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Effects of Anesthesia on Patients Undergoing Surgery for Obstructive Sleep Apnea

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Abstract

Anesthesia and sleep both predispose to upper airway obstruction induced reductions in pharyngeal dilator muscle activation. Symptoms of obstructive sleep apnea are common in patients presenting for surgery and are associated with increased morbidity. Analgesia contributes significantly to postoperative respiratory depression and obstruction. Screening for sleep apnea should be done for all surgical patients. Every patient diagnosed with OSA, or with clinical suspicion of OSA, should be considered to have a difficult airway, and consequently has increased risk of anesthesia. The possible problems may arise during tracheal intubation, extubation, or with postoperative analgesia, since opioids increase the incidence of pharyngeal collapse. Patients with known or suspected obstructive sleep apnea need careful postoperative management. Postoperative monitoring for apnea, desaturation, and dysrhythmias is a necessity in sleep apnea patients.

Keywords: Obstructive sleep apnea; Anesthesia; Analgesia; Upper airway obstruction; Apnea; Postoperative management

Introduction

Obstructive sleep apnea (OSA) is a common complex medical disorder, characterized by repetitive upper airway collapse during sleep, affecting all ages whose etiology is multifactorial and incompletely understood [1].

Maintenance of upper airway patency is a critical issue during unconsciousness patients. The relationship between OSA and complications from anesthesia and postoperative analgesia has wider attention [2]. Anesthesia predisposes to upper airway obstruction as its depressant effects on upper airway muscle activity. In the postoperative period, obstructive apneas may be caused by a decrease in neural drive. Depressant effects of anesthetics on neural control of respiration seems more important and resulting in upper airway obstruction, which is one of the principle mechanisms contributing to hypoxemia during this period [3].

Sleep apnea is often undiagnosed in patients presenting for surgery. The role of OSA as a risk factor for anesthetic morbidity and mortality is considerable. The most serious risk for patients with OSA is loss of the airway owing to anaesthetic, opioid drugs and sedatives [4]. Few data exist in the literature about the effects of anaesthesia on postoperative complications and management of OSA.

Presentation and definition sleep apnea

Apnea is the absence of airflow and tidal volume for 10 seconds or longer. Hypopnea is suboptimal airflow when compared with a normal tidal volume.

There are generally three different types of apnea. The first is central sleep apnea resulting from withdrawal of central drive, which is apnea without ventilatory effort. This occurs in a small portion of the population [5]. The second type is obstructive sleep apnea characterized by repeated episodes of upper airway closure at sleep. OSA is characterized by repetitive episodes of partial or complete upper airway obstruction during sleep that causes arterial hypoxaemia, which leads to a reduced quality of sleep. Despite persisting respiratory efforts, the airway obstruction results in either greatly reduced (hypopnea) or absent (apnea) ventilation.

When excessive daytime sleepiness (EDS) ensues, the condition is referred to as obstructive sleep apnoea syndrome (OSAS). Typically, there is a history of worsening snoring often causing patients and partners to sleep separately. Airway obstruction may arise from a number of anatomical features in the upper airway [4,6]. The primary pathology that resulting in ineffective or absent respiration is associated with OSAS involves the collapse of soft tissue in the oropharynx [7]. During these periods, carbon dioxide does not rise to a significant level while arterial oxygen partial pressure falls rapidly and hypoxemia is the major stimuli for brief awakening (arousal) [8]. After arousal, the patient usually falls asleep quickly with no awareness of the events accompanying cardiac arrhythmias. These multiple episodes of apnea reduce arterial oxygen saturation to less then 80% [9].

The third type of sleep apnea is combination of OSA and central apnea, as called mixed apnea. There is initially no ventilator effort and involves brief periods of central apnea followed by longer periods of OSA [5,9].

The diagnosis of OSA is established by an overnight sleep study or polysomnography. The apnea hypopnea index (AHI) is the number of abnormal respiratory events per hour of sleep [10].

Classically, the accepted minimal clinical diagnostic criteria for OSA are an AHI of 10 plus symptoms of excessive daytime sleepiness (American Academy of Sleep Medicine Task Force 1999). Sleep is not a homogenous state and severity varies with sleep stage, body posture,

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and neck position. The American Academy of Sleep Medicine defines mild OSA as AHI between 5 and 15, moderate OSA as AHI between 15 and 30, and severe OSA as AHI more than 30 [10].

Factors predisposing to OSA

Maintenance of upper airway patency is a critical issue during unconsciousness, anesthesia or sleep. There are a number of factors that predispose to sleep apnea either through their effects on morphology or neural control of the upper airway.

Through several mechanisms obesity also increases OSA risk, including increase in upper airway extra luminal tissue pressure, and decreased functional residual capacity [11,12]. Patient factors such as age, male gender, menopause, obesity, macroglossia, increased neck circumference, micrognathia, retrognathia, nasal congestion, oropharyngeal crowding (high Mallampati scores, decreased pharyngeal width), mandibular or maxillary hypoplasia and hypertension. These morphological changes may be present to varying degrees in part of a disease syndrome, such as Down's syndrome, acromegaly, glycogen storage diseases, Treacher Collins syndrome, Pierre-Robin syndrome or other syndromes associated with craniofacial abnormality. Familial predispositions are common [4,12].

Neuromuscular disorders involving upper airway muscles also predispose to obstruction, as do endocrine (Examples of endocrine diseases associated with increased risk of OSA include hypothyroidism and acromegaly), connective tissue, and storage diseases decreasing upper airway calibre [12,13].

Airway obstruction may arise from a number of anatomical features in the upper airway. Specific abnormalities in the upper airway include nasal obstruction, tonsillar and adenoidal hypertrophy, pharyngeal and laryngeal tumors, foreign bodies, edema and laryngomalacia hematomas are predispose to upper airway obstruction. Other factors predisposing to sleep apnea include alcohol and sedative consumption (which also depress muscle tone and arousal responses) postural changes and snoring itself may cause pharyngeal edema [4,12,13].

A narrow airway is more vulnerable to collapse. Relaxation of the pharyngeal constrictor and genioglossus muscles during sleep allows collapse of the airway. Compliance of the airway wall, upper airway muscle activation, transluminal pressure gradient are another important factors for OSA. Head injury and stroke can increase vulnerability to OSA by depressing muscle tone and arousal responses. The factors that predispose to OSA are prone to difficulties with tracheal intubation also predispose to obstruction and those are prone to difficulties with airway maintenance under anesthesia. Difficult intubation and OSA are related significantly [14,15]. It is important for anesthesiologists and ENT surgen to understand this conditions that may be vulnerable to obstruction under the influence of anesthetic, narcotic analgesic, or sedative drugs.

The upper airway, anesthesia and OSA

The upper airway begins at the nose and lips and ends at the larynx including soft tissue and the bony framework. In patients with obstructive apnea oral cavity, oropharynx, endolarynx and the nasal examination is important for a variety of reasons. The nose serves as an inlet for airflow during sleep and increase in nasal resistance may result in greater negative pressure in the pharynx thus contributing to pharyngeal collapse [12,15].

Information concerning the patient's general physiology and potential sites of airway obstruction can be applied to surgical and nonsurgical treatment of the upper airway. Accurate recording of this information may assist in tracking changes in the examination and assisting research into the causes and treatments of OSA [16].

As with sleep, the administration of medications commonly used in anesthesia, predisposes to upper airway obstruction. Patients with OSA are particularly at risk [7]. Life-threatening problems can arise with to tracheal intubation, tracheal extubation, and providing satisfactory postoperative analgesia. The most serious risk for patients with OSA is loss of the airway owing to anaesthetic, sedative and opioid drugs, and increased risk of anaesthesia. The possibility of OSA should be considered in all patients who present difficulties with airway management under anesthesia [17].

Between the posterior nares and the larynx the upper airway is unprotected by bone or cartilage and is susceptible to obstruction. There are three collapsible pharyngeal segments; the retropalatal pharynx, the retroglossal pharynx and the retroepiglottic pharynx . These segments are collapsible due to the anterior and lateral walls lack of bony support [18-20]. Upper airway activity is inhibited during both sleep and anaesthesia. Because of reduced central drive, it is largely attributable to decreased cortical influences and chemosensitivity seen in both states. Anesthesia predisposes to upper airway obstruction through its depressant effects on upper airway muscle activity and lung volume [12,21].

Anesthesia have depressant effects both directly on the nucleus and on its sources of input through activation of inhibitory GABA(A) and glycine-ergic pathways and inhibition of stimulatory on serotonergic, cholinergic, and glutamate pathways. The hypoglossal nucleus is filled with such receptors as are its sources of input [12,22]. There are also look likes in lung volume changes during anesthesia and sleep. Both effects contribute to increased upper airway collapsibility. In both states functional residual capacity decreases by approximatel 20%. The upper airway longitudinal traction decrease and extra luminal tissue pressure adjacent to the thoracic inlet is increase [12,23].

Where OSA is suspected, special investigations may be needed as part of the perioperative work-up and planning for surgery should begin before admission. Previous anaesthetic records are invaluable. All patients using CPAP at home should be instructed to bring their device with them and the staff looking after them on the ward [4]. During non-REM sleep, some phasic and tonic muscle activity is retained. In REM sleep, this is profoundly depressed and the conditions most closely approximate anesthesia [21].

The number of adult obese patients with obstructive sleep apnea (OSA) is very high. Obstructive sleep apnea in the adult obese patient has increased risk of intubation and extubation difficulties because of increased amount of pharyngeal tissue. Pain management can be expected to be complicated by narcotic/sedative-induced pharyngeal collapse [19]. Following extubation the risk of airway obstruction is increased in OSA patients [24].

According to EMG measurements demonstrated that, during postoperative obstructive apneas, the tonic mode of activity of the geniohyoid muscle was dramatically reduced, but its phasic component was increased [25].

Effect of anesthetic drugs on airway patency patient with OSA in the postoperative period

In the postoperative period, the residual depressant effects of anesthetics on neural control of respiration is one of the principle

mechanisms contributing to hypoxemia which resulting in upper airway obstruction [25].

Patients who have apnea should be given special attention during extubation. Anesthesia, postoperative pain medications and antiemetics dramatically affect sleep architecture.

The agents used perioperatively for sedation and analgesia increase upper airway collapsibility, reduce functional residual capacity, predispose to atelectasis and reduce ventilatory response to hypoxemia and hypercapnia [26].

General and local effects of anesthetics, sedatives (such as diazepam and midazolam), subanaesthetic concentrations of inhalational anaesthetic agents, and analgesic agents on the human respiratory system; depress consciousness, reduce arousal response, diminish neural input to upper airway tone, decrease skeletal muscle tone and inhibit respiration [25-28].

Sedation and analgesics can decrease arousal and ventilatory response to respiratory events and induce sleep apnea in patients do not have clinically significant OSA, anesthetic, sedative and analgesic related disturbances may persist into the postoperative period in patients who have OSA [9,10,28]. Phasic activity of the pharyngeal muscle is diminished significantly by the administration of this drugs [29].

In the postoperative period tonic pharyngeal muscular support modulates airway patency. Benzodiazepine-induced general anesthesia may be associated with low tonic pharyngeal muscular support, resulting in postoperative upper airway obstruction [25]. Cardiovascular complications related to hypoxemia are significantly increased peri and postoperatively [25,26]. A randomized prospective study was performed to compare the recovery patients undergoing uvulopalatopharyngoplasty (UPPP) with either thiopentone-isoflurane-nitrous oxide-fentanyl or propofol-nitrous oxide-fentanyl anaesthesia. They concluded that propofol is preferable to thiopentone-isoflurane in UPPP operations, because physiologic respiratory control recovers faster and postoperative pain is less intense [30].

Topical oropharyngeal anesthesia (TOPA) increases obstructive sleep apnea (OSA) frequency in both loud snorers and normal subjects Central and mixed apneas demonstrated similar frequencies on nights. These data support an impairment of upper airway (UA) protective reflexes among patients with OSA [31].

In several case report the dangers of opioid use in patients with evidence of a compromised upper airway have been highlighted. The use of morphine in patients with OSA has been associated with severe respiratory depression and even death [32].

Combination of analgesics from different classes is used. Medications such as nonsteroidal anti-inflammatory drugs, acetaminophen, tramadol, pregabalin, clonidine, ketamine, gabapentin and dexamethasone are used to alleviate the opioid-related adverse effects of respiratory depression in susceptible patients with OSA. Because of the lack of respiratory depression and opioid-sparing effects in the perioperative period dexmedetomidine has been purported in several case reports to have beneficial effects in patients with OSA [10,33].

Dexmedetomidine may be a useful anesthetic adjunct for patients susceptible to narcotic-induced respiratory depression. In this morbidly obese patient both intraoperatively and postoperatively narcotic-sparing effects of dexmedetomidine are evident [34].

Optimal analgesia for children undergoing adenotonsillectomy for OSA is controversial. Postoperative respiratory and analgesic effects of dexmedetomidine and morphine had compared in children with sleep apnoea having adenotonsillectomy. Zhuang et al. [35] showed postoperatively, dexmedetomidine produced less respiratory depression than morphine, but less effective analgesia. A study by Hullett et al. [36] showed that tramadol is suitable drug for children undergoing adenotonsillectomy for OSA.

In the obese patient with OSA, the goal of postoperative pain management is early mobilization, provision of comfort and improved respiratory function without causing inadequate sedation and respiratory compromise. During the postoperative period, with regard to monitoring, sedation scoring is most relevant, but there should be a low threshold for continuous pulse oxymetry, arterial blood pressure measurement and placement in a high-dependency area for the postoperative period [37].

Postoperative management

All patients presenting for surgery should be controlled for the possibility of OSA. Patients with OSA must be informed and referred to a sleep physician for further investigation as a result of pre-, intra-, or postoperative events. Premedication with opioids or sedatives is avoided wherever possible where OSA is known or suspected [12].

The potential for upper airway obstruction remains high in the recovery room because of the effects of residual anaesthetic or sedative drugs or narcotic analgesic. During the postoperative period patients with severe obstructive sleep apnea have increased risk [2,3,12,38]. In patients suspected or with known OSA particular care must be taken to ensure that neuromuscular blockade is fully reversed [12].

The patient's CPAP device should be in the recovery room for use immediately on emergence from anaesthesia. Under no circumstances should the patient be left unattended and even then consideration should be given to the enterohepatic circulation of any sedative or opioid drugs [4]. The administration of oxygen by facemask is almost universal in the early postoperative period. Alone, this is an inadequate treatment for OSA because the problems of recurrent arousals in response to obstructive episodes and of carbon dioxide retention remain untreated. Furthermore preventing recurrent episodes of desaturation may mask the presence of obstructive episodes [12]. The tendency to obstruction in OSA can be aggravated by postoperative edema after upper airway surgical procedures. Close observation is required until stability is established.

Morbidly obese patients are greatly at risk due to postoperative respiratory insufficiency. CPAP improves respiratory function in morbidly obese patients and accelerates reestablishment of postoperative pulmonary function [39].

On preoperative polysomnography patients with extreme apnea should perhaps be identified as a high risk group who would benefit from concurrent tracheostomy regardless of the proposed sleep surgery. The most commonly performed sleep surgeries do not necessarily effect immediate relief of obstruction, staff may be aided through temporary or longer relief with a tracheostomy [40].

Apneas are most common in postoperative patients, independent of level of consciousness, age, opioid dosage and route of administration an appropriate postoperative nursing environment is a crucial consideration for OSA patients.

References

1. Eckert DJ, Malhotra A, Jordan AS (2009) Mechanisms of apnea. Prog Cardiovasc Dis 51: 313- 323.
2. Hartmann B, Junger A, Klasen J (2005) Anesthesia and sleep apnea syndrome. Anaesthesist 54: 684.
3. Dhonneur G, Combes X, Leroux B, Duvaldestin P (1999) Postoperative obstructive apnea. Anesth Analg 89: 762- 767.
4. Peter J.H. Venn (2011) Obstructive sleep apnoea and anaesthesia. Anaesthesia & Intensive Care Medicine 12: 313- 318.
5. Kabeli C (2005) Obstructive sleep apnea and modifications in sedation. Crit Care Nurs Clin North Am 17: 269- 277.
6. Robinson A, Guilleminaut C (1999) Obstructive sleep apnea. In: Chokroverty S, editor. Sleep disorders medicine: basic science, technical considerations, and clinical aspects. 2nd edition. Boston7 Butterworth-Heinemann; p. 331– 353.
7. Moos DD, Cuddeford JD (2006) Implications of obstructive sleep apnea syndrome for the perianesthesia nurse. J Perianesth Nurs 21: 103-115.
8. Chokroverty S (1999) Physiologic changes in sleep. In: Chokroverty S, editor. Sleep disorders medicine: basic science, technical considerations, and clinical aspects. 2nd edition. Boston7 Butterworth-Heinemann p. 95–125.
9. Ostermeier AM, Roizen MF, Hautkappe M, Klock PA, Klafta JM. (1997) Three sudden postoperative respiratory arrests associated with epidural opioids in patients with sleep apnea. Anesth Analg 85: 452- 460.
10. Seet E, Chung F (2010) Obstructive sleep apnea: preoperative assessment. Anesthesiol Clin 28:199-215.
11. Dancey DR, Haly PJ, Soong C, Lee B, Hoffstein V (2001) Impact of menopause on the prevalence and severity of sleep apnea. Chest 120: 151-155.
12. Hillman DR, Platt PR, Eastwood PR (2007) Sleep, anesthesia, and the upper airway. Seminars in Anesthesia, Perioperative Medicine and Pain 26: 65- 72.
13. Guilleminault C, Stoohs R, Quera-Salva MA (1992) Sleep-related obstructive and non obstructive apneas and neurologic disorders. Neurology 42: 53- 60.
14. Hiremath AS, Hillman DR, James AL, Noffsinger WJ, Platt PR, et al.(1998) Relationship between difficult tracheal intubation and obstructive sleep apnoea. Br J Anaesth 80: 606- 611.
15. Hillman DR, Platt PR, Eastwood PR (2010) Anesthesia, sleep, and upper airway collapsibility. Anesthesiol Clin 28: 443- 455.
16. Goldberg AN, Schwab RJ (1998) Identifying the patient with sleep apnea: upper airway assessment and physical examination. Otolaryngol Clin North Am 31: 919- 930.
17. Hiremath AS, Hillman DR, James AL, Noffsinger WJ, Platt PR, et al. (1998) Relationship between difficult tracheal intubation and obstructive sleep apnoea. Br J Anaesth. 80: 606- 611.
18. Barsh CI (1999) The origin of pharyngeal obstruction during sleep. Sleep Breath 3: 17– 21.
19. Benumof JL (2002) Obstructive sleep apnea in the adult obese patient: implications for airway management. Anesthesiol Clin North America 20: 789-811.
20. Goldberg AN, Schwab RJ (1998) Identifying the patient with sleep apnea: upper airway assessment and physical examination. Otolaryngol Clin North Am 31: 919–930.
21. Eastwood PR, Szollosi I, Platt PR, Hillman DR (2002) Collapsibility of the upper airway during anesthesia with isoflurane. Anesthesiology 97: 786-793.
22. Liu X, Sood L, Liu H, Nolan P, Morrison JL, et al. (2003) Suppression of genioglossus muscle tone and activity during reflex hypercapnic stimulation by GABA(A) mechanisms at the hypoglossal motor nucleus in vivo. Neuroscience 116: 249-259.
23. Rehder K, Marsh HM (1986) Respiratory mechanics during anesthesia and mechanical ventilation, in Macklem PT, Mead J (eds). Handbook of Physiology: The Respiratory System. Bethesda, MD, American Physiological Society, pp 737-752.
24. Rennotte MT, Baele P, Aubert G, Rodenstein DO (1995) Nasal continuous positive airway pressure in the perioperative management of patients with obstructive sleep apnea submitted to surgery. Chest 107: 367– 374.
25. Dhonneur G, Combes X, Leroux B, Duvaldestin P (1999) Postoperative obstructive apnea. Anesth Analg. 89: 762-767.
26. Pelosi P, Croci M, Ravagnan I, Vicardi P, Gattinoni L (1996) Total respiratory system, lung, and chest wall mechanics in sedated-paralyzed postoperative morbidly obese patients. Chest 109: 144-151.
27. Meoli AL, Rosen CL, Kristo D, Kohrman M, Gooneratne N, et al. (2003) Upper airway management of the adult patient with obstructive sleep apnea in the perioperative period avoiding complications. Sleep 26: 1060–1065.
28. Sundman E, Witt H, Sandin R (2001) Pharyngeal function and airway protection during subhypnotic concentrations of propofol, isoflurane, and sevoflurane: volunteers examined by pharyngeal videoradiography and simultaneous manometry. Anesthesiology 95:1125–1132.
29. Tierney NM, Pollard BJ, Doran BR (1989) Obstructive sleep-apnea. Anaesthesia 44: 235–237.
30. Hendolin H, Kansanen M, Koski E, Nuutinen J (1994) Propofol-nitrous oxide versus thiopentone-isoflurane-nitrous oxide anaesthesia for uvulopalatopharyngoplasty in patients with sleep apnea. Acta Anaesthesiol Scand 38: 694-698.
31. Sériès F, Sériès I, Cormier Y (1992) Effects of enhancing slow-wave sleep by gamma-hydroxybutyrate on obstructive sleep apnea. Am Rev Respir Dis. 145: 1378-1383.
32. Lofsky A (2002) Sleep apnea and narcotic postoperative pain medication: a morbidity and mortality risk. APSF Newsletter 17: 24–25.
33. Bamgbade OA, Alfa JA (2009) Dexmedetomidine anaesthesia for patients with obstructive sleep apnoea undergoing bariatric surgery. Eur J Anaesthesiol 26: 176–177.
34. Hofer RE, Sprung J, Sarr MG, Wedel DJ (2005) Anesthesia for a patient with morbid obesity using dexmedetomidine without narcotics. Can J Anaesth 52: 176-180
35. Zhuang PJ, Wang X, Zhang XF, Zhou ZJ, Wang Q (2011) Postoperative respiratory and analgesic effects of dexmedetomidine or morphine for adenotonsillectomy in children with obstructive sleep apnoea. Anaesthesia. doi: 10.1111/j.1365-2044.2011.06817.x.
36. Hullett BJ, Chambers NA, Pascoe EM, Johnson C (2006) Tramadol vs morphine during adenotonsillectomy for obstructive sleep apnea in children. Paediatr Anaesth 16: 648-53.
37. Schug SA, Raymann A (2011) Postoperative pain management of the obese patient. Best Pract Res Clin Anaesthesiol 25: 73-81.
38. Loadsman JA, Hillman DR (2001) Anaesthesia and sleep apnoea. Br J Anaesth 86: 254–66.
39. Pelosi P, Ravagnan I, Giurati G, Panigada M, Bottino N, et al. (1999) Positive end-expiratory pressure improves respiratory function in obese but not in normal subjects during anesthesia and paralysis. Anesthesiology 91: 1221–1231.
40. Johnson JT, Braun TW (1998) Preoperative, intraoperative, and postoperative management of patients with obstructive sleep apnea syndrome. Otolaryngol Clin North Am 31:1025-1030.

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