

Obstructive sleep apnoea syndrome: underestimated and undertreated

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The obstructive sleep apnoea syndrome (OSAS) was first identified only 40 years ago and its clinical importance is increasingly recognized. Although now acknowledged as a worldwide problem, which in Western countries affects 2–4% of middle-aged men and 1–2% of middle-aged women, the majority of affected individuals remain undiagnosed. OSAS is strongly associated with obesity but is also increasingly identified in the less obese, in whom a particular craniofacial structure is an important contributory factor. The prevalence of OSAS is likely to be increasing in parallel with the epidemic of obesity currently occurring in many countries. The common presenting complaints are excessive daytime sleepiness and loud snoring. The sleepiness impairs social functioning, work performance and driving ability, and accounts for a large socio-economic burden on the community. Hypertension is an important independent association. The treatment of choice is nocturnal continuous positive airway pressure which is highly effective and is also cost effective.

Introduction

It is remarkable that a condition as common and disabling as the obstructive sleep apnoea syndrome (OSAS) has only come to the fore in the last 30 years. Before the 1960s there were occasional reports in the medical literature of cases which in retrospect were probably OSAS, but it was not until 1966 that obstructive sleep apnoea was clearly documented. The initial report¹ was in patients with the obesity hypoventilation syndrome (previously known as the Pickwickian syndrome). Such patients have chronic hypercapnic respiratory failure and most are extremely obese. With increasing use of sleep investigations (polysomnography) it subsequently became clear that OSAS was common and that, unlike patients with obesity–hypoventilation syndrome, the great majority of individuals with OSAS have normal blood gases by day, even though many have severely disabling symptoms. Recognition of the nature and size of the problem was driven initially by advances in technology, firstly

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the electroencephalogram (EEG), which was being applied in nocturnal monitoring of patients with neurological conditions such as epilepsy, and then the increasing availability of reliable oximeters for continuous recording of arterial oxygen saturation (SaO_2).

Gastaut *et al.*¹ described three types of apnoea: 'obstructive', in which air flow ceases but movement of the chest wall (rib cage and abdomen) persists, implying respiratory effort in the face of a closed upper airway; 'central', in which both flow and movement cease, apparently because of cessation of the drive to breathe; and 'mixed', a combination of the previous two patterns with features suggesting initially a central and then an obstructive event. It later became clear that mixed apnoeas are essentially obstructive, with the respiratory efforts undetected early in the apnoea.

Central apnoea syndromes are much less common than obstructive syndromes; central apnoea is seen in infants with an immature respiratory control system, while in adults it may occur with cerebrovascular or neuromuscular disease or, most commonly, accompanying Cheyne–Stokes breathing. The waxing and waning pattern of Cheyne–Stokes breathing tends to be exaggerated during sleep and central apnoeas may occur with each cycle. It is seen particularly in patients with advanced heart disease and a low cardiac output. The present review is confined to obstructive sleep apnoea and the obstructive sleep apnoea syndrome as seen in adults.

An apnoea is defined conventionally as cessation of breathing for >10 s but, like most other definitions in this field, this is purely arbitrary. It is important to distinguish between obstructive sleep apnoea (the physiological phenomenon) and the obstructive sleep apnoea syndrome (the phenomenon plus attributable symptoms). Epidemiological studies in the general population detect many subjects with periods of apnoea during sleep but no symptoms, and interpretation of the literature depends critically on whether the population is a community sample or is drawn from the highly selected symptomatic individuals who attend sleep clinics.

OSAS was first defined in terms of the apnoea index (AI), i.e. the average frequency of apnoeas per hour of sleep; one popular definition was an AI >5. Unfortunately, with the passage of time, it has become clear that such a simple arbitrary definition is a gross oversimplification and of little practical use. Firstly, many asymptomatic and apparently healthy subjects in the community have an AI >5. Secondly, it became apparent that the full-blown syndrome with all the typical symptoms could also be seen in patients with periodic hypopnoea rather than complete apnoea.² This led to the concept of the apnoea–hypopnoea index (AHI), a measurement which remains in general use. The distribution of AHI in the general population is continuous rather than bimodal and

it increases with age.³ Studies of patients attending sleep clinics have shown correlations between AHI and the severity of daytime sleepiness, but the relationship is not close.⁴ This is hardly surprising in light of the high prevalence of sleepiness in the population and the numerous other everyday causes (sleep deprivation, shift work, insomnia, etc.). Since both episodic sleep apnoea (or hypopnoea) and daytime sleepiness are common, some subjects will inevitably have both without necessarily a cause-and-effect relationship.

It is assumed that the most disabling symptom of OSAS, daytime sleepiness, is in some way related to impaired sleep quality, sleep deprivation or sleep disruption. Consequently, attention turned to whether sleep-related indices might explain daytime symptoms. Various studies have examined whether the EEG structure of sleep, frequency of awakenings or transient partial arousals relate better to daytime sleepiness, but the results have been disappointing and none has been shown to correlate better with sleepiness than the rather crude AHI. Assessment of sleepiness itself presents difficulties and is usually self-reported rather than objective (see below). Thus neither the consequences nor the proximate cause of OSAS are measurable with the desirable precision. However, such a situation is not unusual in medicine; a precise quantitative definition of asthma has eluded numerous experts over a much longer period than applies to sleep apnoea. In everyday pragmatic terms the critical question is whether a patient with sleep-disordered breathing has attributable symptoms which are likely to be improved by appropriate treatment, in particular by continuous positive airway pressure (CPAP). Again, this situation is not dissimilar to that pertaining elsewhere, for example in chronic airway disease [asthma or chronic obstructive pulmonary disease (COPD)], where therapeutic decisions often depend on an empirical trial of treatment. For practical purposes, OSAS is currently defined as evidence of sleep-disordered breathing plus relevant symptoms (most frequently excessive daytime sleepiness, but also others as described below).

Epidemiology

The best estimates of OSAS based on several studies suggest a prevalence in middle age of 2–4% of men and 1–2% of women.^{3,5} Even in countries where the condition is widely recognized, a large proportion of symptomatic individuals remain undiagnosed. For example, in the USA in 1997 it was estimated from a community survey of 4925 adults that as many as 82% of the men and 92% of the women likely to have moderate or severe OSAS had not yet been diagnosed.⁶ Although this proportion may have declined somewhat since then, it is likely that in other countries, where

the level of recognition is generally less than in the USA, the proportion undiagnosed remains very high.

The prevalence of OSAS in men is two to three times that in women. The reasons are not entirely clear, although hormonal factors clearly play a part. The prevalence is much lower in premenopausal women, and it is less in those postmenopausal women receiving hormone replacement therapy.⁷ Nevertheless, the prevalence remains lower in postmenopausal women than in men, even after controlling for age and body mass index (BMI). The gender difference may also be related to the more central distribution of body fat in men than in women; deposition of fat in the neck is a particularly important risk factor for narrowing and closure of the upper airway.⁸ Numerous studies have shown correlations between the prevalence of OSAS and obesity. Most epidemiological studies to date have been cross-sectional, but longitudinal data are starting to appear.⁹ These highlight the importance of weight gain, which is clearly associated with an increase in AHI (and weight loss is associated with a decrease), supporting the strong clinical impression that symptoms commonly appear in association with a recent increase in weight. This important relationship inevitably implies that the prevalence of OSAS is increasing in parallel with the epidemic of obesity currently occurring in many countries.

Awareness of the association between OSAS and obesity is now so strongly ingrained that it may actually inhibit recognition in the less obese. Most sleep clinics find initially that the great majority of patients referred are obese, but with increasing awareness the problem also becomes more apparent in the less obese. Only about 50% of patients diagnosed at a large sleep clinic in Edinburgh in recent years were obese.⁸

Data on the effects of age are rather confusing. In general population studies, the frequency of sleep-disordered breathing increases progressively with age, but the age distribution of patients diagnosed with OSAS generally peaks in the fifties and sixties with lower rates among older individuals.³ Snoring (a cardinal feature of OSAS) is also less commonly reported in the elderly.¹⁰ The cause(s) of this apparent paradox remains unclear: it may reflect reduced awareness by elderly people living alone, and there may also be a 'survivor effect' in view of the likely adverse consequences of untreated OSAS.

Most of the epidemiological and clinical studies have been performed in predominantly white populations in Europe, North America and Australasia. However, recent reports have extended the populations studied to non-whites in various parts of the world and have shown a similarly high prevalence of OSAS in several other countries,¹¹⁻¹⁴ although the dominant contributory factors vary. In individuals of Chinese¹³ and other Far Eastern¹² origin, BMI appears to be relatively

less important and variations in craniofacial bony structure more relevant. Consequently, in these countries many individuals have severe OSAS with relatively normal, or only mildly increased, body weight. Similarly, in Polynesian men in New Zealand, variations in the craniofacial skeleton have a major contributory effect¹¹ and may interact with obesity. In community studies in the USA the prevalence of OSA in the black population is at least as high as in the white population after controlling for obesity.³

Pathogenesis

The pharynx is a very compliant ('floppy') structure which responds readily to dilating and compressing forces. During inspiration, several small muscles (pharyngeal dilators) contract synchronously with the main inspiratory muscles with the effect of supporting the wall of the pharynx and countering the tendency to narrow which results from the subatmospheric intrapharyngeal pressure. During sleep the activity of these pharyngeal dilators (as all other muscles) is relatively suppressed, and consequently there is a greater tendency for the upper airway to narrow during inspiration. Why obstructive apnoeas develop in some individuals and not in all is determined largely by the initial size of the pharynx, with any structural features which reduce its static dimensions predisposing to more severe narrowing or closure during sleep.¹⁵ The most common of these contributory factors are obesity (with excess adipose tissue increasing the load against which the pharyngeal dilator muscles have to act), enlargement of the tonsils and the relative position of the upper and lower jaws. In relation to the latter, even a mild degree of retrognathia is associated with a more vertically oriented mandible and an intrinsically smaller pharynx. Combinations of these factors may interact in individuals, increasing the likelihood or severity of OSAS. Sometimes a familial tendency to develop sleep apnoea is recognized, probably due in part to heritability of the bony structure of the face.¹⁶

Narrowing or closure of the airway leads to transient reduction or cessation of ventilation with consequent hypoxaemia and partial arousal. The mechanism of arousal is not entirely clear but may relate to the exaggerated intrathoracic pressure swings which accompany the increased resistance to inspiratory air flow. Sometimes, patients awaken fully during an apnoea, in which case they experience a choking sensation which usually subsides within a few seconds. More often, however, full consciousness is not reached but repetitive partial arousal is associated with an abnormal electrophysiological sleep structure, in particular a reduction in the amount of 'slow-wave' or deep sleep. The consequences are akin to sleep deprivation, with daytime sleepiness, inattention, and

impaired concentration and memory. The combination of hypoxaemia and partial arousal with each apnoea or hypopnoea is accompanied by a surge of adrenergic activity with consequent tachycardia and transient systemic hypertension.

Clinical features

The common and less common symptoms of patients with OSAS are listed in Table 1. Most often the patient complains of troublesome daytime sleepiness, which interferes with social functioning, work performance, and driving.⁴ The severity of sleepiness is most easily assessed by the Epworth Sleepiness Score (ESS) (Fig. 1) based on a simple eight-point self-administered questionnaire in which the patient reports the likelihood of falling asleep in a number of everyday situations.¹⁷ Although relatively crude, the ESS (which has been translated into several languages) is usually easy to understand and complete, and it offers a very useful guide to the severity of symptoms and the response to treatment. More objective EEG-based indices of alertness, such as the Multiple Sleep Latency Test or Maintenance Of Wakefulness Test are very labour intensive and unsuitable for routine use in the large numbers of patients attending most sleep clinics. Physical examination of the patient with possible OSAS focuses particularly on visualization of the pharynx, including the uvula, tongue and tonsils, simple inspection of the shape of the bony structure of the face, looking particularly for any degree of retrognathia, and measurement of the blood pressure.

Investigations

In the early days of investigation for OSAS, virtually all patients underwent detailed in-hospital polysomnography, a technique which developed from neurological practice in investigation of patients with

Table 1 Symptoms of obstructive sleep apnoea

Typical	Snoring Unrefreshing sleep Daytime sleepiness
Frequent	Nocturnal choking Witnessed apnoeas Nocturia Morning headaches
Less common	Reduced libido Enuresis

THE EPWORTH SLEEPINESS SCALE

How likely are you to doze off or fall asleep in the following situations, in contrast to feeling just tired?
Use the following scale to choose the most APPROPRIATE NUMBER for each situation:-

- 0 = would NEVER doze
- 1 = SLIGHT chance of dozing
- 2 = MODERATE chance of dozing
- 3 = HIGH chance of dozing

<u>Situation</u>	<u>Chance of Dozing</u>
Sitting and reading
Watching TV
Sitting, inactive in a public place (eg a theatre or a meeting)
As a passenger in a car for an hour without a break
Lying down to rest in the afternoon when circumstances permit
Sitting and talking to someone
Sitting quietly after a lunch without alcohol
In a car, while stopped for a few minutes in the traffic

Fig. 1 The Epworth Sleepiness Scale. Each question is scored from 0 to 3 giving a maximum score of 24. Scores exceeding 10/24 are usually taken to imply excessive daytime sleepiness.

narcolepsy or nocturnal epilepsy. However, it has become obvious that this approach is both impracticable and unnecessary in most patients. At the other extreme, simple recording of overnight SaO_2 by a pulse oximeter allows recognition of the more severe cases, but suffers from a lack of both sensitivity and specificity.¹⁸ The high false-negative rate implies that oximetry cannot exclude the diagnosis.¹⁹

In most sleep clinics these days, patients are investigated using different forms of 'limited sleep study' in which four to six signals are recorded by a simple recorder at the bedside or attached to the patient. Several varieties of device are now available. They usually allow recording of SaO_2 , air-flow, and chest and abdominal movement, sometimes with the addition of heart rate and snoring but with no EEG recording. Increasingly, such techniques are being applied in the patient's home. With this type of equipment the sleep period itself is not recorded and the severity of sleep apnoea, quantitated as AHI, is related to the study time rather than the precise sleep time. In practice, however, this is adequate for clinical purposes in most patients.^{19,20} Overall, the technology employed for diagnosis of OSAS is less important than the experience of the interpreter of the results.

As explained above, a diagnosis of OSAS is made by finding sleep-disordered breathing (usually an AHI >10 or >15) in an individual with attributable symptoms, most often daytime sleepiness plus snoring.

Table 2 Classification of severity of obstructive sleep apnoea (American Academy of Sleep Medicine)

Sleep-related obstructive breathing events	
Mild	5–15 events/h of sleep
Moderate	15–30 events/h of sleep
Severe	>30 events/h of sleep

A severity grading has been proposed by the American Sleep Disorder Association,²¹ but the cut-off points are inevitably arbitrary (Table 2).

Complications and associations

With increasing interest in OSAS the number of recognized associations has increased considerably. These are listed in Table 3 in terms of bodily systems. With many of these, however, debate continues on whether they are truly independent associations or are due to confounding factors, especially obesity. Most interest has been in the associations with cardiovascular disease. This started with the recognition that systemic hypertension is common in patients with OSAS, but shared risk factors (particularly obesity) are also common and for many years it was unclear whether the association was independent. The transient surges

Table 3 Conditions associated with obstructive sleep apnoea

Neuropsychological		Excessive daytime sleepiness Impaired concentration, memory Headache Depression Nocturnal epilepsy
Cardiovascular:	Acute/nocturnal	tachycardia Episodic hypertension Dysrhythmias Heart block Angina
	Chronic	hypertension Ischaemic heart disease Congestive heart failure Stroke
Respiratory		Pulmonary hypertension Cor pulmonale
Metabolic		Diabetes 'Metabolic syndrome'
Genitourinary		Nocturia Enuresis Impotence
Haematological		Polycythaemia

of nocturnal blood pressure which accompany hypoxaemia and partial arousal were not a matter of dispute; the question was whether over a period of years this led to sustained systemic hypertension. Several large community studies have effectively answered the question and have convincingly shown an independent association.³ Furthermore, effective treatment of OSAS is accompanied by a reduction in blood pressure.

Interestingly, community studies which focus on the physiological phenomenon of apnoeas and hypopnoeas rather than necessarily on the syndrome as exhibited by symptomatic individuals, suggest a dose-response relation between AHI and the severity of hypertension, suggesting that the association may not necessarily require the presence of symptoms.³ The potential implications of these findings for the health of the community are considerable. In general, it appears that the association with hypertension is actually more marked in those with less 'classical' OSAS, i.e. it tends to be stronger in younger individuals and in the less obese, presumably because in older subjects and the more obese other factors become more dominant in the genesis of hypertension. OSAS is also unusually prevalent in subjects whose hypertension is particularly refractory to conventional drug treatment.

Several studies have evaluated the associations of OSAS with stroke and cardiac disease (both ischaemic heart disease and cardiac failure). Here, of course, there is the further 'confounder' of hypertension itself, as the association of OSAS with hypertension makes a relationship between OSAS and stroke or myocardial infarction almost inevitable. However, some studies, particularly in patients with stroke, suggest that additional factors may be involved. Several possible mechanisms have been proposed.²² This remains an area of active research and no definitive picture has yet emerged.

Of related interest is the association of OSAS with the 'metabolic syndrome', i.e. central obesity, glucose intolerance and insulin resistance. Again, common contributory factors are relevant, but some data suggest that insulin resistance is disproportionately common in patients with OSAS, even after taking account of obesity,²³ and possibly mediated by increased adrenergic activation.

For a condition where the prime abnormality is cessation of breathing, it is perhaps surprising that daytime respiratory features are relatively uncommon. A minority of patients with OSAS develop pulmonary hypertension and this increases with BMI. Likewise, a small proportion have diurnal respiratory failure with persistent hypoxaemia and hypercapnia. These are the individuals who formerly would have been diagnosed as having the Pickwickian syndrome (now the obesity hypoventilation syndrome). They are often recognizable during nocturnal studies by a low baseline SaO_2 , which, because of the shape of the haemoglobin-oxygen dissociation curve, leads to an exaggerated fall in SaO_2 with each apnoea or hypopnoea. The other situation where diurnal respiratory failure is

seen is in patients with coexistent chronic lung disease, particularly COPD. Here, the two conditions are synergistic in the genesis of the blood gas abnormalities and their consequences such as cor pulmonale. When patients with both COPD and OSAS were first recognized, the combination acquired the unfortunate name of the 'overlap syndrome'. While some early studies suggested that this association was greater than might be expected, more recent data from larger studies have shown that it is a chance occurrence,²⁴ but as both conditions are common, finding them together in one individual is not unusual. In summary, therefore, when patients with OSAS have daytime respiratory failure, severe pulmonary hypertension or evidence of cor pulmonale, they are likely to have coexistent extreme obesity or COPD (or both). The COPD may not be very severe and can go unrecognized unless evaluated by spirometry.

Neuropsychological complications of OSAS are common, particularly those related to sleepiness and its consequences, such as inattention and poor concentration. Some studies have suggested an association with depression, but this has not been shown conclusively. A history of epilepsy which is only nocturnal should raise the suspicion of OSAS (as should a history of purely nocturnal angina) and stimulate enquiry about relevant symptoms such as daytime sleepiness and snoring.

Several endocrine abnormalities have been described in patients with OSAS. These include a diminution of sexual function, which may be associated with low testosterone levels and can improve with treatment.²⁵ A common but under-recognized symptom of patients with untreated OSAS is nocturia, which in middle-aged and elderly men is often assumed to be due to prostatic problems. Occasionally the nocturia is so severe as to cause enuresis, particularly in younger individuals. Patients with untreated OSAS excrete large amounts of sodium and urine overnight, probably because of increased secretion of atrial natriuretic peptide (factor) mediated by stimulation of right atrial receptors exposed to the exaggerated intrathoracic pressure swings which accompany narrowing and obstruction of the upper airway. The natriuresis and polyuria are completely suppressed by effective treatment of OSAS.²⁶

Polycythaemia is seen occasionally, especially in individuals with a degree of diurnal hypoxaemia such as those with obesity-hypoventilation syndrome or coexistent COPD.

Socio-economic effects

The major impact of sleepiness in the community has been the subject of several reports in recent years. Of course, in many individuals the explanations are sociological rather than medical and depend on lifestyle

factors, shift work etc. However, OSAS is the most common 'medical' contributor, with various studies suggesting a prevalence 20 or more times greater than that of narcolepsy, with which it is sometimes confused.

It has been shown that patients with OSAS are a heavy burden on health care systems before the diagnosis is made, with greater average medical costs in the year before diagnosis than age- and sex-matched controls. Estimates in the USA suggest that untreated sleep apnoea may have additional medical costs of as much as \$3.4 billion per annum.²⁷ Sleepiness due to OSAS has also been shown to be associated with poor performance in the workplace.²⁸ There have been several reports showing a high risk of road traffic accidents in individuals with untreated OSAS, and epidemiological studies suggest a particularly high rate of OSAS in truck drivers. Controlled studies of treatment have shown improved performance on driving simulators.¹⁹ Licensing authorities in many countries require evidence that a patient is being treated effectively, although practice varies appreciably between countries.²⁹

Two disease-specific questionnaires have been developed for the evaluation of the effects of OSAS on health-related quality of life of individuals: the Sleep Apnoea Quality Of Life Index (SAQLI)³⁰ and the Functional Outcomes Of Sleep Questionnaire (FOSQ).³¹ Both have proved useful in clarifying how excessive sleepiness affects the subject's ability to perform his normal daily activities and the extent to which this is improved by treatment.

Treatment

In the early years after initial recognition of OSAS the only effective treatment available was tracheostomy. This completely bypasses the segment of the upper airway which narrows or obstructs during sleep. Tracheostomy is now used only very occasionally in patients with severe symptoms refractory to other forms of treatment, but has many obvious disadvantages. The treatment of OSAS was revolutionized by the introduction of nasal CPAP by Sullivan *et al.*³² The principle is very simple; by applying a positive pressure via the nose (sometimes via the nose and mouth) (Fig. 2) any tendency to narrowing and collapse of the airway can be overcome, the airway wall is stabilized and consequently snoring is suppressed, sleep quality is normalized and daytime symptoms are alleviated. The pressure required to overcome the hypopnoeas and apnoeas varies between individuals and, within an individual, may depend on several factors including posture, sleep stage and recent alcohol consumption. In most centres a simple 'titration' study is performed over a single night with the therapeutic pressure determined as that which overcomes 90% or 95% of sleep-related events. Some modern



Fig. 2 CPAP generator and three types of interface (demonstrated by a normal subject): nasal mask, oronasal mask and nasal 'pillows'.

CPAP machines respond rapidly to the state of the upper airway and adjust the pressure automatically (auto-CPAP). This may allow a slight reduction in the average nocturnal pressure and improve compliance in some individuals, particularly those requiring higher pressures,³³ but is not necessary for most patients.¹⁹

One major advantage of CPAP treatment of OSAS over the management of most chronic medical conditions treated with pharmacological agents is the availability of information on usage. This can be averaged over weeks or months to give a nightly figure for 'compliance'. Such information can be very revealing in evaluating the response to treatment as it is clear that many of those who fail to respond use the treatment for

a very limited period. However, the minimum period required to alleviate symptoms is not known and probably varies in a complex way between individuals and, indeed, within individuals. Many patients, especially those with more severe symptoms who therefore perceive the greatest benefit, use the treatment comfortably all night and every night (i.e. averaging 6 or 7 h per night). Others may derive benefit from more limited use, but most authorities question the value of treatment once it falls below an average of 2 h per night.¹⁹ Overall, about 70% of symptomatic patients use CPAP effectively and sufficiently to have a major impact on symptoms. There is no doubt that many thousands of lives have been transformed, marriages saved, jobs preserved and (probably) road accidents prevented by this treatment. The effects are frequently so dramatic that for some years no randomized controlled trials were performed, leading to some criticisms of the treatment and, indeed, doubts about whether OSAS really existed.³⁴ Unfortunately, such criticism did a great disservice to many patients as it was used by health commissioners as a reason not to acknowledge the magnitude of the problem or to fund it adequately. However, the criticism stimulated appropriate randomized controlled clinical trials, which have now shown quite clearly the great benefits of CPAP¹⁹ and the magnitude of the effect of treatment, which is much greater than that seen with the pharmacological treatment of many other chronic medical conditions. Unfortunately, however, the publicity associated with the initial criticism has coloured some attitudes which remain difficult to change.

Effective treatment with CPAP demands considerable time spent on educating the patients, encouraging them in its use, and dealing with problems of mask-fitting etc. Despite the best intentions of the patients concerned, a minority prove to be quite intolerant of this type of treatment. This may be due to claustrophobia associated with the mask, noise from the machine (much less than with earlier models) or, more commonly, side effects such as nasal stuffiness and drying of the nasal and buccal mucosa, particularly in patients in whom the mouth opens during sleep leading to a continual flow of air across the mucosa. The latter symptoms are usually improved by use of an oronasal mask and/or heated humidification of the air. Use of a nasal steroid also helps some patients, particularly those with pre-existent rhinitis.

Not surprisingly, many patients do not wish to be encumbered with an inconvenient and somewhat unaesthetic form of treatment which may be lifelong, and alternative forms of treatment have been explored. Various surgical procedures have been advocated, including uvulopalatopharyngoplasty and laser-assisted uvulopalatoplasty. All are inferior to CPAP in patients with symptomatic OSAS and are not currently recommended in this population.¹⁹ However, simple

tonsillectomy has a role in those with large tonsils and may sometimes completely relieve the syndrome, obviating the need for long-term CPAP treatment.

The second-line therapeutic approach is use of a mandibular advancement device, of which there are several varieties. The device is moulded to sit over the upper and lower teeth and fitted with the jaw partly protruded in order to maximize the size of the pharynx and thereby reduce the likelihood of its collapse during sleep. These devices have been shown to reduce the AHI, but less predictably than CPAP does. Their role is mainly in patients with mild to moderate sleep apnoea or those intolerant to CPAP.^{19,35} Simple nasal-dilating devices have no useful role in patients with OSAS. Supplementary oxygen at night as sole treatment is contraindicated as it may prolong apnoeas. However, in conjunction with CPAP, it has a useful role in the management of patients with chronic hypoxaemia caused, for example, by coexisting COPD or the obesity hypoventilation syndrome.

Weight reduction programmes are strongly advocated in patients who are overweight or frankly obese, but success rates in most clinics are depressingly low. Even when weight reduction is achieved it is often not sustained. More radical treatment by bariatric surgery is sometimes used in the severely obese, and it has been shown in case series that OSAS can resolve if considerable weight reduction is achieved.³⁶

Conflict of Interest Statement

Professor Gibson's department has received support for research and clinical service from Res Med (UK), one of the manufacturers of CPAP equipment.

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