Obstructive Sleep Apnea (OSA) — Symptoms and Treatment

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Obstructive sleep apnea (OSA) is defined as episodic apnea, or cessation of breathing, during sleep, in which the period of apnea should last for more than 10 seconds. It is usually due to partial or complete collapse of the upper airway and is associated with snoring, restlessness, daytime headache and somnolence. The diagnosis relies on history and requires polysomnography for confirmation. Several medical as well as surgical treatment modalities are currently recommended and essential to prevent complications.

Definition and Categories of Sleep Apnea

OSA is defined as repeated cessation of respiration for more than ten seconds during sleep with sleep interruption and daytime somnolence.

Categories

- **Obstructive sleep apnea** is caused by the collapse of the oropharyngeal airway in obese adults/children with hypertrophied adenotonsillar tissue.
- **Central sleep apnea** is due to lack of respiratory effort following central nervous system injury, strokes, congestive heart failure and opioid
intoxication.

- **Mixed sleep apnea** is a combination of obstructive and central sleep apnea.
- **Obesity hypoventilation syndrome**: morbid obesity with BMI > 30 kg/m² leads to hypoventilation, retention of carbon dioxide during the day and during sleep with hypoxemia.

**Epidemiology and Etiology of Obstructive Sleep Apnea**

It is estimated that in the United States, **2-6% of the women and 4-13% of the men**, especially in middle age, have sleep disordered breathing with excessive daytime somnolence. Approximately 50% of the men in a Swiss community sample were reported to have OSA.

This increasing incidence may be partly due to the obesity pandemic, modern lifestyle, improvements in diagnostic methods, awareness about the importance of the disorder and several other factors.

**Etiology**

The cause of OSA can be multifactorial, ranging from **anatomic neuromuscular factors** to associated **genetic predisposition**. Congenital or acquired craniofacial abnormalities (e.g., micrognathia, retrognathia, large tongue or tonsils, neck circumference > 17 inches), aging, alcohol, use of sedatives, postmenopausal status, acromegaly and hypothyroidism are some of the risk factors associated with OSA. A common risk factor for OSA is **obesity**, and it often co-exists with **hypoventilation syndrome**.

During sleep, the individual is recumbent, and muscle activity is diminished, leading to the **collapse of the upper airway**. Repeated episodes of this lead to cessation and diminished airflow. The apneic and hypopneic episodes stimulate respiratory efforts against the narrowed upper airway until the individual is awakened.

Studies have reported EMG patterns in OSA patients suggestive of **genioglossus neuromuscular dysfunction** similar to denervation-renervation. This may be related to systemic inflammation, intermittent hypoxia or vibrational trauma during snoring.

**Presentation of Obstructive Sleep Apnea**

The majority of patients with OSA are unaware of their symptoms. Often it is their sleep partners and spouses who alert them about their disruptive sleep and apnea. Symptoms may include:
Diagnosis of Obstructive Sleep Apnea

The diagnosis of OSA is based on a thorough history from the individual as well as their sleep partners, spouses or roommates. The history should include age and weight of the patient, other medical co-morbidities, daytime sleepiness and fatigue.

Physical examination should rule out nasal obstruction, tonsillar enlargement, craniofacial anomalies and features of acromegaly/hypothyroidism.

Routine laboratory tests like complete blood count, blood sugar levels and glycosylated hemoglobin levels as well as thyroid stimulating hormone levels are recommended based on clinical findings.

The gold standard for diagnosis of OSA is the polysomnography. It assesses the respiratory, cardiac and sleep parameters of the individual during sleep, either at home or in a hospital.

During sleep, the patient is monitored and ECG-recorded to determine the number of apneic episodes and arrhythmias, if any, during the apneic episodes. Other variables such as body position (supine position may predispose to apnea) and limb muscle activity to look for restless leg syndrome and periodic lung movement disorder can also be evaluated.

The severity of OSA can be determined from the Apnea/Hypopnea index (AHI), which
is calculated as the **number of apneic episodes per hour of sleep**. The AHI values can be calculated for the different sleep stages.

### Degree of OSA

- \( \text{AHI} > 5 = \text{OSA} \)
- \( \text{AHI} > 15 = \text{moderate OSA} \)
- \( \text{AHI} > 30 = \text{severe OSA} \)

If EEG monitoring is used, then the **arousal index** (AI) can be calculated as the number of arousals per hour of sleep. Although AI can be correlated with AHI, approximately 20% of apneas are not associated with arousals.

**Drug-induced sleep endoscopy (DISE):** This procedure is useful to evaluate the dynamic upper airway in a sleeping patient with OSA. It is especially helpful prior to surgical intervention to detect the level of obstruction and tailor the surgery for the individual patient.

**Radiological investigations** like lateral radiographic cephalometry, computed tomography (CT) and magnetic resonance imaging (MRI) being static tests are **not of much value** in diagnosing the level of obstruction in patients with OSA.

### Prognosis

Undiagnosed and untreated OSA has been linked to high incidence of reduced workplace productivity, motor vehicle accidents, cognitive impairment, **hypertension**, fatal and non-fatal cardiovascular events, **ischemic stroke**, sexual dysfunction and cancer mortality.

The nocturnal hypoxia can be severe enough to cause pulmonary or systemic hypertension, secondary polycythemia, **arrhythmias** and sudden death. The all-cause mortality of OSA is significantly higher in untreated patients with moderate to severe OSA.

### Differential Diagnosis

- **Insomnia**: nocturnal inability to sleep, especially in depression or restless leg syndrome.
- **Hypersomnia**: daytime sleepiness, e.g., narcolepsy.
- **Paroxysmal nocturnal dyspnea**: dyspnea on lying in the supine position, especially at night when it presents in congestive cardiac failure and **asthma**.

### Treatment of Obstructive Sleep Apnea

The aim of treatment is to improve the quality of life and sleep by reducing the apneic episodes during sleep. OSA is considered to be cured when the AHI decreases to below threshold values, i.e., <10/hour of sleep.

The principles of managing OSA include:

**Manage the risk factors**

Initially, the treatment is directed towards improving the modifiable risk factors:
treatment of hypothyroidism/acromegaly/diabetes; obesity management and eliminating sedative/alcohol use. It is often difficult for patients to lose weight, and the morbidly obese may require bariatric surgery. However, bariatric surgery or weight loss alone is not a cure for OSA.

CPAP or other devices

![Continuous positive airway pressure machine](https://example.com)

**Continuous positive airway pressure** (CPAP) is the most effective method for managing OSA. It has been documented to improve sleep symptoms, snoring, cognition, daytime somnolence and quality of life. Unfortunately, most patients find its usage cumbersome, and compliance is often inadequate.

Other devices like the **mandibular advancement device**, or others which help to pull the tongue forwards, are also available and may help to alleviate symptoms in mild to moderate OSA.

**Surgery**

The role of surgery in the treatment of OSA has always been **controversial**. Surgery aims to detect sites of redundant upper airway tissue and then surgically remove it to widen the upper airway. Common sites of airway obstruction are the nose (nasal polyps, deviated nasal septum), macroglossia, low hanging uvula and enlarged tonsils. Depending upon the level of obstruction, diverse surgeries can be recommended.

**Nasal septal deviation correction, reduction of hypertrophied inferior turbinate/s and polypectomy** are recommended in OSA patients with nasal obstruction. In children with OSA, **tonsil-adenoid removal** is useful to alleviate the symptoms.

**Uvulopalatopharyngoplasty** (UPPP), either conventional or laser assisted (LAPP), involves removal of the uvula, part of the soft palate and excess oropharyngeal tissue and is combined with tonsillectomy. Although post-procedure there is improvement in snoring and AHI, the efficacy of the procedure decreases over time.

**Maxillomandibular advancement** is a surgical procedure recommended for OSA patients with retrognathia or micrognathia. Maxillary and mandibular osteotomies with their advancement help to displace the soft palate and tongue anteriorly, with the resultant widening of the oropharyngeal airway. This is a very effective treatment
modality in selected patients.

**Tracheotomy** is the most effective surgical treatment for OSA but is only recommended for patients with severe refractory life-threatening OSA.

**Hypoglossal nerve stimulation** is a rescue treatment modality for patients with moderate to severe OSA. An implanted device stimulates the hypoglossal nerve.

**Bariatric surgery** is recommended for OSA patients with morbid obesity.

## Prevention of Obstructive Sleep Apnea

**Patient education**, highlighting the **risk factors** and **intensive lifestyle changes** are important in the management of OSA. Patients should be advised to maintain optimal weight, avoid alcohol, sedatives (especially in the evening) and smoking. It is also important to inform patients about the adverse effects of untreated OSA and improve their compliance by educating them about the nocturnal use of CPAP.

## References

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