Waiting to exhale

Surgical intervention can play a key role in obstructive sleep apnoea.

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TIREDNESS and fatigue are common reasons for presenting to GPs. Obstructive sleep apnoea (OSA) is one of the more important causes of daytime somnolence because of the potentially serious systemic effects it has on multiple organ systems, as well as its effect on quality of life.

The ENT surgeon has a role to play in the surgical management of both children and adults with this syndrome — for primary surgical treatment of an obstructive lesion or to improve nasal airflow for CPAP.

In the Australian adult population, the prevalence of moderate to severe OSA is estimated to be 4.7%, with a higher prevalence in men than women.[1,2] This figure underestimates the true burden of the disease, with many more people having symptoms of undiagnosed OSA. In children, the prevalence is estimated to be around 2%.[3]

Pathophysiology

The pathophysiological process that causes OSA is upper airway collapse while sleeping. The mechanism is a combination of an easily collapsible upper airway with the relaxation of pharyngeal dilator muscles that normally occurs during sleep.

Anatomical factors that can contribute to upper airway collapse include obesity, soft tissue hypertrophy (including tonsillar, adenoid and lingual tissue), macroglossia and micrognathia (relatively short mandible).

These factors will all increase the extraluminal tissue pressure surrounding the airway

Anatomical factors are not always the cause of OSA, however, and patients without obvious risk factors may have a problem with reflex pathways from the central nervous system to the pharyngeal dilator muscles.

Anatomically, the three major areas of obstruction are the nose, oropharynx and hypopharynx. Nasal obstruction may be caused by nasal septum deviation, turbinate hypertrophy and/or adenoid hypertrophy and can lead to open-mouth breathing during sleep.

Mouth breathing, in turn, increases upper airway collapsibility, destabilises the oral tongue and may decrease the effect of the dilator muscles. In children, adenotonsillar hypertrophy is the major cause of OSA. However, other structural factors such as craniofacial abnormalities can also play a role in upper airway collapse.

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OSA diagnosis in adults

Daytime hypersomnolence and loud snoring are the most common presenting complaints of adults with OSA. Other symptoms and signs include morning headache, restless sleep, or waking unrefreshed despite a good duration of sleep.

The Epworth Sleepiness Scale is a commonly used tool to assess daytime sleepiness, with a score greater than 10 raising suspicion of OSA.

General physical examination includes BMI calculation, blood pressure and assessment of body habitus. The nose should be assessed for external deformity and septal deviation. Assessing the oropharynx includes examination for tonsillar hypertrophy, macroglossia or an elongated soft palate.

Sleep laboratory polysomnography is the diagnostic investigation of choice. Home sleep studies provide similar information to laboratory polysomnography and are less expensive. However, they may underestimate the severity of sleep apnoea.[4]

The severity of OSA corresponds to the Apnoea–Hypopnoea Index (AHI). In adults, mild OSA is classified by an AHI of 5-15. Patients with mild OSA may either be relatively asymptomatic or have symptoms of daytime sleepiness that do not significantly affect their daily life.

Moderate OSA corresponds to an AHI of 15-30, and patients will generally be aware of daytime somnolence and take steps to avoid falling asleep at inappropriate times.

Patients with an AHI greater than 30 are classified as having severe OSA. These patients tend to have significant but often unrecognised neurocognitive deficits.

OSA diagnosis in children

Children with OSA most frequently present with a history of snoring and difficulty breathing during sleep. Parents may report restlessness, nocturnal sweating and unusual sleeping positions, most commonly with the neck extended.

In contrast to adults, daytime hypersomnolence is less frequently a presenting complaint in children, although a child will often complain of tiredness on waking or in the early afternoon. Screening for snoring in children is recommended and further investigation should proceed if there are other risk factors for OSA.

Physical examination should include general assessment of height and weight and for the presence of craniofacial abnormalities or tonsillar hypertrophy. Reduced nasal airflow, hyponasal speech ('blocked nose' voice) and an open mouth breathing posture are all signs of adenoid hypertrophy. It is difficult to distinguish clinically between primary snoring and OSA. As in adults, polysomnography is the diagnostic investigation of choice.

Complications in adults

OSA has been shown to result in significant morbidity and reduction in quality of life. Untreated OSA

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results in increased mortality, cardiovascular disease and neurocognitive difficulties. OSA has also been reported to increase the risk of fatal and non-fatal motor vehicle accidents 2.5-fold.[5]

The cardiovascular effects of OSA include hypertension, coronary heart disease, congestive heart failure, arrhythmias, pulmonary hypertension and stroke. Untreated OSA has been shown to be a risk factor for insulin resistance and may contribute to the development of diabetes.

Patients with moderate to severe obstructive sleep apnoea have a physiological age 10-15 years greater than their true age. Neurocognitive effects of OSA include problems with attention, working memory, executive reasoning and high-end problem solving.

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Complications in children

OSA can have deleterious effects on a child's development, including reduced growth or failure to thrive, and behavioural or learning problems. OSA may lead to poor memory, reduced attention, poor behavioural and emotional regulation, hyperactivity, aggressiveness, poor school performance and alterations in IQ.[6]

Cardiovascular complications in children, such as pulmonary hypertension and cor pulmonale, may occur as a result of severe, untreated OSA.

Treatment in adults

Treatment for OSA needs to be tailored to the individual patient. In patients who are overweight or obese, weight loss should be the first step. Patient-controlled factors that promote upper airway collapse, such as alcohol consumption, should be discouraged.

Treatment for moderate to severe OSA has traditionally been continuous positive airway pressure. CPAP has been shown to reduce the apnoea–hypopnoea index and to subjectively improve sleep quality and quality of life, and reduce the risk of cardiovascular events and motor vehicle accidents.[7,8]

Compliance with CPAP is an important issue, as many people do not tolerate this treatment modality. If nasal airway obstruction is an issue, surgical correction usually improves compliance for other treatments by improving nasal airflow.[9]

Mandibular advancement splints may be used in some patients, but it can be difficult to determine who will benefit prior to a trial. Reports have suggested high rates of adherence for oral appliance therapy.[10] CPAP, however, has been shown to be more effective in reducing the AHI than mandibular advancement.[11]

The surgical treatment for obstructive sleep apnoea syndrome is a staged stepwise multilevel approach as there are often multiple areas contributing to the obstruction — anywhere from nasal tip to larynx. Careful patient selection is crucial.

Surgical treatment options for obstruction of the nose or nasopharynx include septoplasty, inferior turbinectomy, adenoidectomy and reconstruction of the nasal valve.

Palatal surgery includes tonsillectomy and modified forms of uvulopalatopharyngoplasty.

Hypopharyngeal surgery includes lingual tonsillectomy, partial midline glossectomy, radiofrequency tongue base ablation or open tongue base resection.

Emerging surgical treatments for OSA include hypoglossal nerve stimulation. This involves a pulse generator that is implanted similarly to a cardiac pacemaker and turned on and off by the patient. The pulse generator is connected to an electrode cuff attached to the hypoglossal nerve. Stimulation of the hypoglossal nerve results in tonic contraction of the genioglossus muscle leading to tonic contraction of the base of the tongue and widening of the retrolingual airspace.

Hypoglossal nerve stimulation therapy may provide an alternative surgical treatment option for patients who do not benefit from or tolerate CPAP, as well as patients who get only partial improvement from other surgical therapies.

Treatment in children

As mentioned, OSA in children is often due to adenotonsillar hypertrophy. For these patients, adenotonsillectomy is the first-line treatment.[12] The procedure has been reported to improve growth and decrease energy expenditure during sleep.

Obese children, those with syndromic features and those with severe OSA are less likely to be cured by adenotonsillectomy alone. Weight loss plays an important role in the management of these children.[13]

Children with neuromuscular dysfunction who are at high risk for persistent obstruction after adenotonsillectomy may be considered for other reconstructive techniques. In children with craniofacial disorders, surgical treatments such as mandibular advancement, maxillary expansion, tongue reduction and tongue-hyoid suspension may be used.

In contrast to adults, CPAP is mainly used for children who fail to respond or are not candidates for surgical intervention.[12]

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