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Some degree of obstructive sleep apnoea (OSA) is a common finding in 5–20% of adult men, although only about one in five of these individuals will have associated daytime sleepiness (obstructive sleep apnoea syndrome (OSAS)). Much has been discovered about this condition since the publication of the first widely-read account of the syndrome in 1976. Improvements in our understanding of the pathophysiology of OSAS, and the effects of even minor upper airway obstruction on sleep architecture, have refined our approach to treatment which should now be based on the severity of daytime symptoms rather than on nocturnal respiratory disturbance. This article outlines current approaches to diagnosing and managing OSAS, including issues related to driving and cardiovascular disease. The evidence for the efficacy of nasal continuous positive airway pressure (CPAP) is presented, and other management approaches discussed, including dental appliances and surgery.

Definition

The original definitions of OSAS were specific, requiring an arbitrary number of respiratory events (apnoeas or hypopnoeas) per hour of sleep (apnoea index or apnoea/hypopnoea index) to confirm a diagnosis. It is now recognised that increases in upper airway resistance alone, without an apnoeic, hypopnoeic or even hypoxic event can cause recurrent arousal from sleep, so this index is no longer considered ideal either for defining sleep apnoea or for grading its severity. A clinical definition of OSAS is more appropriate: for example, significant daytime symptoms (eg sleepiness) in conjunction with evidence of sleep-related upper airway obstruction and sleep disturbance.

Diagnosis by sleep study

The aims of a sleep study are to:

- recognise upper airway obstruction
- identify whether this is responsible for substantial sleep disturbance
- exclude other conditions that may also cause daytime hypersomnolence.

Many different physiological signals can directly or indirectly provide this information, and these will not be discussed in detail in this article. No single signal can reliably produce all the necessary information and a ‘montage’
of several is needed. There is no evidence to suggest that any one collection of signals is optimal and complex polysomnographic sleep studies are not essential. Much attention is often paid to which is the ‘best’ system but, in reality, the training and experience of the interpreting physician are overwhelmingly more important.

**Daytime sleepiness**

One of the main symptoms of OSAS, and the main indication for treatment with nCPAP, is daytime sleepiness. A useful, verified instrument to estimate the degree of sleepiness, including the documentation of changes on therapy, is the Epworth Sleepiness Scale which asks patients to estimate the likelihood of falling asleep in a variety of different situations (Fig 1).

**Driving**

Sleepiness while driving is a major cause of motor accidents. Depending on the methodology used, the accident rate in patients with OSAS is 5–10 times normal. This accident rate falls once treatment has been commenced. Driving simulator data suggest that sleepiness due to OSAS is associated with a marked driving impairment comparable to that produced by a blood alcohol level above the legal limit for driving in the UK. This impairment was shown to respond to nCPAP therapy in a controlled study.

In the UK, the Driver and Vehicle Licensing Agency (DVLA) regulations state that driving must cease if the driver is excessively sleepy (from any cause). Group 1 drivers (ordinary licence holders) with a diagnosis of OSAS should notify the DVLA, but can drive provided their sleepiness is controlled by treatment. Group 2 drivers (public service and heavy goods vehicles) should cease driving until the DVLA has received confirmation by a specialist that their condition is adequately treated.

**Cardiovascular disease**

OSA causes recurrent rises in nocturnal blood pressure. Due both to increasing respiratory effort and to arousal from sleep, which produce a characteristic beat-to-beat blood pressure profile virtually diagnostic of OSA (Fig 2). It is unproven whether the increases in nocturnal blood pressure caused by these acute haemodynamic responses alone lead to systemic vascular
Patients with OSAS have a high prevalence of daytime systemic hypertension, but it has been difficult to disentangle how much of this is due to the disease itself and how much to the confounding central obesity and smoking which are common in adult sufferers of this disease. Recent studies attempting to control for these confounding factors suggest that if there is an effect of OSAS on hypertension, it is only small and not a clinical indication for treatment.

**Management**

The main clinical indication for treating OSAS is to reduce daytime sleepiness and improve quality of life. Management should be flexible and staged, depending on symptom severity and complications. For mild or only minimally symptomatic cases, simple measures that may improve snoring and mild OSA can be suggested (Table 1). More specific therapies are discussed below.

**Dental appliances**

The recent development of dental appliances (or mandibular/jaw advancement devices) has provided an alternative treatment option for patients with mild to moderate OSAS, whose symptoms are not severe enough to warrant nCPAP. These devices, worn only at night, open up the retroglossal space by holding the tongue and/or mandible forward. They range in complexity from a simple double gum shield (similar to anti-tooth grinding devices) to elaborate devices that can vary the degree of mandibular protrusion (Fig 3). For the control of OSA, most appliances should be fitted by a dentist under the supervision of a respiratory sleep clinic. Common side effects include an aching jaw and impaired proprioception of the mandible for a couple of hours after the device has been removed. Little is known about the long-term effects on dental health.

**Nasal continuous positive airway pressure**

For patients with symptomatic OSAS, by far the most effective treatment is nCPAP. It immediately and completely corrects upper airway obstruction (snoring, hypopnoeas and apnoeas) (Fig 4), and hence the consequent sleep disruption. In contrast to surgical treatment options, nCPAP can be given on a trial basis and withdrawn if symptoms do not improve or if treatment is not tolerated. Throughout treatment, in particular at the start of treatment, it is...
Table 1. Treatment options for mild to moderate obstructive sleep apnoea.

| Management option                      | Objective                                                                 |
|----------------------------------------|---------------------------------------------------------------------------|
| Bolsters                               | To avoid sleeping on back                                                 |
| No alcohol after 18.00 hours           | Improves pharyngeal muscle tone                                          |
| No sedatives                          | Improves nasal patency and reduces pharyngeal congestion                 |
| Stop smoking                           | (Very difficult to achieve)                                               |
| Lose weight                            | To maintain nasal patency                                                |
| Elevate bedhead, nasal steroids,      | To move jaw forward during sleep                                         |
| nasal surgery                          | (If tonsils are significantly enlarged)                                  |
| Dental appliance                      |                                                                           |
| Tonsillectomy                         |                                                                           |

vital for patients to have access to experienced staff, so that any physical or technical problems can be addressed promptly. The Royal College of Physicians therefore recommends that an nCPAP service be best run from a specialist centre.

In 1997, the British Medical Journal published an evidence-based review article, which concluded that there was no good evidence to support the use of nCPAP for OSAS, despite many positive uncontrolled studies and one positive randomised placebo-controlled study. The review concentrated on the evidence for an effect of OSAS or its treatment on cardiovascular disease rather than on daytime symptoms.

The issue of whether OSAS causes cardiovascular disease is controversial, hence the large volume of data on this subject. Physicians experiencing treating OSAS know that nCPAP is not prescribed to reduce the incidence of future cardiovascular events, but to relieve disabling daytime symptoms. The one randomised controlled trial published at that time, which demonstrated clear beneficial effects on daytime symptoms, was dismissed with no sound argument raised against the validity of the study – apart from suggesting that a tablet placebo was not an appropriate control for a therapy which involved wearing a mask over the face. Since this review, a larger parallel, mask-placebo controlled study of 107 men with symptomatic OSAS has also shown large, positive effects of nCPAP on both daytime sleepiness and quality of life (Table 2).

Surgery

The appeal of surgical procedures for OSAS is that they offer a single treatment, compared to the possible lifetime need for nCPAP. They are, however, not uniformly successful and should be recommended only in a small number of carefully selected cases. Few OSAS patients will therefore require surgery, so small centres will not accumulate much experience and most patients considering surgery should be reviewed in a specialist centre.

Tracheostomy. Tracheostomy is a highly effective treatment, but nCPAP has diminished its role in the management of OSAS. It should still be considered in selected cases, most commonly for patients with life-threatening ventilatory failure who have failed a thorough trial of nCPAP.

Nasal surgery. Nasal surgery has only a limited part to play in the treatment of severe OSAS. If there is only partial pharyngeal collapse with snoring, upstream nasal resistance may be contributing. In this situation, improving nasal patency can be useful – and it is

Fig 3. An example of a commercially available adjustable dental appliance. This device is made from thermoplastic material that can be directly moulded to the patient’s teeth.
sometimes necessary before nCPAP can be effective.

**Uvulopalatopharyngoplasty.** The operation of uvulopalatopharyngoplasty (UPPP) is designed to increase the volume of the pharynx by resecting pharyngeal wall tissue and the soft palate. It has significant morbidity, particularly peri-operative pain and nasal fluid regurgitation which affect up to 20% of patients, and occasional mortality. It also makes subsequent nCPAP less tolerable, with significantly lower compliance. Data on the effect of UPPP in OSA are variable, and its current role in the management of OSA is unclear. The consensus approach is to consider UPPP for OSAS only in non-obese patients without retrognathia, who cannot tolerate nCPAP after a concerted effort.

**Other surgical procedures.** There have been various other experimental surgical approaches to OSAS, including attempts to advance the maxilla, mandible and hyoid bone to enlarge the space behind the tongue. Some success has been obtained in highly selected patients by advancing both the mandible and maxilla, resulting in sustained improvement in OSAS.

Gastroplasty is occasionally performed for very obese patients; although surgery on such obese patients can be hazardous, the effect on weight and OSAS is often dramatic.

**Conclusion**

OSAS is a common condition which causes daytime sleepiness, impaired quality of life and an increased risk of driving accidents. The syndrome is more common in obese patients, but is by no means confined to this group. nCPAP is a highly effective and cheap treatment, but is usually required long-term and somewhat cumbersome for the patient.

Newer treatments are being developed, including hypoglossal nerve stimulation and advances in maxillo-facial surgical techniques. The exact role of these newer treatments is yet to be determined.

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Erratum
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CME Neurology – II

Anette Schrag and Niall Quinn

Disorders of the basal ganglia and their modern management

The first complete sentence of page 326, column 3 should read: 'It is a dominantly inherited disorder (with incomplete penetrance) caused by a mutation in the GTP cyclohydrolase-1 gene (DYT 5) on chromosome 14'.