



# Pre-operative screening for sleep disordered breathing: obstructive sleep apnoea and beyond

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A review of different types of sleep disordered breathing and available screening methods in pre-operative assessment, as well as peri-operative management strategies <https://bit.ly/3oh7gZU>

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## Abstract

Sleep disordered breathing describes an important group of conditions that causes abnormal nocturnal gas exchange, with important implications in the peri-operative management plan. An understanding of the pathophysiology behind obstructive sleep apnoea and other disorders that may lead to hypoventilation can help to prevent complications. Patients with these disorders may be minimally symptomatic and it requires careful screening in the pre-operative assessment process for a diagnosis to be made. Decisions regarding initiation of therapy, such as positive airway pressure, and delay of the operation need to be carefully weighed up against the urgency of the surgical intervention. Planning of the peri-operative care, including the use of positive airway pressure therapy and appropriate post-operative monitoring, can help to avoid respiratory and cardiovascular morbidities and improve clinical outcomes.

## Educational aims

- To review different types of sleep disordered breathing and available screening methods in pre-operative assessment.
- To understand the pathophysiology behind sleep disordered breathing and how it can lead to complications in the peri-operative setting.
- To review the planning and treatment strategies that should be considered as part of peri-operative management.

## Introduction

Sleep disordered breathing (SDB) describes a heterogeneous group of nocturnal breathing disorders causing abnormalities in the gas exchange during sleep. There are important implications due to insufficient gas exchange while asleep, as this is mimicked in the peri-operative setting during sedation and is predictive of possible peri-operative complications. The International Classification of Sleep Disorders describes several conditions, including obstructive sleep apnoea (OSA), central sleep apnoea, sleep-related hypoventilation syndromes, sleep-related hypoxaemia and isolated symptoms such as snoring [1]. In the peri-operative context, there are two important considerations that cause pathophysiological changes to neural respiratory drive and abnormalities in the gas exchange that are of relevance: 1) isolated hypoxaemia, or 2) hypoxaemia accompanied by hypoventilation. Patients with SDB are at an increased risk for peri-operative respiratory and cardiovascular complications [2–4].

The most common type of SDB is OSA, which is characterised by recurrent partial or complete upper airway closure during sleep. Moderate–severe OSA is a common disorder, with epidemiological estimates that prevalence in the adult male population in the USA may be up to 13%; most of these cases are



minimally symptomatic and undiagnosed [5, 6]. OSA may be associated with nocturnal hypoventilation when carbon dioxide levels increase during sleep because of diminished ventilation due to airway closure. In its most severe form in obese patients, it can lead to changes in neural respiratory drive resulting in ongoing hypercapnia, which is termed obesity hypoventilation syndrome (OHS) [6–9]. Pathological changes in the respiratory control can also lead to other types of SDB in the form of central sleep apnoea, which may be idiopathic or secondary to other disease processes. Finally, the respiratory muscle pump may be limited in its function due to neuromuscular or chest wall disease that cause sleep-related hypoventilation. It is important to note that comorbid cardiorespiratory disease can lead to both sleep-related hypoxaemia and hypoventilation, the most common comorbidities being COPD and heart failure.

Pre-operative assessment should identify patients who may be at risk for SDB and refer them for appropriate diagnostic tests and therapies. A successful screening programme requires a high sensitivity and specificity based on information that is readily available. The identification of patients with possible SDB in the pre-operative setting is often difficult, as the only reported symptoms, such as snoring and sleep disruption, are common and unspecific. There is a body of work on identifying patients at risk for significant OSA, with various proposals for protocolised approaches based on screening questionnaires followed by diagnostic testing [10, 11]. However, the time required for a diagnostic work-up needs to be carefully weighed up against the urgency of surgery. The screening for hypoventilation syndrome is reliant on a high index of clinical suspicion, although the use of serum bicarbonate can help to identify patients who may require further diagnostic work-up.

In patients who are diagnosed with significant SDB, peri-operative complications can be avoided by appropriate treatment and planning. Patients with OSA can be treated with positive airway pressure therapy, which has been shown to prevent peri-operative respiratory and cardiovascular complications [12–15]. Furthermore, the anaesthetic risk associated with hypoventilation syndromes is well recognised and patients can be carefully monitored peri-operatively with individualised respiratory support plans.

### **Pathophysiology of sleep disordered breathing**

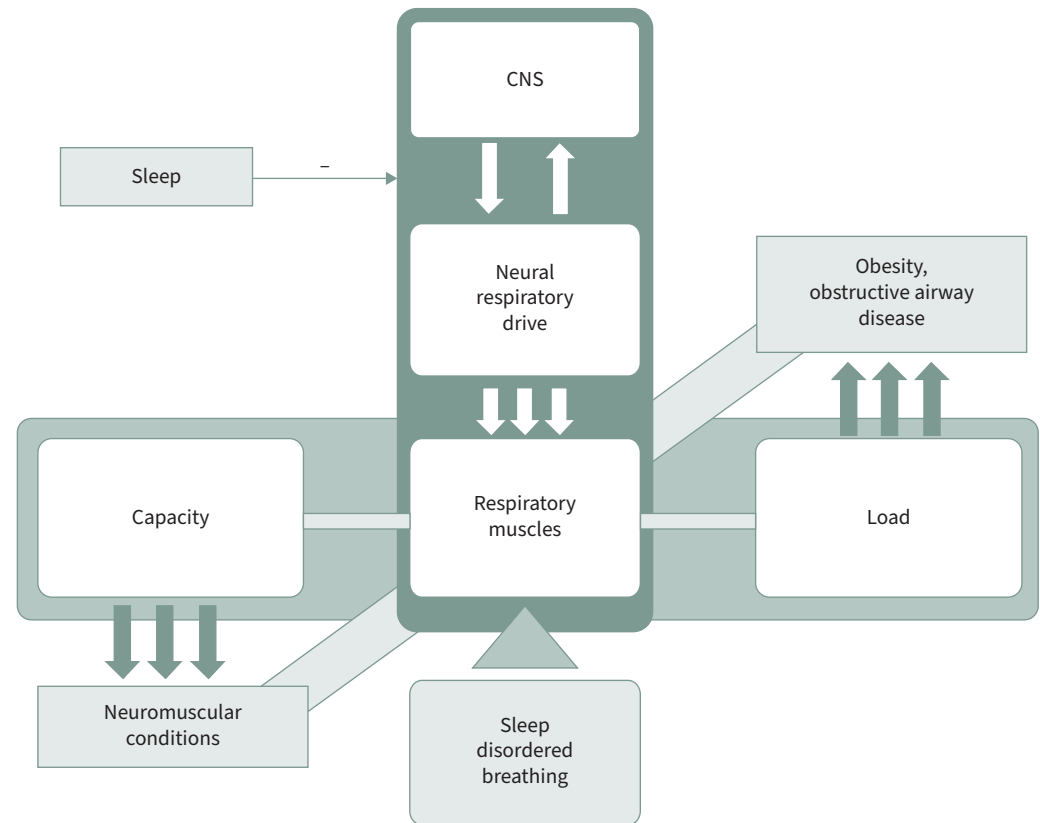
There are various pathophysiological conditions that may occur in isolation or in combination and that can cause SDB, leading to an imbalance of the load–capacity ratio of the respiratory muscle pump with abnormalities in the gas exchange (figure 1). The most common type of SDB encountered is upper airway obstruction causing OSA. However, severe OSA associated with significant obesity can lead to a pathological response in neural respiratory drive that is associated with OHS. Changes to the central chemoreceptor control also underpin different forms of central sleep apnoea. Pathological changes to the respiratory muscle pump, due to either neuromuscular or chest wall disease, can also cause hypoventilation. Finally, comorbid cardiorespiratory disease (*e.g.* COPD and heart failure) can lead to abnormal gas exchange that is exacerbated during sleep and causes either hypoxaemia or hypoventilation.

#### ***Upper airway obstruction***

Patients with significant OSA suffer from recurrent partial or complete airway closure during sleep that leads to decreased airflow and intermittent hypoxaemia, typically resulting in arousal from sleep. The cause is multifactorial and due to a combination of anatomical predisposition interacting with other risk factors, such as obesity (which increases the tissue burden around the neck), sex and age, leading to tissue laxity [9, 16]. The anatomical phenotype associated with upper airway narrowing is worth considering as it can be associated with a difficult airway. There may be a large tongue or increased pharyngeal tissue, such as large tonsils or uvula, which may decrease the airway calibre. The presence of retrognathia with the position of the mandible set back relative to the opposing maxilla is also important, as it predisposes to the narrowing of the oropharynx.

#### ***Pathological changes in neural respiratory drive***

Changes in the central control of breathing can cause a variety of sleep-related breathing disorders [17]. Pathological changes in the central drive to breathe can be idiopathic or secondary to another disease process. The concept of loop gain response characterises the central chemoreflex response to carbon dioxide levels on the ventilatory system. A high loop gain explains various forms of central sleep apnoea. However, abnormal central control of breathing may cause different types of SDB. A high loop gain is seen in idiopathic central sleep apnoea, while a low response may be found in patients with chronic hypercapnia, or medications such as opiates and benzodiazepines. Additionally, there may be a waxing and waning, seen in Cheyne–Stokes respiration, which can be associated with congestive heart failure.



**FIGURE 1** Simplified schematic illustration of the load-capacity ratio of the respiratory muscle pump. The central efferent output, neural respiratory drive, needs to increase to account for an elevated load on the respiratory muscle pump (e.g. obesity, obstructive sleep apnoea), or with diminished capacity (e.g. neuromuscular conditions, kyphoscoliosis). However, sleep inhibits central efferent output and an imbalance in the load-capacity ratio results in sleep disordered breathing with the development of respiratory failure and nocturnal hypoventilation. These conditions are replicated under sedation or with use of centrally active drugs (e.g. opioids). CNS: central nervous system.

OHS (body mass index (BMI)  $>30 \text{ kg} \cdot \text{m}^{-2}$  and daytime carbon dioxide level  $>6 \text{ kPa}$ ) is the most prevalent cause of chronic hypercapnic respiratory failure due to changes in neural respiratory drive requiring noninvasive ventilation [18]. The most common phenotype of OHS is found in an overlap with OSA. The pathophysiology that leads to chronic hypercapnic respiratory failure in these patients is illustrative of the pathological changes in nocturnal gas exchange that may lead to alterations in neural respiratory drive. OSA leads to periodic reductions in ventilation due to intermittent apnoeas and hypopnoeas causing a temporal ventilation-perfusion mismatch and shunting of blood, ultimately resulting in hypoventilation and carbon dioxide accumulation [8, 19]. Consistent hypercapnia leads to a compensatory retention of bicarbonate in the kidneys; over time, as the baseline bicarbonate level increases, patients develop a “blunted drive” due to central chemoreceptor regulation with a neutral pH despite increased carbon dioxide levels. This explains daytime hypercapnia despite a patent upper airway. It is important to recognise that there is a second phenotype of isolated OHS related to mechanical restriction, hypo-inflation of the chest wall and increased intrinsic positive end-expiratory pressures in supine posture, leading to respiratory failure without upper airway obstruction [20, 21].

#### **Neuromuscular/restrictive chest wall disease**

Neuromuscular disorders cover a wide range of congenital and acquired diseases that may affect any of the structures between the central nervous system and the respiratory muscle pump (e.g. brainstem, neurons, neuromuscular junction, muscle). Acquired neuromuscular disorders that may be of relevance include stroke, spinal cord injuries, phrenic nerve injuries, chest wall deformities such as kyphoscoliosis, and anterior horn cell disorders such as motor neuron disease and poliomyelitis [18]. In each of these disorders, there is the potential for hypoventilation due to respiratory muscle involvement, particularly when the diaphragm is affected, causing decreased effectiveness of breathing mechanics that may manifest

with hypoventilation in sleep without obvious symptoms while awake, as well as possible pathological changes to the neural respiratory drive.

### *Sleep-related hypoxaemia and hypoventilation due to comorbid pathology*

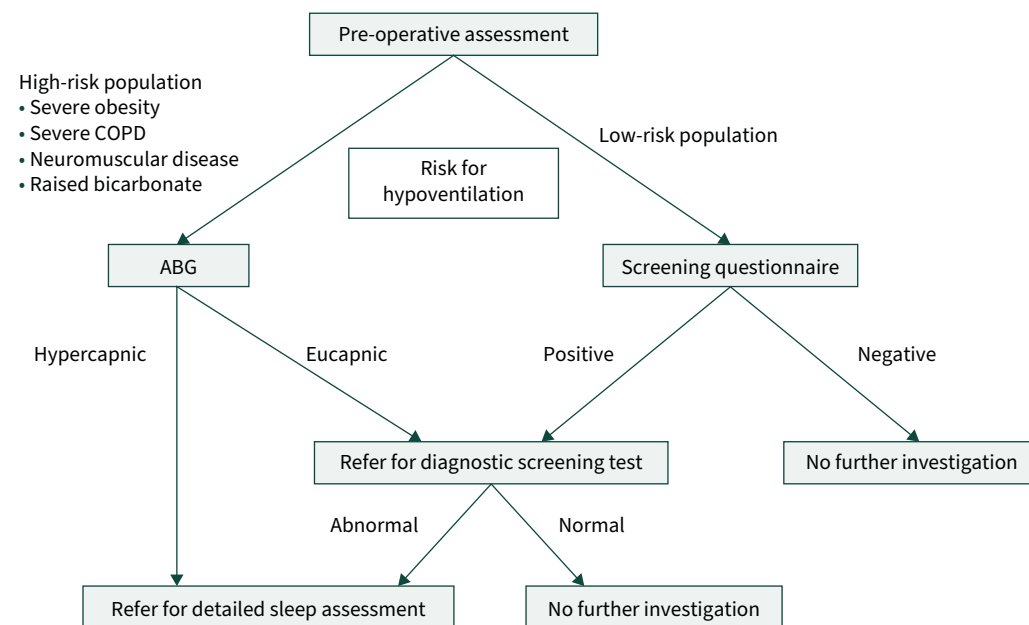
Cardiorespiratory disease can also lead to SDB. Increased levels of neural respiratory drive during wakefulness fall with sleep onset and exacerbate the pathophysiology of altered gas exchange during sleep, particularly during rapid eye movement (REM) sleep, due to the associated physiological peripheral muscle atonia. Chronic heart failure can lead to Cheyne–Stokes respiration with the central neural respiratory drive being affected. However, the most common respiratory disease affecting the lower airway while asleep is COPD, causing both sleep-related hypoxaemia and hypoventilation. COPD is associated with expiratory flow limitation, leading to a rise in end-expiratory lung volume, hyperinflation and increased levels of neural respiratory drive while awake. Expiratory flow limitation worsens during sleep due to decreased neural respiratory drive, diminished respiratory muscle activity and lower operating lung volumes, leading to worsening of the ventilation–perfusion mismatch, which can be exacerbated by supine sleep posture [22, 23]. At an early stage, COPD leads to lower baseline oxygen saturation and, as the airway disease progresses, to nocturnal hypoventilation at a more progressed stage. Patients with COPD–OSA overlap syndrome represent a cohort of patients with an increased BMI and more profound nocturnal hypoxaemia [24].

### **Pre-operative assessment**

The aim of pre-operative assessment is to identify patients who have a clinically high suspicion of peri-operative risks for complications, and to optimise modifiable risk factors to minimise morbidity and mortality. The key is to identify relevant medical diagnoses and develop a plan for optimising peri-operative clinical outcomes while balancing the urgency of the surgery [10, 11, 25]. The difficulty with SDB in this setting is that many clinical assessments have a limited diagnostic accuracy; therefore, a two-step process with patients referred for diagnostic testing is required (figure 2). However, there may be waiting lists for diagnostic testing and subsequent treatment, which may delay surgery. In the setting of acute emergency surgery, there may be a need to proceed with caution but without the confirmatory diagnostic testing and with appropriate peri-operative modification of anaesthetic strategy and post-operative monitoring.

### *Clinical review*

Patients typically undergo a pre-operative assessment consisting of history taking, clinical examination and review of relevant investigations, before a plan for anaesthesia and peri-operative management is made. An important consideration is the patient's functional capacity, in the form of metabolic equivalents (METs) or



**FIGURE 2** Proposed pre-operative assessment pathway for sleep-disordered breathing. ABG: arterial blood gas.

other equivalent scoring systems such as the Duke Activity Status Index (DASI), which is based on the ability to perform daily tasks and exercise level [26]. The aim is to estimate the patient's peak oxygen uptake and, as a surrogate, whether there may be relevant undiagnosed comorbid disease. This is particularly relevant in screening for patients who may be at risk for ventilatory abnormalities, as an abnormal functional capacity would prompt further investigation. Patients identified with specific comorbid diseases such as a neuromuscular disorder, severe airway disease (COPD Global Initiative for Chronic Obstructive Lung Disease (GOLD) stage 3/4) and/or severe obesity (BMI >40 kg·m<sup>-2</sup>) may have higher risk of developing hypoventilation and/or chronic hypercapnic respiratory failure. In such cases, further investigation should be considered, and careful peri-operative planning may be required, in case of emergency surgery.

The clinical identification of patients who may have OSA is somewhat more complex. Well known risk factors including anthropometric, comorbid cardiovascular diseases and common symptoms have been identified but are nonspecific. Studies have shown that unstructured clinical reviews perform poorly at identifying patients who are at risk of SDB, which has prompted a more protocolised screening process with the use of questionnaires to identify patients at risk before arranging diagnostic testing [27, 28]. The Mallampati score is a commonly used tool to assess the upper airway and identify patients with oropharyngeal features that make intubation difficult, many of which overlap with the anatomical predisposition to OSA; however, the score has limited use in the screening for OSA [29].

### Questionnaires

Questionnaires based on anthropometrics, symptoms and comorbidities that are already routinely collected in assessment clinics are recommended in most anaesthetic guidelines to screen for OSA (table 1) [30–34]. The questionnaires have a high sensitivity but relatively low specificity; this means that patients still need to undergo confirmatory diagnostic testing. The most commonly used questionnaire is the STOP-BANG questionnaire, which is validated to screen for surgical patients undergoing pre-operative assessments. The specificity and sensitivity are reported as 30.7% and 87.3% for the diagnosis of moderate–severe OSA [34]. The high sensitivity makes it a good tool to identify at-risk patients in the general pre-operative setting; however, the low level of specificity means that there is limited value in high-risk populations such as those undergoing bariatric surgery, where prevalence has been reported at up to 60%. The recommendation for these patients is to proceed directly to diagnostic testing [35].

### Serum bicarbonate levels and arterial blood gas

Arterial blood gas assessment is the gold standard in diagnosing acute and chronic respiratory failure, but it is not accepted as a routine screening test due to a degree of patient discomfort and availability of testing equipment in outpatient settings. In chronic hypercapnic respiratory failure, there is a metabolic compensation by the kidneys retaining bicarbonate to buffer the carbon dioxide retention. Measurement of serum bicarbonate has good sensitivity and specificity in screening for hypoventilation in obesity, although there are limitations in settings with multi-comorbid patients and polypharmacy, which may influence the accuracy of the measurement [36]. It is often, but not always, performed as part of the renal function and electrolyte biochemistry panels, so it can be easily checked as part of the pre-operative blood screening. Patients with elevated serum bicarbonate may require further investigation, including arterial blood gas assessments and sleep studies.

**TABLE 1** Commonly used questionnaires for obstructive sleep apnoea (OSA) screening and parameters included

Questionnaire	Validated population	Parameters included	Specificity/sensitivity %
STOP-BANG	Pre-operative screening	Nocturnal symptoms, daytime somnolence, hypertension, BMI, neck circumference, age and sex	31/87
ASA OSA checklist	Pre-operative screening	BMI, neck circumference, airway assessment, nocturnal symptoms and daytime somnolence	37/79
Berlin	General population	Nocturnal symptoms, daytime somnolence, hypertension and BMI	44/77
NoSAS	General population	Nocturnal symptoms, BMI, neck circumference, age and sex	49/80
Sleep Apnoea Clinical Score	General population	Nocturnal symptoms, hypertension and neck circumference	54/76
Epworth Sleepiness Scale score	General population	Daytime somnolence symptoms	71/39

Data from [30–34]. ASA: American Society of Anesthesiologists; NoSAS: neck circumference, obesity, snoring, age, sex; BMI: body mass index.

It is important to point out that serum bicarbonate level does not pick up isolated nocturnal hypoventilation with daytime eucapnia. This may be observed indirectly through sustained nocturnal hypoxaemia without oxygen saturations returning to baseline (which is suggestive of hypoventilation) on oximetry studies or early morning arterial blood gases that demonstrate carbon dioxide retention when the patient first wakes up. Alternatively, some sleep laboratories will also perform paired nocturnal and early morning blood gases to check for carbon dioxide rise. While prognostically not as significant as daytime hypercapnia, which is reflective of a pathological change to the patient's neural respiratory drive, significant nocturnal hypoventilation is important in predicting risk for acute peri-operative respiratory failure.

#### **Diagnostic screening test**

Patients at risk for SDB require diagnostic screening testing. The gold standard for the diagnosis of SDB is an in-laboratory polysomnography [37]. This involves overnight monitoring of at least seven parameters (electroencephalogram, electro-oculogram, chin electromyogram, airflow, respiratory effort, oxygen saturation and electrocardiogram). The recording of a polysomnography provides information on sleep stages, upper airway patency, work of breathing and respiratory effort (*e.g.* apnoea/hypopnoea), arousal from sleep and possible alternative causes for daytime somnolence symptoms. However, it is resource intensive and accessibility can be limited. Other types of nocturnal sleep studies are available and classified according to the degree of nocturnal monitoring involved (table 2). The choice of screening method should be individualised and based on an understanding of the information required to guide clinical care. Sleep studies using respiratory polygraphy (type 2 or 3) are recommended for SDB screening as they provide sufficient data to understand causes of hypoxaemia, differentiate between central and obstructive apnoeas, and interpret causes of hypoventilation, as well as detect artefactual traces [38]. However, accessibility of respiratory polygraphy may be limited, as these studies are more resource intense and require manual scoring. In the setting of pre-operative assessment, a type 4 nocturnal pulse oximetry study may be adequate, as it provides information on the degree of hypoxaemia (both in terms of duration and nadir) that may be encountered in the post-operative period; therefore, the degree of risk for possible respiratory complications can be estimated. This has been used in universal screening programmes for patients undergoing assessment for bariatric surgery [39] (figure 3).

#### **Peri-operative management**

##### **Peri-operative anaesthetic approach**

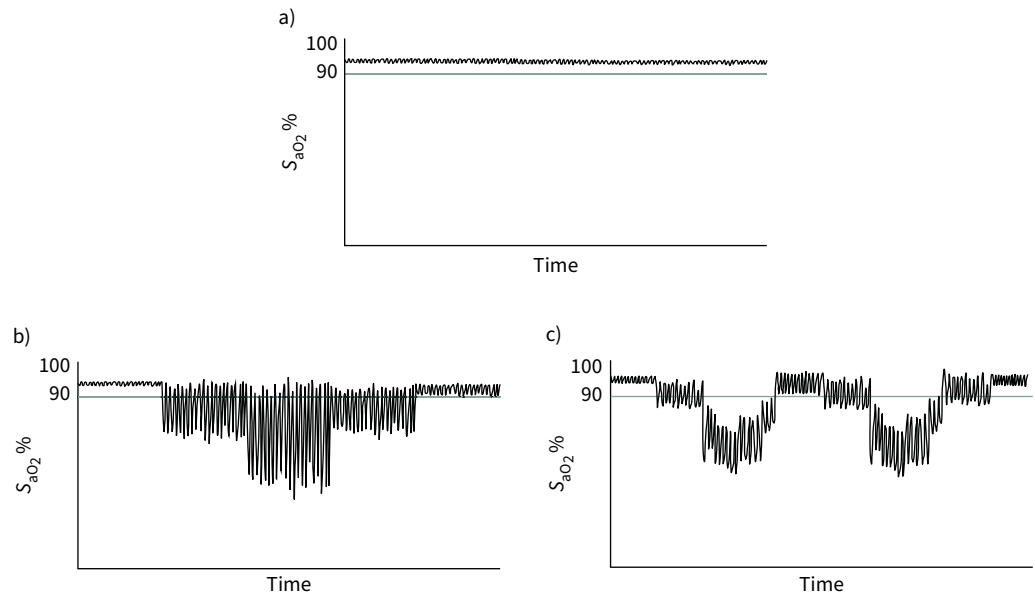
The peri-operative anaesthetic risk associated with OSA is recognised in the American Society of Anesthesiologists (ASA) risk scoring system, with patients scored at least class III if they have severe OSA. Patients who have been either identified to be at risk of SDB or diagnosed with SDB need to have specific plans made for their peri-operative management [2–4, 12–15]. The therapy of choice is the use of positive airway pressure therapy, either continuous positive airway pressure (CPAP) or, in more severe cases with hypoventilation, noninvasive ventilation. There may be benefits to disease modification by implementing positive airway pressure therapy for a few months pre-operatively, but this comes at the cost of delays to surgery. If surgery cannot wait, an understanding of the possible complications may still help in the planning for the anaesthetic and peri-operative management.

There are some important anaesthetic considerations in patients with SDB, including choice of anaesthesia technique, approach to airway management and, finally, peri-operative monitoring [10]. The choice of anaesthesia will involve decisions such as strong consideration for regional anaesthetic techniques over general anaesthesia and, importantly, avoiding deep sedation without a secure airway. Airway management considerations include appropriate positioning of the patient's neck and jaw, and preparation for difficult intubation. When patients are being prepared for extubation, full reversal of neuromuscular blockade needs to be checked and patient extubation during full wakefulness in a non-supine position could be considered. The use of positive airway pressure therapy during induction

**TABLE 2** Sleep study classification and parameters assessed

Type of study	Parameters assessed
<b>Type 1</b> In-laboratory polysomnography	EEG, EOG, EMG, airflow, respiratory effort, oximetry, snoring, ECG
<b>Type 2</b> Non-laboratory polysomnography	EEG, EOG, EMG, airflow, respiratory effort, oximetry, snoring, ECG
<b>Type 3</b> Cardiopulmonary study	Airflow, respiratory effort, oximetry
<b>Type 4</b> Oximetry study	Oximetry

EEG: electroencephalogram; EOG: electro-oculogram; EMG: electromyogram; ECG: electrocardiogram.



**FIGURE 3** Schematic traces of pulse oximetry showing a) normal oxygenation, b) obstructive sleep apnoea (OSA) and c) OSA with hypoventilation.  $S_{aO_2}$ : arterial oxygen saturation.

prior to intubation and in the immediate post-operative period following extubation may be an important part of the anaesthetic approach. It is important to consider the choice of anaesthetic drugs during the operation and post-operative analgesia, due to the effects on the neuromuscular tone of the upper airway in patients predisposed to airway closure and their potential impact on neural respiratory drive and the respiratory muscle pump. The preferred choice of analgesia sedation in patients with SDB is short-acting agents wherever possible.

Finally, post-operative monitoring will be crucial, with consideration for admission to a high dependency unit. Patients with OSA have a higher risk for post-operative respiratory failure with increased incidence of re-intubation and re-admission to intensive care units. There is also an increased risk of cardiac events in patients with OSA (*e.g.* myocardial ischaemia, tachyarrhythmias) that may be related to the increased incidence of post-operative hypoxaemia and high sympathetic tone [4, 12–14]. Importantly, these complications are limited to the immediate post-operative period.

#### **Continuous positive airway pressure therapy**

The gold standard therapy for OSA is CPAP therapy, which provides a pneumatic splinting of the upper airway to maintain its patency during sleep. This therapy is delivered *via* a noninvasive interface, providing a seal over the nasal passage and/or the mouth. Physiologically, with the correct pressure titration and application, it maintains upper airway patency and prevents occlusion and reductions in oxygen delivery, as demonstrated by the improvement in the apnoea–hypopnoea index to normal range in polysomnography studies.

The initiation of CPAP therapy at different pre-operative time-points provides various physiological effects for disease modification. Although the optimal period to initiate treatment prior to elective surgery remains unclear, there is an argument to consider up to 3 months in order to optimise upper airway function, as the airways can often be swollen due to turbulent airflow and recurrent micro-trauma during nocturnal occlusions, and for the patient to acclimatise to therapy [40]. However, CPAP adherence can be problematic, particularly in a minimally symptomatic patient population. In a study of patients newly diagnosed as part of pre-operative screening, only 40% of the patients were adherent, with a median usage of 2.5 h during the first 30 days after CPAP commencement [41].

In the peri-operative period, the application of CPAP therapy helps to stabilise the upper airway, inflate the chest, reduce work of breathing, optimise neural respiratory drive and normalise the sympathetic tone, which is important in the prevention of peri-operative cardiovascular and respiratory complications. The use of CPAP therapy in the post-operative period prevents upper airway closure in patients who may

be susceptible because of centrally acting anaesthetic medications that impact on their neural respiratory drive and/or pharyngeal neuromuscular tone. It has been shown that the extubation of patients onto CPAP therapy after laparoscopic bariatric surgery in morbidly obese patients helps to prevent atelectasis and recruits lung volume [42]. This effect is lost when patients are commenced on CPAP therapy later, in the recovery area or on the ward. There is further evidence for cardiovascular stabilisation with the use of peri-operative CPAP therapy in these patients, who often have significant cardiac risk factors, with a decrease in the incidence of peri-operative myocardial infarctions being described in observational studies [4, 12–14]. Potentially, the risk for tachyarrhythmias, such as atrial fibrillation, is also decreased by limiting the sympathetic tone and adrenaline surges associated with upper airway obstruction in hypoxic conditions, although this still remains a theoretical rather than proven advantage from observational studies.

Although there have been postulations on possible risks of aerophagia with the use of post-operative CPAP, this has been largely rebutted in studies, which have found no significant increase in post-operative nausea and vomiting, and no increase in anastomotic leak in patients undergoing bypass surgery [43, 44].

### **Noninvasive ventilation**

Patients with hypoventilation and hypercapnic respiratory failure require noninvasive ventilatory support for their breathing. It improves work of breathing by providing pressures that reduce the trans-diaphragmatic pre-load, increase minute ventilation, optimise respiratory muscle function, and overcome any intrinsic positive end-expiratory pressure. There is an augmentation of the alveolar ventilation and consequent improvement in gas exchange, leading to lower carbon dioxide levels and improved oxygenation. In the post-extubation period, the use of noninvasive ventilation supports alveolar ventilation during a phase when respiratory mechanics may be compromised.

The initiation of noninvasive ventilation for a pre-operative period can have several positive effects on the respiratory and cardiovascular systems. By improving ventilation and lowering the nocturnal carbon dioxide levels, there are two important effects. First, there is a resetting of the neural respiratory drive, with increased central sensitisation to elevated carbon dioxide levels, decreasing the likelihood of peri-operative acute respiratory failure [45]. Secondly, in patients who may have developed pulmonary hypertension due to alveolar hypoventilation, treatment will lead to improved peri-operative cardiovascular stability with diminished pulmonary congestion and oedema, which is beneficial for airway patency [46]. With nocturnal noninvasive ventilatory support, respiratory muscles may also be supported by avoiding fatigue; there is sleep consolidation and improvement in sleep quality. Specifically, in its use to support patients with severe COPD, maintaining airway patency above the closing volume of the airway during the end-expiratory phase, noninvasive ventilation can lead to decreased respiratory rate and increase the available expiratory time to support better chest deflation. This leads to diminished levels of air trapping and improvement in hyperinflation.

### **Conclusion**

The importance of screening for SDB in the peri-operative assessment is increasingly acknowledged, as it represents a modifiable risk factor for prevention of cardiorespiratory complications. SDB describes a spectrum of disorders ranging from upper airway obstruction to altered neural respiratory drive, failure of the respiratory muscle pump, and gas exchange disorders that are frequently related to comorbid cardiorespiratory disease. It remains difficult to find a consensus on how to systematically identify patients at risk in a timely and sensitive setting; benefits of pre-operative diagnostics need to be balanced against the risk of delaying surgery. Once SDB is identified, appropriate treatment (*e.g.* CPAP, noninvasive ventilation) needs to be commenced, with appropriate planning for the peri-operative management of the patients, in order to prevent complications.

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