

Key points

- ▶ OSAS, with a prevalence of 4% in adults, represents the most common chronic respiratory disorder after asthma.
- ▶ The condition is fundamentally caused by an inability of the UA-dilating muscles to withstand the collapsing forces generated during inspiration.
- ▶ Patients typically present with a triad of symptoms: snoring, witnessed apnoea and daytime sleepiness, in addition to other clinical features.
- ▶ The most common physical morbidity of OSAS is cardiovascular disease, and successful therapy improves these outcomes.
- ▶ While the gold-standard diagnosis is sleep laboratory-based polysomnography, increasing attention is being focused on limited cardiorespiratory studies, particularly in the home.
- ▶ Most patients with OSAS are best managed by nasal CPAP, particularly in moderate to severe cases, and the improvements achieved can be dramatic, particularly in daytime alertness.

Sleep apnoea syndrome

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Educational aims

- ▶ To provide a broad overview of OSAS, particularly from a clinical perspective.
- ▶ To review the basic mechanisms of the disorder and provide a brief introduction to some of the emerging concepts concerning molecular mechanisms of cardiac dysfunction in OSAS patients.
- ▶ To provide practical information for the clinician on the optimum approach to diagnosis and treatment of the disorder.

Summary

OSAS is characterised by recurring episodes of UA obstruction during sleep. Apnoeas are usually terminated by brief arousals, which result in marked sleep fragmentation, and contribute significantly to the symptoms of EDS and cognitive dysfunction. OSAS is also an independent risk factor for cardiovascular disease. The prevalence of OSAS is in the region of 2–4% of the adult population, and this high prevalence creates logistical difficulties in diagnosis.

This review aims to give a broad overview of OSAS for the clinician, whilst providing practical information concerning the diagnosis and treatment of the disease, including nasal CPAP for the management of moderate to mild OSAS, and weight loss, alcohol avoidance, relief of nasal congestion and keeping the patient off their back in milder cases. The possibility of using a mandibular-advancement device is also discussed.

Obstructive sleep apnoea/hypopnoea syndrome (OSAS) is a clinical syndrome marked by recurring episodes of upper airway (UA) obstruction that lead to markedly reduced (hypopnoea) or absent (apnoea) airflow at the nose/mouth. These episodes are usually accompanied by loud snoring and hypoxaemia, and are typically terminated by brief arousals, which result in marked sleep fragmentation and diminished amounts of slow-wave and rapid eye movement (REM) sleep [1]. Patients with OSAS are usually unaware of such arousals, but the resulting deterioration in sleep quality contributes significantly to the prominent symptom of

excessive daytime sleepiness (EDS). However, despite having significant breathing problems during sleep, most patients have no readily detectable respiratory abnormality while they are awake.

OSAS represents something of a paradox in clinical medicine. On the one hand, the clinical disorder has only been widely recognised in recent decades, although the sleeping characteristics of obstructive apnoea have been observed in the medical and classical literature for centuries [2]. On the other hand, OSAS is now recognised to be a very common clinical condition, with prevalence figures ranging up to at least 4%, depending on the diagnostic

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criteria used [3, 4]. Furthermore, there is increasing evidence that OSAS is associated with a considerable number of adverse sequelae, both behavioural and physical. Behavioural consequences include daytime sleepiness, impaired concentration and neuropsychological dysfunction, whereas physical consequences include cardiovascular disorders, particularly hypertension.

Physiology and pathophysiology

The stability and patency of the UA are largely dependent upon the action of oropharyngeal dilator and abductor muscles, which are normally activated in a rhythmic fashion coordinated with each inspiration. The pharynx is subjected to collapse when the force produced by these muscles, for a given cross-sectional area, is exceeded by the negative airway pressure generated by inspiratory activity of the diaphragm and intercostal muscles [5]. A narrowed UA is very common among sleep apnoea patients, which leads to increased pharyngeal resistance and the generation of more negative pharyngeal pressures during inspiration.

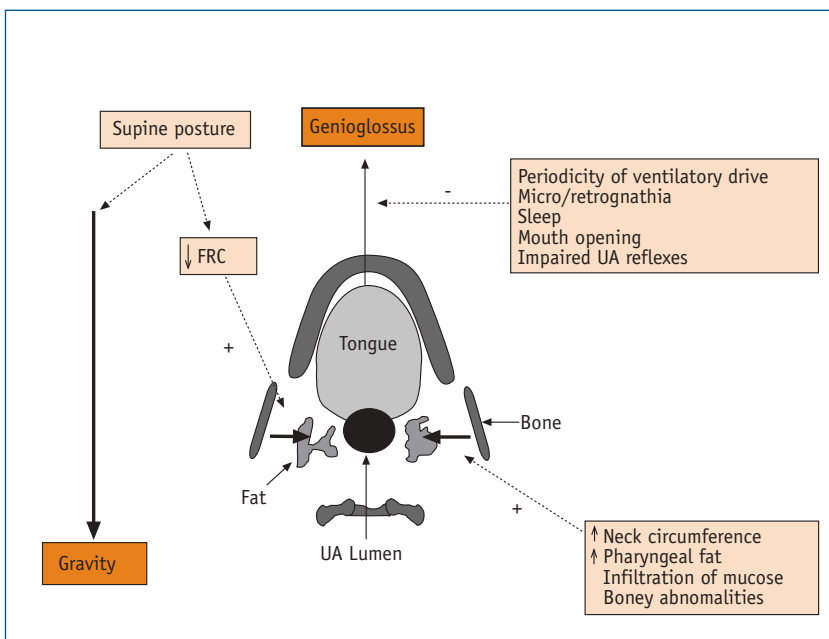
UA narrowing requires an increase in pharyngeal dilator muscle contraction to maintain airway patency, and there is evidence that patients with OSAS have more forceful contraction of these muscles during wakefulness than normal subjects, but show a larger decrement in

contraction during sleep, thus contributing to the development of obstructive apnoeas [6]. However, OSAS patients still show greater force of pharyngeal dilator muscle contraction during sleep than control subjects, which reinforces the fact that an imbalance between pharyngeal collapsing forces and dilator muscle contraction is responsible for obstruction, rather than a primary deficiency in muscle contraction. However, the sustained increase in dilator muscle contraction in OSAS may predispose to fatigue of these muscles, which might aggravate the tendency to pharyngeal occlusion. To date, this aspect of the pathophysiology of OSAS has been little studied.

The importance of negative intrapharyngeal pressure as a stimulus for dilator muscle contraction is reinforced by studies of the impact of nasal continuous positive airway pressure (CPAP) on pharyngeal muscle function. Nasal CPAP results in a marked decrement in both tonic and phasic contraction of the genioglossus muscle, an effect that can be blocked by topical anaesthesia of the pharynx, indicating that the effect is reflex in origin [7]. Local UA neuromuscular reflexes also play a significant role in maintaining pharyngeal patency and there is evidence of defects in these reflexes in patients with OSAS. In addition, interference with these reflexes by topical anaesthesia in normal subjects can result in obstructive apnoeas during sleep [5].

Figure 1 shows, in diagrammatic form, the main mechanical forces acting on the UA at tongue level in the supine position (FRC: functional residual capacity) and the factors influencing their relative contribution (+: enhancing effect; -: inhibitory effect). The tendency for gravity to cause dorsal movement of the tongue is counteracted by the genioglossus, which pulls the tongue forwards. Encroachment on the lateral walls of the UA (horizontal arrows) can result from bony abnormalities and/or an increase in soft tissue.

Figure 1
The main mechanical forces acting on the UA at tongue level in the supine position and the factors influencing their relative contribution.



Predisposing factors

Factors predisposing to OSAS are outlined in table 1. These factors represent anatomical or pathophysiological abnormalities that tilt the balance towards increased collapsibility of the UA. Anatomical factors generally result in UA narrowing, whereas pathophysiological factors generally impair the efficiency of UA dilating muscle contraction. For example, obesity, which is probably the most common predisposing factor, results in UA narrowing by increased fat deposition in the

pharyngeal walls and external compression by superficially located fat masses [8]. The supine position predisposes to OSA by gravitational forces that result in posterior displacement of the tongue. Ethanol ingestion is well known to increase the frequency and duration of apnoeas. This effect results from a combination of a reduction in UA-dilating muscle activity and a depressant effect on the reticular activating system, which impairs arousal [9, 10]. Similar effects have been reported with diazepam.

Definition

Early definitions of the syndrome were based on an apnoea frequency without reference to symptoms [1]. However, as epidemiological data made it clear that apnoeas during sleep are very common in the general population, the definition of a clinical syndrome was revised to include certain symptoms, particularly daytime sleepiness. Furthermore, it was recognised that hypopnoeas, which are events of shallow breathing causing oxygen desaturation, have similar clinical significance as apnoeas [11] and, subsequently, the disorder began to be referred to as the obstructive sleep apnoea/hypopnoea syndrome. A further development in the definition of the syndrome was the description of patients who presented clinical features of OSAS but who did not have obstructive apnoeas or hypopnoeas on polysomnography [12]. It was suggested that these events, characterised by increasing negative oesophageal pressure during inspiration and terminating with an arousal, reflected an UA resistance syndrome. Central sleep apnoea is a

much rarer form of sleep apnoea and may include particular forms of sleep apnoea, such as Cheyne-Stokes breathing and high-altitude periodic breathing [13].

An important development in the definition of various sleep apnoea syndromes was the report of a Working Group of the American Academy of Sleep Medicine, which laid out the clinical criteria necessary for the diagnosis of a clinically significant sleep apnoea/hypopnoea syndrome and also proposed a grading of severity [14]. The European Respiratory Society participated in this Working Group, including the present author. In the report, OSAS requires that the individual must fulfil criterion A or B, plus criterion C.

- A→ EDS that is not better explained by other factors.
- B→ Two or more of the following that are not better explained by other factors:
 - choking or gasping during sleep
 - recurrent awakenings from sleep
 - unrefreshing sleep
 - daytime fatigue
 - impaired concentration.
- C→ Overnight monitoring demonstrates five or more obstructed breathing events per hour during sleep; these events may include any combination of obstructive apnoeas, hypopnoeas or respiratory effort-related arousals.

Epidemiology

The prevalence of OSAS has been extensively studied over the past two decades, and has been variously estimated at between 1 and >6% of the

Table 1 Factors contributing to the development of OSA

General factors	Anthropometric (male sex, age, obesity) Drugs (ethanol, hypnotics) Genetics
Reduced UA calibre	Specific anatomical lesions (enlarged tonsils, micrognathia) Neck flexion Nasal obstruction
Mechanical factors	Supine posture Increased UA resistance Increased UA compliance
UA muscle function	Abnormal UA dilator muscle activity Impaired relationship of UA muscle and diaphragm contraction
UA reflexes	Impaired response to negative pressure Feedback from the lungs
Central factors	Reduced chemical drives Increased periodicity of central drive
Arousal	Impaired arousal responses Post-apnoeic hyperventilation leading to reduction in respiratory drive



adult population. One of the most comprehensive studies of OSAS prevalence to date is the Wisconsin cohort study, which studied employed males and females, aged 30–60 years, by means of full polysomnography [15]. While 9% of females and 24% of males were found to have an apnoea index (AI) >5 per hour, this estimate of prevalence fell to 2% of females and 4% of males when an AI >5 was combined with symptomatic daytime sleepiness. These findings underline the importance of not viewing OSAS in terms of AI or apnoea/hypopnoea index (AHI) alone. In a Spanish study, the prevalence of males having an AHI >5 was 15.3%, while 9.1% had an AHI >20. However, only 6.5% of the males met the minimal diagnostic criteria for OSAS with an AHI >5 combined with daytime sleepiness [4].

It has long been recognised that sleep apnoeas are very common in the elderly. However, the clinical significance of this finding remains unclear. While many of these subjects are otherwise asymptomatic for OSAS, there is evidence that sleep apnoea in the elderly has an adverse prognosis [16]. Children may develop a sleep apnoea syndrome similar to that seen in adults, and several epidemiological reports suggest a relatively high prevalence, although somewhat less than in adults. The aetiology of OSAS in children differs from adults in that adenotonsillar hypertrophy is the most common cause and can be cured by surgical removal in many cases.

Clinical features

When interviewing a patient with suspected OSAS, it is highly desirable to also interview the partner, who can usually provide important additional information based on direct observation of the patient while asleep. The clinical features of OSAS are given in table 2.

Table 2 Clinical features of sleep apnoea syndrome

Day	Night
Excessive sleepiness	Snoring
Impaired work/school performance	Witnessed apnoeas
Morning headache	Restless sleep
Intellectual deterioration	Insomnia
Impaired memory	Nocturnal choking
Impaired concentration	Recurrent arousals
Depressive symptoms	Nocturia
Heartburn	Enuresis (particularly children)
	Reduced libido/impotence
	Sweating
	Cardiac arrhythmias

Nocturnal symptoms

Snoring is almost universally present in patients with OSAS, because it reflects the basic pathophysiology underlying the disorder, namely narrowing of the UA. In population surveys, 25% of males and 15% of females are habitual snorers. The prevalence of snoring increases progressively with age: 60% of males and 40% of females aged 41–65 years habitually snore [17]. Thus, while most patients with OSAS snore, only a small proportion of habitual snorers have OSAS.

Witnessed apnoeas have been shown to be a good diagnostic predictor of OSAS, but do not help in predicting severity of the disorder.

Many patients with OSAS report waking with nocturnal choking or gasping sensations, which can be quite frightening and presumably reflect an episode of outright waking during an obstructive apnoea. The choking almost invariably passes within a few seconds of waking.

OSAS patients may experience insomnia. Patients rarely have difficulty falling asleep, although many report recurring awakenings during the night, which probably reflect the disturbing effect of recurring arousal on sleep.

Other nocturnal symptoms may be reported by patients or their bed partner, such as nocturia, enuresis, frequent arousals, diaphoresis and impotence [18].

Daytime symptoms

Although OSAS is the most common cause of EDS, it has not been found to be very useful as a clinical feature to discriminate between patients with and without the disorder. Many studies have found that severity of EDS and OSAS do not correlate, which may reflect the fact that many other sleep disorders also cause EDS.

OSAS is reported to be associated with many other daytime symptoms, such as fatigue, memory impairment, poor concentration, intellectual deterioration, personality changes, morning nausea, morning headaches, automatic behaviour and depression [18]. Although these features may be important in assessing the impact of OSAS on a patient and the effectiveness of therapy, there has been no systematic study of the capacity of these features to predict the presence or absence of OSAS.

Physical characteristics

Obesity is very common in OSAS, particularly upper body obesity, and there is evidence that patients with OSAS are particularly prone to having fat necks.

Craniofacial anatomical factors that predispose to UA narrowing should be sought in the physical examination of a patient suspected of having OSAS. These include retrognathia, micrognathia, tonsillar hypertrophy, macroglossia and inferior displacement of the hyoid [5]. However, the most common physical finding in patients with OSAS is a non-specific narrowing of the oropharyngeal airway, with or without an increase in soft tissue deposition.

A link between sleep apnoea and hypertension has been consistently demonstrated in many studies, and the finding of hypertension in a patient with symptoms suggestive of OSAS increases the likelihood of the disorder.

Combined features

In practice, the clinical suspicion of OSAS is based on a combination of supportive features, but it is generally not possible to predict the severity of the disorder based on clinical features, except at the extremes of the clinical spectrum. Thus, some form of objective monitoring during sleep is necessary to confirm and grade the severity of the disorder.

Diagnosis

The gold standard for the diagnosis of OSAS is full polysomnography, which provides detailed information on sleep state, respiratory and gas exchange abnormalities, in addition to a range of other variables, including body position, heart rate and rhythm, and muscle tone and contraction. An example of an obstructive apnoea is given in figure 2 (EEG: electroencephalogram; EOG: electrooculogram; ECG: electrocardiogram). Initially there is normal non-obstructed breathing

with ribcage and abdominal movements in phase. With the onset of apnoea in REM sleep (arrow), there is paradoxical indrawing of the ribcage, with absent tidal ventilation as indicated by the flat sum tracing. As the apnoea proceeds, there is increased abdominal effort culminating in an arousal. This coincides with relief of UA obstruction followed by post-apnoeic hyperventilation.

However, these studies are resource intensive, since they generally require the facilities of a full sleep laboratory and a trained technician. Thus, it is common to encounter long waiting lists for diagnostic sleep studies in sleep centres throughout the world. The high prevalence of OSAS makes it necessary to consider other simplified approaches to the diagnosis. Furthermore, the prevalence of OSAS is so great that the clinical assessment of these patients will probably involve clinicians outside major sleep centres who may not have as detailed an understanding of the syndrome as clinicians who have undertaken specific training in sleep medicine, and the availability of simplified limited diagnostic systems further increases this likelihood. It is important, therefore, that clear-cut guidelines and criteria be available for the assessment and management of patients with suspected OSAS.

The development of many limited diagnostic systems in recent years represents recognition of the logistical problems described previously. Unfortunately, there is no uniformity among these devices, and the only consistent variable common to these systems is oxygen saturation (S_{a,O_2}). Detailed S_{a,O_2} analysis is often sufficient to diagnose OSAS in severe cases because of the characteristic pattern of repetitive transient

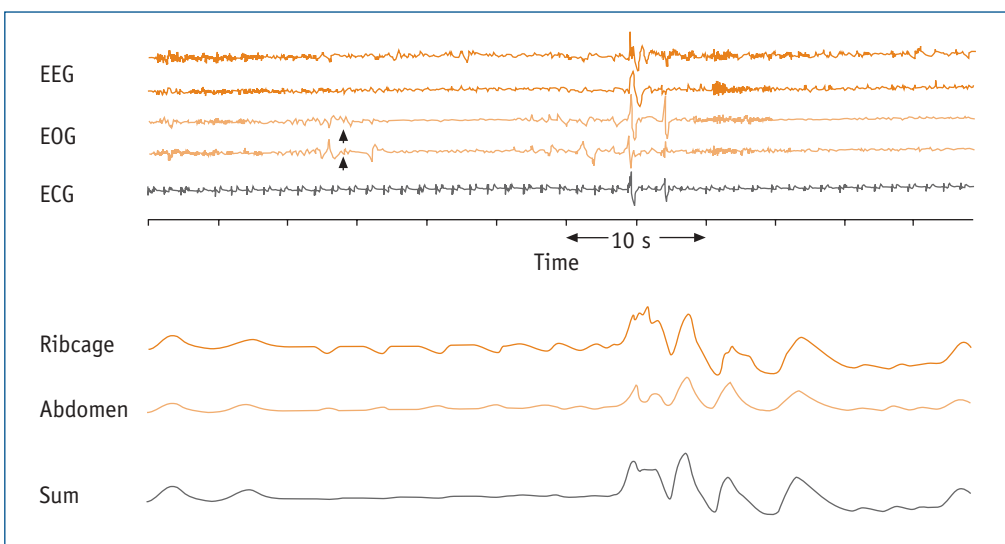
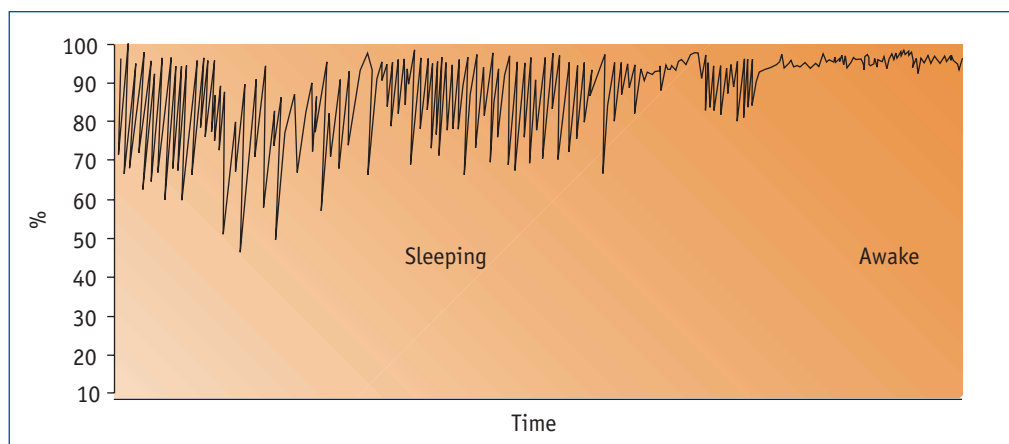


Figure 2
Polysomnographic record of an obstructive apnoea during REM sleep.

Figure 3
S_aO₂ tracing demonstrating the transition from wakefulness to sleep in a patient with OSAS.



desaturations that is unique to sleep apnoea, as can be seen in figure 3. However, more complete sleep studies are often necessary in patients with mild to moderate disease.

The very large numbers of patients presenting for assessment of possible OSAS have also focused attention on the role of home-based sleep studies. While there are obvious advantages of such studies (particularly improved sleep quality and cost-savings), there are also disadvantages, *i.e.* the lack of technician supervision, means that leads that become dislodged are not replaced during the study and, consequently, the likelihood of technically unsatisfactory studies is higher.

Morbidity and mortality

The morbidity of OSAS relates principally to the cardiovascular system. Compared with the general population, OSAS patients appear to have at least twice as much hypertension, ischaemic heart disease and cerebrovascular disease [19, 20]. However, this population of patients has a high incidence of other co-existing cardiovascular risk factors, such as obesity, hyperlipidaemia, increased age, male sex, smoking history and excessive alcohol intake, which makes the identification of a clear independent association of OSAS with cardiovascular disease more difficult [21]. Nonetheless, there is now convincing evidence from many studies of an independent association of OSAS with hypertension, and this association has been reinforced by recent studies demonstrating a reduction in blood pressure levels with nasal CPAP therapy [22, 23]. A growing, but not yet conclusive, body of evidence points to an independent link between OSAS and both ischaemic heart disease and stroke.

The mechanisms by which OSAS predispose

to cardiovascular disease are not fully understood, but probably include elevated sympathetic drive secondary to recurrent hypoxias and arousals from sleep [24], and increased oxidative stress secondary to recurring oxygen desaturation and resaturation [25]. There is emerging evidence that the intermittent hypoxia (IH) that is characteristic of OSA particularly predisposes to the selective activation of pro-inflammatory transcription factors, such as nuclear factor- κ B, with consequent increased production of proinflammatory cytokines, such as tumour necrosis factor- α , and interleukins-6 and -8. These cytokines have been linked to the pathogenesis of atherosclerosis and hypertension. However, IH is less effective than sustained hypoxia in activating the protective hypoxia inducible factor (HIF)-1-dependent pathway. HIF-1 activates transcription of genes encoding proteins that will either increase oxygen delivery (vascular endothelial growth factor, erythropoietin and endothelin-1) or achieve metabolic adaptations under conditions of reduced oxygen availability (glucose transporter-1 and glycolytic enzymes). These findings raise the possibility that the IH associated with OSAS may selectively activate molecular pathways that are pro-atherogenic.

Early studies evaluating the impact of treating OSAS on cardiovascular morbidity have suffered from sub-optimal design, being uncontrolled, not randomised or underpowered. However, recent randomised placebo-controlled trials have demonstrated clear objective benefits of CPAP therapy on blood pressure levels. Apart from hypertension, there are limited data to support a significant impact of treatment on cardiovascular morbidity, although there is emerging evidence of a reduction in death rate from cardiovascular disease among OSAS patients successfully treated with CPAP, including a recent report from the current author's unit [26].

Management

Management options for OSAS are outlined in table 3. The most widespread and effective treatment modality is CPAP, but milder cases of OSAS can often be managed by conservative measures. These include weight loss (where appropriate), sleep hygiene, alcohol avoidance, relief of nasal congestion and measures to keep the patient off their back (since most cases of sleep apnoea tend to be worse when the patient is supine). Only a minority of adult cases have a correctable anatomical lesion obstructing the UA, but the majority of children with OSAS have enlarged tonsils and adenoids, and surgical removal of these tissues can resolve the condition in many cases. The surgical technique uvulopalatopharyngoplasty (UPPP) can be effective in the management of simple snoring, but its role in the management of sleep apnoea is controversial, and most respiratory experts in this field regard UPPP as being contraindicated in anything more than mild cases.

Obesity

Approximately 60–70% of OSAS patients are obese, that is having a body mass index $>28 \text{ kg}\cdot\text{m}^{-2}$ or a body weight $>20\%$ above the ideal weight. Obesity is one of the most commonly recognised risk factors for OSAS, particularly upper body obesity. Weight loss has definite beneficial effects, and results in improvement, or occasionally disappearance, of sleep-related breathing disturbances in many patients. Weight loss is difficult to achieve and especially to maintain. However, weight loss should be encouraged in all obese OSAS patients. Weight loss also has beneficial effects on snoring, but not as consistently as with OSA.

Sleep posture

It has been long recognised that snoring patients do so most loudly in the supine position. Similarly, it has been demonstrated that a large proportion of unselected OSAS patients have a higher AHI in the supine compared with the prone or lateral positions. A tennis ball sewn into the pyjama top at the mid-thoracic level was one of the first means used to inhibit sleeping in the supine position and is based on the principle of inducing conditioned behaviour. Two other different strategies have been used, namely sleep-position training, using a posture alarm device, and a tongue-retaining device, in order to prevent tongue retrolapse when the patient sleeps in the

Association of OSAS with road traffic accidents

The link between OSAS and road traffic accidents has been recognised for over a decade. Various studies have demonstrated an increase in accident rate between 3- and 7-times that of the general population among untreated OSAS patients, which falls to normal levels after successful therapy with CPAP [27]. There is also evidence that occupations, such as long-haul truck driving, are particularly associated with a risk of sleepiness while driving and an increased risk of accident, particularly where there is evidence of associated OSAS. These findings may not be surprising given the relative sedentary and monotonous nature of this occupation, and the fact that long-haul truck drivers frequently drive for many hours at a time. These findings assume particular significance given the likelihood of a fatal accident where an articulated truck driven by a sleepy driver is involved. The risk of accident also calls into question the suitability of untreated OSAS patients to hold a driving licence, and several European countries have introduced regulations in this regard [27]. However, patients with OSAS who are successfully treated do not pose an accident risk.

supine position. The positional factor is likely to be more important in mild to moderate OSAS patients.

Table 3 Management options for OSAS

Moderate and severe

Nasal CPAP is the definite treatment of choice

Other therapies should be considered if CPAP fails or is not tolerated

Measures to advance the mandible can benefit some severe cases:

Oral appliances

Maxillomandibular advancement osteotomy and variations

Surgery to the UA, i.e. UPPP and radiofrequency ablation, should be a last resort

Mild and moderate OSAS

Nasal CPAP benefits patients with mild to moderate OSAS; however, compliance is poor

Weight loss improves OSAS, but is difficult to achieve

Sleep posture measures may benefit patients with predominantly supine OSA

Oral appliances are an alternative to CPAP in mild OSAS, and possibly UARS, although there are no reports of efficacy in UAR

Surgery can be considered in selected cases with mild OSAS, but is more suited to non-apnoeic snoring patients; the role of UPPP in patients with UARS is unclear

Various pharmacological agents have been evaluated in OSAS, with generally disappointing results

Suggested further reading

Breathing Disorders in Sleep. McNicholas WT, Phillipson EA, eds. London, WB Saunders, 2002.

This multi-authored text, written by internationally renowned experts in the field, is primarily directed towards the clinician and provides a comprehensive overview of sleep apnoea, in addition to the impact of sleep on other chronic respiratory disorders. The style is somewhat similar to Breathe with liberal use of Key point summaries in the text.

Public Health and Medicolegal Implications of Sleep Apnoea. Report of an ERS Task Force. Krieger J, McNicholas WT, Levy P, et al. *Eur Respir J* 2002; 20: 1594–1609.

This report particularly focuses on the impact of sleep apnoea on driving accidents and the approach taken by various European countries to issuing driving licenses to OSAS patients. This issue is likely to become increasingly important in future years for clinicians treating patients with OSAS.

Report of a Task Force of the American Academy of Sleep Medicine. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. *Sleep* 1999; 22: 667–689.

This report provides comprehensive information and recommendations concerning the clinical criteria required for diagnosis and optimum measurement techniques in sleep studies.

CPAP therapy

The current management of moderate to severe OSAS is largely dependent on nasal CPAP, which acts to splint the UA open during sleep, and, thus, counteracts the negative suction pressure during inspiration, which promotes UA collapse in these patients [28]. Nasal CPAP is highly effective and usually has a dramatic effect on the patient's awake performance because of the normalised sleep pattern. However, the device is cumbersome and compliance data is only moderately satisfactory. Compliance relates positively to severity of OSAS and level of daytime sleepiness. Thus, CPAP therapy is particularly appropriate for patients with moderate to severe levels of OSAS. There is evidence that compliance is improved by careful patient education and support. Partners of OSAS patients treated with CPAP also report a significant improvement in quality of life.

Oral appliances

The goal of therapy with oral appliances is to modify the position of UA structures in order to enlarge the airway, to reduce resistance and, presumably, to reduce UA collapsibility. The effects on UA muscle function may also be important, due to the changes in direction of muscle fibres. The effects on AHI have been widely studied, including several comparisons with nasal CPAP [29]. Overall, oral appliances have been found to be less effective on breathing abnormalities and sleep structure than nasal CPAP, but better accepted by the patients. Side-effects of treatment have also been reported. Excessive salivation and transient discomfort after awakening are commonly experienced with initial use and may hinder early acceptance of the device. Later complications include temporomandibular joint discomfort and changes in dental occlusion.

UPPP

The objectives of UPPP are to enlarge the oropharyngeal airway and to reduce the collapsibility of this particular segment of the UA. The results of the procedure have been summarised in a meta-analysis carried out by Sher *et al.* [30]. For many authors, the criteria for a good response are defined as a 50% drop in AI or AHI and the consequent achievement of an AI value of <10 or an AHI of <20. When this last criteria was taken into account, the response rate in the meta-analysis was only 41% (137 out of 337 patients). In OSAS patients, UPPP indications should be restricted to those with mild disease and evidence of retropalatal narrowing, and careful patient assessment and selection is very important. It should also be remembered that, by increasing mouth leaks, UPPP may compromise CPAP therapy and reduce the maximal level of pressure that can be tolerated.

Other management strategies

A number of other therapeutic strategies have been developed for selected patients with OSAS. These include sophisticated maxillo-facial surgical procedures designed to move the mandible forward and enlarge the UA. Radiofrequency ablation of the posterior tongue has also been proposed, in addition to electrical stimulation of the hypoglossal nerve. These techniques are either highly sophisticated and, thus, confined to highly specialised centres, or poorly validated and beyond the scope of the present review.

Conclusion

OSAS is a highly prevalent disorder that can have major adverse consequences on the health and quality of life of the affected patient. However, the disorder is eminently treatable, and successfully treated patients generally report a dramatic transformation in their quality of life.

Educational questions

1. What is the fundamental pathophysiological abnormality in OSA?
2. Is laboratory-based polysomnography required to confidently diagnose OSAS?
3. Some (cynical) observers have proposed that obese patients with OSAS should be managed by weight reduction alone. Do you agree with this comment?
4. Does effective therapy of OSAS with CPAP remove the recognised increase in driving accident risk and is it reasonable to allow such patients to continue driving?
5. What is the most common cardiovascular morbidity of OSAS?

Suggested answers

1. Increased pharyngeal resistance, as it has been shown that pharyngeal dilator muscle activity is increased in OSAS as a compensatory mechanism. While muscle tone falls in sleep, the actual levels remain at least as high as in normal subjects, but insufficient to withstand the high collapsing forces in the UA that develop as a consequence of increased pharyngeal resistance.
2. No. Simplified cardiorespiratory monitoring studies are sufficient to diagnose OSAS in moderate to severe cases and to exclude significant OSAS in non-apnoeic snorers. Polysomnography may be necessary in intermediate or atypical cases, or where limited studies are inconclusive.
3. This approach is inappropriate and equivalent to suggesting that obese type-2 diabetic patients should be managed by weight reduction without recourse to oral hypoglycaemic agents. While obesity plays an important role in the pathophysiology of OSAS, the disorder is more complex, and specific therapy is recognised to greatly improve alertness, cognitive function and also cardiovascular morbidity/mortality. Furthermore, untreated OSAS patients find it difficult to lose weight because of the behavioural effects of the disorder.
4. Untreated sleepy OSAS patients are at a substantially increased driving-accident risk and should be warned of this. However, this risk is largely normalised with successful therapy. There is considerable variation between European countries in regulations for the issuing and retention of a driving license to OSAS patients, but countries that do specify OSAS in their regulations also allow the return of a licence to a patient who is being successfully treated.
5. Hypertension. There is now very convincing evidence of a strong association between OSAS and hypertension that is independent of confounding factors such as obesity and hyperlipidaemia, and CPAP therapy lowers blood pressure in hypertensive OSAS patients. There is also good evidence from large-scale studies, such as the Sleep Heart Health Study in the USA, of an independent association with ischaemic heart disease, but probably not as strong an association as with hypertension. While OSAS is common in patients with stroke, the relationship is complex and not fully understood.

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