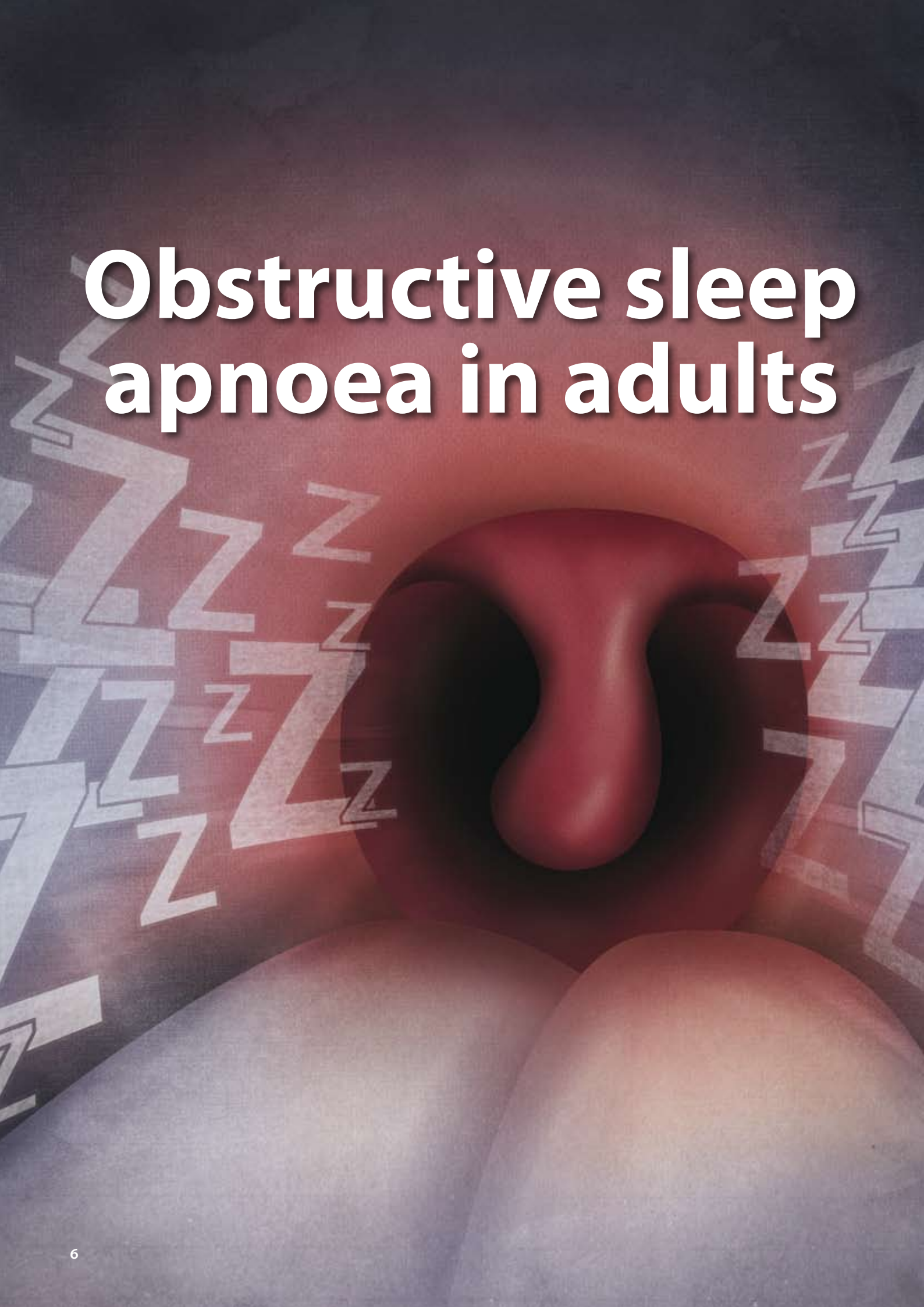
Obstructive sleep apnoea in adults



Obstructive sleep apnoea is reported to affect 4% of adult males and 2% of adult females. In New Zealand, it is twice as common in Māori adults males compared to non-Māori. Moderate to severe obstructive sleep apnoea is associated with a significantly increased risk of cardiovascular morbidity and mortality. People with mild symptoms and an absence of risk factors can often be managed with lifestyle interventions. However, people with more significant symptoms, such as excessive daytime sleepiness, will usually require treatment with a continuous positive airway pressure (CPAP) device.

Obstructive sleep apnoea is common in adults

Obstructive sleep apnoea is a sleep-related breathing disorder resulting in recurrent, partial (hypopnoea) or complete (apnoea) obstruction of the upper airways. It is caused by relaxation of airway muscles during sleep which allows soft tissue in the pharynx to collapse and block the upper airway. As a result of not breathing, oxygen saturation levels in the blood rapidly fall, and carbon dioxide increases, and eventually the brain triggers a brief arousal to resume breathing. Pauses in breathing usually last between ten and 30 seconds, but may persist for one minute or more. This cycle of events can occur hundreds of times a night and leads to broken, poor quality sleep. This night time "marathon" causes daytime sleepiness, which is the characteristic feature of obstructive sleep apnoea syndrome (OSAS).[1] Recurrent hypoxaemia also results in negative health consequences such as cardiovascular morbidity and mortality.

Upper airway resistance syndrome and central sleep apnoea are less common than obstructive sleep apnoea, but may be associated with similar symptoms. People with upper airway resistance syndrome do not experience airway blockage, however, they are frequently aroused from sleep due to the increased work required to breathe. Central sleep apnoea is caused by instability or imbalance in the control mechanisms that drive respiration. This causes respiration to cycle between apnoea and hyperpnoea (deep breaths). Referral to a sleep physician is generally required to differentiate upper airway resistance syndrome and central sleep apnoea from OSAS.

Apnoea-hypopnoea index

The severity of obstructive sleep apnoea is quantified by recording the number of pauses in breathing each hour that last longer than ten seconds. This is referred to as the apnoea-hypopnoea index (AHI).

Table 1: Classification of the severity of obstructive sleep apnoea[2]

Obstructive sleep apnoea severity	Apnoea-hypopnoea index (AHI)
Normal	Less than 5
Mild	5 – 15
Moderate	16 – 30
Severe	> 30

Traditionally, the AHI is determined following a full sleep study carried out in an attended sleep laboratory (polysomnography). However, partial studies conducted by appropriately trained sleep technicians in the patient's home can accurately diagnose obstructive sleep apnoea in the majority of patients.

How common is obstructive sleep apnoea in New Zealand?

The prevalence of OSAS in New Zealand is reported to be 4% in adult males and 2% in adult females.[1] However, rates are elevated among Māori and Pacific peoples. Obstructive sleep apnoea is twice as common in Māori males compared to non-Māori males.[3] Māori and Pacific peoples also tend to present with more severe forms of OSAS and increased co-morbidities.[3] Higher rates of obesity among Māori and Pacific peoples is thought to be the principle reason for the increased prevalence of OSAS in these ethnic groups.[4]

Obesity is a major risk factor for obstructive sleep apnoea

Between 40 – 90% of people with OSAS are obese.[2, 5] Obesity increases the risk of OSAS because excess fat tissue around the neck exerts pressure on the upper airways, increasing the likelihood of upper airway collapse occurring during sleep.[3] Abdominal obesity has also been shown to reduce lung volumes, which can further increase the risk of upper airway collapse.[6] It has been estimated that a 1 kg/m^2 increase in BMI in a person who is obese, results in a 30% increase in the relative risk of clinically significant sleep apnoea occurring in the next four years.[7] Sleep loss caused by OSAS is also likely to further contribute to obesity.

Smoking is also associated with an increased prevalence of OSAS, and **alcohol** use can increase sleep apnoea duration, possibly by reducing muscle tone.[2]

The incidence of OSAS is increased in people with **hypothyroidism** and females with **polycystic ovary syndrome**. The severity of untreated sleep apnoea may be worsened in males using **testosterone supplementation**.

Severe obstructive sleep apnoea increases mortality risk

Moderate to severe obstructive sleep apnoea is independently associated with an increased risk of all-cause mortality. A study of more than 77 000 patients found that increasing OSAS severity was associated with increasing all-cause mortality in patients aged younger than 50 years, after adjustments were made for BMI and age.[8] This elevated risk has been estimated to be equivalent to an increase in age of 17.5 years or a 29 mmHg increase in mean arterial blood pressure.[9]

Obstructive sleep apnoea is associated with increased cardiovascular risk. A predominant feature of OSAS is chronic, intermittent hypoxia, which is associated with the development of hypertension and hypertensive cardiomyopathy. Co-existing coronary artery disease, diabetes and obesity add to this risk.[10] A large study found that, following adjustment for known risk factors, e.g. BMI, people with mild and moderate obstructive sleep apnoea had an approximately two and three-fold respectively, increased risk of hypertension compared to people without sleep apnoea.[11] Treatment of OSAS in people with severe disease has been reported to result in a reduction in arterial blood pressure of approximately 4 mmHg, and may improve some cardiac dysfunction, although further trials are needed.[10, 12] Insulin resistance and abnormal lipid metabolism have also been independently associated with obstructive sleep apnoea.[5] Both of these factors are likely to further increase the cardiovascular risk of people with OSAS.[5]

Motor vehicle and occupational accidents are increased in people with OSAS due to impaired cognitive function caused by disrupted sleep. A study of over 900 adults undergoing sleep assessment in the United States found that males with mild sleep apnoea had a four-fold increased risk of having a motor vehicle accident compared to people without a sleep-breathing disorder.[13] A small study of 40 injured drivers admitted to the Wellington Hospital Emergency Department (mean age 44 years) found that over one-third had obstructive sleep apnoea.[14] The rate of traffic accidents involving people with OSAS has been reported to be significantly reduced after treatment for OSAS.[12]

Clinical features of obstructive sleep apnoea

The clinical features of OSAS can be divided into symptoms that occur when the patient is either awake or asleep (Table 2).

Table 2: Symptoms of OSAS

Awake	During sleep
Excessive day time sleepiness	Snoring
Lack of concentration	Witnessed apnoeas
Cognitive deficits	Non-refreshing sleep
Changes in mood	Choking
Morning headaches	Restlessness
Dry mouth	Vivid dreams
Decreased libido or impotence	Gastroesophageal reflux
	Insomnia and frequent awakenings
	Nocturia
	Hypersalivation
	Diaphoresis (sweating)

Generally all people with obstructive sleep apnoea snore.[2] But not all people who snore have sleep apnoea (see "Snoring alone is not a good predictor").

Excessive daytime sleepiness and witnessed apnoeas are the symptoms most suggestive of obstructive sleep apnoea. Partners who have witnessed apnoeas report a sudden halt to snoring followed by a loud snort and resumption of snoring.[2] Sleep apnoeas are more commonly reported by the partners of males.[2] Females with obstructive sleep apnoea often report constant fatigue and lack of energy.[2]

Assessment tools for suspected obstructive sleep apnoea

The Epworth Sleepiness Score (ESS) is a subjective assessment of daytime sleepiness. It is recommended that this score is calculated prior to referral for sleep testing. Patients are asked to assign a numerical value to each of the following situations to indicate the likelihood that they might fall asleep:

1. Sitting and reading
2. Watching television
3. Sitting in a public place, e.g. a theatre or meeting
4. As a passenger in a car for an hour
5. Lying down to rest in the afternoon
6. Sitting while conversing
7. Sitting after lunch (without alcohol)
8. As a passenger in a car stopped in traffic for a few minutes

Score: 0 = would never sleep, 1 = a slight chance of sleeping, 2 = a moderate chance of sleeping, 3 = a high chance of sleeping. A total score greater than ten is considered abnormal and a score greater than 16 indicates pathological daytime sleepiness.

The British Lung Foundation has an online version of the Epworth Sleepiness Score available at: **www.blf.org.uk/Page/Obstructive-Sleep-Apnoea**

The Mallampati score can be used to assess the degree of airway obstruction. Decreased upper airway size is associated with increased sleep apnoea prevalence and is more likely in people who are obese or who have anatomical abnormalities such as a large tongue, or enlarged tonsils, airway walls or soft tissue.[5] To determine the Mallampati score, ask the patient to protrude their tongue, assess the visibility of the tonsils, uvula and soft palate and classify according to the degree of obstruction (Figure 1). A Mallampati score of class III or IV is useful for predicting obstructive sleep apnoea severity due to soft palate and tongue abnormalities.[17]

Snoring alone is not a good predictor of obstructive sleep apnoea

Snoring is caused by the vibration of the soft palate and throat during inspiration. It affects between 20 – 60% of the adult population, and is a frequent cause of relationship strain.[15] However, because snoring is so common among otherwise healthy people, it is not a good predictor of OSAS. Snoring may also be associated with obesity, nasal congestion, craniofacial abnormalities, hypothyroidism, acromegaly and soft tissue hypertrophy within the palate. Severe snoring has been linked to hypertension, cardiovascular disease and cerebrovascular disease. However, it is not known if snoring itself is an independent risk factor for cardiovascular disease.

Snoring can often be reduced with lifestyle interventions, such as weight reduction (if overweight), avoiding alcohol, smoking and avoiding sleeping on the back. If lifestyle measures such as these do not result in reduced snoring, oral appliances such as a mandibular splint or assessment for surgical tightening of the soft palate may be considered.[16]

Excessive daytime sleepiness can be a symptom of other disorders

Simple causes should be considered first when encountering a patient with excessive daytime sleepiness. Lifestyle factors in the patient's history should be explored to uncover insufficient sleep, primary insomnia and secondary causes of insomnia, such as depression or anxiety. Circadian rhythm disorders, e.g. jet lag, shift work or delayed sleep phase syndrome, and sedating medicines should also be excluded.

Chronic conditions such as cardiac, respiratory or neuromuscular diseases can cause fatigue, hypoventilation and daytime sleepiness. Carbon dioxide retention due to hypoventilation associated with severe obesity, central hypoventilation syndrome, or chronic obstructive pulmonary disease can result in increased daytime sleepiness, which may or may not be accompanied by breathlessness.

Unusual causes of excessive daytime sleepiness are considered last and are usually suggested by specific clinical features. Endocrine disorders, e.g. hypothyroidism, adrenal insufficiency or diabetic ketoacidosis, and neurological causes of drowsiness, e.g. subdural haematoma, encephalitis or intracranial neoplasm, may be a consideration, especially when the presentation does not fit well with OSAS.

OSAS frequently occurs in conjunction with other sleep disorders such as periodic limb movement of sleep (including restless leg syndrome), primary insomnia and narcolepsy.

For further information see: "Sleep disturbances: managing parasomnias in general practice" Page 16.

Sleep apnoea prediction tools

There are several tools available to stratify the likelihood of a patient having severe obstructive sleep apnoea. However, these should be used as a guide only, as they do not provide sufficient information to make definitive decisions about referral or treatment urgency for individual patients.

The adjusted neck circumference calculation (Table 3) can be used to assess the probability of a patient having obstructive sleep apnoea, but it does not categorise potential severity.

The OSA50 screening questionnaire combined with overnight pulse oximetry can be used in patients in a primary care setting to rule out moderate to severe obstructive sleep apnoea. This tool relies on access to an electronic pulse oximeter with data recording functionality (or referral to a private sleep clinic). The process involves asking the patient specific questions (Table 4), and for patients who score five or more, arranging overnight pulse oximetry (equivalent to a level IV sleep study – see "Sleep studies"). Patients who experience 16 or more occurrences per hour of a 3% drop in oxygen saturation, are suspected to have moderate to severe sleep apnoea.[19] The positive predictive value for the OSA50 tool (questionnaire and oximetry) is 55 – 65%, i.e. the patient has moderate to severe sleep apnoea, and the negative predictive value is 97 – 99%, i.e. they have mild sleep apnoea or no sleep apnoea.[19] The greatest value of this approach is therefore in ruling out moderate to severe obstructive sleep apnoea.[19]

Refer patients with suspected obstructive sleep apnoea for a sleep study

For most patients, an overnight sleep study (see "Sleep studies") is required to confirm a diagnosis of obstructive sleep apnoea. In New Zealand, there is an absence of nationally agreed referral criteria for this service and availability is the limiting factor in determining when a patient receives a publicly funded sleep assessment.

For information about availability of sleep clinic services, contact your local DHB.

Patients at high-risk should be promptly referred for testing, e.g. sleepy drivers with a history of a sleep-related accident or near-miss, occupational drivers or machine operators. Sleepiness should be assessed using the Epworth Sleepiness Score (Page 9). Co-morbidities such as hypertension, cardiovascular disease and diabetes should also influence the urgency of referral.

Patients who do not report daytime sleepiness may not benefit from referral as treatment is aimed primarily at

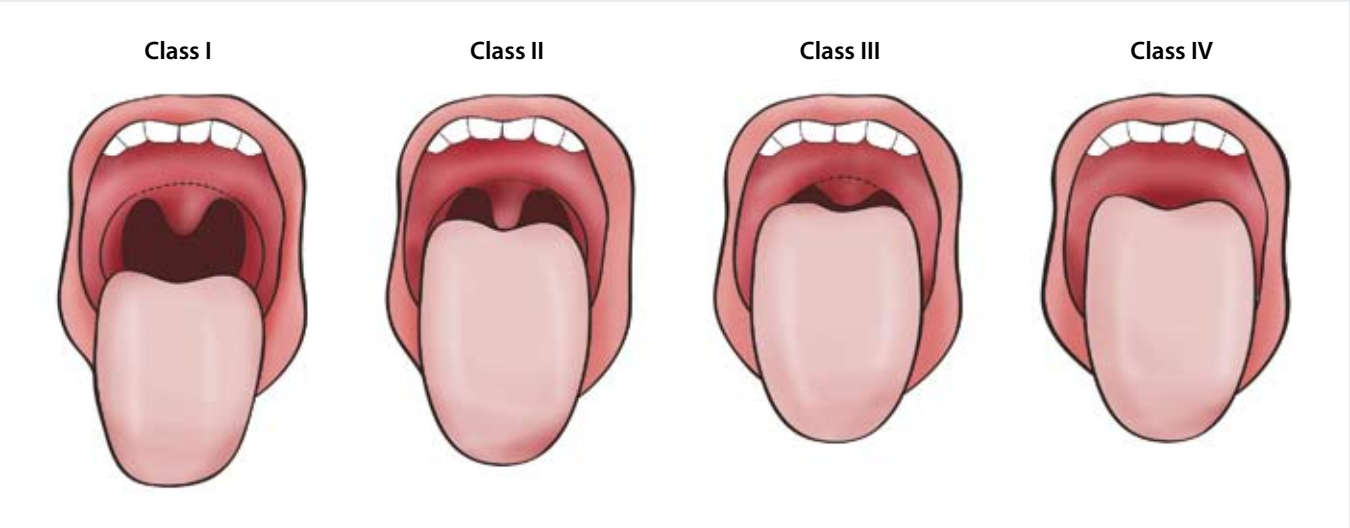


Figure 1: The Mallampati classification for airway obstruction

Table 3: Adjusted neck circumference calculation to assess probability of sleep apnoea, adapted from Skjodt, 2008[18]

Measure	+	Add	=	Adjusted neck circumference*
Neck circumference in cm		■ 3 cm for snoring history ■ 3 cm for history of witnessed apnoeas ■ 4 cm for history of hypertension		■ < 43 cm is low risk ■ 43 – 47.9 cm is intermediate risk ■ ≥ 48 cm is high risk

* Low risk equals a 17% probability of sleep apnoea. High risk equals 81% probability of sleep apnoea.

Table 4: OSA50 screening questionnaire for moderate to severe obstructive sleep apnoea[19]

Criteria		If yes, SCORE
Obesity:	Waist circumference* – Males >102cm or Females >88cm	3
Snoring:	Has your snoring ever bothered other people?	3
Apnoeas:	Has anyone noticed that you stop breathing during your sleep?	2
50:	Are you aged 50 years or over?	2
	TOTAL SCORE	/10 points
	If score = ≥ 5, proceed to overnight pulse oximetry	

* Waist circumference to be measured at the level of the umbilicus

Sleep studies

Level I studies, referred to as polysomanography, are performed in a sleep laboratory with a technician present and are the diagnostic gold standard. These studies include monitoring and recording of EEG (electroencephalography), EOG (electro-oculography), EMG (electromyography), heart rate, blood oxygenation, nasal flow, thoracic and abdominal movement, limb movements and body position.

Level II studies are unattended sleep studies recording the same parameters as Level I studies.

Level III studies record airflow, respiratory effort, blood oxygenation and heart rate. This level should be regarded as the minimum standard when diagnosing patients with obstructive sleep apnoea.

Level IV studies record two parameters, typically blood oxygenation and heart rate. Some level IV studies also record nasal flow. This level of sleep study should be used with caution as oximetry only detects apnoea-hyponoea episodes leading to oxygen desaturation and are not sensitive enough to detect some instances of obstructive sleep apnoea, e.g. mild sleep apnoea. For this reason a negative result will usually require a higher level study if obstructed sleep apnoea continues to be suspected.

improving daytime symptoms. Treatment of OSAS is usually with continuous positive airway pressure (CPAP), which is often poorly tolerated by people without significant daytime sleepiness.[12] Daytime sleepiness due to insufficient sleep should be excluded prior to referral (see: "Consider the presence of other sleep disorders", Page 10).

Additional referral information that is likely to be required to receive a publicly funded sleep assessment includes:

- Patient history, e.g. snoring, witnessed apnoeas
- Details of any previous otolaryngological assessments or procedures
- Co-morbidities, including cardiovascular, metabolic and psychiatric
- Social circumstances, including any current psychosocial stressors
- Occupational risk, especially if sleepiness is threatening the patient's employment
- Current medicines
- BMI and history of any recent weight change

Private sleep clinics and/or home-based partial sleep studies are an alternative to referral to specialist sleep laboratories where there are significant service delays. In New Zealand some private clinics are resourced by respiratory physicians. However, considerable debate exists about the validity of portable testing for the diagnosis of OSAS, compared with testing in dedicated sleep clinics. The diagnostic value of private studies is dependent on the variables that are measured. The 2007 American Academy of Sleep Medicine guidelines recommend level III sleep studies (see "Sleep studies") as a diagnostic minimum.[20] It is important for clinicians referring to private services to be aware of the level of sleep study used. Interpretation of the results from home-based partial sleep studies should be undertaken with the full clinical context of the individual patient.

Suggested contraindications to unattended home diagnostic sleep studies include:[18]

- Congestive heart failure
- Stroke
- Cor pulmonale
- Chronic obstructive pulmonary disease
- Hypoventilation
- Other serious medical disorders, e.g. seizures, psychosis, valvular heart disease, asthma, kidney or liver failure

Treatment for obstructive sleep apnoea

Lifestyle interventions are the simplest approach to reducing OSAS severity and improving other health parameters. Studies have shown that weight loss in patients with mild, moderate and severe OSAS results in improvements in OSAS symptoms.[21] A weight loss programme and a healthy lifestyle alone may provide clinical benefit for patients with mild OSAS. In patients with more severe OSAS, life style interventions should be used in conjunction with CPAP or other interventions.

All patients with OSAS who smoke should be offered smoking cessation advice in the ABC format (Ask, Brief advice, Cessation support).

Avoidance of medicines or drugs that may contribute to OSAS. There is no conclusive evidence that alcohol, benzodiazepines or opioids exacerbate OSAS. However, in theory, these substances could relax upper airway dilatory muscles, reduce ventilator drive or diminish the likelihood of arousing from an apnoea. If required, opioids and benzodiazepines should be prescribed at the lowest effective dose. Alcohol should be avoided, especially in the few hours prior to bedtime.

Continuous positive airway pressure (CPAP)

CPAP treatment is reported to be 100% effective at eliminating obstructive sleep apnoea, if tolerated. It is recommended as first-line treatment for people with moderate or severe OSAS.[12] A meta-analysis of 23 randomised controlled trials found that CPAP significantly reduced daytime sleepiness in patients with moderate or severe OSAS.[12] CPAP treatment in people with OSAS has also been found to improve cognitive function, reduce arterial blood pressure and reduce the rate of motor vehicle accidents by up to 83%.[12, 22] Some studies have demonstrated improved glycaemic control in patients following CPAP initiation.[23] However, CPAP treatment is not associated with weight loss.[21]

Generally, CPAP treatment is not considered to be appropriate for people with milder OSAS without significant symptoms, as the inconvenience of the device is thought to outweigh the benefits, resulting in poor adherence to treatment.[12]

CPAP prevents upper airway collapse by delivering positive air pressure through a mask which is worn during sleep. There are two forms of CPAP; fixed CPAP delivers constant pressure throughout the night, and auto-titrating CPAP devices adjust the pressure delivery as the patient breathes. Auto-titrating devices can be used to monitor treatment effectiveness and titrate CPAP pressure in patients commencing treatment. Both forms of CPAP have been shown to effectively control OSAS. Patient preference and economic considerations often determine which form of CPAP is provided.

Correct mask selection and treatment adherence are essential. Many patients find masks uncomfortable and may experience a sensation of claustrophobia. CPAP mask leakage can result in treatment failure. Patients should be encouraged to persevere with treatment for at least four weeks, as treatment acceptance often improves over time. Patients should also be encouraged to use the device throughout their sleep as increased treatment time is associated with increased benefit.[24] Patients may report difficulties with adherence due to nasal dryness or bleeding, throat irritation, skin irritation, local sweating, pressure intolerance or a poor mask fit.[12] It is essential that the operation of the device is understood, and any issues that may affect adherence are addressed. CPAP machines with built-in humidifiers and the use of silicon gels or plasters and facial shaving may alleviate some of these adverse effects. It has been reported that adherence to CPAP treatment among Māori with OSAS may be less than adherence among New Zealand Europeans.[25]

Mandibular advancement devices

Mandibular advancement devices can prevent sleep apnoeas by widening the upper airway and pushing the pharyngeal fat pads laterally and moving the tongue base muscles anteriorly. These may be a suitable treatment for patients with mild to moderate disease, or as a second-line treatment for patients who cannot tolerate CPAP. There are many types of mandibular device available. Adverse effects, including teeth and jaw pain, are likely to influence treatment adherence. A New Zealand study assessing the use of a mandibular advancement splint in 18 males and one female, with varying severities of obstructive sleep apnoea, found that treatment resulted in significant improvements in the apnoea-hypopnoea index and other indices associated with OSAS, including snoring volume.[26] However, the device was poorly tolerated with approximately one-quarter of participants reporting that adverse effects prevented regular use of the device.[26] In general, mandibular splints can only be fitted in patients with all or most of their own teeth.

A Cochrane review found that use of a mandibular advancement device improved subjective sleepiness and sleep disordered breathing in people with OSAS, compared to control.[27] Several studies have also reported significant decreases in blood pressure in the order of 2 – 3 mmHg following four to ten weeks of treatment with a mandibular advancement device.[28]

Obstructive sleep apnoea and driving risk

People with OSAS can drive without restriction if their condition is well-managed with satisfactory control of symptoms.[31] However, in some cases the New Zealand Transport Agency may impose licence conditions, with regular medical follow-up by the patient's General Practitioner.

People with OSAS should be advised not to drive if:[31]

- There is a high level of concern regarding sleepiness when driving while awaiting diagnosis from a sleep study
- They have a history of sleep-related motor vehicle accidents and report daytime sleepiness
- They have a diagnosis of severe OSAS which is either untreated, or they are unwilling to accept treatment

Treatment with a mandibular advancement device appears to be more successful in people with milder forms of OSAS, females, younger or leaner people, or people with supine-dependent sleep apnoea.[28]

Mandibular advancement devices can be purchased from some community pharmacies and individually adjusted by a dentist, or custom-made by dentists or orthodontists. Custom-fitted devices are often more effective and better tolerated than more generic devices. If the degree of advancement is too small then the device will not be effective, and if it is too large, adverse effects are increased.

Tongue-retaining devices are designed to widen the upper airway by pulling the tongue forward by suction. There is evidence that these devices may be as effective as mandibular advancement devices if worn, however, they appear to be poorly tolerated by patients.[29]

Pharmacological treatment of obstructive sleep apnoea

The use of medicines for the treatment of obstructive sleep apnoea is controversial because they can mask diagnostically useful symptoms and reduce adherence to CPAP treatment.[30] Modafinil, a respiratory stimulant, is licensed in New Zealand for the treatment of obstructive sleep apnoea, although it is only subsidised under Special Authority for the treatment of narcolepsy. Modafinil may be considered in limited circumstances only, and usually after consultation with a respiratory physician.

Surgical treatment of obstructive sleep apnoea

Surgery may be appropriate for the treatment of OSAS in patients who have clear upper airway anatomical abnormalities, such as enlarged tonsils. Other patients are less likely to benefit from upper airway surgery, especially if obesity or other co-morbidities are present.

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