Obstructive sleep apnoea

Guillermo Martinez MD
Peter Faber MD, PhD, FRCA

Revalidation FOR ANAESTHETISTS
RCA Revalidation matrix
Matrix reference 3I00

Key points

Patients with obstructive sleep apnoea (OSA) are at an increased risk of perioperative complications.

Any pre-existing continuous positive airway pressure therapy should be continued in the post-operative period.

OSA is strongly associated with obesity.

The prevalence of obesity is increasing and so will OSA.

The best treatment strategy for OSA is weight management.

Guillermo Martinez MD
Specialist Registrar
Anaesthetic Department
Papworth Hospital
Papworth Everard
Cambridge CB23 3RE
UK

Peter Faber MD, PhD, FRCA
Consultant Anaesthetist
Anaesthetic Department
Papworth Hospital
Papworth Everard
Cambridge CB23 3RE
UK
Tel: +44 01 480 830 541
Fax: +44 01 480 364 406
E-mail: peter.faber@papworth.nhs.uk
(for correspondence)

Obstructive sleep apnoea (OSA) affects 5–10% of the adult population with estimates of twice that in patients scheduled for surgery.[1] When accompanied by excessive day-time sleepiness, OSA is termed OSA syndrome (OSAS). The prevalence of OSA is highest in obese middle-aged people. It is also associated with smoking, metabolic syndrome, diabetes, alcoholism, and sedentary lifestyle. OSA affects males twice as often as females. OSA also affects young children in whom it is usually associated with pharyngeal obstruction because of adeno-tonsillary hypertrophy, craniofacial abnormality or both. In adolescence, obesity is a more important factor. Table 1 summarizes the most common factors associated with OSA.

From the association with modifiable lifestyle factors, interventions aimed at weight loss, cessation of smoking, lowering of alcohol intake, and increased physical activity may ameliorate the symptoms of OSA.[2 3] However, in the surgical population, time constraints may not allow the implementation of such measures. It is therefore important to recognize patients with, and at risk of OSA, in order to provide safe and professional care. As the prevalence of obesity has trebled during the past 25 yr, and is predicted to increase further,[4] it is no longer the norm to be of normal weight for height. Obesity is the major risk factor for OSA; therefore, healthcare teams will inevitably have to manage an increasing number of these patients.

Pathophysiology of OSA

During inspiration, the negative pressures produced by the diaphragm and intercostal muscles promote a collapse of the oropharynx. However, the time co-ordinated contraction of the oropharyngeal dilator and abductor muscles maintains the patency of the upper airway. A change in balance towards narrowing of the airway, increased inspiratory pressures, and decreased tone of the oropharyngeal muscles will contribute to obstruction of the upper airway. Among obese subjects, increased pharyngeal adipose tissue deposits will cause further narrowing of the airway, demanding an increase in oropharyngeal muscle tone in order to maintain upper airway patency. Perturbations in the balance between the collapsing forces of the narrowed airway and the dilator muscles may contribute to the obstruction experienced by OSA patients. Complete collapse results in obstruction and a period of apnoea, whereas partial collapse results in snoring and hypopnoea. Breathing resumes when there is arousal from sleep, because of increased oxygen and carbon dioxide chemo-receptor activity and an increased oropharyngeal muscle tone in response to an increased inspiratory effort.

Diagnosis of OSA

The diagnosis of OSA can often be established based on clinical history and examination alone. Predisposing conditions combined with a history of snoring, restless sleep, headaches, and daytime sleepiness should alert to the possibility of OSAS. Additional diagnostic screening tools include a range of questionnaires. These screening tools, however, contain numerous items with an often-confusing scoring system and have not all been validated in a hospital setting. The most suitable diagnostic aid for surgical patients is the STOP-BANG questionnaire,[5] which is summarized in Table 2.

For standardization and a qualitative diagnosis, more formal investigations are required. A sleep study incorporating polysomnography (PSG) will establish the extent and severity of OSA. PSG examinations include recordings of heart rhythm (ECG), electroencephalography (EEG), eye movements, and electromyography. Snoring volume, oro-nasal airflow, and peripheral pulse oximetry are usually also recorded. In the morning, at the end of the test, recordings are analysed in 30 s intervals called epochs. EEG is studied to establish the sleep pattern and distribution between REM and non-REM sleep (Stages 1, 2, 3, and 4). The time-synchronized PSG recordings, including periods of 'arousal' (sudden changes in brain wave activity) and observations of body position during sleep, are subsequently analysed to assist in diagnosing specific sleep disturbances.

doi:10.1093/bjaceaccp/mkq042 Advance Access publication 10 November, 2010

Table 1 Predisposing conditions for obstructive sleep apnoea

Obesity
Age 40–70 yr
Male gender
Excess alcohol intake
Smoking
Pregnancy
Low physical activity
Unemployment
Neck circumference >40 cm
Surgical patient
Tonsillar and adenoidal hypertrophy
Craniofacial abnormalities (e.g. Pierre Robin, Down's syndrome)
Neuromuscular disease

Table 2 STOP-BANG questionnaire. Yes to ≥3 questions=high risk of OSA. Yes to <3 questions=low risk of OSA

STOP		
S (snore)	Loud snoring	Yes/no
T (tired)	Daytime tiredness	Yes/no
O (observed)	Anyone observed cessation of breathing during sleep	Yes/no
P (blood pressure)	Have or being treated for high blood pressure	Yes/no
BANG		
B (body mass index)	BMI >35 kg m^{-2}	Yes/no
A (age)	Age >50 yr	Yes/no
N (neck)	Neck circumference >40 cm	Yes/no
G (gender)	Male	Yes/no

The apnoea/hypopnoea index (AHI) is calculated from the number of apnoea and hypopnoea periods lasting 10 s or longer per hour of sleep. Apnoea is defined as total airflow obstruction despite respiratory efforts. Currently, periods of hypopnoea are not universally defined. The American Academy of Sleep Medicine recommends adopting definitions of hypopnoea to include (i) ≥30% airflow reduction and ≥4% desaturation or (ii) ≥50% reduction in nasal pressure signal excursions with associated ≥3% desaturation or arousal, respectively. Such uncertainty in the definition of hypopnoea may explain the often-large discrepancies in the prevalence of OSA between cohorts of patients. Characteristically, OSA includes repetitive obstruction of the upper airway five or more times per hour of sleep. The severity of OSA is categorized from an AHI of ≥5, ≥15, and ≥30 designating mild, moderate, and severe OSA, respectively. In settings without access to PSG, OSA may be assessed by the number of peripheral oxyhaemoglobin desaturations >4% h^{-1} (oxygen desaturation index, ODI). An ODI of ≥5 is considered significant. The sleep study in Figure 1 demonstrates typically recorded parameters from a patient with OSA. In the UK, oximetry monitoring during sleep time is often the starting point. However, if symptoms are suggestive of OSA but oximetry is negative, then either a respiratory polysomnogram or extended PSG is necessary.

Medical consequences of OSA

OSA is an independent risk factor for serious neuro-cognitive, endocrine, and cardiovascular morbidity and mortality in all age groups. OSAS is furthermore associated with low socioeconomic status and unemployment.

Patients with OSA are at increased risk of cerebro-vascular accidents with a poorer outcome compared with patients without sleep disordered breathing (SDB). The excessive day-time sleepiness observed in OSAS is linked to a reduced quality of life comprising psychosocial problems, decreased cognitive function and depression. These findings may be directly linked to an irreversible decrease in brain grey matter as MRI and oximetry studies have demonstrated morphological changes in brain grey matter[6] and a failure of cerebral autoregulatory blood flow. Worryingly, sufferers of childhood OSA are comparatively disadvantaged with reports of reduced IQ, memory, and learning skills.[7]

The endocrine consequences of OSAS include the development of impaired glucose tolerance and dyslipidaemia; even after adjustment for BMI, age, and gender.[8] OSA patients have perturbed hypothalamic–pituitary–adrenal response with increased adrenocorticotropic hormone and cortisol concentrations and, in both male and female patients, OSAS is associated with testicular and ovarian dysfunction. Females with OSA are more likely to suffer from poly-cystic ovarian syndrome and hypothyroidism.

Most studies on the medical consequences of OSAS have been conducted on cardiovascular morbidity and mortality. Hypertension, brady- and tachyarrhythmias occur more frequently in OSA patients than in the general population. Biventricular dysfunction, pulmonary hypertension, and congestive heart failure significantly increase the risk of haemodynamic instability in the perioperative period. Coupled with concomitant dyslipidaemia, activation of the inflammatory system with endothelial dysfunction, increased platelet aggregating ability, a decrease in circulating anti-oxidants and increase in pro-inflammatory cytokines, it is hardly surprising that OSAS patients are at an increased risk for post-operative cardiovascular complications.[9] Even after controlling for known coronary risk factors, OSA remains an independent risk factor for myocardial infarction.

Treatment of OSA

The complex cause–consequence relationship between OSA and the associated lifestyle and health consequences become apparent in studies examining treatment options. The two most widely available treatments for OSA are continuous positive airway pressure (CPAP) set between 5 and 20 cm H_2O and weight loss. Studies have consistently reported a significant decrease in AHI and improved patient-reported quality of life during CPAP therapy. Long-term use of CPAP has been shown to decrease the frequency of cardiac arrhythmias and improve cardiac function. With parallel reductions in platelet aggregation and improvements in endothelial function, CPAP treatment may reduce the risk of cardiac and cerebrovascular events. These beneficial effects of CPAP are most prominent in the non-obese OSA patient and those suffering from severe OSA. However, lasting effects of isolated CPAP treatment is disputed in obese OSA patients, where reports on CPAP

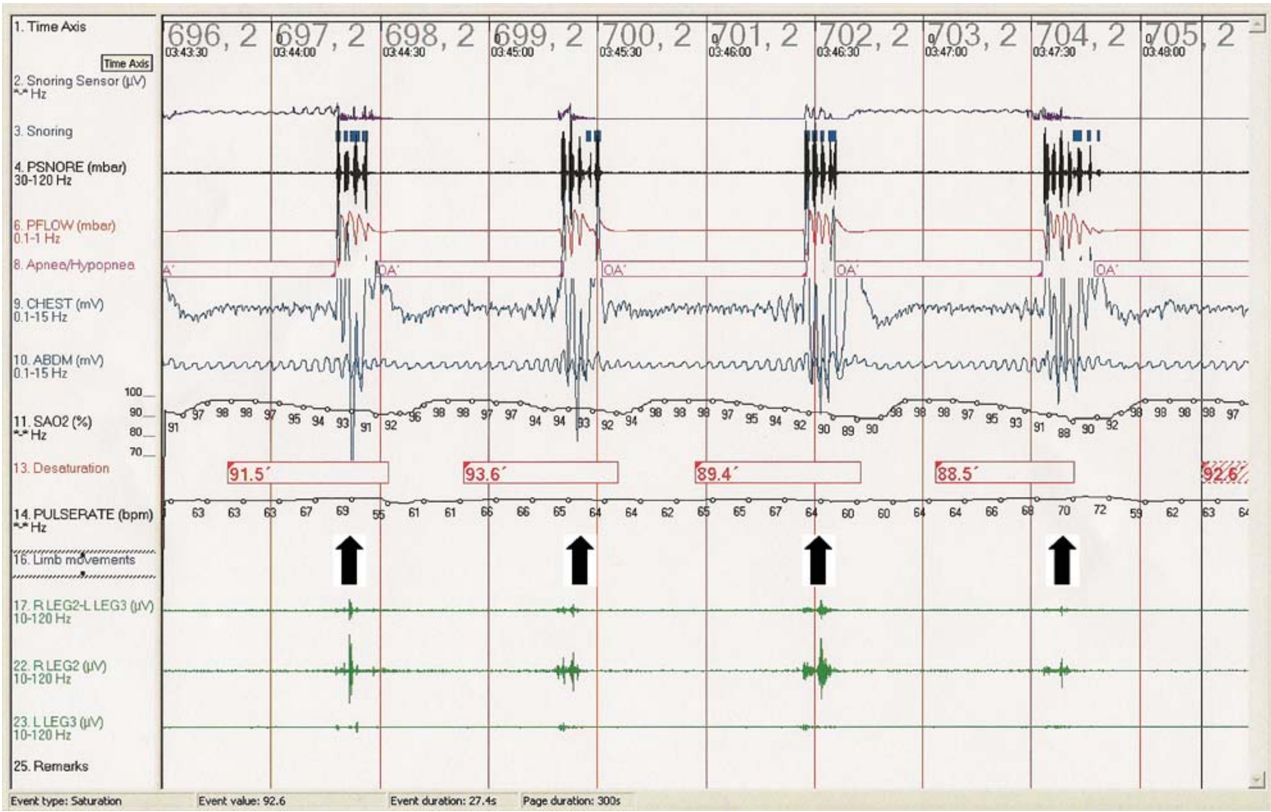

Fig 1 Sleep study in patient with OSA. The black arrows point to episodes of apnoea/hypopnoea.

treatment have yielded equivocal results in hypertension, dyslipidaemia, and insulin resistance. Such conflicting results may originate from differences in the contribution of OSA to cardiovascular risk factors among the studied patient groups. In the obese with metabolic syndrome, OSA may simply constitute an added burden or be a consequence of obesity, whereas in other cohorts of patients the causal relationship between OSA and cardiovascular risk may be stronger. Therefore, although obese OSA patients receiving CPAP therapy may report improvements in daytime sleepiness and quality of life; sustained weight loss, cessation of smoking, and decreased alcohol intake should be encouraged as part of a long-term management strategy. Alternatives to weight loss and CPAP include other non-invasive ventilation modalities mainly used when higher pressures are needed, for patients struggling to tolerate CPAP, or where there is associated hypoventilation with airflow obstruction as a result of obesity. Surgical uvulo-palato-pharyngoplasty and various supportive airway devices promoting mandibular advancement can be offered to selected patients. However, these latter therapies are less prevalent and of lower efficacy compared with weight loss and CPAP.

Anaesthetic considerations

Patients at risk of, or with known OSA presenting for surgery should be comprehensively assessed and investigated for the associated risk factors. Those presenting with congestive heart failure or hypercapnia ($P_{CO_2} > 6.5$ kPa) should have surgery re-scheduled until they have been medically managed and received CPAP therapy for a pre-operative period of $\sim$3 months. CPAP therapy should be continued during hospital admission. In the perioperative period, CPAP support may need adjusting because of operative and anaesthetic factors (e.g. immobilization and opioid analgesia).

Patients with OSA are susceptible to the central respiratory depressant effects of benzodiazepines, neuroleptics, and opioids. Additionally, by causing an enhanced relaxation of pharyngeal muscles during sleep, these drugs will compound the symptoms of OSA. Unless the patient can be safely monitored, sedative pre-medication should be avoided.

Although difficult intubation is strongly associated to OSA, its incidence varies according to different reports. Ideally, a regional or a local anaesthetic technique should be considered in conjunction with multimodal analgesia, including only short action opioids. However, should a patient require a general anaesthetic, it is wise to plan for a difficult intubation in order to ensure a safe induction and continued ventilation. General anaesthesia (GA) preceded by adequate pre-oxygenation, and with tracheal intubation and mechanical ventilation, is preferred to GA with spontaneous ventilation or a sedative technique. Residual neuromuscular block should be judiciously monitored and extubation was performed only in a fully awake patient. CPAP therapy should be recommenced in the recovery room. In the post-operative period, supplementary oxygen should be administered and monitoring of peripheral pulse oximetry continued during immobilization. Monitoring of end-tidal carbon dioxide concentration provides an additional facility for the early detection of airway obstruction or subclinical degrees of respiratory depression. Patients should be individually assessed on the need for postoperative care in a high-dependency unit.

Special patient groups

Weight loss surgery

Surgery for weight loss, also known as bariatric surgery, is offered to adult patients with a BMI of $\geq$40 kg m^{-2} or of 35–40 kg m^{-2} accompanied by serious health conditions. Considering the benefits to patients from bariatric surgery and the increase in the prevalence of obesity, surgical weight loss procedures are almost certain to increase from a current annual level of ~4000. Approximately 70–80% of patients scheduled for weight loss procedures suffer from OSA. These patients have a significantly higher incidence of post-surgery anastomotic leak and prolonged hospital stay. Even with supplementary oxygen therapy, patients having had weight loss surgery experience frequent episodes of postoperative hypoxaemia.[10] Therefore, they should be nursed with facilities in place to offer CPAP therapy and alarmed pulse oximetry in order to detect and minimize the number of hypoxaemic episodes. Before surgery, consideration should be given in this group to routine sleep studies to detect and instigate CPAP therapy. Although sustained weight loss has been demonstrated to significantly reduce AHI in successful dieters and patients after bariatric surgery, a large proportion of patients still require CPAP 12 months after surgery.

Pregnancy

An increasing number of pregnant women are obese and both mother and baby suffer associated health issues. In the most recent Centre for Maternal and Child Enquiries (CEMACH) report, excess body weight was attributed as a major risk factor for maternal mortality with more than half of the women who died classified as either overweight or obese. During pregnancy, intermittent SDB occurs in more than 50% of women. Snoring and OSA are independently linked to hypertension in pregnancy. CPAP therapy has been demonstrated to be a safe and acceptable adjunct for blood pressure management in the group of women with OSA and associated hypertension. Some reports have associated fetal growth retardation with OSA. However, a firm causal relationship has yet to be established between OSA, fetal compromise, and pregnancy outcome.

Day-case surgery

Within the NHS, the government has called for the number of surgical procedures performed as day-cases to increase to 75%. With the expected increase in the number of overweight and obese patients, the anaesthetist will more frequently have to manage patients with suspected or proved OSAS within the day-case unit. Most professional healthcare organizations have issued guidelines on patient selection for day-case surgery and, in general, patients with a BMI of 35 kg m^{-2} are accepted. Although, no studies have examined specifically the suitability and outcome of day-case surgery in OSA patients, intuitively, they ought to be offered regional or local anaesthesia suitable for the planned procedure. Sedatives and opioid analgesia should be avoided.

Acknowledgement

The authors would like to thank Dr T. Quinnell, RSSC, Papworth Hospital for his valuable comments and provision of clinical material.

Conflict of interest

None declared.

References

1. Young T, Peppard PE, Gottlieb DJ. Epidemiology of obstructive sleep apnoea. A population health perspective. *Am J Respir Crit Care Med* 2002; **165**: 1217–39
2. Johansson K, Neovius M, Lagerros YT *et al.* Effect of a very low energy diet on moderate and severe obstructive sleep apnoea in obese men: a randomised controlled trial. *Br Med J* 2009; **339**: b4609
3. Peppard PE, Austin D, Brown RL. Association of alcohol consumption and sleep disordered breathing in men and women. *Clin Sleep Med* 2007; **3**: 265–70
4. Brown M, Byatt T, Marsh T, McPherson K. A prediction of obesity trends for adults and their associated diseases. Analysis from the Health Survey for England 1993–2007. Report by the National Heart Forum. *National Heart Forum: Micro Simulation of Obesity Trends* 2006–2050; **2010**: 1–19
5. Chung F, Yegneswaran B, Liao P *et al.* STOP questionnaire: a tool to screen patients for obstructive sleep apnea. *Anesthesiology* 2008; **108**: 812–21
6. Joo EY, Tae WS, Lee MJ *et al.* Reduced brain grey matter concentration in patients with obstructive sleep apnea syndrome. *Sleep* 2010; **33**: 235–41
7. Halbower AC, Degaonkar M, Barker PB *et al.* Childhood obstructive sleep apnea associates with neuropsychological deficits and neuronal brain injury. *PLoS Med* 2006; **3**: 1391–02
8. Attal P, Chanson P. Endocrine aspects of obstructive sleep apnea. *J Clin Endocrinol Metab* 2010; **95**: 483–95
9. Hung J, Whitford EG, Parsons RW, Hillman DR. Association of sleep apnoea with myocardial infarction in men. *Lancet* 1990; **336**: 261–4
10. Ahmad S, Nagle A, McCarthy RJ, Fitzgerald PC, Sullivan JT, Prystowsky J. Postoperative hypoxemia in morbidly obese patients with and without obstructive sleep apnea undergoing laparoscopic bariatric surgery. *Anesth Analg* 2008; **107**: 138–43

Please see multiple choice questions 4–7.