Obstructive sleep apnoea

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Abstract

Obstructive sleep apnoea occurs during sleep when there is repeated pharyngeal collapse that obstructs the airway and causes repeated awakenings from sleep. Sufferers complain of unrefreshing sleep and daytime somnolence, and bed partners usually report snoring with frequent apnoeic attacks. Problems with tasks requiring concentration, such as driving, are common, and in severe sleep apnoea the rate of road traffic accidents secondary to sleepiness is known to be high. Investigation with a sleep study is required to confirm the diagnosis and severity before the decision is made to initiate life-long treatment with nasal continuous positive airway pressure treatment. Milder cases may respond to a jaw advancement device and weight loss.

Keywords Continuous positive airway pressure; daytime somnolence; jaw advancement device; obstructive sleep apnoea; snoring

Key points

- Obstructive sleep apnoea (OSA) is common, and its prevalence will increase as obesity becomes more common
- OSA should be suspected in all sleepy snorers
- Investigation should include an assessment of somnolence using the Epworth Sleepiness Scale and an overnight sleep study, recording markers of sleep disruption (e.g. pulse rate rise, movement), snoring and oxygen saturation
- Abnormalities of thyroid function and tonsillar enlargement should be excluded
- Treatment with continuous positive airway pressure is effective in improving somnolence and quality of life, and reducing the risk of road traffic accidents to baseline
- Jaw advancement devices can treat mild forms of OSA and snoring, particularly if retrognathia is present

Introduction

Obstructive sleep apnoea (OSA) is caused by recurrent obstruction of the upper airway during sleep, preventing airflow into the lungs and resulting in repeated falls in oxygen saturation. OSA is one end of a spectrum that includes snoring, snoring-induced arousals and upper airways resistance syndrome.

Definition

Apnoeas have traditionally been arbitrarily defined as a cessation of airflow for 10 seconds (briefer interruptions are known as hypopnoeas), and their severity is calculated according to the number of events per hour. For example, severe OSA can be defined as having an apnoea–hypopnoea index (AHI) of more than 35/hour throughout the night, and normal sleep as having an AHI of less than 5/hour. However, these thresholds are now regarded as arbitrary, and most sleep physicians favour an approach that takes symptoms into account. Thus, significant OSA can be defined as sleep-induced upper airways obstruction leading to symptomatic sleep disturbance.

Epidemiology

In various population studies, the prevalence of OSA among middle-aged men is 1–4%, and in middle-aged women it is 1.2–2.5%.[1] The reason for the difference is not fully known, but different patterns of fat deposition and responsiveness to inspiratory loading are thought to contribute. In adults, the prevalence of snoring and OSA increases with age, reaching a peak at 40–60 years.

Pathogenesis

The airway is usually kept patent by a balance between the opening force of dilator muscle activity and initial size of the airway, and the collapsing force of negative intraluminal pressure and the external mass around the neck. Thus, apnoeas are more likely to occur when obesity, retrognathia or a supine posture is present. The site at which the obstruction occurs varies: usually, the posterior aspect of the tongue comes to rest on the posterior pharyngeal wall. In patients with OSA, the pharyngeal airway is usually small and, on average, more compliant, than in healthy individuals. Obstruction can occasionally be laryngeal, when the noise is usually stridor rather than snoring, and associated with neurological or physical laryngeal problems.

OSA is usually more severe in rapid eye movement (REM) sleep than in non-REM sleep because the loss of muscle tone in REM sleep affects the upper airway muscles and the arousal threshold is higher. Sleeping in a supine position can worsen airway narrowing by exerting gravitational effects on the tongue base and soft palate.

Repeated arousals, in order to resume breathing, cause sleep disruption and are a major determinant of daytime sleepiness, but other factors are also known to contribute. Obesity is a leading cause of OSA so there is a close association between OSA, obesity and increased cardiovascular risk as well as metabolic associations (insulin resistance, dyslipidaemias).

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Cardiovascular risk, oxidative stress and obstructive sleep apnoea

There is accumulating evidence from human studies that untreated OSA causes oxidative stress and that effective continuous positive airway pressure (CPAP) therapy can reverse these abnormalities. The first studies demonstrating the presence of oxidative stress in humans with OSA observed that circulating neutrophils and monocytes from OSA patients exhibited markedly enhanced *in vitro* release of superoxide radical anions. Patients with OSA can exhibit endothelial dysfunction, that is, a reduction of endothelial-dependent vasorelaxation. This has been shown by various techniques investigating vasoreactivity, for example venous occlusion plethysmography and measurements of flow-mediated vasodilatation of the brachial artery. Endothelial dysfunction is a precursor of both atherosclerosis and arterial hypertension and can already be detected in OSA patients who do not have clinically overt cardiovascular disease.

Causes of obstructive sleep apnoea

The most common factors leading to OSA are obesity and a small or posteriorly positioned lower jaw (retrognathia). In obesity, deposition of fatty tissue around the airway causes compression of the airway during sleep when muscle tone is reduced. There are many other rarer causes that should be borne in mind when examining a patient (Table 1). All are worsened by alcohol consumption and smoking.

Symptoms of OSA

Sleepiness

The principal symptom of OSA is excessive daytime sleepiness (EDS). The degree of sleepiness varies significantly between individuals for a given severity of OSA (and indeed some individuals do not manifest sleepiness). It is vital to establish how severe the EDS is and how it is affecting quality of life. EDS can lead to feelings of fatigue, poor memory and concentration, loss of mental flexibility, mood swings and irritability. Secondary depression can occur. Poor performance at work can lead to lack of promotion, loss of employment or work-related accidents. Effects on social and family life can be significant, with sufferers often sleeping in the evening before going up to bed, and having to sleep apart from their partner because of the snoring. There can be significant concerns about safety when driving or operating machinery. If possible, a history should also be obtained from a bed partner with regard to the presence of apnoeas at night (often unknown to the sufferer) and to the level of symptoms during the day (often downplayed by the sufferer).

Causes of upper airway narrowing and OSA

Anatomical malformations
- Retrognathia
- Congenital malformations

Structural pharyngeal lesions
- Enlarged tonsils and adenoids
- Webbed pharynx
- Pharyngeal tumours
- Laryngeal webs
- Cricoarytenoid arthritis

Hormonal causes
- Hypothyroidism
- Menopause
- Acromegaly

Neuromuscular weakness
- Arnold–Chiari malformation
- Stroke
- Muscular dystrophies
- Myasthenia gravis
- Shy–Drager syndrome

Infiltration of pharyngeal tissue
- Obesity
- Mucopolysaccharidoses

Table 1

Other symptoms

Common symptoms occurring in more than 60% of sufferers are:
- loud snoring
- EDS
- choking or shortness of breath sensations during sleep
- restless sleep
- unrefreshing sleep
- changes in personality
- nocturia.

Less common symptoms, occurring in 10–60% of sufferers, are:
- morning headaches
- enuresis
- reduced libido
- nocturnal sweating.

Rare symptoms reported by less than 10% of sufferers are:
- symptomatic oesophageal reflux
- recurrent arousals or insomnia
- nocturnal cough.

Symptoms can differ in children, in whom OSA can cause hyperactivity, poor concentration, difficulty learning and poor academic performance.

Differential diagnosis

Other disorders also lead to daytime somnolence and, in the presence of snoring, can lead to referral for investigation of possible OSA. These include:
- poor sleep hygiene (inadequate length of sleep, irregular bedtimes, shift work, excessive caffeine ingestion, excessive bedtime stimuli such as a TV or computer)
- depression (early morning wakenings)
- periodic leg movements during sleep, which is part of restless legs syndrome and causes recurrent leg and arm movements and arousals from sleep
- less commonly, narcolepsy, caused by damage of the hypocretin/orexin pathways in the brain, probably from a viral infection in genetically susceptible individuals (HLA type DQB1*0602).

Clinical examination

Clinical examination in a patient with suspected OSA should include: