

# **OBSTRUCTIVE SLEEP APNOEA AND ANAESTHESIA**

## **ANAESTHESIA TUTORIAL OF THE WEEK 152**

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### **QUESTIONS**

Before continuing, try to answer the following questions.  
The answers can be found at the end of the article.

1. Regarding obstructive sleep apnoea (OSA), which of the following are true?
  - a. Approximately 40% of patients are undiagnosed
  - b. It is more common in women
  - c. It is positively correlated with difficult intubation
  - d. Obesity is the most commonest cause of OSA in children
  - e. It diminishes quality of life
  
2. Which of the following are screening tools for obstructive sleep apnoea?
  - a. Epworth Sleepiness Scale
  - b. Berlin questionnaire
  - c. STOP-BANG model
  - d. Montreal questionnaire
  - e. ASA checklist
  
3. In relation to the peri-operative management of patients with OSA, which of the following are true?
  - a. Have a low threshold for awake intubation
  - b. Extubate in the supine position
  - c. Minimise opioid administration
  - d. Children under two years should go to Intensive Care postoperatively
  - e. Nasal CPAP is not useful postoperatively

### **INTRODUCTION**

Obstructive Sleep Apnoea (OSA) is a sleep-related breathing disorder characterized by repeated episodes of apnoea and hypopnoea during sleep. Apnoea is defined as complete cessation of airflow for more than 10 seconds, and hypopnoea as airflow reduction more than 50% for more than 10 seconds. This repetitive upper airway obstruction often results in oxygen desaturation and arousal from sleep. 'OSA syndrome' refers to the clinical entity of OSA resulting in excessive daytime sleepiness and other symptoms such as unrefreshing sleep, poor concentration, fatigue and morning headaches.

### **EPIDEMIOLOGY**

Population based epidemiological studies conducted over the last decade have uncovered a high prevalence of OSA worldwide, both in developed and developing countries. In middle age, the prevalence of overt OSA is approximately 4% in men and 2% in women, with these figures expected to

increase as the population becomes older and more obese. It is estimated that 80% of patients are undiagnosed, with sleep study data estimating sleep disordered breathing having a prevalence of 24% in men and 9% in women.

OSA is strongly correlated with obesity, in particular morbid obesity (Body mass index  $>40 \text{ kg/m}^2$ , or a BMI  $>35 \text{ kg/m}^2$  with significant co-morbidities). It is found in 40% of obese females and 50% of obese males. Other predisposing conditions include age  $>50$  years (incidence in the elderly may be more than 80%), male gender, neck circumference  $>40\text{cm}$ , nasal/pharyngeal/laryngeal obstruction, craniofacial abnormalities (such as Down's Syndrome, micrognathia and achondroplasia), neuromuscular disorders, and the use of alcohol, sedatives and cigarettes.

## **PATHOPHYSIOLOGY**

Apnoea occurs when the pharyngeal airways collapse. The body is in its most relaxed state during rapid eye movement sleep, which is when there is decreased tone of the pharyngeal dilator muscles (musculus genioglossus and musculus geniohyoideus). In obese patients, increased adipose tissue in the neck and pharyngeal tissues narrows the airway further, predisposing to airway closure during sleep. In non-obese patients, tonsillar hypertrophy or craniofacial skeletal abnormalities may lead to airway narrowing and sleep apnoea.

In OSA patients, neural control mechanisms generating pharyngeal dilator muscle tone are active in the awake state to overcome the relatively smaller upper airway. Sleep and anaesthesia substantially attenuate pharyngeal dilator muscle activity. It is this combination of anatomical structure and neural compensatory mechanisms that is responsible for the development of OSA.

With airway obstruction, inspiratory efforts increase as arterial oxygen desaturation progresses, leading to a partial arousal from sleep and a sudden opening of the airway. A short period of hyperventilation follows, until sleep deepens and airway obstruction recurs, repeating the cycle. The result is blood gas oscillation and sleep fragmentation.

Physiological changes arising from repetitive airway obstruction include arterial hypoxaemia, arterial hypercarbia, polycythaemia, systemic hypertension, pulmonary hypertension, cardiac rhythm disturbances and right ventricular failure. There is an increased incidence of heart disease, cerebrovascular events and sudden death. Disturbance of good sleeping patterns during the night manifests as excessive sleepiness during the day, with poor concentration, fatigue and an increased risk of accidents. Overall, OSA is a serious condition that diminishes quality of life. Studies have found that even mild OSA is associated with significant morbidity.

## **RISKS ASSOCIATED WITH ANAESTHESIA**

OSA is associated with increased peri-operative morbidity and mortality. The peri-operative risk increases in proportion to the severity of OSA. Patients with OSA are at high risk of developing a range of complications when they receive sedation, analgesia or anaesthesia. The most serious of these risks being the potential loss of the airway caused by the use of anaesthetic, sedative and opioid drugs. Difficult intubation and postoperative respiratory depression and airway obstruction are also possible. These risks exist whether or not the surgery is related to treatment of OSA.

## PREOPERATIVE MANAGEMENT

### Preoperative Screening

The American Society of Anesthesiologists (ASA) recommends that patients should be screened for risk of OSA prior to surgery. Numerous clinical screening tests have been developed and validated for this purpose, including the Berlin questionnaire, the ASA checklist, Flemons criteria and the Epworth Sleepiness Scale. There is no consensus about the best screening test. One useful and easy to remember screening tool is the STOP-Bang model, consisting of 8 items.

- S** do you **S**nore loudly, enough to be heard through closed doors?
- T** Do you feel **T**ired or fatigued during the daytime almost every day?
- O** Has anyone **O**bserved that you stop breathing during sleep?
- P** Do you have a history of high blood **P**ressure, with or without treatment?
  
- B** **B**ody mass index (BMI) greater than 35 kg/m<sup>2</sup>
- A** Age over 50 years
- N** Neck circumference greater than 40 cm
- G** Male **G**ender

Patients are considered to be high risk of having OSA if they have three or more of the STOP-BANG criteria, and low risk if they have less than three of the items.

Of the many clinical prediction formulas that can be used to identify patients at high risk of OSA preoperatively, the majority have good sensitivities (>85%). In particular, severe OSA can be predicted with a high degree of accuracy. However the screening tools have low specificities (<55%), leading to a significant false negative rate. This means that most of the clinical screening tests will miss a significant proportion of patients with OSA.

### Preoperative diagnostic workup

The gold standard for the diagnosis of OSA is an overnight sleep study (polysomnography). This may be undertaken prior to surgery in any patient with a history suggestive of OSA and findings of obesity, short bulky neck and a large tongue. Full polysomnography includes monitoring of the chest movement, airflow dynamics, heart rate, blood pressure, SaO<sub>2</sub>, and the electroencephalogram during sleep. It is expensive to perform, labour intensive, and often sleep centres have long waiting lists. As such, formal sleep studies on all patients suspected of having OSA may not be appropriate. If not available, patients with signs or symptoms of OSA should be treated as if they have at least moderate sleep apnoea.

Polysomnography provides classification of severity of patients with OSA based on an apnoea-hypopnoea index (AHI). This refers to the average number of obstructive apnoea and hypopnoea episodes measured per hour, reflecting the departure from the normal physiology of breathing during sleep. A score of 5-15 is 'mild OSA', 15-30 'moderate', and 'severe OSA' is over 30.

### Preoperative treatment

The gold standard of treatment for OSA is the nocturnal use of nasal continuous positive airway pressure (nCPAP) delivery devices. Bi-level Positive Airway Pressure (BiPAP) or Variable Positive Airway Pressure (VPAP) devices can also be used. A nasal mask provides positive airway pressure to the pharynx throughout the breathing cycle to overcome the obstructive forces due to pharyngeal collapse. Pressure requirements range from 5 to 20 cmH<sub>2</sub>O, depending on the severity of the obstruction. Preoperative use of nasal CPAP may lead to improvement in condition prior to surgery and better postoperative compliance with the device.

Mandibular advancement devices may be used for mild OSA. These devices position the mandible forward, pulling the tongue away from the posterior pharyngeal wall. Uvulopalatopharyngoplasty is no longer performed for the surgical management of OSA, and patients who have had this procedure previously should still be considered to have OSA and remain at risk of peri-operative complications. Preoperative weight loss can also be recommended.

### **Preoperative planning**

Elective surgery should be postponed until the patient has been fully investigated and treated. Review of previous anaesthetic notes grading ease of direct laryngoscopy and intubation should be sought. A thorough history and examination is required, with particular attention to assessment of the airway. Patients using nasal CPAP are required to bring their device to hospital, and staff involved in their care from the recovery room to the ward need to be familiar with its use.

## **INTRAOPERATIVE MANAGEMENT**

### **Premedication**

The use of benzodiazepines as premedication relaxes upper airway musculature, reducing the pharyngeal space. This can lead to hypopnoea, and consequently hypoxia and hypercapnia preoperatively. Ideally all sedative premedications should be avoided, or used extremely cautiously.

### **Choice of anaesthetic technique**

All central depressant drugs diminish pharyngeal tone predisposing to upper airway collapse. Common anaesthetic drugs that have been shown to cause pharyngeal collapse include propofol, thiopentone, opioids, benzodiazepines, neuromuscular blockers and nitrous oxide. Local or regional anaesthesia should therefore be the preferred technique whenever possible. If moderate sedation is necessary, continuous capnography monitoring is required, and the use of intraoperative CPAP may be helpful. General anaesthesia with a secured airway is preferred to deep sedation without a secure airway. Laryngeal Mask Airways are generally not recommended.

### **Intubation technique**

A history of OSA is positively correlated with difficult intubation. In one study, Cormack laryngoscopy grade III and IV views were present in 90% of patients with OSA. The ASA difficult airway algorithm offers guidance regarding the difficult to intubate patient. Equipment necessary to handle a difficult airway should be readily available prior to induction, and attention to adequate pre-oxygenation is important. Laryngoscopy should be performed in the optimal 'sniff' position. Consideration of an awake fiberoptic intubation should be made if an airway problem is suspected. Ventilation and oxygenation must be adequately maintained to avoid haemodynamic changes associated with hypoxia and hypercapnia. Adverse effects associated with difficult airway management include death, brain injury, cardiorespiratory arrest, airway trauma and damage to teeth.

### **Extubation**

Tracheal extubation should be carried out with the patient conscious, communicative, and breathing spontaneously with an adequate tidal volume and oxygenation. Extubation should be performed in the semi-upright or lateral position, after complete reversal of neuromuscular blockade. Non-supine positions should then be maintained throughout the recovery period. Reversal of neuromuscular blockade with sugammadex, if available, could be considered preferable to neostigmine. The use of antihypertensive drugs such as beta-blockers or alpha-2 agonists can be used prior to extubation to avoid excessive hypertension, especially in patients with cardiovascular disease.

## **POSTOPERATIVE MANAGEMENT**

### **Respiratory depression and upper airway obstruction**

Respiratory depression and repetitive apnoeas are common immediately following extubation in patients with OSA. Careful cardiorespiratory monitoring in the postanesthesia care unit (PACU) may identify patients who are at risk for further postoperative respiratory events. Vigilance for apnoeas, hypopnoeas, desaturations and pain-sedation mismatch in PACU should lead to the institution of more intensive monitoring of a high risk patient following discharge from PACU.

Supplemental oxygen should be administered continuously to all OSA patients until they are able to maintain their baseline oxygen saturation whilst breathing room air. Oxygen administration will not prevent apnoea (especially in those dependent on a hypoxic respiratory drive), but it may reduce the duration of apnoea and the degree of resulting oxygen desaturation.

The use of preoperative and postoperative nasal continuous airway pressure in patients with OSA undergoing non-airway related procedures has been shown to reduce the risk of developing complications. Patients undergoing upper airway surgery may benefit from a prolonged period of tracheal intubation postoperatively, in view of the potential for significant airway swelling.

During the first three days after surgery, pain scores are highest, resulting in increased analgesic requirements with an associated risk of respiratory depression. Reestablishment of sleep patterns occurs three to four days postoperatively, with the potential for a "REM rebound" and exacerbation of respiratory depression. Overall the 'at risk' period for patients with OSA may extend to one week post surgery.

### **Postoperative Analgesia**

Adequate pain relief remains a priority in OSA. Regional anaesthesia techniques are the preferred methods of postoperative analgesia. Otherwise a multi-modal approach to analgesia is useful. Agents such as paracetamol, non-steroidal anti-inflammatory drugs, tramadol, ketamine, clonidine or gabapentin could be administered. If opioids are required, titration of a short-acting opioid analgesic (such as a half-normal dose fentanyl Patient Controlled Analgesia) under close observation with continuous oxygen therapy is advised.

### **Monitoring**

Continuous pulse oximetry should be provided for all OSA patients in hospital whilst at increased risk of respiratory compromise. The ASA recommend OSA patients should be monitored for three hours longer than usual before being discharged from a facility. Monitoring should continue for at least seven hours after the last airway obstruction or hypoxaemic episode. Consideration of where to discharge a patient from recovery to a regular ward, high dependency unit or intensive care unit, will depend on the type of surgery and the clinical state of the patient. There is minimal evidence available for discharge protocols.

## **PAEDIATRIC OBSTRUCTIVE SLEEP APNOEA**

Adenoidal or tonsillar hypertrophy is the usual cause of OSA in children, and therefore may often be encountered on ENT operating lists for surgical correction. Questioning parents about a history of snoring and breathing pattern in their child is important. Snoring tends to be continuous and mouth breathing is common, whilst hypopnoeas are more frequent than apnoeas. Daytime manifestations of sleep disruption in children may include aggressive or distractible behaviour. Overnight pulse oximetry may be a cheaper, easier alternative to polysomnography in evaluation of children suspected of having OSA.

Children with OSA have a diminished ventilatory response to CO<sub>2</sub>, requiring extra vigilance in the postoperative period. They also have a heightened sensitivity to opioids. It is recommended that children under 2 years of age be monitored overnight in intensive care postoperatively, and that all children under 3 years should be admitted to the ward. Only children over three years old could be considered for discharge on the day of surgery.

## **PERSONAL ASPECTS OF OBSTRUCTIVE SLEEP APNOEA**

Sufferers of OSA, due to their fragmented poor quality sleep, are a potential danger to themselves and to others who may be dependent upon their judgement and vigilance. Being a common yet often unrecognised condition, awareness of the potential of OSA in ourselves must be considered if we are to perform our anaesthetic duties without endangering the lives of our patients.

## CONCLUSION

Obstructive sleep apnoea is largely an undiagnosed yet very prevalent disorder. A high index of suspicion should be made with obesity. People with OSA are at high risk of developing postoperative complications following general anaesthesia – being vulnerable to sedation, anaesthesia and analgesia. Useful management strategies include using alternative non-opioid methods of analgesia, using nasal continuous positive airway pressure before and after surgery, and transfer to intensive care if necessary – particularly after upper airway surgery. Being prepared for and managing a difficult airway with a low threshold for awake intubation is another essential step in the management of OSA in anaesthesia. Further research is required in all areas relating to OSA and peri-operative management.

## ANSWERS TO QUESTIONS

1. FFTFT
2. TTTFT
3. TFTTF

## REFERENCES and FUTHER READING

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