Obstructive sleep apnoea is a disease of increasing importance because of its neurocognitive and cardiovascular sequelae. Abnormalities in the anatomy of the pharynx, the physiology of the upper airway muscle dilator, and the stability of ventilatory control are important causes of repetitive pharyngeal collapse during sleep. Obstructive sleep apnoea can be diagnosed on the basis of characteristic history (snoring, daytime sleepiness) and physical examination (increased neck circumference), but overnight polysomnography is needed to confirm presence of the disorder. Repetitive pharyngeal collapse causes recurrent arousals from sleep, leading to sleepiness and increased risk of motor vehicle and occupational accidents. The surges in hypoxaemia, hypercapnia, and catecholamine associated with this disorder have now been implicated in development of hypertension, but the association between obstructive sleep apnoea and myocardial infarction, stroke, and congestive heart failure is not proven. Continuous positive airway pressure, the treatment of choice for obstructive sleep apnoea syndrome, reduces sleepiness and improves hypertension.

Definitions
Severity of obstructive sleep apnoea is measured as the apnoea-hypopnoea index (AHI). An apnoea, defined as cessation of airflow for at least 10 s, is classified as obstructive or central on the basis of presence or absence of respiratory effort. A consensus conference (Chicago criteria) provided a definition of hypopnoea as including one of three features: substantial reduction in airflow (>50%), moderate reduction in airflow (<50%) with desaturation (>3%), or moderate reduction in airflow (<50%) with electroencephalographic evidence of arousal.20 Traditionally, oronasal thermistors were used to estimate airflow; however, measurement of nasal pressure can detect much smaller changes in airflow.21,22 Previously, the definition of obstructive sleep apnoea syndrome was restricted to patients with increased AHI and symptoms such as excessive daytime sleepiness.23 However, because obstructive sleep apnoea has been shown to increase cardiovascular risk, many investigators classify non-sleepy patients with high AHI as having this disease. Thus, the statistics about prevalence of this disease vary depending on the definitions used.

Epidemiology
Young and colleagues23-24 showed that 4% of men and 2% of women in a middle-aged North American population had symptoms of obstructive sleep apnoea and an AHI of greater than 5 events per hour of sleep. 24% of North American men and 9% of women had an AHI greater than 5 events per hour of sleep, but only those with excessive daytime sleepiness were included in the former statistics. However, recently cardiovascular risk has been associated with AHIs that were previously deemed to be within the normal range—ie, less than 5 events per hour of sleep.1 Such findings may lead to a new notion of obstructive sleep apnoea—defining a spectrum of disease...
rather than an abrupt cutoff between normal and abnormal. However, a dose-response relation between AHI and its sequelae has been modest at best.25 Thus, the definition and epidemiology of obstructive sleep apnoea are evolving, and a final definition must await development of new methods to measure severity.

Previously, the risk factors for obstructive sleep apnoea have been obesity, male sex, and increasing age (panel 2). Of these factors, obesity is the most important because it is present in roughly 70% of patients with this disorder; it is reaching epidemic proportions, especially in developed countries; and it is the only major reversible risk factor. Despite the importance of this relation between obesity and obstructive sleep apnoea, the underlying mechanisms are unclear. However, pharyngeal airway size is probably diminished with increased weight, thereby increasing the propensity for apnoea.

Male sex is also a risk factor for obstructive sleep apnoea, but the magnitude of the effect varies between studies.26 Sleep laboratory data suggest a five or six-fold increased risk of obstructive sleep apnoea in men compared with women, whereas results of community based studies suggest only a two or three fold increased risk of this disorder in men versus women. This discrepancy may arise because of an underappreciation of this disease in women by physicians and patients alike. On the other hand, women may report different symptoms or have been obesity, male sex, and increasing age (panel 2). Of these factors, obesity is the most important because it is present in roughly 70% of patients with this disorder; it is reaching epidemic proportions, especially in developed countries; and it is the only major reversible risk factor. Despite the importance of this relation between obesity and obstructive sleep apnoea, the underlying mechanisms are unclear. However, pharyngeal airway size is probably diminished with increased weight, thereby increasing the propensity for apnoea.

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The relation between ageing and obstructive sleep apnoea is complex. First, in children, obstructive sleep apnoea after adenotonsillar hypertrophy is well described, and can lead to poor performance at school.27 Second, in adults, prevalence of this disorder rises with increasing age.28 Third, a survivor effect might exist in elderly people, with many middle-aged patients with apnoea not surviving to old age.29 Finally, some data also suggest that apnoea has a protective effect against mortality in elderly people.30

Some newer risk factors for apnoea have been identified, including menopausal status and ethnic origin. Only recently have epidemiological data been available with sufficient power to define the effect of menopausal status independent of changes in age. In a prospective follow-up study of many individuals of the Wisconsin sleep cohort, Young and colleagues31 identified an important increase in frequency of apnoea in postmenopausal women compared with those who had not reached menopause, independent of their age. Thus, physicians should be aware of the effect of ageing and menopause when stratifying risk of obstructive sleep apnoea.

Apnoea is more frequent in African Americans than in white people. Specifically, African Americans develop obstructive sleep apnoea at a younger age than white people, independent of confounding variables.32 Although the anatomical and physiological mechanisms underlying this predisposition are unclear, differences in the soft tissue and bony structure of upper airways are the most likely explanations.

Finally, some social habits have also been identified as risk factors for obstructive sleep apnoea, including cigarette smoking and alcohol consumption. Data for smoking are largely epidemiological, with the presumed mechanism being upper airway oedema.33 The effect of alcohol intake is almost certainly due to suppression of pharyngeal dilator muscle activation.34 Although the effects of alcohol and tobacco need further study, avoidance of these habits is probably helpful.

### Clinical presentation

Sleep apnoea should be suspected in individuals with the signs and symptoms outlined in panel 3.19 Measures of obesity, witnessed apnoeas, and prominent snoring are the strongest of these associations. Although patients with obstructive sleep apnoea are usually obese older men, more subtle presentations can occur. For example, roughly 30% of patients with obstructive sleep apnoea are not obese, but many physicians do not pursue this diagnosis in individuals who are not overweight. In view of the prevalence of sleep apnoea, physicians should include in a general review of body systems, questions about snoring and sleep quality.

### Pathophysiology—the pharyngeal airway

Although most mammals have rigid skeletal support of the pharyngeal airway, patency of the human upper airway is maintained mostly by muscle activation and soft tissue structures.10–12 The evolution of speech is thought to have needed substantial laryngeal motility, leading to a hyoid

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Panel 1: **Consequences of obstructive sleep apnoea**

<table>
<thead>
<tr>
<th>Effect</th>
<th>Magnitude (odds ratio)</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurocognitive</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Motor vehicle accidents</td>
<td>7</td>
<td>Teran-Santos1</td>
</tr>
<tr>
<td>Occupational accidents</td>
<td>2:2</td>
<td>Lindberg14</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prevalent hypertension</td>
<td>1:4</td>
<td>Nieto6</td>
</tr>
<tr>
<td>Incident hypertension</td>
<td>2:9</td>
<td>Peppard6</td>
</tr>
<tr>
<td>Coronary disease</td>
<td>1:3 to 23</td>
<td>Shahar, Hung6,122</td>
</tr>
<tr>
<td>Stroke</td>
<td>1:6</td>
<td>Shahar6</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>2:4</td>
<td>Shahar6</td>
</tr>
</tbody>
</table>

Panel 2: **Risk factors, and mechanisms and effect on obstructive sleep apnoea**

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Magnitude</th>
<th>Possible mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male sex</td>
<td>++</td>
<td>Anatomy, vent control</td>
</tr>
<tr>
<td>Ageing</td>
<td>++</td>
<td>Anatomy, neural reflex impairment</td>
</tr>
<tr>
<td>Obesity</td>
<td>+++</td>
<td>Anatomy, vent control stability</td>
</tr>
<tr>
<td>Menopausal status</td>
<td>+</td>
<td>Unknown, possibly anatomy</td>
</tr>
<tr>
<td>Black race</td>
<td>+</td>
<td>Unknown, possibly anatomy</td>
</tr>
<tr>
<td>Alcohol</td>
<td>++</td>
<td>Impaired dilator muscle activity</td>
</tr>
<tr>
<td>Smoking</td>
<td>+</td>
<td>Airway inflammation, oedema, or both</td>
</tr>
</tbody>
</table>

Panel 3: **Signs and symptoms suggestive of sleep apnoea**

1. Snoring
2. Witnessed apnoeas, gasping, or both
3. Obesity (especially neck circumference)
4. Hypertension
5. Excessive daytime sleepiness
6. Family history
7. Previous tonsillectomy
8. Non-restorative sleep
Variables tend to promote pharyngeal collapse include negative pressure within the airway (eg, during inspiration) and positive pressure outside the airway (eg, fat deposition, small mandible) (figure 1). Conversely, patency is preserved by activation of the pharyngeal dilator muscle (eg, genioglossus) and by increases in lung volume, which tend to keep the airway open by longitudinal traction. As a result, dilating forces (muscle activation) have a complex interaction with collapsing forces (anatomy, airway negative pressure). Most models of the pathogenesis of obstructive sleep apnoea are based on pharyngeal imaging (CT and MRI) during wakefulness, which indicate reduced upper airway size in patients with this disorder when compared with age and weight-matched controls. To control for differences in pharyngeal dilator muscle activity, Isono and colleagues assessed pharyngeal airway size in patients with apnoea and healthy controls under paralysed, anaesthetised conditions. Using endoscopic techniques, the investigators recorded a smaller pharyngeal airway and increased upper airway dilator muscles when a rapid pulse of suction (negative) pressure is applied through the nose. Such activation is presumably a protective reflex that allows the pharynx to resist closure during a collapsing perturbation. Second, when a healthy individual is placed in a negative pressure ventilator (an iron lung) and normal respiratory effort and central modulation is reduced or eliminated, or the individual entrains to this manner of ventilation, negative pressure in the airway has a linear relation with volume, which tend to keep the airway open by longitudinal traction.37–39 As a result, dilating forces (muscle activation) have a complex interaction with collapsing forces (anatomy, airway negative pressure).

Figure 1: The balance of forces

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<tr>
<td>Negative pressure on inspiration</td>
<td>Pharyngeal dilator muscle contraction (genioglossus)</td>
</tr>
<tr>
<td>Extraluminal positive pressure</td>
<td>Lung volume (longitudinal traction)</td>
</tr>
<tr>
<td>Fat deposition</td>
<td></td>
</tr>
<tr>
<td>Small mandible</td>
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In healthy people during wakefulness, pharyngeal patency is carefully protected by these pharyngeal dilator muscles, with negative airway pressure (collapsing pressure) probably the most important local stimulus to their activation. First, the negative pressure reflex describes the robust activation of pharyngeal dilator muscles when a rapid pulse of suction (negative) pressure is applied through the nose. Such activation is presumably a protective reflex that allows the pharynx to resist closure during a collapsing perturbation. Second, when a healthy individual is placed in a negative pressure ventilator (an iron lung) and normal respiratory effort and central modulation is reduced or eliminated, or the individual entrains to this manner of ventilation, negative pressure in the airway has a linear relation with volume, which tend to keep the airway open by longitudinal traction.37–39 As a result, dilating forces (muscle activation) have a complex interaction with collapsing forces (anatomy, airway negative pressure).

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To overcome compromised pharyngeal anatomy, the upper airway dilator muscles of a patient with apnoea must be much more active during wakefulness than those of healthy controls. Using a new technique allowing quantitative electromyographic comparisons between individuals, substantially greater genioglossal activation was recorded during wakefulness in patients with obstructive sleep apnoea than in healthy controls. With the negative pressure ventilation model mentioned above, increased genioglossal activity in patients with apnoea has two components. First, the negative pressure in the upper airway is greater (more negative) in patients than in controls—almost certainly a product of the smaller pharyngeal lumen and the need for greater intra-pharyngeal pressure to generate adequate airflow. As the slope of the relation between negative pressure and
muscle activation is similar in patients with obstructive sleep apnoea and healthy people, this increased negative pressure drives greater muscle activation. Second, the tonic (basal, non-inspiratory) activity of these muscles is also greater in patients with apnoea than in healthy people. The mechanism or mechanisms underlying the increase in tonic activation are unclear, but are probably the result of a plasticity of the neural system. Thus, the airway muscles compensate very precisely for the deficient anatomy of the apnoea patient while awake and ventilation is maintained.

Even in patients with very severe apnoea, disordered breathing events occur only during sleep, emphasising the importance of state (sleep) in the pathogenesis of this disorder. This state effect is probably mediated by a loss of neuromuscular reflexes.37,38 We have known for decades that postural neuromuscular reflexes are reduced or absent during sleep. In addition, the ability of the pharyngeal dilator muscles to respond to negative pressure is substantially attenuated during sleep even in healthy people. Although the neurochemical explanation for this process is unknown, the state-sensitive neuromodulatory systems (cholinergic, adrenergic, serotonergic, and orexinergic) may have a role. Loss of these excitatory inputs to the hypoglossal motorneurons may greatly decrease the ability of the genioglossus and other upper airway dilator muscles to respond to negative pressure and other stimuli that reliably activate these muscles during wakefulness.

To the extent that pharyngeal airway patency is maintained by reflex-driven activation of dilator muscles during wakefulness in patients with apnoea, loss of this reflex mechanism during sleep would be expected to precipitate large decrements in muscle activity and subsequent airway closure. As a result, if an individual’s pharyngeal anatomy needs reflex activation of dilator muscles to maintain patency while awake, this airway will be vulnerable to collapse during sleep.

Despite the importance of pharyngeal dilator muscle control in the pathogenesis of apnoea, other variables are also probably important. Based mostly on data from animals, increases in lung volume seem to promote pharyngeal patency.36,39 Direct mechanical effects and neuromuscular reflexes might be important in mediating this effect. Sleep-induced decrements in lung volume can lead to important reductions in longitudinal traction on the airway, yielding an increasingly collapsible pharynx.40,41 In theory, some individuals may be quite dependent on this mechanism to maintain airway patency while awake and lose it during sleep.

The importance of individual variability in ventilatory control mechanisms in the pathogenesis of sleep apnoea has, until recently, been controversial. However, Younes and colleagues61,62 developed a technique of proportional assist ventilation to assess loop gain (ventilatory control stability) during sleep. Loop gain describes the propensity of a feedback control system to oscillate (become unstable) on the basis of its intrinsic properties. For the ventilatory control system, extended circulation time, small lung volumes, and high ventilatory drive (responsiveness to hypoxia, hypercapnia, or both) tend to destabilise the system and thus increase loop gain. Such results have shown that, independent of upper airway collapse, the loop gain of individuals with obstructive sleep apnoea is much higher than that of controls, suggesting that the ventilatory control system in patients with apnoea during sleep is intrinsically less stable. Although a cause-and-effect relation has not been clearly established, the implication of these observations is that unstable ventilatory control may contribute to sleep disordered breathing in some patients.

**Alternative hypotheses**

Although such pathophysiological mechanisms are generally accepted, other hypotheses have also been developed. First, the pharyngeal dilator muscles of patients with apnoea might become injured or fatigued over time by the repetitive collapse with high negative pressures. Muscle dysfunction or afferent nerve injury could thus evolve and have a perpetuating role in the cause of apnoea.63-66 Although abnormalities have been identified in certain palatal muscles, dysfunction of the entire upper airway muscle system seems unlikely in view of the overall effectiveness of these muscles in maintaining patency during wakefulness. Therefore, the extent to which pharyngeal neuropathy or myopathy perpetuates apnoea is unclear.

Second, apnoea may develop as a result of timing abnormalities between the contraction of upper airway dilator muscles and the diaphragm or pump muscles. Generally, inspiratory pharyngeal dilator muscles activate before diaphragmatic contraction, in theory preparing the upper airway for the collapsing effect of inspiration. Abnormalities in the timing of this system could thus contribute to the propensity for upper airway collapse. Although some supportive data do exist, these are limited and this mechanism is not generally considered important.67

**Consequences**

Once apnoea or hypopnoea develops, arousal from sleep is generally needed to stop the event (figure 2).68 Although the activity of the dilator muscle activity increases as apnoea progresses, the increases are generally insufficient to re-establish pharyngeal patency.69,70 Thus, the patient repeatedly arouses from sleep throughout the night. The precise stimulus to arousal is debated, with most investigators suggesting some combination of increasing respiratory effort in association with hypoxia or hypercapnia as the cause.69 The neurocognitive consequences of recurrent arousals are well established and include sleepiness, reduced performance in neuropsychological tests, lengthened reaction times, reduced creativity, decreased quality of life, and increased accidents (panel 1).67,71 Furthermore, results of randomised controlled trials have recently shown a substantial improvement in cognitive performance and daytime function after treatment with CPAP. 67,72-74

The cardiovascular consequences of obstructive sleep apnoea can be acute and chronic. Acutely, many mechanisms probably contribute to the haemodynamic response to obstructive apnoea. Reductions or cessations in breathing can be associated with large reductions in the partial arterial pressure of oxygen (PaO2), increases in the partial arterial pressure of carbon dioxide (PaCO2), and increases in sympathetic activity. In addition, ongoing respiratory effort during pharyngeal collapse can lead to substantial decreases (ie, more negative) in intrathoracic pressure. Negative intrathoracic pressure contributes to increases in cardiac preload and left ventricular afterload. At the termination of apnoea, increased stroke volume (augmented preload) in the setting of a vasoconstricted circulation (sympathetic activation) can lead to repetitive profound acute increases in systemic blood pressure during the night. Chronically, obstructive sleep apnoea can lead to sustained periods of high blood pressure. Several lines of evidence support this observation, including rigorous investigations in animals,
large well controlled epidemiological studies, and most interventional clinical trials.\textsuperscript{3,4,8} The mechanisms are under assessment, but probably include sustained sympathetic excitation, reduced parasympathetic activity, and release of endothelin.\textsuperscript{76,77} Finally, in cross-sectional studies, the combination of acute and chronic haemodynamic effects in obstructive sleep apnoea have been associated with increased risk of myocardial infarction, cerebrovascular accidents, and congestive heart failure.\textsuperscript{78} An ongoing study (Sleep Heart Health Study) will prospectively define the attributable risk of obstructive sleep apnoea in these adverse cardiovascular outcomes.

**Diagnosis**

A frequently used strategy for diagnosis of obstructive sleep apnoea is overnight polysomnography in a sleep laboratory, which generally incorporates recording of electroencephalogram, electro-oculogram, chin electromyogram,\textsuperscript{26} snoring (microphone), thermistor, electrocardiogram, pulse oximetry, and tibialis anterior electromyogram.\textsuperscript{79} Measurement of nasal pressure may also be helpful in identification of high inspiratory resistance and more subtle respiratory events.\textsuperscript{21,22,80} Results of many studies suggest that the sensitivity of nasal pressure is better than that of a thermistor in detection of respiratory events during sleep. What remains unclear, however, is whether the specificity of this device is also better. Some have questioned whether these subtle events detected only by measurement of nasal pressure are clinically important. Definitive data will need outcome-based studies comparing diagnosis on the basis of nasal pressure with that made by thermistor. Thus, although polysomnography remains the gold standard for diagnosis of obstructive sleep apnoea, the specifics of equipment and technique are evolving.

The combination of diagnosis and treatment in a single night through split night studies has increased in popularity. The idea underlying this strategy is to initially monitor the patient for the first 3 h of the night. If unequivocal apnoea is observed, a nasal CPAP titration is then undertaken. This approach has many advantages and disadvantages. First, the cost of one study is certainly less than that of two, and more patients can be diagnosed and treated if sleep laboratory time is a limiting resource. Second, Epstein and colleagues\textsuperscript{81} showed that the initial patient impression of CPAP is an important predictor of long-term adherence. Thus, some have argued that a full night of therapeutic titration leads to better sleep consolidation than a half-night approach. However, the available data do not suggest important differences in adherence to CPAP based on split versus full night titration.\textsuperscript{82} Third, a therapeutic CPAP level might not be reached during split night titration because of insufficient time, occasionally leading to the need for a second night in the laboratory, but this situation is rare. Fourth, if autotitrating positive pressure devices become well accepted in the home, then the advantages of split-night studies would be reduced, leading to more home-based diagnosis and treatment. However, split-night studies are an effective approach at this time.

Use of overnight oximetry in screening for sleep disordered breathing is controversial.\textsuperscript{83} Although repetitive desaturation in the appropriate clinical context is highly specific for obstructive sleep apnoea, oximetry frequently does not have enough sensitivity for detection of more subtle disordered breathing, with sensitivity and specificity ranging from 50–90%. As a result, a negative oximetry recording generally needs a follow-up sleep study to improve diagnostic sensitivity, leading many to question the usefulness of the original oximetry test. In cases where marked desaturations are recorded, an in-laboratory CPAP titration is generally needed. As a result, neither a positive nor a negative oximetry obviates the need for subsequent polysomnography. Thus, overnight oximetry may be of little clinical use.\textsuperscript{84}

Many home diagnostic methods have been, or are now under investigation, with most simply measuring respiratory signals.\textsuperscript{85–87} The current systems vary substantially from two-channel (snoring and oximetry) to four-channel (oximetry, airflow, effort, position), to full polysomnogram. In general, the diagnostic accuracy of such tests is about 80%, with increasing channels improving accuracy, but adding complexity. However, the role and use of such devices is controversial.

The most cost-effective approaches to diagnosis and treatment varies between countries and within regions of the same country. In some regions, home diagnosis and treatment are the standard, whereas in others, polysomnography in the laboratory remains the preferred approach. Finally, some practitioners use only respiratory monitoring in the laboratory for diagnosis of sleep apnoea and CPAP titration. Thus, many approaches yield satisfactory diagnostic information.\textsuperscript{88–90}

**Management**

CPAP

CPAP treatment remains the treatment of choice for obstructive sleep apnoea because of its effectiveness in elimination of apnoea and improvements in apnoea.
sequelae.3,4,7,9 Results of randomised trials have shown substantial improvements in both sleepiness and neurocognitive performance of patients on nasal CPAP compared with those on placebo or subtherapeutic CPAP. In addition, decrements in blood pressure have been shown with CPAP treatment. Thus, treatment of obstructive sleep apnoea should include a trial of CPAP in most cases since it is effective in elimination of episodes of apnoeas and hypopnoeas in essentially all patients who choose to use it. However, CPAP adherence is still a difficulty, with the best compliance in patients with severe obstructive sleep apnoea and substantial sleepiness. Strategies including heated humidification or nasal decongestants, steroids, or both to alleviate nasal symptoms48 plus intensive support with regular follow-up improve CPAP adherence.90,91

Other (non-CPAP) treatments should be considered in three situations. First, individuals with clearly reversible causes for obstructive sleep apnoea (eg, anatomical deformities such as adenotonsillar hypertrophy) should be considered for surgery. Although obesity is a reversible risk factor, substantial weight loss has been associated with poor success rates.101 Second, individuals who have failed or refuse CPAP treatment should be considered for other approaches (oral appliances or upper airway surgery).102,103 Third, treatment of mild apnoea is a subject of much debate.104–106 In such patients, conservative measures should be emphasised, including maintenance of nasal patency, avoidance of depressants including alcohol, and the goal of 7–8 h sleep per night. In addition, individuals with documented positional apnoea should be encouraged not to sleep on their backs. Despite the potential advantages of conservative measures, treatment with CPAP should also be considered for patients with mild apnoea. Results of randomised trials have shown improvements in daytime symptoms after CPAP treatment in these patients. In addition, the correlation between AHI and the associated complications is poor, making arbitrary designations of mild versus moderate or severe potentially problematic. Thus, although CPAP is the treatment of choice for obstructive sleep apnoea, other treatments can be considered in some situations.

Newer positive pressure devices have gained popularity and include bilevel positive airway pressure machines (different inspiratory and expiratory pressure) and auto-titrating devices (which provide variable pressure levels (different inspiratory and expiratory pressure) and also have a role in treatment of obstructive sleep apnoea. Investigators from one trial20 reported 4-year follow-up data of clinical trials107 do not support use of bilevel positive airway pressure over CPAP, since patient adherence is generally similar. However, patients needing high CPAP pressures to eliminate disordered breathing events who complain about the expiratory work of breathing often prefer bilevel positive airway pressure. Results of studies assessing auto-titrating devices have shown improved adherence in only a few trials, with most finding little effect. Thus, routine use of such devices is difficult to justify because of their increased costs.108–111 Simple CPAP therefore remains the mainstay of apnoea treatment.

Surgery

The most common surgical procedure for obstructive sleep apnoea is uvulopalatopharyngoplasty (UPPP), in which the uvula and redundant soft tissue of the soft palate is resected. Only about 41% of patients who undergo the procedure obtain an AHI of fewer than 20 events per hour.111 In addition, 20 events per hour is not always judged an adequate surgical outcome, especially in view of the uncertain correlation between AHI and apnoea complications. Furthermore, imaging or physical examination procedures have not been shown to improve patients’ selection for surgery. Thus, the role of UPPP for treatment of obstructive sleep apnoea is rather limited. Overnight polysomnography 3–4 months after surgery is clearly indicated. In many cases, snoring will stop after the operation, but disordered breathing continues, leading to silent apnoeas and hypopnoeas in essentially all patients who choose to use it. However, CPAP adherence is still a difficulty, with the best compliance in patients with severe obstructive sleep apnoea and substantial sleepiness. Strategies including heated humidification or nasal decongestants, steroids, or both to alleviate nasal symptoms48 plus intensive support with regular follow-up improve CPAP adherence.90,91

At least one group112–114 has reported success with more aggressive surgery (eg, genioglossal advancement and maxillomandibular advancement). Decrements in AHI to fewer than 10 events per hour have been reported in about 60% of patients after genioglossal advancement and in 90% after maxillomandibular advancement. However, the generalisability these results is unclear and these procedures have not gained widespread acceptance. Randomised controlled trials are needed.

Newer surgical approaches such as laser-assisted palatal procedures, and radiofrequency ablation techniques have also been disappointing. Neither of these techniques has been effective in treatment of obstructive sleep apnoea.115–117 However, for patients whose main complaint is snoring, with little or no apnoea found on formal testing, these procedures may be considered. Snoring is a common problem, which is generally not associated with important adverse medical sequelae in the absence of obstructive apnoeas. Snoring can, however, be a social issue for the bed partners of prominent snorers. In this situation, treatment for snoring with laser-assisted uvuloplasty or somnoplasty (radiofrequency ablation) can be considered. However, longer term follow-up after these procedures does not suggest sustained alleviation of snoring. Thus, caution should be exercised in recommending their use.

Although surgical approaches specifically targeting the upper airway are rather poorly studied, data are emerging for the role of surgical treatment of obesity. The enthusiasm for these procedures has been largely driven by the poor success of diet and exercise in achieving sustained weight loss. Results of several studies118–121 have shown long-term improvements in AHI after gastric stapling. However, some results have shown recurrence of apnoea after surgery for weight loss in the absence of substantial weight gain.122 Thus, the role of surgery for obesity in management of obstructive sleep apnoea is unclear, even though surgery is increasing in popularity.

**Oral appliances**

Mandibular advancing and tongue retaining devices are mechanical devices designed to prevent retroglottal collapse.123,124 Although CPAP is clearly more effective than such devices, especially for severe disease, oral appliances also have a role in treatment of obstructive sleep apnoea. Investigators from one trial125 reported 4-year follow-up data in patients randomly assigned to receive oral appliances versus UPPP in treatment of mild obstructive sleep apnoea. Although oral appliances seemed to be better, the substantial number of drop-outs from this trial limits any definitive conclusions.125 When directly compared in randomised trials, oral appliances are generally preferred by patients over CPAP, even when only partly successful in elimination of disordered breathing events. Thus, oral appliances should be considered for patients who have failed or refused CPAP treatment, for those with snoring or mild obstructive sleep apnoea, and for those who do not respond to surgery. Such surgery probably reduces retroglottal collapse, whereas the oral appliance decreases retroglottal collapse.124 However, more data are needed for neurocognitive and cardiovascular outcomes after treatment with oral appliances.
Conclusion

The rising number of people with obesity will probably make obstructive sleep apnoea an increasingly important public-health problem, especially in view of the neurocognitive and cardiovascular sequelae associated with this disorder. Furthermore, CPAP has been established as the treatment of choice for obstructive sleep apnoea syndrome on the basis of randomised controlled trials, and improvements in our understanding of the underlying mechanisms of obstructive sleep apnoea will hopefully lead to improved therapeutic strategies.

Contributors

Both authors contributed equally to the writing of this report.

Conflict of interest statement

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