Snoring and sleepiness are common symptoms presenting to general practice. In the United States of America, 60% of subjects consecutively surveyed in a primary care setting presented with symptoms of an underlying sleep disorder. In Australia, 6% of patients reported visiting a doctor for a problem of sleep apnoea or snoring.

It is estimated that more than 60% of adults occasionally snore and more than 30% regularly snore, and that obstructive sleep apnoea (OSA) occurs in approximately 10% of females and 25% of males, of whom 2 and 4% respectively have OSA with sleepiness – the OSA syndrome. In children, 12% snore regularly and 2% have OSA. Given the rise in obesity and reductions in sleep time, the prevalence of sleep related symptoms, snoring, and OSA are likely to have increased in both children and adults.

The term ‘OSA syndrome’ is used to describe the constellation of recurrent apnoeas (ie. absence of airflow for >10 seconds) or hypopnoeas (ie. reductions in airflow >10 seconds, sufficient to cause a fall in oxygen saturation or arousal from sleep) occurring at least five times per hour and associated with snoring and symptoms of daytime sleepiness or fatigue. On a sleep study report, the total number of apnoeas and hypopnoeas in an hour is given as the apnoea-hypopnoea index (AHI). An AHI ≥5 is considered significant.

Pathophysiology

Upper airway collapse due to sleep related loss of muscle tone (or inadequate muscle tone due to elevated nasal resistance) associated with a small oropharyngeal area (Figure 1) is considered the cause of most cases of OSA. In recent years, greater consideration has been paid to subconscious arousals and an altered central ‘controller’ of respiration causing transient reductions in respiratory drive. This culminates in an oscillation of apnoeas and/or hypopnoeas with hyperpnoeas which, with an unstable upper airway, will appear ‘obstructive’, whereas in other circumstances, with a patent upper airway, these may contribute to ‘periodic breathing’. This latter form of sleep apnoea is seen at high altitude, in premature infants and in...
adults with cardiac failure (Cheyne-Stokes respiration), stroke, renal failure, chronic narcotic use and/or diabetic ketoacidosis.

Snoring with less than five events per hour (AHI <5) has often been classified as benign, however there is evidence suggesting it may contribute to premature atherosclerosis of the carotid artery – presumably due to local vibrational damage, as the femoral artery does not appear to be affected. In the long term, this may contribute to cerebrovascular events. Moreover, regardless of the AHI, other family members may be disturbed by the snoring, which therefore may warrant treatment.

**When to investigate**

In the primary care setting, the following groups should be considered for a sleep study.

**Loud noisy snoring**

Loud noisy snoring defined as noise sufficiently loud enough to cause disruption to another person’s sleep on a regular basis. Snoring is associated with greater divorce rates – no small matter when one considers the value of the family unit in terms of cost effective delivery of housing, education and health care. Patients with any of the following snoring habits should be referred for assessment by a sleep physician:

- sufficient to disturb partner more than three nights per week
- audible in other rooms
- occurs despite alcohol abstinence
- occurs in lateral sleep position, or
- occurs >10% of the night.

### Table 1. Epworth Sleepiness Scale

The following questions refer to sleepiness or the tendency to doze off when relaxed.

How likely are you to doze off or fall asleep in the following situations, in contrast to just feeling tired? This refers to your usual way of life in the past 3 months. Even if you haven’t done some of these things recently, try to work out how they would have affected you.

Choose the most appropriate number for each situation by putting an X in one box for each question.

<table>
<thead>
<tr>
<th>Situation</th>
<th>(0) Would never doze</th>
<th>(1) Slight chance of dozing</th>
<th>(2) Moderate chance of dozing</th>
<th>(3) High chance of dozing</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Sitting and reading</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>2. Watching TV</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>3. Sitting, inactive in a public place (e.g. at the theatre or a meeting)</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>4. As a passenger in a car for an hour without a break</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>5. Lying down to rest in the afternoon when circumstances permit</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>6. Sitting and talking to someone</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>7. Sitting quietly after a lunch (without having had alcohol)</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
<tr>
<td>8. In a car, while stopped for a few minutes in traffic</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
<td>☐</td>
</tr>
</tbody>
</table>

Total = _______________/24__________

Score: 1–6 = adequate sleep; 7–8 = average sleep; >9 = abnormal sleep
Obesity
Obesity is the most significant risk factor for OSA. Increasing body mass index, neck circumference and waist-to-hip ratio are all associated with increased prevalence of OSA. Importantly, obesity is a controllable risk factor and weight reduction results in improvement of OSA, especially in those patients undergoing bariatric surgery. Obesity is also associated with many cardiovascular risk factors which, combined with OSA, can result in significant morbidity and mortality.

Unstable general medical conditions
Hospital patients in whom to consider OSA are those presenting multiple times for the same problem, such as acute exacerbation of COPD or chronic heart failure (CHF), particularly if present with hypercapnia and responsive to noninvasive ventilation. Patients assessed as ‘difficult to intubate’ by an anaesthetist should also be considered (Figure 2). Unexplained polycythaemia is another common sequelae of untreated OSA.

Clinical examination
The clinical examination of patients with possible OSA should include:
- estimate sleep duration
- estimate Epworth Sleepiness Scale (Table 1)
- assess nasal patency and sinus disease
- assess upper airway – Mallampati score (Figure 2), state of dentition, hard palate
- measure neck circumference/shirt collar size (≥42 cm in males, ≥39 cm in females increases the risk of OSA)
- check blood pressure
- reassess medications.

Which investigation?
The investigation for OSA should include the monitoring of sound, airflow, respiratory effort, sleep, body position and cardiac function. Unfortunately a monitor that would do all these accurately, inexpensively and with simplicity and durability does not as yet exist. While there has been a rapid development of diagnostic tests for OSA, many are devoid of quality assurance and comparative testing.

Levels of investigation
There are four levels of investigation. A diagnosis of OSA should be made from a combination of history, examination, a test (one of level 1–4 below), and occasionally, a trial of therapy.

Level 1
Laboratory ‘attended’ polysomnography: useful to confirm a diagnosis absolutely. Also assists in determining other forms of sleep disordered breathing (eg. Cheyne-Stokes respiration, hypoventilation disorder) and is helpful in patients who are less coordinated. Is often considered the ‘gold standard’ and additional monitoring (eg. infrared video monitoring, transcutaneous CO₂, continuous systemic BP, oesophageal pressure, multiple sleep or wakefulness latency...
testing) can be utilised. Currently funded by Medicare Australia. Test failure rate = <0.01%.
(NB: Test failure rate = percentage of tests performed in which inadequate information is collected to make a diagnosis – usually the result of technical difficulties, eg. electrodes dislodging, battery failure, patient error in managing equipment.)

Level 2

Home ‘unattended’ polysomnography: basic polysomnography with patient setting themselves up with the aid of detailed instructions (eg. DVD). Currently, interim Medicare funding is available. Test failure rates are modestly high (<15%), thus requiring repeat testing.

Level 3

Cardiopulmonary monitoring: airflow, respiratory effort, oximetry and electrocardiogram (ECG) are monitored either at home or in the hospital setting. Ideally suited to moderately high risk OSA patients or in patients in whom Cheyne-Stokes respiration or cardiac arrhythmias associated with OSA are suspected. No Medicare funding is available. Test failure rate = <5%.

Level 4

Single channel cardiopulmonary monitoring (eg. oximetry [SpO₂ and heart rate] and/or airflow): ideal to ‘rule in’ OSA for patients in whom OSA is highly suspected. As false negative results can occur, it should not be used to ‘rule out’ OSA. No Medicare funding is available. Test failure rate = 1%.

Management

A patient’s life stage will often give a good indication of the underlying cause of their OSA (Table 2). The majority of patients are middle aged, for whom initial lifestyle management should include:

- caution about alcohol intake (<7 drinks per week)
- antismoking advice
- avoidance of sleep deprivation
- initiation of nasal steroids for a 4 week trial
- consideration of positional therapy: sleep in nonsupine position (recovery position), use one pillow and raise head of bed 10 cm, and
- initiation of a weight loss program.

A referral for a sleep study should be considered if signs or symptoms of OSA persist after a period of lifestyle management.

Surgery

In children, tonsillectomy with adenoidectomy has a role as the soft tissues are disproportionately greater than the bony airway size (Figure 3). In adults, bariatric surgery may have a role in reducing obesity – before and after studies suggest amelioration of OSA rather than abolition. A multicentered trial is underway in Australia with results expected in 2011.

Nasal decongestive surgery or tonsillectomy with adenoidectomy should be considered where soft tissue abnormalities exist.

Table 2. Lifestyle indications of causes of OSA

<table>
<thead>
<tr>
<th>Age</th>
<th>Cause</th>
<th>Best treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Children/adolescents</td>
<td>Anatomical</td>
<td>Surgical</td>
</tr>
<tr>
<td>Middle aged patients</td>
<td>Lifestyle/weight</td>
<td>• Lifestyle change</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Weight loss</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Mandibular advancement splints</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• CPAP</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Surgery</td>
</tr>
<tr>
<td>Older patients</td>
<td>• Drugs</td>
<td>• Rationalise drugs if possible</td>
</tr>
<tr>
<td></td>
<td>• Medical conditions</td>
<td>• Treat underlying condition</td>
</tr>
<tr>
<td></td>
<td>• Lifestyle/weight</td>
<td>• Lifestyle change/weight loss</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• CPAP</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Mandibular advancement splints</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Surgery</td>
</tr>
</tbody>
</table>

Figure 3. Graph of upper airway and soft tissue dimensions in 41 children, followed with sequential lateral cephalograms, and indicating the small upper airway size relative to soft tissue before 11 years of age, suggesting a relative predisposition to airway closure with tonsillar and adenoid hypertrophy.

Jaw advancement and maxillary expansion surgery should be considered where bony abnormalities exist.

Uvulectomy, which showed much promise in the 1980s, has not proven to be of long term benefit and has significant complications, most importantly difficulty tolerating CPAP later in life.

Overall, surgery has the greatest impact in the young where an anatomical abnormality is present.

Dental splints

Designed to either hold the mandible forward or to widen the hard palate, there are a range of dental splints available and these are smaller than a tennis ball in size. Suitable patients should have mild to moderate OSA, minimal hypoxemia, adequate nasal patency, good dentition, healthy temporomandibular joints, and an absence of significant cardiovascular disease. In selected populations, the success rate can be as high as 70%. However, splint cost ($1500–2000), longevity (~2 years) and side effects (dental pain and movement, excessive salivation, temporomandibular joint pain) may limit their use.
Continuous positive airway pressure

Developed in the early 1900s and rediscovered and made portable for domiciliary use in the 1980s, continuous positive airway pressure (CPAP) has been effective in managing moderate to severe OSA. It has also been used to treat acute pulmonary oedema in the hospital environment.

Several CPAP variants have been developed, such as bilevel positive airway pressure, to treat acute and chronic hypercapnic respiratory failure from many causes such as neuromuscular disease, COPD, and kyphoscoliosis. The engineering behind CPAP has also been developed to assist patient comfort, eg. auto-titrating CPAP, expiratory pressure relief, humidification and pressure ramping, as well as to treat more complex sleep apnoeas such as Cheyne-Stokes respiration.

Most pumps have inbuilt hour meters to assess usage objectively; others have built in computers which analyse mask leak, night-to-night usage, snoring, and apnoeas and hypopnoeas. Thus adherence and compliance to treatment can be measured objectively. Pumps are lightweight (<2 kg), small (<2000 cc) and operationally quiet (<35 dB). Developments in mask technology have produced external nasal, oral, oro-nasal, facial and total head (hood) masks.

Circumstances in which CPAP users might require special attention include while camping, on overseas travel and while travelling on an aeroplane. Most pumps are multi-voltage and can be used anywhere that has an electricity supply. Travelling patients should be reminded to take an extension cord and a local power point adapter. Inflight use of CPAP varies with airlines and patients should be advised to contact their airline before travel. Continuous positive airway pressure pumps can be carried as hand luggage.

Complications of CPAP are not uncommon: pump breakdown (after 5–10 years use) as well as mask and tubing decay (after 1–2 years) occur, therefore it is advisable that patients have their pump checked by a CPAP distributor or sleep centre at least annually. Acute rhinitis, dermatitis and pressure sores do occur (Figure 4). Humidification and/or nasal corticosteroids and alteration of CPAP pressure and/or mask should be considered respectively. Occasionally nasal decongestive surgery can assist adherence to CPAP. Most patients can cope if given support, and GPs play an important role in both recognising a need for, and providing, this support. Further support can be provided via a multidisciplinary approach.

Conclusion

Management of OSA is a long term commitment for both patients and their clinicians. It is imperative that the physician takes an ongoing interest in determining that the patient’s (or partner’s) concerns have been adequately dealt with. Importantly, more than one diagnosis may be apparent and, if so, a multi-pronged approach may be required.

Conflict of interest: none declared.

References