AANA Journal Course

Update for nurse anesthetists

The Starling resistor: A model for explaining and treating obstructive sleep apnea

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Recent epidemiological research places the incidence of obstructive sleep apnea as high as 16% in the general population. Serious postoperative respiratory complications and death have been reported in this population. Anesthetic drugs contribute to these complications secondary to acute and residual influences on the complex orchestration of airway muscles and reflexes involved in airway patency.

The Starling resistor model is a theoretical model that has application in explaining upper airway dynamics and the treatment and management of obstructive sleep apnea. The model postulates the oropharynx as a collapsible tube. The oropharynx remains open or partially or completely closed as a result of pressure upstream at the nose and mouth, pressure downstream at the trachea and below, or tissue pressure surrounding the oropharynx.

This AANA Journal course provides an overview of the Starling resistor model, its application to obstructive sleep apnea, and preoperative and postoperative anesthetic considerations.

Key words: Airway, anesthesia complications, obstructive sleep apnea, Starling resistor.

Introduction

Considerable attention has been given to the management and treatment of patients with obstructive sleep apnea (OSA) during the last decade. The earliest description of OSA may have been as early as fourth century BC: Dionysius, a tyrant who lived during the era of Alexander the Great, manifested obesity, hypersomnolence, and snoring. In 1836 Charles Dickens published the *Pickwick Papers*, in which Joe the Fat Boy manifested symptoms of what later was termed pickwickian syndrome. Salvador Dali had OSA, which inspired his painting “Sleep” in 1937 (C. Biddle, CRNA, PhD, written communication, September 2003).

Until recently the incidence of OSA was described as 2% to 4%. More recent epidemiological research places the incidence in the general population as high as 16%. Due to the widespread distribution of OSA in the general population, anesthesia providers frequently encounter surgical patients with the comorbid condition, OSA.

The postoperative period is a particularly critical time for patients with OSA. There have been reports of serious respiratory complications and even death in this population. Anesthesia providers should have a thorough understanding of the acute and residual influences of anesthetic drugs on the complex orches-

Objectives

At the conclusion of this course, the reader should be able to:

1. Identify the incidence and definition of obstructive sleep apnea.
2. Describe how the Starling resistor model facilitates understanding of the mechanism of obstructive sleep apnea and its treatment.
3. Identify the risk of anesthetic drugs for the patient with obstructive sleep apnea.
4. Outline patient management during the postoperative period for the obstructive sleep apnea patient.
5. Understand the benefits of using nasal continuous positive airway pressure during the postoperative period for the patient with obstructive sleep apnea in preventing potential life-threatening complications.

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tration of airway muscles and reflexes involved in airway patency. The Starling resistor model has application in explaining upper airway dynamics and the treatment and management of OSA.

**The Starling resistor model**

The Starling resistor model represents theoretical phenomena that can be applied to air flow in the upper airway and to fluid flow through the coronary arteries, the cerebral circulation, and the epidural space. The model can be described in terms of a potentially collapsible tube within some type of chamber. In considering airway patency issues occurring in OSA or during anesthetic management, imagine the oropharynx as a collapsible tube and the surrounding tissues of the neck as the chamber (Figure 1). Here the oropharynx remains open, or is partially or completely closed, as a result of a variety of forces including the following: (1) the proximal or opening pressure at the level of the nose and mouth, (2) the distal or end pressure present at the trachea and below, and (3) the pressure that is applied to the oropharynx as a result of tissue surrounding it.

As shown in Figure 1, the tissue pressure (Ptissue) corresponds directly to the pressure with which the surrounding tissues of the oropharynx constrict the upper airway. A high opening pressure (Pproximal) will assist in overcoming the collapsing pressure of the surrounding tissue (Ptissue). A low or negative pressure below the vocal cords (Pdistal) that occurs as a result of inspiratory efforts will contribute to airway collapse if Ptissue is elevated. When sufficient muscular tone is operative in tissues surrounding the collapsible tube, in effect “lifting tissue” away from the collapsible element, Ptissue does not contribute to airway collapse.

Because the pharynx is a collapsible segment situated between the upstream oronasal segment and the downstream tracheal segment, flow is dependent on upstream pressure and the pressure surrounding the collapsible segment. Ineffectual driving pressure, excessively negative inspiratory pressure, and excessive tissue pressure can promote collapse. In a complex orchestration of timed nerve and muscular coordination, pharyngeal muscles have inspiratory phasic activity that just precedes diaphragmatic activity, thus preparing the pharyngeal airway for the ensuing negative pressure during inspiration. Ideally this results in an exquisite symphonic performance of coordinated inspiratory and airway muscle contraction reflexes, ensuring airway patency regardless of body position or sleep state.

**Obstructive sleep apnea**

Obstructive sleep apnea is defined as a cessation of airflow for more than 10 seconds, despite continuing ventilatory effort, occurring 5 or more times per hour of sleep. It is associated with a decrease in arterial oxyhemoglobin saturation of 4% or greater from baseline.

Sleep in patients without OSA is associated with pharyngeal narrowing and an increased negativity of inspiratory pressure, particularly during rapid eye movement (REM) sleep. In healthy subjects, pharyngeal dilator muscles maintain airway patency as negative pressure is generated by the diaphragm.

Patients with OSA have sufficient pharyngeal muscle dilator activity during wakefulness to maintain airway patency. However, that muscle activity is lost with the onset of sleep, and apneic episodes are most likely to occur during REM sleep. Pharyngeal dilator muscle activity then increases at the termination of the apnea, thereby reestablishing effective ventilation.

Patients with OSA generally have larger neck circumferences than patients who do not have OSA, although this does not, by itself, explain the pathophysiology of OSA. Excessive tissue in the surrounding wall of the oropharynx and nasopharynx compresses the compliant airway by redundant or fatty tissue and may make intrinsic muscular activity less effective. Exacerbation of upper airway narrowing may occur as a result of recurrent vibratory trauma from snoring and excessively negative inspiratory pressure.

Arousal during apnea is necessary for survival. The following mechanisms are responsible for arousal. Hypoxemia stimulates the carotid body receptor output. Hypercarbia stimulates central nervous system receptors. Ventilatory effort progressively increases as the apnea ensues as a function of the worsening hypoxemia and hypercarbia. As a result of a combination of these reflex responses and discharge of pressure-sensitive receptors, arousal occurs, activating pharyngeal dilator muscles and opening the collapsed airway, resuming effective ventilation.

Hypopnea is an abnormal decrease in the depth and
rate of breathing. The *apnea-hypopnea index* is the number of apneic episodes per hour of sleep. The apnea-hypopnea index can range from 6 to 50, and, as the index increases, so do the symptoms of OSA. Symptoms experienced by patients with OSA may include loud snoring, daytime somnolence, headaches, memory problems, anxiety, sexual dysfunction, impotence, and nocturia. Potentially life-threatening complications include arrhythmias, systemic hypertension, pulmonary hypertension, left ventricular hypertrophy, ischemic heart disease, stroke, pulmonary hypertension, and depression. Figure 2 illustrates the pathophysiology of OSA.

**Preoperative management**

Until recently, the incidence of OSA was thought to be higher in men at all age groups. However, recent research indicates that the predominance of OSA in men decreases with increasing age and that the effect of body mass index also decreases with increasing age. Body habitus and sex alone are not strongly predictive of OSA, and all patients should be asked preoperatively about their sleep habits.

Appropriate questions to ask the patient and his or her bed partner are as follows:
- Do you experience chronic, loud snoring?
- Do you experience gasping or choking during sleep?
- Do you experience excess daytime sleepiness?
- Have you had accidents during periods of wakefulness due to sleepiness?

The “gold standard” for establishing a diagnosis of OSA is polysomnography, which involves an overnight stay in a sleep laboratory. Evaluations of sleep staging, airflow and ventilatory effort, arterial oxyhemoglobin saturation, electrocardiogram, body position, and periodic limb movements are conducted. In the absence of polysomnography, an informal diagnosis of OSA can be made based on the preoperative assessment.

**Influence of anesthetic drugs on the upper airway**

Control of the pharyngeal dilator muscles involves central and reflex components. Central control of pharyngeal dilator muscles is diminished by virtually all drugs used in anesthetic care, including propofol, thiopental, opioids, benzodiazepines, nitrous oxide, and the halogenated inhalation agents.

The sensitivity and vulnerability of airway reflex components were demonstrated by Fogel et al., who applied local anesthesia to the nasopharynx and oropharynx of awake patients with OSA. In response to the application of the local anesthetic, partial airway obstruction occurred as a result of obtundation of airway pressure-sensing reflexes, a decrease in genioglossus muscle activity, and an increase in pharyngeal airflow resistance. This simple clinical manipulation is a classic representation of the Starling resistor model.

Similar findings were demonstrated by Eastwood et al. in studying the collapsibility of the upper airway.
with isoflurane. When the depth of anesthesia was decreased, the pharynx was less collapsible. Eastwood et al. also found that the primary site of airway collapse was the soft palate, challenging the widespread view of the tongue as the primary site of upper airway obstruction.

Postoperative management

The postoperative period is a particularly critical time for patients with OSA. Postoperative airway obstruction in the patient with OSA occurs due to the residual effects of sedatives, opioids, neuromuscular blockers, and halogenated inhalation agents. These drugs cause a decrease in central and reflex control of the pharyngeal dilator muscles. A potentially lethal situation occurs when an unmonitored patient with OSA with an unprotected airway receives sedatives or opioids.

As discussed, REM sleep is associated with an increased number of apneic episodes. Postoperative pain scores are the highest and REM sleep is most disturbed during postoperative day (POD) 1. An increase in REM sleep occurs after POD 1. Therefore, the patient with OSA is at risk for increased apneic episodes on POD 1 due to increased need for pain medication and thereafter due to increased REM sleep.

Ostermeier et al. reported several sudden postoperative arrests in patients with sleep apnea who were receiving epidural opioids. The patients received varying concentrations of bupivacaine, from 0.06% to 0.5%, and fentanyl, 10 µg/mL, at continuous infusion rates of 7 to 9 mL/h during their stay on an unmonitored unit. Each of the patients was found in cardiac or respiratory arrest on POD 2 or 3. None survived the arrest despite resuscitation measures and treatment with naloxone. It was thought that the arrests occurred on POD 2 or 3 due to the effective epidural analgesia, less monitoring, and an increase in REM sleep.

The findings of this study were alarming because epidural opioids were thought to be “safer” than parenteral opioids for postoperative pain control in patients with OSA. Opioids diminish central control of the pharyngeal dilator muscles. These drugs, administered epidurally and parenterally, diminish the arousal response to hypoxemia and hypercarbia.

Gupta et al. reported postoperative complications in patients with OSA undergoing hip or knee replacement. Three groups of patients were studied, two with OSA and a control group. All patients received parenteral morphine for postoperative pain control. The patients in the OSA groups had a higher incidence of reintubation, acute hypercapnia, episodic hypoxemia, and unplanned intensive care unit admissions.

A recent article in the Anesthesia Patient Safety Foundation newsletter reported an increased number of “unexplained” postoperative cardiopulmonary arrests in patients receiving parenteral opioids who ultimately were given a diagnosis of OSA. Many were diagnosed with OSA only after the patient’s history was discussed with family members after the arrest. Adequate preoperative assessment indicating OSA may have influenced postoperative treatment.

It seems that increased monitoring and vigilance during the postoperative period are vital. Remote pulse oximetry became available to hospitals in 2001. It provides pulse oximetry and heart rate data via pager and at a centralized monitoring station. Following epidural or parenteral opioid administration, patients with OSA should be considered at high risk for respiratory and cardiovascular complications. In the absence of remote pulse oximetry monitoring or an observational unit, patients with OSA should be monitored in the intensive care unit.

If continuous monitoring is not available, it is necessary to moderate opioid pain medication during the postoperative period. Moderate pain relief in this population may be an acceptable goal because the attenuation of arousal responses caused by complete pain relief may be the cause of respiratory complications. Alternatives to opioids such as regional anesthesia with local anesthetics, mixed agonist-antagonist drugs, partial agonist drugs, and nonsteroidal anti-inflammatory drugs should be considered.

Postoperative use of nasal continuous positive airway pressure

The use of nasal continuous positive airway pressure (n-CPAP) may be a valuable tool during the postoperative period for the patient with OSA. It works by delivering CPAP that “pneumatically splints” the upper airway. By delivering a set positive airway pressure during inspiration, n-CPAP increases proximal positive airway pressure above Ptissue, thereby preventing upper airway collapse. As shown in Figure 3, the clinical efficacy of n-CPAP can be explained entirely on the basis of the Starling resistor model.

Gupta et al. found that patients who used n-CPAP postoperatively had a lower incidence of serious complications and a shorter length of hospital stay. The complications found in the patients not using n-CPAP were reintubation, acute hypercapnia, episodic hypoxemia, and myocardial infarction.

In another study, conducted by Rennotte et al., patients with diagnosed OSA were studied during the postoperative period. The majority of these patients used n-CPAP continuously for 24 to 48 hours postop-
Anesthesia providers need to be aware of the acute and residual influences of anesthetic drugs on the complex orchestration of airway muscles and reflexes involved in upper airway patency in the patient with OSA. Preoperatively, all patients should be asked about their sleep habits. A high index of suspicion should be given to patients who have symptoms of OSA without a formal diagnosis. Patients with OSA who use n-CPAP at home should be advised during a preoperative evaluation to bring the CPAP device with them to the hospital.

The anesthesia provider should consider several factors when deciding to extubate the patient with OSA in the operating room. Nondepolarizing neuromuscular blocking drugs should be reversed fully to avoid their potential adverse postoperative effects on pharyngeal dilator muscles. The patient with OSA should be fully awake before extubation. Consideration should be given to using an indwelling airway exchange catheter such as the Cook catheter, which provides a mechanism to provide oxygenation and also facilitates rapid reintubation should the need arise.42

Close monitoring during the postoperative period is essential. If remote pulse oximetry is available, it should be used. Intensive care unit admission may be necessary to provide closer monitoring. N-CPAP should be used during the postoperative period. Persons caring for the patient with OSA during the postoperative period should have a high index of suspicion of respiratory complications. The risks and benefits to the patient with OSA having ambulatory surgery must be considered carefully. Moderation of opioids both parenterally and epidurally during the postoperative period is an important consideration. The risk of postoperative respiratory complications in the OSA population can be attenuated with meticulous care and communication by the anesthesia provider.

REFERENCES


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