

Obstructive Sleep Apnoea

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Introduction

Sleep-disordered breathing (SDB) disorders include: central sleep apnoea (Cheyne-Stokes respiration), obstructive sleep apnoea and mixed or complex sleep apnoea.¹ Obstructive sleep apnoea (OSA) is the most common of these three disorders and is defined as airway obstruction during sleep, accompanied by at least five episodes of apnoea or hypopnoea per hour. Each episode is often associated with a decrease in arterial oxygen saturation of > 4%.

Pathophysiology

Normally, the negative pressure produced within the chest by the diaphragm and intercostal muscles, when transmitted to the oropharynx, is counteracted by the co-ordinated contraction of the oropharyngeal dilator and abductor muscles. This keeps the oropharyngeal airway patent. If this co-ordination is impaired, or if there is an excess of additional soft tissue present, then the oropharynx may become obstructed during inspiration, causing a total (apnoea) or partial (hypopnoea) airway obstruction.² In addition, the supine position itself contributes to airway obstruction in the OSA patient, by gravity pulling the tongue and soft palate down as well as reducing the functional residual capacity (FRC) of the lungs.³

- Apnoea is a total airway obstruction with no airflow for 10 seconds or longer.
- Hypopnoea is a partial airflow obstruction causing an airflow reduction of >50% for 10 seconds or longer.^{2,4}

The severity of OSA is determined by the number of episodes of apnoea and/or hypopnoea a patient has per hour of sleep, using the apnoea-hypopnoea index (AHI).

- Mild: an AHI of 5-14 episodes per hour.
- Moderate: an AHI of 15-30 episodes per hour.
- Severe: an AHI of > 30 episodes per hour.

(An AHI of < 10 is unlikely to be associated with a clinical problem or sleep disorder).⁴

When OSA is accompanied by excessive daytime somnolence, it is called obstructive sleep apnoea syndrome (OSAS). The hypersomnolence is caused by

poor sleep quality, especially during deep sleep (stages 3 & 4 of sleep), with frequent arousals due to hypoxia or hypercarbia.^{2,4,5} The prevalence of moderately severe sleep apnoea is estimated to be around 5% in women and 10% in men. The prevalence does increase in older or obese patients.^{2,6,7} While OSA & OSAS are common in obese patients, this does not mean that all obese patients have OSA/ OSAS and similarly not all patients who have OSA/OSAS are necessarily obese.

OSA diagnosis

A high index of suspicion is required to make the diagnosis. It is estimated that up to 90% of patients may be undiagnosed.⁸ In addition to patients who have the predisposing conditions listed below; any history of excessive snoring, restless sleep, headaches, a morning dry mouth and sore throat, or daytime somnolence should act to alert one to investigate further.²

Predisposing conditions for obstructive sleep apnoea include:^{2,4,5}

- Obesity.
- Age 40-70 years.
- Male gender or postmenopausal women.
- Excess alcohol intake or other sedative agents.
- Smoking.
- Pregnancy.
- Low physical activity.
- Unemployment.
- Neck circumference > 40 cm.
- Surgical patient.
- Tonsillar and adenoidal hypertrophy. (Most common cause in children).
- Craniofacial abnormalities (e.g. Pierre Robin, Down's syndrome, retrognathia, micrognathia, brachycephaly).
- Neuromuscular disease.
- Diabetes mellitus.
- Nasal congestion.
- Family history of OSA.

The polysomnogram sleep study (PSG) is the gold standard diagnostic tool which also provides information

as to the extent and severity of OSA.² This study is done in a sleep laboratory and includes recordings of the electrocardiogram (ECG), electro-encephalogram (EEG), eye movements, electromyography (EMG), pulse oximetry (SpO₂) and measurements of oro-nasal airflow and snoring volume.² A PSG test will give an exact AHI index number for the patient, making it possible to exactly classify the patient as having mild, moderate or severe OSA. Clearly a PSG is a costly resource, not freely available, and is usually reserved for patients in whom daytime somnolence severely interferes with quality of life.^{8,9}

Home sleep study units which measure ECG, SpO₂, respiration rate and record snoring volume are used as a cheaper alternative. Neither of these study methods is routinely used in the perioperative setting. A screening questionnaire is more routinely used perioperatively. In a retrospective review; Chung et al (2013) showed that it is clinically safe to proceed with elective surgery, without delay for formal Polysomnography confirmation, with no increase in postoperative complications, if the patient is managed with a screening checklist and a perioperative risk reduction protocol.¹⁰

Three such checklist questionnaires have previously been used:

- The Berlin (2003).
- The American Society of Anaesthesiologists (ASA) checklist (2006).
- The STOP questionnaire.

These all showed moderately high sensitivity for detecting OSA when compared to PSG results. The STOP checklist is the simplest to use, making it the most popular clinically.

By adding the "BANG" questions to the "STOP" questionnaire the sensitivity for moderate to severe OSA increased to 93% for moderate and 100% for severe OSA, if the patient scores 3 or higher.

Interpretation:

- Yes to ≥ 3 questions = high risk of OSA (93% & 100% sensitive for moderate & severe OSA respectively)
- Yes to < 3 questions = low risk of OSA

However, at the cut-off level of three, the STOP-BANG checklist only has a specificity of 47% for moderate and 37% for severe OSA.^{8,11,12} (In other words a very high false-positive rate).

In a prospective study Chung et al (2012) revisited the STOP-BANG checklist. They found that with a score of:¹²

- 0-2 they could confidently rule out OSA.
- 5 the checklist had sufficient sensitivity & specificity to confirm moderate OSA.
- 8 the checklist had sufficient sensitivity & specificity to confirm severe OSA. (Common sense would suggest that a female patient with a score of 7 should also be considered to have severe OSA).

Table 1: STOP-BANG Questionnaire (Chung et al, 2008)¹¹

STOP		
S (snore)	Do you snore loudly (Louder than talking or loud enough to be heard through closed doors)	Yes/ No
T (tired)	Do you often feel tired, fatigued or sleepy during daytime?	Yes/ No
O (observed)	Has anyone observed you stop breathing during sleep?	Yes/ No
P (blood pressure)	Do you have or are you being treated for high blood pressure?	Yes/ No
BANG		
B (body mass index) (BMI)	BMI > 35 kg/m ² ?	Yes/ No
A (age)	Age > 50 years?	Yes/ No
N (neck)	Neck circumference > 40 cm?	Yes/ No
G (gender)	Gender male?	Yes/ No

This is extremely helpful because it is important clinically, to be able to distinguish between mild, moderate and severe OSA.

Importantly all checklists only provide an estimation of the patient's AHI index classification, and not an actual AHI index result like the PSG. For perioperative purposes however, this is sufficient.

OSA consequences^{2,11,13}

Cardiovascular

- Sudden cardiac death, especially nocturnally during periods of hypoxia.
- Hypertension.
- Dysrhythmias (Brady- and tachydysrhythmias).
- Pulmonary hypertension & cor pulmonale.
- Biventricular dysfunction and congestive cardiac failure (CCF).
- Myocardial infarction.

Respiratory

- Chronic intermittent hypoxia and hypercarbia.
- Pulmonary hypertension.

Neuro-cognitive

- Depression, decreased IQ and memory loss.
- Increased risk of cerebrovascular accidents.

Endocrine

- Metabolic syndrome.
- Prediabetes or diabetes.
- Disturbed hypothalamic-pituitary-adrenal response with increased ACTH and cortisol levels.
- Testicular and ovarian dysfunction.
- Hypothyroidism.

OSA treatment options^{2,4}

Known OSA patients may be offered the following treatment options;

- Lifestyle changes, notably to lose weight, stop smoking, limiting alcohol or other sedative intake and to sleep on the side.
- Continuous positive airway pressure (CPAP). This has been shown to reduce the risk of hypertension and stroke by 40% and the risk of other cardiac complications by 20%. A pressure of 5-20 cmH₂O is used. Most effective in non-obese patients and patients with severe OSA. CPAP via nasal mask (nCPAP) is more effective than full face mask CPAP. (Induction of anaesthesia with nCPAP in place may reduce the incidence of difficult mask ventilation).³
- Bi-level Positive Airway Pressure (BIPAP) or Automatic Positive Airway Pressure (APAP) are more advanced forms of CPAP where the pressures are varied between inspiration and expiration to be more comfortable to the patient than a constant high pressure. This also causes less aerophagia, resulting in less flatulence and consequently better patient compliance.
- Mandibular responding splint (MRS). A device which is worn over the teeth like a gum guard, designed to hold the mandible and tongue forward during sleep.
- Surgery. May be specifically indicated for patients with a deviated nasal septum, enlarged tonsils and adenoids or a receding mandible. Bariatric surgery in appropriate cases could also improve OSA. Uvulopalatopharyngoplasty is rarely recommended as it is not shown to be more effective than CPAP.
- Soft palate implants inserted under local anaesthetic to stiffen the soft palate and reduce vibration of the soft palate.
- Playing the didgeridoo. Apparently regular playing of this Australian wind instrument strengthens the upper airway muscles and helps improve mild to moderate OSA.

Anaesthetic considerations^{2,6,8,11,14,15}

Pre-operative

- Patients with CCF or hypercapnoea (PaCO₂ > 6.5 kPa) should be delayed if possible and offered CPAP for 3 months prior to elective surgery.
- As little as 4 hours of CPAP preoperatively may provide benefit.
- Prescribe anxiolytic premed with caution or omit altogether, to avoid complications, especially during the unmonitored period pre-op.
- α_2 -agonist (clonidine, dexmedetomidine) premedication may reduce intraoperative anaesthetic requirements and have an opioid-sparing effect.

Intra-operative

- Because of chronic adrenergic stimulation, OSA patients may develop down-regulated α - and β -receptors. This will cause an attenuated response to vasopressors used and may also lead to more dramatic haemodynamic swings intraoperatively.
- Try to use regional anaesthesia as far as possible in conjunction with multimodal analgesia (NSAID's, paracetamol, tramadol, ketamine, dexamethasone) and short-acting opioids.
- If a general anaesthetic is performed, then intubation and mechanical ventilation is recommended in preference to spontaneous ventilation or deep sedation techniques.
- Use short-acting agents as far as possible.
- Minimise opioid use as far as possible.
- OSA patients have an 8-times higher incidence of difficult intubation than non-OSA patients. They may also typically be difficult to ventilate with face-mask ventilation. Conversely a patient with an unexpectedly difficult airway is very likely to have undiagnosed OSA.
- Position the patient carefully in the "sniffing" position, and "ramped-up" with pillows from scapula to head, to align the earlobe with the sternal notch.

Post-operative

- Increased rate of postoperative complications, mostly on the first day after surgery, but can occur up to days 4 or 5. Notably a two times higher incidence of desaturations, respiratory complications and myocardial ischemia than seen in patients without OSA.
- Respiratory complications include: Aspiration pneumonia, adult respiratory distress syndrome and the need for re-intubation and post-op ventilation. Orthopaedic patients in particular are also more prone to pulmonary embolism.
- OSA is an independent predictor of the patient being likely to develop post-op delirium.
- Initiating CPAP immediately post-op for the first time ever may be difficult to titrate and prove unhelpful.
- Continue with CPAP during hospitalisation for those already using it. The CPAP apparatus should be brought to the recovery room for use immediately post-op.
- Extubate fully awake and after fully reversing any muscle relaxant used.
- Position semi-upright for extubation and recovery.
- Patient controlled analgesia may not be appropriate.
- All patients with severe OSA, who have undergone major surgery with parenteral opioids, should receive supplemental oxygen with pulse oximetry and end tidal CO₂ monitoring post-op, preferably in a high care setting.
- The danger of supplemental oxygen is that oxygen alone does not improve airway patency & OSA. Supplemental oxygen may actually mask the hypoxia

that OSA patients normally experience, and these patients may be at risk of developing significant CO₂ retention.

- Monitor with pulse oximetry post-op until room-air SpO₂ remains > 90% during sleep.

Conscious sedation

- No increased periprocedural risk could be shown in several studies, however common sense dictates extra caution be observed.¹⁵

Ambulatory Surgery

- Patients with severe OSA should not be offered ambulatory surgery.
- Patients with mild OSA, undergoing minimally invasive or superficial procedures, who will not require post-op opioids, may be offered ambulatory surgery.
- OSA patients, who are offered ambulatory surgery, should preferably be offered local or regional anaesthesia and be monitored in the facility for at least 3 hours longer than their non-OSA counterparts before being discharged. These patients must be able to continue CPAP at home. Their pain should be able to be managed with non-opioid analgesics.³

Children with OSA³

- Mostly occurs in age group 2-6 years.
- Most common and effective treatment is adenotonsillectomy.
- These children commonly experience severe desaturations during sleep (low 50s and 60s).
- Sedative premed is usually omitted or drastically reduced, especially if the child will be in an unmonitored environment for any period pre-op.
- Almost all these children will obstruct their airway during induction of anaesthesia. A guedel airway should always be used.
- OSA children have a much higher incidence of airway obstruction postoperatively following adenotonsillectomy than non-OSA children. All OSA children should be hospitalised overnight following an adenotonsillectomy.
- Chronic hypoxemia in childhood may result in upregulation of opioid receptors. These children may be more sensitive to opioids and have decreased opioid requirements.

Conclusion

OSA is commonly diagnosed for the first time when a patient presents for surgery. As such the Anaesthetist often provides a valuable first medical contact point for

diagnosis and long-term management in these patients. While clear peri-operative guidelines do exist for most mild or severe cases, there are still significant gaps in the data, necessitating a common sense approach being followed in the management of these patients.

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Further reading

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