Management of Obstructive Sleep Apnoea Syndrome



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Obstructive sleep apnoea syndrome affects about 4% of men and 2% of women, most of whom are currently undiagnosed and untreated. The general practitioner plays an important role in assessing the need for specialist investigation in a sleep centre.

Obstructive sleep apnoea (OSA) involves repeated episodes of reversible obstruction to the pharyngeal airway during sleep. This occurs when the pharyngeal and lingual dilator muscles of the airway are so relaxed during sleep that they are unable to withstand the negative air pressure on inspiration, and the airway collapses inwards (Fig. 1). If the consequent airway obstruction is complete and lasts for more than 10 seconds it is called an obstructive apnoea; with partial obstruction it is called a hypopnoea.

OSA is not the same as the obstructive sleep apnoea syndrome (OSAS). This syndrome has three hallmarks:

- Being heard to stop breathing or make choking noises and loud gasps when asleep (apnoeas/hypopnoeas)
- · Persistent snoring
- Excessive daytime sleepiness (EDS).

OSAS is present in about 4% of men and 2% of women. There is substantial but not yet conclusive evidence linking OSAS with hypertension, myocardial infarction, stroke and increased mortality. [11] The syndrome is certainly associated with significant morbidity. The social and domestic upheaval that persistent snoring can cause should not be underestimated. The importance of EDS in the community is only now being realised, particularly as a cause of motor vehicle and industrial accidents, even as a major contributing factor in such disasters as Chernobyl and the Exxon Valdez oil spill. [2]

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The first and perhaps the most important step in the management of OSAS is for the general practitioner to suspect it and to ask about the three hallmark symptoms routinely. Each of these symptoms, considered separately, is surprisingly common in the community. They have seldom been adequately

SALIENT POINTS

- Enquiries about snoring, observed apnoeas and daytime sleepiness should form part of any thorough medical history-taking
- There is substantial but not yet conclusive evidence linking obstructive sleep apnoea syndrome with hypertension, myocardial infarction, stroke and increased mortality
- It is possible to kill yourself and others and to create havoc on a massive scale simply by dozing off at an inappropriate time
- The social and domestic upheaval that persistent snoring can cause should not be underestimated
- Snoring and OSA are made worse by obesity, nasal obstruction due to rhinitis, a deviated septum or nasal polyps, a relatively small oropharynx with a large tongue, a recessive mandible, a long soft palate or enlarged tonsils
- Nasal continuous positive airway pressure (CPAP) is the mainstay of treatment for obstructive sleep apnoea syndrome

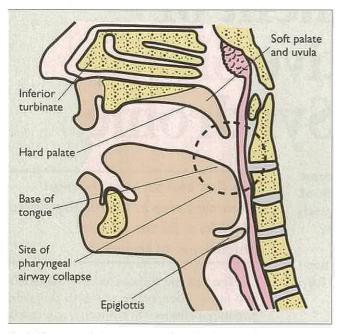


Fig. 1. Pharyngeal obstruction causing obstructive sleep apnoea.

assessed in the past. The focus of the discussion that follows will be on their clinical assessment rather than on technicalities of the treatment of OSAS which, of necessity, is carried out by specialists in sleep disorders centres.

Snoring

Snoring is the noise generated by vibration of the relaxed soft palate, uvula and faucial pillars on inspiration during sleep. About 75% of all men and 40% of women are reported by their spouse/partner to snore sometimes. For occasional snorers this may only be when lying on their back or after drinking alcohol in the evening, both of which increase snoring. Persistent snorers snore for at least part of every night or most nights, lying on their sides as well as their back, with and without alcohol. About 20% of all men and 10% of women are persistent snorers. They usually disrupt the sleep of their partners who often sleep in a separate room for at least part of the night. This room is another bedroom for the fortunate ones, but it may be a couch or a mattress on the floor of the living room for others.

One or both parents of most persistent snorers have also been troublesome snorers

Some persistent snorers who do not have clinically significant OSA nevertheless have EDS, presumably because of he increased work of breathing when snoring. One or both parents of most persistent snorers have also been troublesome snorers. Snoring and OSA are made worse by obesity, nasal obstruction due to rhinitis, a deviated septum or nasal

polyps, a relatively small oropharynx with a large tongue, a recessive mandible, a long soft palate or enlarged tonsils. Snoring and OSA increase with age up to about 70 but may then decrease, at least in some patients. Whether or not snoring is the subject of complaint from the partner depends to some extent on the perceived annoyance value of the noise for that person (we respond more to offensive noises). This, in turn, depends on the partner's hearing acuity and usual depth of sleep.

Observed Apnoeas

Being heard to stop breathing or make choking noises and loud gasps when asleep (i.e. having 'apnoeas') is reported by a surprising number of ostensibly healthy adults in the community (about 30% of men and 15% of women). Objective recordings in patient's own houses and in sleep laboratories are consistent with the subjectively reported epidemiological data.

Overnight polysomnography in a specialist sleep centre is essential for making the diagnosis of OSAS

It is the duration of these apnoeas, their frequency per hour and the degree of oxygen desaturation and of sleep fragmentation that they cause that makes them important. This cannot be accurately assessed by the patient or partner. Overnight polysomnography in a specialist sleep centre is essential for making the diagnosis of OSAS, as described elsewhere. [3] It involves continuous recordings of sleep, breathing (air flow and respiratory movements), arterial oxygen saturation, snoring and other noises, sleeping position, leg movements, heart rate, etc. Such polysomnography is now widely available in Australia.

It is common in a sleep laboratory to record apnoeas lasting longer than 60 seconds associated with arterial oxygen desaturation to 50% or less. Many apnoeas and hypopnoeas end with a brief arousal, often with body and limb movement that makes the sleep restless and fragmented although the patient is seldom aware of this. It is the distressed partner who witnesses these events, wondering whether and when apnoeas will end.

The more frequently apnoeas and/or hypopnoeas occur per hour of sleep the more likely it is that the patient will have excessive daytime sleepiness

The more frequently apnoeas and/or hypopnoeas occur per hour of sleep the more likely it is that the patient will have EDS although this is not a close relationship. That

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frequency is measured by the apnoea-hypopnoea index (AHI), otherwise known as the respiratory disturbance index (RDI). Apnoeas and hypopnoeas are usually but not always more frequent and longer lasting in rapid eye movement (REM) sleep than in non-REM sleep.

It seems that to have occasional brief apnoeas or hypopnoeas is a benign condition. At what frequency these episodes begin to be clinically significant is not yet generally agreed, some centres using a lower cut-off frequency of five events per hour, others 10 or even more per hour. However, the RDI by itself cannot be the single measure of the severity of OSAS nor can the level of arterial oxygen desaturation or the frequency of arousals from sleep although all are involved. In a patient with coronary artery disease, a few apnoeas per hour, each causing the arterial oxygen saturation to fall below 80%, may be sufficient to cause myocardial ischaemia and potentially fatal arrhythmias.

Fig. 2. The Epworth Sleepiness Scale Name: Today's date: Your sex (Male = M, Female = F): ___ Your age (Yrs): · How likely are you to doze off or fall asleep in the following situations, in contrast to feeling just tired? This refers to your usual way of life in recent times. . Even if you haven't done some of the 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 I = slight chance of dozing 2 = moderate chance of dozing 3 = high chance of dozing It is important that you put a number (0 to 3) in each of the 8 boxes. Situation Chance of Dozing (0-3) Sitting and reading Watching TV Sitting, inactive in a public place (e.g. a theatre or a meeting)..... As a passenger in a car for an hour without a break..... Lying down to rest in the afternoon when circumstances permit Sitting and talking to someone Sitting quietly after a lunch without alcohol..... In a car, while stopped for a few minutes in the traffic

Excessive Daytime Sleepiness

This symptom has been difficult to assess in the past. It is not synonymous with hypersomnia, which is the tendency to sleep for much longer than the average 7.5 hours per 24 hours. Nor is it synonymous with fatigue or tiredness that are subjective feelings, especially of 'heavy' muscles. Sleepiness is a measure of how likely a person is, relative to other people, to doze off while engaged in particular activities. This sleep propensity at a particular time is markedly influenced by posture and by psychological and behavioural state, so sleepiness can be measured only in relation to particular activities. Some situations involve relative inactivity (such as sitting quietly and watching television) and are consequently much more soporific than other activities such as standing, walking or talking. Some patients who suffer from EDS avoid soporific situations and dozing by keeping busy all day, perhaps not even sitting down. They

> may not consciously recognise or complain about sleepiness. Nevertheless, the sleepy brain is generally slowed down and has more difficulty with tasks that involve divided attention (driving a car is one such task).

The Epworth Sleepiness Scale measures a person's average level of daytime sleepiness

There is now a simple self-administered questionnaire - the Epworth Sleepiness Scale - that measures a person's average level of daytime sleepiness across a variety of situations that are commonly met in daily life (Fig. 2). This is standardised and used in several countries. The Epworth score, which can vary from zero to 24, is the sum of eight numbers (0-3) chosen as answers to eight questions. Normal Epworth scores lie within the range zero to 10 (mean $4.6 \pm 2.8 s$). Scores above 10 represent EDS that may be mild (scores of 11 or 12), moderately severe (13-15), and severe (16 and above). At least 10% of the adult population have EDS, defined by an Epworth score > 10. The wide range of normal Epworth scores seems to represent long-term differences in a psychophysiological trait that makes us



Table II. Symptoms and clinical features of obstructive sleep apnoea syndrome

Hallmarks

Persistent snoring

Observed apnoeas

Excessive daytime sleepiness

Associated symptoms

Unrefreshing sleep

Restlessness when asleep

Dull head and dry mouth in the morning

Gastro-oesophageal reflux during sleep

Nocturia, enuresis

Irritability and depression

Poor concentration and short-term memory

Impotence

Predisposing factors

Obesity

Sleeping on one's back

Family history of snoring

Anatomically small airway, e.g. recessive mandible, long soft

palate, closely spaced

posterior pillars, large tongue

Adenotonsillar hypertrophy

Nasal obstruction, e.g. deviated septum, polyps, allergic rhinitis

High alcohol intake, especially at night

Musculoskeletal disorders, e.g. kyphoscoliosis, muscular dystrophy

Metabolic disorders, e.g. hypothyroidism

Some drugs, e.g. hypnotics, prazosin

relatively 'sleepy' or 'alert' individuals, even without a sleep disorder.

The effect of a sleep disorder such as OSAS is to increase he average level of daytime sleepiness, which is reflected n higher Epworth scores. However, a variety of other sleep lisorders such as restless legs syndrome, periodic limb novement disorder, narcolepsy and some forms of insomnia can also cause EDS. It is quite common for a particular patient o have more than one sleep disorder contributing to their EDS. Other disorders such as hypothyroidism, renal insufficiency or depression can also be associated with EDS and should be treated on their own merits. Only a minority of people in the community who have EDS have OSAS as its main cause.

Symptoms and Clinical Features Associated with OSAS

Many symptoms are associated with obstructive sleep apnoea syndrome (OSAS), in addition to the three hallmark symptoms (Table II). None of these symptoms, by itself, is specific for OSAS. Obesity is a good example of this. Though the majority of OSAS sufferers are overweight there is no measure of obesity, such as body mass index or neck circumference, that is an accurate predictor of OSAS. OSAS is not synonymous with obesity–hypoventilation syndrome, previously called 'Pickwickian syndrome'.

Who Should be Referred for Polysomnography?

The symptoms and clinical features listed in Table II usually only become indicative of OSAS when some or all of the three hallmark symptoms are also present. It is the hallmark symptoms that should provide the basis for referral to a specialist sleep centre for polysomnography. For example, in someone who snores but does not stop breathing at night and does not have EDS, it is unlikely that morning headaches or hypertension will be caused by OSAS.

Anyone who has excessive daytime sleepiness, particularly if severe, requires polysomnography and expert evaluation

Anyone who has EDS, particularly if severe, requires polysomnography and expert evaluation. Those who snore persistently and are reported to stop breathing at night will also need such investigation even if they do not have EDS. Other patients who snore and have EDS may have episodes of significant arterial oxygen desaturation at night without having apnoeas or hypopnoeas. This is called the 'upper airway resistance syndrome' and cannot be diagnosed without polysomnography. An occasional patient will have significant OSA without much snoring.

Treatment of OSAS

The mainstay of treatment for OSAS is nasal CPAP (continuous positive airway pressure). Seldom is weight loss or reduction in alcohol intake sufficient treatment although patients should be encouraged to achieve both. The clinical need for CPAP and its implementation are best dealt with by a specialist sleep centre. Once a diagnosis of OSAS, or significant OSA without EDS is made, patients come in to the sleep centre again to have their sleep monitored with the CPAP mask on so that the appropriate CPAP pressure can be established. That pressure typically lies between 5cm and 15cm of water and occasionally higher. The air pressure must be sufficient to keep open the nasal and pharyngeal airway, preventing apnoeas and snoring alike. Nasal CPAP is very safe treatment that can be guaranteed to work, provided the patient tolerates it and uses it at home.

Compliance rates after 1 year are about 70%. The main reason for non-compliance is nasal symptoms made worse

by CPAP, such as stuffiness, dry or cold nose and watery discharge during the day. Many such problems subside within a month. Those that persist can often be helped by including a heater—humidifier in the CPAP system. Nasal septoplasty and turbinate trimming may make it possible for some patients to use CPAP in whom it would not otherwise be possible. Feelings of claustrophobia with the CPAP mask on can generally be overcome if enough time is spent in training patients to use it, particularly with the wide range of different masks now available. For most patients with OSAS it is the improvement in the quality of their sleep and relief of their EDS by CPAP treatment that encourages them to comply with it. With OSA alone (i.e. without EDS) CPAP is less frequently tolerated but is still the most effective treatment.

For those who cannot use CPAP, surgery is most suitable for cases of snoring without OSAS

For those who cannot use CPAP, surgery (palatoplasty [often now with laser] combined with nasal septoplasty, turbinate trimming and tonsillectomy where indicated) may offer an alternative. Surgery is most suitable for cases of snoring without OSAS or perhaps for mild OSAS. The results are often very good within the first few weeks but some patients begin to snore again later. After a year, two-thirds of patients have some reduction in the frequency

and loudness of snores, less daytime sleepiness and typically about 50% fewer apnoeas/hypopnoeas. Seldom is snoring or OSAS fully controlled by surgery.

Another alternative to CPAP is the use of oral and dental devices that reposition the tongue and mandible forward when worn at night. They have not been widely used in Australia but are about as effective as surgery and may be used more commonly in the future.

Apart from the relief of rhinitis and avoiding medications that make OSAS worse, drugs have no direct role in the management of OSAS.

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