Diagnosis and management of obstructive sleep apnoea

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Introduction

Obstructive sleep apnoea (OSA) is the most common sleep-related breathing disorder and the most important sleep disorder seen in both primary and secondary care. It is characterised by recurrent partial (=hypopnoea) and complete (=apnoea) upper airway obstruction during sleep. In severe cases apnoeic episodes may last up to 60 seconds or more, and can occur 50 or more times per hour overnight. Upper airway obstruction is typically associated with oxygen desaturation and accompanied by increasing respiratory effort leading to a brief arousal from sleep and re-opening of the upper airway. It is thought that the combination of sleep disruption and recurrent hypoxaemia is responsible for the important negative social and health consequences which are now known to be associated with OSA.

Epidemiology

OSA with associated daytime hypersomnolence (the OSA syndrome) affects 4% of men and 2% of women in the general middle-aged population.[1] Sleep-disordered breathing without symptoms is more common and affects up to 25% of men. In New Zealand, epidemiological studies have shown that OSA symptoms and risk factors are very common, affecting over 15% of the adult male population.[2] Maori men have approximately twice the prevalence of OSA as non-Maori, and Maori and Polynesian patients typically present with more severe disease and greater comorbidities than European patients. OSA is becoming increasingly recognised as a problem in children, where the prevalence may be as high as 2%.

Aetiology

Obesity is the most important risk factor for OSA, and is thought to account for up to 50% of the risk of OSA. In almost 60% of adults with at least moderate OSA it is attributable to excess weight. OSA is approximately twice as common in men and prevalence tends to increase with age. Other predisposing factors include craniofacial abnormalities such as retrognathia, neuromuscular disease and pregnancy. In children OSA is largely due to adenotonsillar hypertrophy but obesity is becoming increasingly recognised as a contributor in children as well as adults.

Pathophysiology

In the normal upper airway there is narrowing during sleep due to loss of muscle tone and reduced compensatory reflexes. In patients with a structurally narrow airway these normal changes in sleep result in greater loss of airway calibre and greater tendency to obstruction. In obese patients, excess tissue surrounding the upper airway results in greater external collapsing forces, which overwhelm normal upper airway dilator muscle activity and lead to partial or complete collapse. Once the airway becomes obstructed, there is increasing ventilatory effort which is thought to eventually trigger a brief arousal which serves to re-establish airway patency. During the obstructed period there is often a fall in arterial oxygen tension and a rise in systemic sympathetic activity with associated transient hypertension and tachycardia. Repetitive arousal from sleep leads to the excessive daytime sleepiness that is characteristic of OSA, as well as a generalised impairment of neurocognitive functioning. The physiological changes which accompany an apnoea and the subsequent arousal are thought to underlie some of the cardiovascular consequences now known to be associated with OSA.

Health consequences of OSA

The most common negative health consequence associated with OSA is

excessive daytime sleepiness. This is thought to result from a combination of frequent arousal, disrupted sleep architecture and recurrent hypoxaemia. However, it is now becoming increasingly recognised that OSA is linked to impairment across a spectrum of neurocognitive functions, including vigilance, executive functioning, and motor coordination.[3] It is thought that the combination of sleepiness and these functional abnormalities accounts for the increased risk of motor vehicle accidents (MVAs) in OSA sufferers. In one study from the US, men with moderate OSA were found to be 3.4 times as likely to be involved in at least one MVA, and 7.3 times more likely to have been involved in multiple accidents.[4] A recent study in New Zealand of drivers admitted to hospital following an MVA found a prevalence of OSA of almost 36%.[5] Current New Zealand Fitness to Drive Guidelines state that driving should be restricted or cease for individuals who are confirmed as or even suspected of having OSA where there is a high level of concern regarding the risk of excessive sleepiness while driving.

There is a large body of evidence now supporting the role of OSA as a risk factor for cardiovascular disease. Cross-sectional data suggests that patients with OSA have a two to threefold increased likelihood of hypertension and heart failure even when controlled for potential confounding factors such as obesity and smoking.[6] A recent long-term observational study found that the rates of both fatal and non-fatal cardiovascular events over a 10 year period were significantly higher in a group of men with OSA (16% and 30% respectively) compared with a group with treated OSA of similar severity (4% and 6%).[7] Patients with OSA have been shown to have a higher likelihood of resistant hypertension, severe heart failure and cardiac arrhythmias. OSA also seems to be linked to an increased risk of developing impaired glucose tolerance and established Type 2 diabetes, independent of obesity.

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Diagnosis of OSA

Most patients with OSA are reported to snore, and bed partners often describe apnoeic episodes. Patients may complain of waking overnight with a choking or gasping sensation, restless sleep, and waking unrefreshed, in addition to daytime sleepiness (Figure 1). Other symptoms may include nocturia, restless sleep and insomnia. Examination of the patient with suspected OSA should include an assessment of nasal patency (especially if CPAP is to be considered), inspection of the pharynx (tonsillar hypertrophy, retrognathia) and screening for complications such as hypertension and cardiac failure. Investigations which should be considered include blood glucose and thyroid function tests, as hypothyroidism can both mimic and cause OSA.

Most of the symptoms and examination findings in OSA are fairly non-specific, and diagnosis of OSA requires formal overnight testing. The gold standard for diagnosing OSA is overnight polysomnography (PSG, Level 1 Study), which measures a range of physiological parameters and is usually performed as an attended study in an inpatient sleep unit setting. PSG allows full sleep staging, assessment of arousal, and characterisation of respiratory events. The most commonly used polysomnographic measure of OSA severity is the apnoea-hypopnoea index (AHI), which gives an indication of the average number of obstructive events per hour. Definitions of severity vary, but generally an AHI <5 is considered normal, while an AHI >40 is considered to represent severe OSA.

Figure 1. Common Symptoms of OSA

Excessive daytime sleepiness
Snoring
Witnessed apnoeic episodes
Waking choking or gasping
Nocturia
Waking unrefreshed

Figure 2. Complications of CPAP therapy

Facial marks from mask
Nasal bridge discomfort
Nasal congestion
Dry nose
Dry or red eyes
Rhinitis
Facial acne under mask

In many countries, including New Zealand, PSG availability is generally restricted to main centres, with limited access for patients from remote areas. Furthermore, there is an inability to meet the demand for sleep studies even in major centres, with increasing pressure on waiting lists. As a result, there has been a move towards undertaking full (Level 2) or partial (Level 3) unattended sleep studies in the home environment. While these have the advantage of greater portability and better patient acceptance, they still require set-up and analysis by experienced sleep staff. Another diagnostic modality, which is being increasingly employed, is overnight oximetry (Level 4 Study), which has the advantages of low cost, greater patient acceptance and portability. While oximetry can rapidly identify severe disease, it has a relatively low sensi-

tivity and may miss up to 30% of cases of OSA,[8] especially in younger patients or where there is less pronounced desaturation. For this reason oximetry is insufficient as a stand-alone test for OSA, and should ideally be performed in conjunction with more detailed testing modalities.

Treatment of OSA

Before specific OSA treatment is commenced, there are a number of lifestyle modifications which may prove beneficial. Given that obesity is the main contributor to OSA, it stands to reason that weight loss will benefit patients with OSA. However, sustained weight loss is only achievable in a minority of patients. There is now evidence to support the role of bariatric (weight loss) surgery in the treatment of OSA but unfortunately this is not widely available in New Zealand. Other lifestyle modifications which may prove helpful include avoidance of precipitants to upper airway obstruction (such as alcohol and nocturnal sedatives), raising the head of the bed, and avoiding supine sleep, especially if there has been shown to be a strong supine influence on OSA.

The most widely accepted and effective treatment for OSA is CPAP (continuous positive airway pressure). CPAP delivers positive air pressure, which provides a pneumatic splint to the upper airway during both inspiration and expiration, maintaining patency and preventing obstruction during sleep. CPAP can be delivered via a number of different mask designs including nasal, nose and mouth ('full-face mask'), oral and nasal prongs/pillows. Common side effects of CPAP (Figure 2) include discomfort from the mask, which can be reduced with proper attention to mask fit, dry mouth which can be alleviated by addition of a humidification circuit, and nasal stuffiness/coryza, which usually responds to nasal corticosteroids or systemic antihistamines. Most studies demonstrate overall CPAP compliance rates of around 70%, which can be optimised by early patient education, attention to mask fit, and early and effective treatment of nasal symptoms.

CPAP has been shown to be effective in improving both the daytime sleepiness and the neurocognitive effects associated with OSA.[9] In addition, MVAs are significantly reduced once CPAP has been commenced,[10] although this is contingent upon satisfactory compliance and ongoing use – driving performance has been shown to deteriorate again even after one week off CPAP.[11] There is now a large body of evidence demonstrating the beneficial effects of CPAP on cardiovascular function. CPAP has been shown to reduce blood pressure by up to 10mmHg, although the size of this effect depends on both severity of OSA and whether hypertension is present in the treatment group.[12] CPAP has also been shown to improve left ventricular function by 30% in a group of patients with OSA and left ventricular failure already on optimal medical therapy.[13] CPAP has also been shown to increase insulin sensitivity in type II diabetes.

The effectiveness of CPAP is now well-established and this should be considered first line therapy, although satisfactory alternatives are available and should be considered if CPAP is not tolerated

Oral appliances such as a mandibular advancement splint (MAS) are most commonly used to treat snoring, but may also be an effective second line treatment when CPAP is not tolerated. While improvement in OSA severity indices is inferior to CPAP, compliance may be better, and there is now evidence that demonstrates a reduction in blood pressure in patients with OSA using a MAS.[14]

Key Points

- Obstructive sleep apnoea (OSA) is the most common sleep-related breathing disorder and the most important sleep disorder seen in both primary and secondary care.
- Obesity is the most important risk factor for OSA, and is thought to account for up to 50% of the risk of OSA.
- The most common negative health consequence associated with OSA is excessive daytime sleepiness.
- There is a large body of evidence now supporting the role of OSA as a risk factor for cardiovascular disease.
- Oximetry is insufficient as a stand-alone test for OSA, and should ideally be performed in conjunction with more detailed testing modalities.
- The most widely accepted and effective treatment for OSA is CPAP (continuous positive airway pressure).

Upper airway surgery also has a role in OSA management, particularly where there is a clearly defined anatomical abnormality.[15] Tonsillectomy and adenoidectomy is recommended in children with OSA where there is evidence of tonsillar or adenoid hypertrophy, or in selected adults if tonsillar enlargement is felt to be sufficient to cause significant upper airway compromise. Nasal surgery is mainly of benefit in improving nasal patency and improving CPAP tolerance, but it may provide some reduction in OSA severity. Surgery to the soft palate such as uvulopalatopharyngoplasty (UPPP) may provide some benefit, but cure of OSA is rare and response may be only seen in the short term. Much interest has recently focussed

on upper airway reconstruction, which involves a series of procedures aiming to modify both soft tissue and bony structures in the upper airway. Long-term data assessing the benefit of this approach are not currently available.

Summary

OSA is a common disorder, which has well-recognised social and medical consequences, including cardiovascular disease and increased risk of motor vehicle accidents. OSA is strongly associated with obesity, and prevalence is likely to increase as rates of obesity rise in New Zealand. Presenting symptoms are easy to recognise, although formal diagnosis requires an overnight sleep study. The effectiveness of CPAP is now well-established and this should be considered first line therapy, although satisfactory alternatives are available and should be considered if CPAP is not tolerated. Most of the medical and social complications associated with OSA can be either reduced or normalised with effective treatment.

Competing interests

Michael Hlavic declares that both Resmed and Fisher & Paykel (CPAP manufacturers) provided funding for a GP education and private practice promotion evening that he organised and spoke at in Christchurch earlier this year.

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Extreme obesity: a new medical crisis in the United States

'The prevalence of obesity has markedly increased in the past few decades, and this disorder is responsible for more health care expenditures than any other medical condition. The greater the body mass index (BMI) (calculated as weight in kilograms divided by the square of height in meters), the greater the risk of comorbidities, including diabetes mellitus, hypertension, obstructive sleep apnea, many cancers, dyslipidemia, cardiovascular disease, and overall mortality. Class III (extreme) obesity, defined as a BMI of 40 kg/m2 or greater, has also increased such that it now affects almost 1 in 20 Americans.'

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