ARTICLE

Diagnosis and management of obstructive sleep apnoea in adults

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Keywords
continuous positive airway pressure, obstructive sleep apnoea, sleep study, snoring

Aust Prescr 2024;47:52–6
https://doi.org/10.18773/austprescr.2024.010

Summary

Obstructive sleep apnoea is the most common form of sleep-disordered breathing. It is characterised by recurrent occlusion of the airway during sleep. Ensuing apnoeas terminate in arousal from sleep and lead to non-restorative sleep, excessive daytime sleepiness and adverse cardiovascular and neurocognitive effects.

A sleep study should be offered to patients reporting witnessed apnoeas or symptoms related to non-restorative sleep. It should also be considered in the presence of predisposing factors for obstructive sleep apnoea (e.g. obesity, tonsillar hypertrophy, retrognathia, refractory hypertension).

Treatment should aim to improve symptoms and reduce cardiovascular and neurocognitive risk. The treatment approach should consider the symptom burden, severity, anatomical factors, and patient preference.

Positive airway pressure is the most effective treatment option, although intolerance and non-adherence are common. Other options include positional therapy, oral appliances and upper airway surgery.

Weight loss and optimisation of cardiovascular disease risk should be considered in selected patients.

Introduction

Obstructive sleep apnoea (OSA) is a common condition, with a global adult population prevalence between 9 and 38%.[1] It is characterised by repetitive collapse of the upper airway, causing increased respiratory effort, transient absences of spontaneous respiration (apnoeas) and oxygen desaturations, terminating in partial or full arousal from sleep. Resulting sleep fragmentation leads to non-restorative sleep and daytime sleepiness.

OSA results from a complex interplay of anatomical factors causing upper airway crowding, in addition to physiological predisposing factors, including reduced pharyngeal muscle tone and abnormal airway reflex responses.[2] It is most common in individuals with obesity or structural narrowing of the upper airway. Non-anatomical contributors, including arousal and ventilatory responses to apnoeas, are believed to propagate the condition. Alcohol and some medications (e.g. hypnotics, opioids, myorelaxants, testosterone supplements and antipsychotics) may worsen OSA, usually by depressing respiratory drive or increasing upper airway muscle collapsibility.

OSA is linked to cardiovascular, metabolic and neurocognitive sequelae. These include myocardial infarction, atrial fibrillation, stroke, hypertension, insulin resistance,[3] mood disorder[4] and reduced cognition.[5] There is an increased risk of motor vehicle accidents,[6] which has implications for public safety.

Treatment of OSA reduces daytime sleepiness, and improves mental health, cognition, quality of life[7] and motor vehicle safety.[6] Despite its association with cardiovascular disease and cognitive decline, studies aiming to demonstrate improved longer-term health outcomes from treatment of OSA have not identified a positive effect.[7]

Screening

Population screening for OSA is not currently recommended. Screening should occur in higher risk groups, including people with predisposing factors or medical conditions associated with OSA (Box 1). Symptoms may be under-reported, so clinicians should consider the possibility of OSA in the absence of symptoms in these higher risk groups. Commonly used validated screening tools include the STOP-BANG,[8] OSA-50[9] and the Berlin Questionnaire.[10] All tools have similarly high sensitivity and low specificity for OSA. There is some overlap in each questionnaire; however, specific items in each tool may result in a higher score in a particular patient.

Clinical assessment

Clinical assessment for suspected OSA should establish the self-reported sleep quality and daytime symptom profile. History from available witnesses informs the assessment. Witnesses should be asked about the frequency and severity of snoring,

This article is peer-reviewed

Box 1 Predisposing factors and medical conditions associated with obstructive sleep apnoea

Anatomical
- obesity
- adenoid/tonsillar hypertrophy
- retrognathia

Metabolic
- polycystic ovarian syndrome
- trisomy 21
- hypothyroidism
- acromegaly

Neuropsychiatric
- depression
- cognitive impairment

Vascular
- refractory hypertension
- atrial fibrillation
- type 2 diabetes
- myocardial infarction
- ischaemic stroke
- heart failure (preserved and reduced ejection fraction)

Other
- pregnancy
- chronic lung disease
- floppy eyelid syndrome

the presence of apnoeas, snorting, gasping and motor restlessness. Other nocturnal symptoms include fragmented sleep and nocturia. Common daytime symptoms include dry throat and daytime sleepiness, fatigue, and poor concentration and vigilance due to non-restorative sleep. The Epworth Sleepiness Scale is a tool that can assist with quantifying symptoms of subjective sleepiness.[11] Many who suffer from OSA will not report sleepiness. Sleepiness may also arise from separate sleep conditions or mood disorders.

It is important to determine safety risks pertaining to driving motor vehicles or operating machinery. Prior accidents or near misses related to sleepiness predict future events.[6] People with a commercial or heavy vehicle driver's licence hold higher public safety requirements and specialist input may be required to evaluate their risk profile. It is the driver's responsibility to alert relevant authorities of an OSA diagnosis.

Examination in suspected OSA assesses for predisposing anatomical factors. This includes body mass index, and waist and neck circumference. Upper airway examination should assess for the presence of retrognathia, pharyngeal crowding and tonsillar enlargement. The Friedman Tongue Position (FTP) is a commonly used scale to grade airway narrowing.[12] It differs from the Mallampati score as the tongue is assessed in the neutral position.

Investigations

An overnight sleep study is used to confirm a diagnosis of OSA. Laboratory or home testing are both acceptable options;[13] the choice is based on logistics and patient capability and preference. Both forms of sleep study collect multiple channels of physiological data, including electroencephalogram, respiratory flow and effort, oximetry, muscle tone and cardiac rhythm. These are the most validated tests in the diagnosis of OSA.

A home sleep study requires the patient to attend a sleep centre to fit sensors, sleep at home and then return the equipment the following day for analysis. It may be preferred in those who struggle to sleep in unfamiliar environments or have irregular sleeping hours. A laboratory sleep study requires a patient to attend a sleep centre, where they will be fitted with similar but additional sensors (including video monitoring). It may identify the presence of other sleep disorders related to observed movements such as parasomnias. Laboratory sleep testing may be more practical for those travelling long distances, for which collecting and returning equipment may be burdensome (e.g. regional patients).

In Australia, Medicare reimbursement for either a laboratory or home-based sleep study is available when requested by a sleep or respiratory specialist, or by any medical practitioner for patients who have an Epworth Sleepiness Scale score greater than or equal to 8, plus any one of the following:

- STOP-BANG score greater than or equal to 3
- OSA-50 score greater than or equal to 5
- Berlin Questionnaire – high risk.

There are other commercially available sleep tests that use fewer channels to record sleep data. In some instances, these can be purchased online or via a community pharmacy without the need for referral from a medical practitioner. Many of these have been validated in the diagnosis of OSA; however, the lack of a personalised treatment plan is a limitation of these clinical pathways.

Medically referred sleep studies are reported by a sleep specialist, and the report outlines the presence and severity of OSA and any other relevant findings or recommendations.

The severity of OSA is graded by the frequency of respiratory events, as represented by the apnoea-hypopnoea index (AHI) (Table 1).[14] The degree of symptoms often correlates poorly with OSA severity. Quantifying the hypoxic burden is another important factor for predicting cardiovascular outcomes.[15]

Treatment options

Treatment goals include improving symptoms and reducing risks to health and safety. Goal setting should be individualised, noting that objectives around reducing longer-term cardiovascular disease risk are presumptive.

A general practitioner (GP) can initiate treatment for symptomatic OSA. Positive airway pressure (PAP) is typically the first-line therapy for moderate to severe OSA, as outlined below, although long-term adherence can be suboptimal. Specialist referral may be considered if the benefits of treatment are unclear, or if further appraisal of treatment choices is required. Modifiable predisposing factors should be addressed.

Weight loss and medication review

Obesity is a factor in many OSA cases. Although weight loss rarely resolves OSA, it can reduce severity and improve symptoms.[16] A 10% reduction in weight has been shown to cause a 26% decrease in the AHI.[17] Weight loss also has cardiovascular benefits.

It is important to minimise alcohol intake and rationalise the use of medications that can worsen OSA or contribute to weight gain.

Positive airway pressure therapy

PAP therapy is efficacious across all OSA severities, and is recommended first line for moderate and severe cases.[7] Applying positive pressure to the upper airway, via a nasal or full-face mask, prevents airway collapse during sleep. Pressure can be delivered at a preset, continuous, fixed level (continuous positive airway pressure; CPAP) or with auto-adjusting positive airway pressure (APAP). APAP uses technology that senses snoring and airflow limitation, and increases the pressure to maintain airway patency. Fixed-pressure CPAP would typically require an initial laboratory titration study to identify the minimum pressure needed to control OSA. APAP device data can be used to select an optimal fixed pressure if preferred. APAP machines are as well tolerated as CPAP, and the difference in cost is diminishing, making APAP the initial choice in most circumstances.

A minimum of 4 hours nightly use is considered sufficient, although further symptom improvement occurs with up to 6 hours of use. Most devices accurately record the residual AHI to confirm treatment success and further sleep studies are often not required. Additional laboratory assessment may still be indicated for those with comorbidities (such as severe chronic obstructive pulmonary disease, hypoventilation, or mixed sleep-disordered breathing).

Table 1 Obstructive sleep apnoea severity

Severity	Apnoea-hypopnoea index (events per hour)
Not present	less than 5
Mild	5 to 14
Moderate	15 to 29
Severe	30 and above

PAP carries minimal risk and contraindications are rare. Adverse outcomes include sleep disruption, mask leak, aerophagia, skin irritation and, occasionally, pressure injuries. Adherence is limited by comfort, cost and perceived social stigma. Most PAP devices can accurately record nightly usage, allowing adherence to be easily tracked. Examples of people who may require evidence of adherence to treatment are commercial or heavy vehicle licence holders.

It is appropriate for a GP to initiate treatment in uncomplicated OSA. This can be established through an experienced CPAP vendor, who will provide patient education and training on the technical aspects of device operation. Many public hospital sleep departments offer subsidised PAP devices to people experiencing financial hardship. Patients will occasionally purchase a PAP device independently without prior medical consultation. It is advised to seek an opinion from a sleep specialist to ensure PAP is medically appropriate and correctly configured.

When initiating a patient on PAP therapy, early engagement is important to establish comfortable settings. This can be done remotely if the device has internet capabilities, and many suppliers will offer this service with a trial prior to purchase. Approximately 50% of patients who start PAP therapy cannot tolerate long-term use.[18] Discomfort can be reduced with optimal fitting of the interface, use of the device's ramp setting (to gradually increase the pressure to the prescribed level each night), and adequate humidification. In some instances, capping the maximum pressure may improve tolerability. Adjustments can be conducted with input from an experienced supplier or sleep specialist. A laboratory PAP titration study can be considered in cases where intolerance overnight is an issue. Real-time assistance with mask fitting and precise pressure assessment can be offered in this way.

Positional therapy

Wearable devices that prevent supine sleep are beneficial in cases of supine-dominant OSA. Options include semi-rigid cushions across the upper back fixed by strapping, or body position sensors that deliver a vibration when supine. Avoidance of supine sleep is reliably achieved by these devices, although long-term adherence is not well demonstrated.[19]

Oral appliance therapy

Mandibular advancement devices are applied to the upper and lower dentition. By advancing the mandible forward, the upper airway cross-sectional area is increased. These devices range from inexpensive non-adjustable 'boil and bite' devices, to custom-fit options usually provided by dental practitioners. They are most effective in mild and moderate OSA.[20] Oral appliance therapy can be considered in combination with weight loss or body repositioning in severe OSA, when PAP is poorly tolerated.

Favourable dental health and the retention of some native teeth are required for successful fitting. Mandibular advancement devices are generally well tolerated and major complications are uncommon. Some individuals experience persistent jaw repositioning and teeth-shift, which may affect bite alignment impermanently. Jaw and temporomandibular joint pain may develop, leading to discontinuation of use. Although major teeth-shift is uncommon, regular dental follow-up is essential to monitor for potential complications.

Upper airway surgery

Surgical management of OSA targets anatomical characteristics of the upper airway. Multiple procedures are available, sometimes in combination to achieve the best results.[21] Specialist assessment and nasendoscopy can help identify those with anatomy that favours surgical intervention. These are most effective in non-obese people and those with mild or moderate OSA,[21] although they can be considered in carefully selected patients with severe OSA.[22]

Nasal CPAP therapy may be poorly tolerated in the presence of an obstructed nasal passage. Septo-turbinoplasty is shown to improve CPAP adherence without significantly modifying underlying OSA severity.[23] OSA can be treated with tonsillectomy in select patients with tonsillar enlargement.[24] Uvulopalatopharyngoplasty may improve snoring and reduce OSA severity in appropriately selected patients. Postoperative symptoms following pharyngeal surgery can last weeks, or uncommonly, months. This includes pain, dysphagia, odynophagia, globus sensation and velopharyngeal incompetence.

Orthognathic surgery involves surgical advancement of both mandible and maxilla. It is most commonly performed in those with craniofacial abnormalities.[25] These procedures may be efficacious even in severe OSA. There is risk of facial numbness, and the postoperative recovery period is protracted.

Conclusion

OSA is a common condition, with a variable clinical presentation. Evaluation for OSA should be offered to those with relevant symptoms or deemed to be at high risk. Multiple treatment options are available, enabling a personalised approach that addresses the patient's goals and disease severity. For complex cases, specialist or multidisciplinary input may be required. Management of associated obesity and its metabolic complications is advised. <

Conflicts of interest: Darren Mansfield is a member of the Sleep Health Foundation board. He has received speaker honoraria from Somnomed, a producer of mandibular advancement splints.

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