormalities: large swings in intra-thoracic pressures associated with

continued thoracic effort during airway obstruction compromise left ventricular filling; intermittent hypoxia results in elevations in levels of reactive oxygen species, oxidative stress, and an inflammatory state; and stimulation of the sympathetic nervous system in response to apnea, hypoxia, and arousal results in increased systemic blood pressure and heart rate.²

Definitions

The criteria most commonly employed to assess the frequency of obstructive events is the apnea–hypopnea index (AHI), which is the number of apneas and hypopneas per hour of sleep that can be noted during a sleep study. An AHI >5 suggests the presence of OSA. In the presence of concomitant symptoms such as excessive daytime somnolence, unrefreshing sleep, snoring, or nocturnal choking, the diagnosis of OSA is established. Detailed diagnostic criteria have been published by the American Academy of Sleep Medicine (AASM).³

Epidemiology

There is considerable variation in the population estimates of OSA due to population heterogeneity, differences in methodology in measuring sleep, and variation in thresholds that differentiate abnormal from normal subjects. Estimates of the prevalence of OSA (defined as AHI >5 events/hour) using laboratory polysomnography range between 9 and 28%.^{4–7} The prevalence of OSA with daytime symptoms is in the region of 5%.⁴ Several risk factors for OSA have been reported, of which obesity is the most convincing (see *Table 1*).

Pathophysiology

Sleep apnea results from narrowing of the upper airway. Factors affecting upper airway patency include those related to upper airway anatomy (craniofacial/soft tissue structures) and neuromuscular tone during wakefulness and sleep.⁸ The upper airway is subject to a balance/imbalance of collapsing/patency-promoting factors. As a result of the absence of a robust support framework of cartilaginous rings, the upper airway is at risk for collapse due to extra-luminal tissue pressure exerted by surrounding structures, and negative intra-luminal pressures associated with inspiratory breaths. In opposition to these collapsing forces are the actions of the pharyngeal dilator muscles, which collectively promote patency when stimulated by neural input in response to negative luminal pressure, respiration, and arousal.⁸

In patients with sleep apnea, the most frequent sites of collapse are the retropalatal and retroglottal regions of the pharynx.⁹ Imaging techniques have demonstrated that OSA subjects have an excess of upper airway soft



Nirav P Patel, MD, is a Post-doctoral Fellow at the Center for Sleep and Respiratory Neurobiology, University of Pennsylvania. Having completed fellowships in pulmonary and critical care medicine, he is also a clinical sleep medicine fellow and a masters student in the Center for Public Health Initiative at the University of Pennsylvania. He moved to Philadelphia in 2001 to begin internal medicine training at the Albert Einstein Medical Center, then moved to Penn in 2004 to begin his specialty fellowships. Dr Patel obtained his medical training in the UK at Guy's and St Thomas' Medical and Dental Schools, London.



Richard J Schwab, MD, is an Associate Professor of Medicine in the Divisions of Sleep Medicine and Pulmonary, Allergy, and Critical Care at the University of Pennsylvania. He is Co-Director of the Sleep Center at the University of Pennsylvania. He also has a secondary appointment in the Department of Radiology. Dr Schwab has emerged as a pre-eminent investigator in the field of upper airway imaging and sleep apnea. He has developed and utilized sophisticated magnetic resonance imaging (MRI) and volumetric analysis paradigms to study the mechanisms leading to sleep apnea. His research has resulted in seminal observations about the genetics, pathogenesis, and treatment of obstructive sleep apnea. He has shown the importance of the lateral pharyngeal walls in the pathogenesis of sleep apnea and that the volume of the upper airway soft tissue structures are larger in patients with sleep-disordered breathing than normal controls. Dr Schwab completed his pulmonary/critical care and sleep fellowship at the University of Pennsylvania in 1991, received his medical degree from the University of Pennsylvania School of Medicine, and received his undergraduate degree from Haverford College.

E: rschwab@mail.med.upenn.edu

Table 1: Risk Factors for Obstructive Sleep Apnea

Risk Factor	Comment
Obesity	Graded increase in prevalence of OSA with increasing BMI. ⁴¹
Male gender	Two- to three-fold increased risk for OSA in men related to anatomical and sex hormone differences. ⁴²
Age	Population studies indicate higher prevalence of OSA in older subjects (aged 60 years and above) compared with middle-aged groups. ⁴²
Craniofacial abnormalities	Imaging techniques have described the craniofacial features of OSA patients. These include a reduction in the length of the mandible, an inferiorly positioned hyoid bone, a retroposition of the maxilla, retrognathia, nasal septal deviation, and narrowing of the hard palate. ¹⁰
Upper airway soft tissue abnormalities	Macroglossia, lateral peritonsillar narrowing, soft palate enlargement, and tonsillar hypertrophy alter the configuration of the luminal airway, increasing the risk of obstructive apnea. ^{10,41}
Genetics	Mendelian and familial inheritance patterns of upper airway/craniofacial structures predisposing to OSA have been reported. ^{2,43}
Endocrine	OSA is more common in hypothyroid (especially myxedema) and acromegaly patients. ⁴⁴
Alcohol	Acute consumption may induce apneic events in normal subjects and worsen the severity of OSA in established patients. ⁴²

OSA = obstructive sleep apnea.

Table 2: Clinical Features of Obstructive Sleep Apnea

Excessive daytime somnolence
Snoring
Witnessed apnea
Gasping/choking events during sleep
Unrefreshing sleep
Morning headache
Nocturia
Mood swings
Difficulty concentrating
Automobile or work-related incidents
Diminished libido
Impotence

Table 3: Types of Polysomnography Studies Available

Type of Polysomnography	Monitoring	Information Derived
Level I Attended in-lab PSG	EEG, EOG, chin EMG Airflow, respiratory effort, oximetry, ECG Anterior tibialis EMG Sleep position	Sleep architecture and stage-related events, such as arousal, hypoxia Obstructive/central apnea Arrhythmia Leg movements
Level II Complete unattended home PSG	Measures the same parameters as a level I study at home	Same as level I study
Level III Partial home PSG	No EEG, EOG, chin EMG, ECG anterior tibialis EMG	Obstructive/central apnea Oxygen desaturation Tachycardia/bradycardia
Level IV Limited home PSG	Oximetry	Oxygen desaturation as a screen for OSA

PSG = polysomnography; EEG = electroencephalogram; OSA = obstructive sleep apnea; EOG = electro-oculogram; EMG = electromyogram; ECG = electrocardiogram.

tissue relative to the craniofacial structures, and that upper airway caliber is reduced compared with controls during wakefulness and sleep.¹⁰ Anatomical findings that have been shown to increase the risk for OSA include increased airway length and increased volumes of the lateral pharyngeal walls, parapharyngeal fat pads, tongue, and soft palate.² These anatomical factors do not cause upper airway collapse during wakefulness since they are opposed by neural input to pharyngeal dilator muscles. During sleep, neural input is attenuated; hence, in anatomically predisposed patients, significant increments in airflow resistance ensue.¹¹ During rapid eye movement (REM) sleep, decrements in airway dilator activity can be further depressed, explaining the common finding of REM-related sleep apnea.

Clinical Manifestation

Excessive daytime somnolence (EDS) reported in various settings is a cardinal symptom of sleep apnea. The severity of sleepiness can be assessed using the Epworth Sleepiness Scale, in which scores range from 0 to 24. A score above 10 is deemed abnormal and indicates a propensity to fall asleep. Patient perception of the degree of sleepiness is subjective and may be clouded by adaptive behavior. Bed partners are important sources for uncovering relevant nocturnal events such as snoring, witnessed apnea, choking, gasping, and arousals. Although EDS is very common in sleep apnea, some patients may present without EDS; the long-term outcome for such patients is not known. Furthermore, EDS may be present in the absence of OSA secondary to sleep deprivation, depression, insomnia, poor sleep hygiene, or another sleep disorder (narcolepsy, periodic limb movements). *Table 2* summarizes the symptoms/complaints associated with OSA. It is important to note that snoring in the absence of apnea is very common. Snoring in epidemiological studies is associated with hypertension, cardiovascular disease, myocardial infarction, stroke, and increased mortality. It is believed that snoring is an antecedent condition to the development of OSA.¹²

The above symptoms/complaints constitute the bulk of the presenting features of OSA patients. However, OSA is associated with many other medical conditions, such as cardiovascular disease, hypertension, stroke, diabetes, metabolic syndrome, pulmonary hypertension, chronic obstructive pulmonary disease, and depression. Therefore, vigilance regarding these conditions and their link with OSA is important for diagnostic and treatment reasons. Detecting and treating unidentified OSA may improve the established medical condition.

Diagnosis

The current gold standard for diagnosing OSA is polysomnography (PSG), which involves an overnight sleep in the laboratory and monitoring of multiple physiological variables. A sleep technician is available throughout the study. There are four types of PSG, dependent on attendance by a technician and equipment utilized (see *Table 3*).

If a patient exhibits apneas/hypopneas during the study that meet criteria set forth by the AASM, continuous positive airway pressure (CPAP) therapy is applied.¹³ The remainder of the night involves optimizing both the CPAP setting to abolish events and the CPAP–patient interface. This split-night paradigm (three hours of a baseline study followed by CPAP treatment) has been viewed as more cost-effective and efficient than the traditional two-night model of a diagnostic PSG followed by second night CPAP titration.¹⁴ The advent of portable monitoring systems (levels II–III) to improve diagnosis procedures and treatment of OSA has posed many questions on a scientific

and public health level. Drawing conclusions about the accuracy of level II–III monitors in detecting OSA from available data is difficult due to variations in definition of respiratory events, parameters measured, and thresholds adopted to diagnose sleep apnea.¹⁴ Portable studies may prove to be more cost-effective and end-user-friendly, but currently Medicare and Medicaid Services do not recognize them as legitimate means of testing.¹⁵ Furthermore, a comprehensive review conducted by the AASM and American College of Chest Physicians (ACCP) concluded that there was insufficient evidence to support the use of portable devices in an unattended setting at the present time.¹⁶

Consequences of Sleep Apnea

There are numerous studies that strongly support the need to treat OSA. Broadly, the consequences of sleep apnea may be categorized into cardiovascular and neurocognitive effects. OSA induces sleep fragmentation and EDS that impair cognitive function, alertness, and quality of life.¹⁷ These secondary effects of OSA explain the increased risk for motor vehicle accidents: a recent meta-analysis reported an odds ratio (OR) of 2.5 for OSA subjects to have a crash.¹⁸

Cardiovascular Effects and/or Associations of Sleep Apnea

Sleep apnea and its association with cardiovascular disease has been an area of considerable research. The main areas of interest have been on elucidating SDB's link with hypertension, coronary artery disease (CAD), congestive heart failure (CHF), arrhythmia, cerebrovascular accident (CVA), pulmonary hypertension, metabolic syndrome, and diabetes, as well as potential mechanisms of association such as endothelial dysfunction, inflammation, and hypercoagulability (see *Table 4*). To this end, a multitude of experimental, cross-sectional, case-controlled, randomized, and longitudinal studies have been undertaken to examine and explain these biological associations. There is compelling evidence of causality between the presence of sleep apnea and the development of hypertension due to chronically elevated sympathetic nervous system activity.^{19,20} The Wisconsin Sleep Cohort Study, a longitudinal investigation, showed that sleep apnea is associated with increased risk of incident hypertension (OR 2.89; 95% confidence interval (CI) 1.46–5.64 for AHI >15 versus AHI = 0).²⁰

Randomized trials employing CPAP and sham CPAP (a placebo version of CPAP) demonstrate that several weeks to months of treatment effect a clinically significant reduction in daytime blood pressure of between 1.3 and 5.3mmHg.^{21,22} OSA is also associated with CHF, CVA, CAD, and arrhythmia. The Sleep Heart Health Study, a cross-sectional evaluation of approximately 6,000 subjects, showed that the presence of OSA (AHI >11) was associated with a 2.30 OR for CHF ($p=0.002$), 1.27 for CAD ($p=0.004$), 1.58 for CVA ($p=0.03$),²³ 4.02 for atrial fibrillation (OR 4.02; 95% CI 1.03–15.74), 3.4 for non-sustained ventricular tachycardia, and 1.74 for complex ventricular ectopy.²⁴ OSA has been associated with both systolic and diastolic heart failure. The prevalence of OSA among CHF patients with diastolic dysfunction and preserved systolic function was found to be 36%.²⁵ Case-control studies matched for body mass index (BMI) indicate that OSA is a risk factor for myocardial infarction/unstable angina.²⁶ Furthermore, a prospective study reported an increased risk of cardiovascular events over a 10-year period in untreated sleep apnea patients.²⁷

Sleep apnea is also a plausible risk factor for atherogenesis. Mechanisms implicated include oxidative stress,²⁸ elevated levels of mediators of inflammation such as C-reactive protein (CRP) and interleukin (IL)-6,²⁹

Table 4: Cardiovascular Disease Associated with Obstructive Sleep Apnea

Hypertension

Coronary Artery Disease

- Acute coronary syndrome
- Angina
- Atherogenesis

Myocardial Dysfunction

- Left ventricular systolic and diastolic dysfunction
- Left ventricular hypertrophy
- Congestive heart failure
- Right ventricular dysfunction

Cardiac Arrhythmia

- Atrial fibrillation
- Sinus bradycardia
- Atrioventricular heart block
- Supraventricular and ventricular tachycardia

Cerebrovascular Accident

Pulmonary Hypertension

Metabolic Syndrome

endothelial cell injury,³⁰ and elevated sympathetic drive.²⁰ Prospective data, spanning 7–10 years, comparing sleep apnea patients with matched controls report decreased cardiovascular events and mortality in CPAP-treated patients.^{27,31} While the exact relationship between SDB and CAD remains unknown, the available evidence suggests that modification of SDB in patients at risk for CAD may be beneficial.

OSA and the metabolic syndrome share common pathophysiology. OSA is associated with all of the components of the metabolic syndrome: central obesity, insulin resistance, and dyslipidemia. A nine-fold increase in prevalence of the metabolic syndrome has been reported in OSA subjects compared with controls.³²

Economics of Sleep Apnea

Sleep apnea is an important public health and economic burden. Despite increasing vigilance among the public and health practitioners, a large proportion of subjects remains undetected.³³ The group of subjects identified as 'at-risk' for sleep apnea are subject to the 'bottle-neck' in ability to diagnose and treat in that the demand for sleep medicine services currently exceeds capacity, despite large growth in infrastructure nationally and internationally. Why is this important? Based on the evidence discussed above, it is clear that on an individual level there are detrimental effects of untreated sleep apnea. Patients have been shown to utilize healthcare resources at approximately twice the rate of controls as far back as 10 years prior to diagnosis.³⁴ Recent economic analysis estimates the burden of sleep disorders in the US to be \$109 billion.³⁵ Reports of cost-effectiveness show that diagnosis and treatment of sleep apnea is economically attractive.³⁶

Treatment

Treatment for most patients is medical, and the cornerstone is CPAP (see *Table 5*). General measures recommended in concert with CPAP can include weight loss (a 1% change in weight is associated with a 3% change in AHI³⁷), sleep hygiene (maintaining regular sleep–wake cycles and avoiding alcohol, sedatives, and hypnotics), and positional therapy. The latter is prescribed if the patient has position-dependent sleep apnea; therefore, the sleep position should be strategically altered to minimize apnea. In general,

Table 5: Treatment of Obstructive Sleep Apnea

General measures	Weight loss Avoid alcohol, sedatives, and hypnotics
Positive airway pressure	Position therapy Continuous positive airway pressure (CPAP) Auto-titrating CPAP
	Bi-level systems
	Intra-oral devices
Intra-oral devices	Tongue-retaining device Palatal-lifting device Mandibular advancing device
	Surgery
	Nasal surgery Tonsillectomy Uvulopalatopharyngoplasty (UPPP) Lingualplasty Genioglossus and hyoid advancement (GAHM) Sliding genioplasty Maxillo-mandibular advancement osteotomy (MMA) Laser-assisted uvulopalatoplasty (LAUP) Radiofrequency volumetric tissue reduction Tracheostomy Bariatric surgery

pharmacological measures have not been efficacious, although modafinil 200–400mg per day may be beneficial for patients with persistent sleepiness in spite of optimal CPAP therapy.

CPAP is a non-invasive therapy that is applied through a nasal mask, nasal inserts, or a full-face-mask. CPAP has been shown to improve apneas, hypoxia, sleepiness, quality of life, driving performance, hypertension, and other cardiovascular outcomes.¹⁷ Bi-level systems and auto-titrating CPAP are alternatives to traditional CPAP and can be used in a range of clinical contexts. Presently, neither bi-level systems nor auto-CPAP have demonstrated superiority in outcomes over traditional CPAP.

Intra-oral devices have evolved tremendously over the past 20–30 years. Their objective is to alter the airway structures so that the caliber of the

upper airway is increased. A number of devices are available, but only a few are US Food and Drug Administration (FDA)-approved.³⁸ Improvement in snoring and sleep apnea has been reported to varying extents. The success is closely related to the titration and adjusting of the device once it is fitted. At present, oral devices are indicated for snoring and mild to moderate OSA where weight loss and CPAP have not been feasible options.³⁹

Surgical intervention is considered when medical therapy has not been successful, or when subjects have anatomical defects amenable to surgical correction. As indicated in *Table 5*, there are a variety of options. Selection of a suitable surgical candidate is crucial. The process involves careful examination of the airway lumen and surrounding tissues and structures. The former is achieved by clinical examination, nasopharyngoscopy, and imaging techniques, such as magnetic resonance imaging (MRI), computed tomography (CT), or cephalography. The surrounding tissues and airway lumen can be assessed during wakefulness and sleep to identify the primary site of obstruction. Uvulopalatopharyngoplasty (UPPP), the most common procedure, involves the removal of excess mucosa and tissue from the palate and palatopharyngeal arch with the aim of widening the oropharyngeal aperture. Success rates range from 40 to 50%.⁴⁰ Detailed descriptions of other surgical procedures can be viewed elsewhere.⁴⁰ Tracheostomy is an extremely successful procedure that is reserved for refractory sleep apnea patients due to its detrimental effect on patient quality of life.

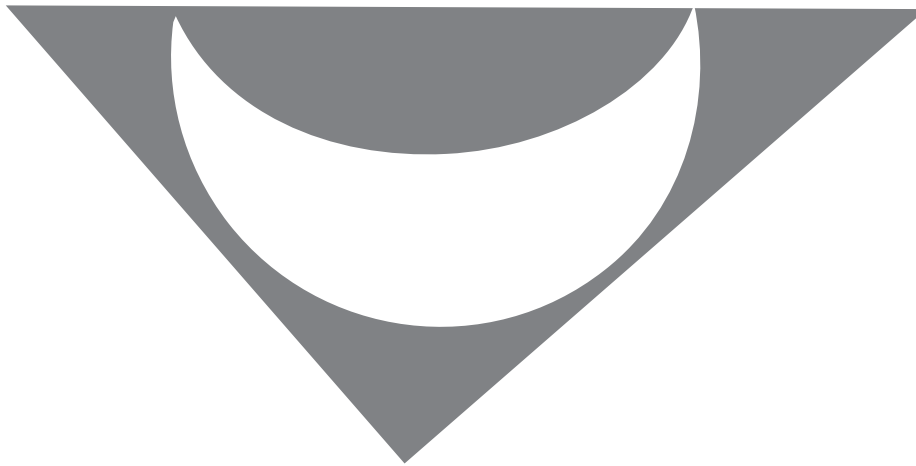
Conclusion

Sleep apnea is a burdensome public health concern. Recent studies have helped to understand the pathogenesis and consequences of OSA. The associations between sleep apnea and important diseases continue to unfold. Cardiovascular risk and mortality in sleep apnea patients are elevated based on current research. There is great disparity between the actual population prevalence and the rate of clinical recognition. Much of this can be explained by lack of awareness and the current infrastructural demand for sleep services exceeding capacity. Nonetheless, sleep apnea is an easy diagnosis to make and CPAP represents a highly efficacious treatment. More work is required to implement strategies to lessen the overall burden of such a treatable disease. ■

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Education, Support and Advocacy



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