

# Obstructive Sleep Apnoea – Missed Diagnosis, A Nightmare

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## ABSTRACT

*Preoperative evaluation forms an important part of perioperative management. Missed diagnosis can increase perioperative morbidity as a chance of preoperative optimisation is missed. Though obstructive sleep apnoea is common and can be easily diagnosed, it remains undiagnosed due to lack of awareness. If identified preoperatively, we can confirm and quantify the diagnosis with a sleep study and prepare an appropriate perioperative management plan, thus reducing perioperative morbidity and mortality. Hence all the anaesthesiologists should have adequate knowledge of the clinical presentation of OSA. Here we describe diagnosis and anaesthesia management of OSA.*

**Keywords:** Obstructive sleep apnoea, Preoperative, Optimisation, Perioperative management undiagnosed

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## INTRODUCTION

Preoperative evaluation forms an important part of perioperative management. Missed diagnosis can increase perioperative morbidity as a chance of preoperative optimisation is missed. Though obstructive sleep apnoea (OSA) is common and can be easily diagnosed, it remains undiagnosed due to lack of awareness. If identified preoperatively, we can confirm and quantify the diagnosis with a sleep study and prepare an appropriate perioperative management plan, thus reducing perioperative morbidity and mortality. Hence all the anaesthesiologists should have adequate knowledge of the clinical presentation of OSA.

## CASE REPORT

A 57 year male, posted for arthroscopic Bankart repair, weighing 78 kg, with no major medical or surgical illness in the past, was a chronic alcoholic. His pulse was 84/min, BP was 130/80 mm Hg. Mouth Opening was adequate with Mallampatti Class I. Neck was short.

In the Operating room, non-invasive blood pressure (NIBP), pulse-oxymeter and cardiocope was attached. He was premedicated with glycopyrrolate, midazolam 1.5 mg and fentanyl 100 mcg IV and then induced with titrated doses of propofol and succinyl choline 2mg/kg. Patient was intubated over bougie (Cormack Lehane III) with a Flexometallic tube of 9 mm internal diameter. He

was maintained on oxygen, nitrous oxide, isoflurane (1%) and repeated doses of vecuronium.

Intraoperatively, after 30 min, BP started rising to 170/110 mm Hg, with heart rate of 84/min. Inj fentanyl 50 mcg was repeated. BP remained high; hence 25 mcg fentanyl was repeated twice, Isoflurane was increased to 1.5%. But BP remained 164/104 mm Hg with a heart rate of 56/min. After starting IV nitroglycerine infusion 2µ/kg/min. BP decreased to 158/98 mmHg with a heart rate of 96/min. IV metoprolol 1mg boluses were repeated thrice. With this the BP was 110/70mm Hg with a heart rate of 60/min.

Inj. diclofenac 75 mg in slow IV infusion, IV paracetamol 1gm and left supraclavicular block with inj. Bupivacaine (0.25%) 10 cc was given for postoperative analgesia. Neuromuscular blockade was reversed with IV neostigmine-glycopyrrolate and tracheal extubation was done when the patient was breathing regularly with adequate respiratory effort. He was sedated but arousable. But 10 minutes after extubation, the patient started desaturating (SpO<sub>2</sub>-65%). Mask ventilation with 100% Oxygen was tried but was inadequate, SpO<sub>2</sub> dropped further, and hence it was decided to reintubate the patient. Airway obstruction appeared out of proportion to the degree of sedation. After stabilization, he was shifted to SICU and mechanically ventilated with SIMV mode, a FiO<sub>2</sub> of 0.5, Tidal volume of 500ml, a respiratory rate of 12/min and a PEEP of 5 cm H<sub>2</sub>O.

On the same evening, patient was fully awake and following commands. He was extubated over a tube exchanger in propped up position. Non-Invasive Ventilation was kept ready. Postoperatively patient was maintained in propped up position throughout the recovery process and his vitals were monitored overnight in the ICU. On probing, his wife gave a history of snoring interrupted by silence and then heroic deep breaths at night since the last 3 years

with a history of waking up 4-5 times at night and daytime somnolence. His height was 156cm, BMI was 32.05 kg/m<sup>2</sup> and neck circumference was 43 cm. With this history, suspected that our patient might be suffering from OSA and hence on discharge he was advised to reduce weight, avoid alcohol, and maintain regular sleep hours and to undergo polysomnography.

## DISCUSSION

Obstructive sleep apnea (OSA) is a sleep disorder that involves cessation or significant decrease in airflow in the presence of breathing effort<sup>[1]</sup>. It is the most common type of sleep-disordered breathing (SDB). It is characterized by recurrent episodes of upper airway collapse during sleep, which is associated with recurrent oxyhemoglobin desaturations causing increased inspiratory effort and arousals from sleep. This results in sympathetic nervous system stimulation causing systemic and pulmonary hypertension, and myocardial ischemia<sup>[2]</sup>. Cycles of sleep, obstruction, arousal, restoration of breathing and falling asleep again results in poor quality sleep and hence increased daytime sleepiness & morning headaches.

Despite being a common disease, an estimated 80% of Americans with OSAS are not diagnosed.<sup>[3]</sup> Udawadia and colleagues have reported the incidence of OSA to be 19.5 % in urban Indian males<sup>[4]</sup>, Sharma and colleagues reported it as 19.7 % in males and 7.4 % in females<sup>[5]</sup>. There are three cardinal symptoms of OSA: snoring, daytime somnolence and spouse apnea report. These were identified in our patient retrospectively. The typical history is snoring interrupted by periods of silent apnoea broken by a 'heroic' deep breath. But not all the snorers have OSA. Normal snoring does not interfere with sleep hence there is no daytime sleepiness. There are different published clinical diagnostic criteria for OSA. In 1999, the task force of the American Academy of Sleep Medicine established the minimal clinical diagnostic criteria for OSA as an AHI of 10 plus symptoms of excessive daytime sleepiness<sup>[6]</sup>. The Canadian Thoracic Society guidelines stipulate the diagnostic criteria for OSA as

having daytime sleepiness not explained by other factors or at least 2 other symptoms of OSA, with an AHI of 5 or more on Polysomnography<sup>[7]</sup>. Perioperative complications related to OSA are increasingly being reported as the central contention of malpractice suits in US.<sup>[8]</sup> Various causes of OSA are central obesity, micro-or retrognathia, maxillary underdevelopment as in Treacher-Collins Syndrome and pharyngeal encroachment due to tonsillar hypertrophy, large tongue, acromegaly, tumors or edema. If a cause is present, we should investigate for OSA. Risk factors for OSA are overweight, >65 years age, male, related to someone who has sleep apnea, smoking.

Compared to normal person, OSA have decreased heart rate variability and increased BP variability<sup>[9]</sup>. Our patient had a highly variable BP. Grading the perioperative risk is important. This is done with the help of polysomnography (Apnea-hypopnea index 5-15 mild, >30 severe)<sup>[6]</sup>, depending on invasiveness of surgical procedure, anesthesia or requirement of postoperative opioids. On examination BMI may be >35kg/m<sup>2</sup>. Neck circumference >17 inches in males is predictor of difficult airway. Assessment for comorbidities, like hypertension, obesity, coronary artery disease, myocardial dysfunction, and arrhythmias, pulmonary arterial hypertension, gastroesophageal reflux, insulin-resistance diabetes and depression should be done.

It has been reported that OSA is associated with postoperative complications including severe respiratory, cardiac, and neurological complications.<sup>[10,11,12]</sup> Hypoxemia associated with OSA predisposes patients to mental dysfunction<sup>[13]</sup> and possibly poor wound healing after surgery<sup>[14]</sup>. Nearly fatal respiratory complications and even unexpected deaths have been reported after surgery in patients with serious OSA that has been unrecognized or inadequately treated in the perioperative period. Bhamra *et al.* have reported more pulmonary morbidity and prolonged duration of hospitalization in OSA patients after CABG as compared to those without OSA.<sup>[15]</sup>

Kenneth et al reported incidence of postoperative complications<sup>[16]</sup> as follows:

Postoperative complication	Number of patients (%)
Mortality	0 (0%)
Postoperative hypoxemia	2 (1.6%)
Respiratory failure requiring mechanical ventilation	2 (1.6%)
Myocardial infarction	3 (2.4%)
New postoperative arrhythmia	3 (2.4%)
Postoperative renal failure (50% increase in creatinine)	5 (4%)
Postoperative renal failure (need for dialysis)	2 (1.6%)
Unplanned hospital admissions for outpatient surgery	2 (1.6%)
Unplanned admission to the ICU	6 (4.8%)
Postoperative wound infection	2 (1.6%)
Pulmonary embolus	0 (0%)
Deep venous thrombosis	0 (0%)

Mild to moderate OSA patients are advised to lose weight, quit tobacco, avoid alcohol and sedatives especially before bedtime, avoid caffeine and heavy meals within two hours of going to bed, maintain regular sleep hours and throat exercises to strengthen muscles. Moderate to severe OSA may be advised CPAP.

OSA patients are exquisitely sensitive to CNS depressants with a potential for upper airway obstruction or apnoea with even minimal doses. Hence premedication with sedatives should be used sparingly, if at all. Mask ventilation may require two anaesthesia providers using bilateral jaw thrust and mask seal. Options to come out of "cannot ventilate, cannot intubate" situations must be immediately available. Not all OSA patients are difficult to intubate. If "Awake" tracheal intubation is planned, thorough topical and nerve block anaesthesia of the upper airway should be utilised rather than sedatives and opioids. If intubation is to be done with the patient asleep, fully preoxygenate the patients obese patients with relatively small functional residual volume and high oxygen consumption desaturates much more rapidly<sup>[17]</sup>.

They have a tendency for upper airway obstruction during the perioperative period. As in our case, the airway obstruction is out of proportion to the apparent degree of sedation. Spontaneous ventilation against an obstructed airway can lead to rapid development of severe negative pressure pulmonary oedema. Unless there is a medical or surgical contraindication, these patients should be extubated while awake in a semi-upright position<sup>[18]</sup>.

Opioids cause pronounced respiratory depression. Opioids cause airway obstruction by pharyngeal collapse and poor ventilatory response to hypoxaemia and hypercapnia. Hence prophylactic multimodal analgesia technique using nonopioid analgesics including local / regional anaesthesia, acetaminophen, NSAIDs, ketamine and alpha-2-agonists should be used for opioids sparing effect. Residual effect of non-depolarizing muscle relaxants should be adequately antagonized, as even minimal residual effect can result in airway obstruction. Postoperative sedatives increase the risk of respiratory depression and airway obstruction and hence should be avoided.

Though the gold standard for the diagnosis of OSA is overnight polysomnography, it is expensive, requiring highly trained personnel, sophisticated equipment and an entire night of recording. STOP or STOP-Bang questionnaire is an easy to use method to predict severe OSA. Only the patients in whom the physician has a high index of suspicion of sleep apnea based in screening tests should be evaluated with polysomnography<sup>[16]</sup>. Thus, polysomnography may be used in a cost-effective way. If possible, the patient should be treated with at

least 4 to 6 weeks of CPAP before surgery, because an increase in pharyngeal size and a decrease in tongue volume have been noted on MRI after 4 to 6 weeks of nasal CPAP therapy<sup>[19]</sup>. During the first three days after surgery, pain scores are highest, with increased analgesic requirements. Hence danger of life-threatening apnoea during drug-induced sleep is increased. Deep stage 3 and 4 NREM and REM sleep suppressed during the first three postoperative days rebounds in the next three days, hence there is a danger of life-threatening natural deep sleep-induced apnoea. Thus risk of prolonged apnoea during sleep is increased for approximately one week for the postoperative OSA patient<sup>[20]</sup>. Postoperatively, patient is kept in 30° propped up throughout the recovery process. Supplemental oxygen is used with caution as it may reduce hypoxic respiratory drive and increase the incidence and duration of apnoeic episodes. Recurrent hypoxaemia is better treated with CPAP along with oxygen rather than oxygen alone. If patient is using CPAP preoperatively, use CPAP postoperatively when patients are awake and alert<sup>[17]</sup>.

ASA "Practice Guidelines for the perioperative management of patients with obstructive sleep apnoea" suggest that monitoring should continue for a median of seven hours after the last episode of airway obstruction or hypoxaemia while breathing room air in an unstimulated environment<sup>[18]</sup>. We observed this patient overnight in ICU using pulse-oxymeter.

Studies have shown that Nasal CPAP started before surgery and resumed immediately after extubation allows safe management of a variety of surgical procedures in patients with OSA, and to freely use sedative, analgesic, and anaesthetic drugs without major complications<sup>[20]</sup>.

## CONCLUSION

In obese patients we should have high index of suspicion for OSA. It is important to have a presumptive clinical diagnosis because if identified preoperatively, we can confirm and quantify the diagnosis with a sleep study and prepare an appropriate perioperative management plan, thus reducing perioperative morbidity and mortality.

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