Obstructive Sleep Apnea

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Introduction

• Sleep apnea is the intermittent cessation of airflow at the nose and mouth during sleep.
• Apneas of at least 10 s duration are important but in most cases the apneas last 20-30 s and can last as long as 2-3 min.
• Sleep apnea is a leading cause of daytime sleepiness and contributes to CVS disorders.
• Prevalence: 2% in middle-aged women and 4% in middle-aged men
Definition

Sleep apneas are divided into:

- Central sleep apnea: neural drive to all respiratory muscles is abolished
- Obstructive sleep apnea: airflow ceases despite continuing respiratory drive because of occlusion of the oropharyngeal airway.
Electroencephalographic tracings recorded from a normal young adult demonstrating the four stages of NREM sleep. In the stage 2 recording, the arrow points to a characteristic K complex and the underlining to sleep spindles. (From Carskadon and Dement, with permission.)
Pathogenesis

• Occlusion of the oropharyngeal airway results in progressive asphyxia until there is a brief arousal from sleep, whereupon airway patency is restored and airflow resumes.

• The patient then returns to sleep and the process is repeated, up to 300-400 x per night – sleep becomes fragmented.
• The immediate factor leading to collapse of the upper airway is generation of subatmospheric pressure during inspiration and which exceeds ability of airway dilator and abductor muscles to maintain airway stability.

• During wakefulness upper airway muscle activity is greater than normal to compensate for airway narrowing and high airway resistance.

• Alcohol is a co-factor – has a depressant effect on airway muscles and the arousal response that terminates each apnea.
Structural abnormalities:

- Oropharyngeal airway may predispose to closure (short neck)
- Structural compromise may be due to anatomic disturbances such as tonsillar hypertrophy, retrognathia and macroglossia.
- In the majority there is only subtle reduction in airway size which can be described as “crowding”
- Obesity may contribute to reduction in upper airway size by ↑ fat deposition in the soft tissues of the pharynx or by compressing the pharynx by superficial fat masses in the neck.
- The airway may also have high compliance – “floppy” and be prone to collapse.
Clinical Features:

• Snoring: may antedate OSA by several years
  Snoring in the absence of other symptoms, does not warrant investigation for OSA but counseling re alcohol and weight gain is required.

• Recurrent bouts of asphyxiation and arousal lead to clinical complications:
  1. Cognitive and behavioural disturbances, excessive daytime sleepiness, intellectual impairment, memory loss and personality changes.
  2. Cardiorespiratory: ↑LV afterload, cyclical bradycardia (30-50/min) during apnea and tachycardia (90-120/min) during ventilation. Sudden death during sleep may occur.
Systemic BP fails to decrease during sleep – BP increases at termination of each apnea due to sympathetic nerve activation and reflex vasoconstriction – 50% of OSA pts have systemic HT and OSA is an independent risk factor for developing Systemic HT.

OSA may precipitate myocardial ischaemia in patients with coronary artery disease.

In patients with congestive heart failure, OSA may acutely or chronically depress LV function
Epworth Sleepiness Scale

In contrast to just feeling tired, how likely are you to doze off or fall asleep in the following situations? (This refers to your usual life in recent times. Even if you have not done some of these things recently, try to work out how they would have affected you.) Use the following scale to choose the most appropriate number for each situation:

0 = Would never doze
1 = Slight chance of dozing
2 = Moderate chance of dozing
3 = High chance of dozing

<table>
<thead>
<tr>
<th>Situation</th>
<th>Chance of Dozing</th>
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<tbody>
<tr>
<td>Sitting and reading</td>
<td></td>
</tr>
<tr>
<td>Watching TV</td>
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<tr>
<td>Sitting inactive a public place</td>
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<tr>
<td>(e.g., in a theater or at a meeting)</td>
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<tr>
<td>As a passenger in a car</td>
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<tr>
<td>for an hour without a break</td>
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<tr>
<td>Lying down to rest in the afternoon</td>
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<tr>
<td>when circumstances permit</td>
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</tr>
<tr>
<td>Sitting and talking to someone</td>
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<tr>
<td>Sitting quietly after lunch without alcohol</td>
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<tr>
<td>In a car, while stopping for a few minutes</td>
<td></td>
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<tr>
<td>in traffic</td>
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</table>
Silhouettes of four obese men. The three on the left had severe chronic alveolar hypoventilation. The most obese (right) weighed 750 lb and had normal blood gases and no evidence of obstructive sleep apnea.
Diagnosis

- Clinical: typical patient is male, 30-60y, snores, has daytime sleepiness, nocturnal choking or gasping, witnessed apneas during sleep, moderate obesity and large neck circumference and mild to moderate hypertension.

- Women with OSA are postmenopausal, snoring is less frequent and daytime fatigue is more common than outright sleepiness.
Diagnostic tests

• Polysomnography = a detailed overnight sleep study with recordings of:
  ECG (arrythmias), EEG (brain waves – level of sleep ), EOG (eye movements – REM sleep) and submental EMG (muscle twitches - REM sleep) to evaluate sleep
  Ventilatory variables: movement of chest wall and airflow at the mouth and nose
  Arterial O₂ saturation (finger/ear oximetry)
  Heart rate
FIGURE 101-8  Sinus arrest in a normal person during REM sleep. Profound changes in the cardiovascular system can occur in association with the phasic events of REM sleep. Although this example is not particularly common, it does show how intense the “normal” cardiovascular changes can be in REM sleep. (From Guilleminault et al,9 with permission.)
Interpretation

- Episodes of airflow cessation or reduction at the nose and mouth despite continuing respiratory effort (chest wall movement) are diagnostic of OSA.
- Test is expensive and time consuming – at home overnight monitoring of arterial $O_2$ saturation can confirm OSA if episodes of desaturation (10-15/hour) are found – can ↓ need for PSG by 1/3.
- When negative, polysomnography is indicated
- ↑ upper airway resistance during sleep may be associated with arousals in the absence of apneas or hypopneas and can still result in clinically important sleep-related syndromes.
FIGURE 102-9  Example of an obstructive apneic episode in a patient with sleep apnea syndrome. The polysomnography traces from the top down are as follows: three EEG channels (C3-A2, C4-A2, OZ-A2); two EOG channels (R and L); submental EMG (EMG); right and left anterior tibialis EMG (RAT, LAT); electrocardiogram (ECG); nasal and oral airflow; chest and abdominal motion (CHEST and ABD). During the apneic episodes, there is abnormal airflow (both oral and nasal), with paradoxical motion of the rib cage and abdomen. At the end of the apneic episode, there is a burst of EMG activity at the arousal. Following arousal, respiration resumes with synchronous movements of the rib cage and abdomen.
FIGURE 101-8  Sinus arrest in a normal person during REM sleep. Profound changes in the cardiovascular system can occur in association with the phasic events of REM sleep. Although this example is not particularly common, it does show how intense the “normal” cardiovascular changes can be in REM sleep. (From Guilleminault et al,9 with permission.)
Severe OSA

- Significant daytime sleepiness
- >30 obstructive events and arousals per hour of sleep
Management

• ↑upper airway muscle tone: mild OSA – avoid alcohol and sedatives

• ↓upper airway lumen size: 1. Mild to moderate OSA – weight reduction, avoid supine position and use oral prosthesis to keep airway patent
  2. Severe OSA: Uvulopalatopharyngoplasty

• upper airway subathmospheric pressure: 1. mild to moderate OSA – improve nasal patency;
  2. severe OSA – nasal CPAP

• Bypass occlusion: severe OSA - tracheotomy
Nasal CPAP in treating obstructive sleep apnea.  

A. Schematic representation of the system used for CPAP: an air compressor, a tight-fitting nose mask, and a ball value resistor to adjust pressure. (From Sanders, 1984.)  

B. The operation of the CPAP. By creating a positive pressure in the oropharynx and nasopharynx, the negative pressure developed in the hypopharynx during inspiration is overcome. (From Sullivan, Issa, Berthon-Jones, Eves, 1981.)
Example of a subject connected to a CPAP machine. The top panel shows the patient wearing a CPAP mask that covers the nose. The bottom panel demonstrates the patient using nasal pillows that insert directly into the nostrils. With both types of device, it is essential to ensure a good seal with no leaks.

Example of a mandibular repositioning device. This device fits on the upper and lower teeth; it is worn during sleep and results in anterior motion of the mandible, with consequent enlargement of the airway.
The primary sequence of events, physiologic responses, and clinical features of obstructive sleep