Understanding obstructive sleep apnoea

DEFINING sleep apnoea can be confusing because it is described in research literature under many guises. This single condition has at least seven different names:

- Obstructive sleep apnoea (OSA);
- Sleep apnoea (SA);
- Obstructive sleep apnoea syndrome (OSAS);
- Sleep apnoea/hypopnoea syndrome (SAHS);
- Obstructive sleep apnoea/hypopnoea syndrome (OSA/H);
- Upper airway resistance syndrome (UARS);
- Obstructive sleep apnoea is specifically characterised by the occurrence of repetitive episodes of partial or complete collapse of the upper airway, which prevents breathing. This is known as apnoea. Episodes of apnoea are usually accompanied by: loud snoring, excessive daytime sleepiness, and a reduction of blood oxygen saturation accompanying apnoea. Physiologically these episodes are generally associated with mini-awakenings, sleep fragmentation, intermittent hypoxaemia, hypercapnia and nocturnal hypertension.

There is also the condition known as central sleep apnoea (CSA), which can be a sign of a disorder in the breathing centres in the brain. The brain ‘forgets’ to initiate breathing during sleep, CSA is a very rare condition and cannot easily be recognised by signs and symptoms that present with OSA. Some patients experience a combination of both OSA and CSA.

Development of OSA

OSA does not develop spontaneously; rather it can best be described as a continuum of snoring. It is estimated to affect around four per cent of men and two per cent of women. The lack of awareness among the general population and physicians means that an estimated 80 to 90 per cent of people with OSA have not received a clinical diagnosis.

Mechanisms of OSA

OSA occurs because of a partial or complete obstruction somewhere in the upper airway, which prevents air from entering the lungs. This obstruction, which can occur anywhere in the region of the nose, throat or mouth, causes a narrowing of the airway and an increase in pharyngeal resistance with the generation of excessive negative pressure during inspiration. This narrowing requires an increase in pharyngeal dilator muscle activity to maintain airway patency and there is evidence that patients with OSA lack this muscle tone (McNicholas, 2002).

Physical properties that may contribute to the narrowing of the airway include:

- Neck obesity (an anatomically short, fat neck);
- Fat deposition in the oropharynx (because of increased body weight/obesity);
- Mandibular/pharyngeal abnormalities (incorrect jaw shape);
- Loss of muscle tone in the pharyngeal dilator muscles during sleep (due to vibratory trauma from long-term snoring, excessive alcohol intake or sedatives);
- Increased size of surrounding soft tissue structures (tongue, soft palate, lateral pharyngeal walls, tonsillar hypertrophy).

Assessment of OSA

There are various terms used to describe the events that occur during sleep and it is generally agreed that a definitive diagnosis of OSA can only be elicited by polysomnography performed in a specialised sleep laboratory (Neven et al, 1998). However, due to the large numbers of patients presenting for assessment it is not possible to perform full polysomnography on every patient and so home-based sleep studies have become more frequent.

Information needed for clinical diagnosis

- The apnoea/hypopnoea index (AHI) – the number of respiratory disturbances per hour of sleep. Hypopnoeas are usually considered as a reduction in airflow, or respiratory movement of 50 per cent with desaturation;
- An AHI of at least five, each lasting for more than 10s;
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A fall in oxygen saturation levels (SaO₂) of more than four per cent.

Degree of excessive daytime sleepiness (EDS) usually defined using the Epworth Sleepiness Scale (ESS) questionnaire. EDS is the most important symptom of OSA and a score of more than 10 would be highly significant.

The severity of OSA is defined by the AHI ranging from mild (<5) to severe (>20).

When a patient presents for assessment there are several signs and symptoms that are often immediately recognisable. Some of these symptoms are better described by the patient’s bed partner than the patient:

- Obesity (a collar size >16inches predisposes to OSA);
- Excessive alcohol intake;
- Heavy smoking;
- Extremely loud, heavy snoring often interrupted by pauses and gasps;
- Excessive daytime sleepiness. For example, falling asleep at work, while driving, during a conversation or when watching TV (this should not be confused with excessive tiredness, which most people experience from time to time);
- Morning headaches;
- Irritability and short temper;
- Forgetfulness;
- Changes in mood or behaviour;
- Anxiety or depression;
- Decreased interest in sex.

The fact that we all experience one or all of these symptoms at some point demonstrates that we need to look carefully at the severity of an individual patient’s symptoms rather than make a diagnosis on whether his or her symptoms are within the clinical definition.

Prevalence of OSA

OSA is estimated to affect around four per cent of men and two per cent of women (Kryger, 2002; Levy et al, 2002; Schwab, 1999). However, Sjostrom et al (2002) reckon that 24 per cent of men and 9 per cent of women in the middle-aged population have OSA, while Neven et al (1998) estimate that at least 45 per cent of men aged 35 or above have clinically significant OSA.

The wide range of these estimates is probably due to differences in: definitions, study design and investigations performed; and in the age, sex and other characteristics of the populations surveyed (Neven et al, 1998).

An estimated 80-90 per cent of people with OSA have not received a clinical diagnosis because of a lack of awareness among the general population and physicians (Hossain and Shapiro, 2002).

The recommended treatment for OSA is continuous positive airway pressure (CPAP) (Kryger, 2002; Levy et al, 2002; McNicholas, 2002). But how clinicians make the decision about whether a patient should receive this treatment raises some dilemma. Should patients be prescribed the treatment regardless of the severity of their symptoms? If their symptoms fall within the clinical criteria, should all patients be prescribed CPAP? Conversely, if a patient’s symptoms fall short of clinically significant SA, but he or she clearly has other symptoms such as EDS, why shouldn’t CPAP treatment be given to alleviate these symptoms? Such dilemmas often prompt patients to contact the office of British Snoring and Sleep Apnoea Association (www.britishsnoring.com).

Treatment of OSA

Since Sullivan et al (1984) introduced CPAP more than 20 years ago it has remained a well-proven treatment in reversing the symptoms of OSA. CPAP therapy acts as a ‘pneumatic splint’ by blowing room air into the airway, normally via a nasal mask, at a positive pressure to keep the airway open and thus preventing collapse. However, according to Malhotra and White (2002), CPAP adherence is difficult, with the best compliance found in patients with severe OSA and substantial sleepiness.

Patients discontinue using CPAP for several reasons. Physical problems include: mask leak, dry throat/mouth, pressure sores and eye infections. However, according to Engleman and Wild (2003): ‘Biomedical investigations of determinants of CPAP adherence have shown largely weak relationships between symptomatic/polysomnographic disease severity and subsequent CPAP use. They believe that ‘cognitively derived perceptions of health status, health beliefs and attitudes may underlie CPAP adherence behaviour.’ Based on this evidence it would be advantageous for providers to offer a long-term support service to increase compliance. Engleman and Wild’s paper (2003) offers a series of practical solutions to ensure greater compliance for their patients.

Alternative treatments

In mild OSA there are alternative treatments. Oral appliances such mandibular advancement splints (MAS) have become popular, not least because they are far less obstructive than CPAP. However, individuals with clearly reversible causes should first be advised to make lifestyle changes to alleviate their symptoms. Malhotra and White (2002) suggest ‘conservative measures should be emphasised, including maintenance of nasal patency, avoidance of depressants including alcohol, and the goal of seven to eight hours’ sleep per night. In addition, individuals with documented positional apnea should be encouraged not to sleep on their backs.’

CPAP does not always resolve EDS and additional treatments may be of benefit for regular users of CPAP therapy. In a recent study the use of modafinil significantly reduced symptoms of EDS and is now recognised as a useful adjunct treatment for the management of residual daytime sleepiness in patients with OSA/hypopnoea syndrome who are regular users of nasal continuous positive airway pressure therapy.

Kingshott et al (2001) point out that this is not a first-line therapy in SA as it does not treat the underlying upper airway collapse or the resulting mini awakenings and blood pressure changes. From their study the benefits and limitations should be carefully considered before administration.

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REFERENCES


