PBLD – Table #9

You want airway obstruction on purpose!?  
Deep sedation with an unprotected airway for a 3 year old with SEVERE OSA.

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Goals:

1. Review the interpretation of sleep studies in children.

2. Appreciate the subtleties of different levels of sedations versus general anesthesia in children.

3. Appreciate the pharmacodynamics of respiratory depressant drugs in children with OSA.

4. Discuss the importance of an open dialogue between the anesthesia provider and the practitioners who request procedural conditions that may put your patient in jeopardy.

5. Review airway management and airway rescue approaches in children, particularly those with OSA.
Case Description:
A 3-year-old 23 kg male with severe OSA presented, on an outpatient basis, for flexible endoscopy of the upper airway. Despite having had a “routine” tonsillectomy and adenoidectomy 3 months prior in hopes of improving the symptoms of OSA, the patient continued to have snoring, excessive daytime somnolence, nocturnal choking episodes and observed nocturnal apneas.

Questions:
What additional history or physical exam findings would you seek before proceeding with the anesthetic? What specific history questions would you ask the parents in regard to his OSA? What signs and symptoms do children with OSA present? What are the anesthetic implications of a pediatric patient with OSA?

Case history and physical exam:
The only significant past medical history was obesity and chronic snoring. His parents deny any cardiac, respiratory, renal, neurologic, hepatic, or endocrine disorders, however they report excessive daytime somnolence and they have observed nocturnal choking and apnea episodes. On exam he is alert and interactive, obese at 23.5kg, HR 137, RR 26, BP 123/91, O2 sat 100% on RA. His airway appears normal for age.

Questions:
What is the expected post operative course following T&A? How does this affect you as the anesthesiologist? What elements of the physical exam are you most interested in? How reliable is an airway exam in a 3-year-old child? What additional information would you request, if any?

Sleep study results:
The sleep study obtained demonstrated severe obstructive sleep apnea that was associated with severe desaturation. Multiple obstructive apneas and hypopneas were observed. The apnea/hypopnea index (AHI) was 102 and the respiratory disturbance index (RDI) was 108. The lowest oxygen saturation during baseline sleep was 63% and the oxygen saturation was below 90% for 28% of the study. He also had significant sleep fragmentation because of multiple micro arousals due to respiratory events. There were no cardiovascular changes in relation to these obstructive apneas. The end-tidal CO2 tended to rise during the study and was above 50mmHg for 2% of the total sleep time.

Questions:
How do you interpret these results? What specific elements of the study might affect you as the anesthesiologist? What implication does OSA have for your perioperative anesthetic management?

Procedural Plan:
Following the T&A there was no significant improvement of the symptoms of OSA; therefore the child was referred to the pediatric pulmonary service for a flexible endoscopic exam of the
upper airway. The pulmonologist performing the study has requested “sleep-like” sedation in order to observe the anatomy of the supraglottic airway under conditions as close to normal sleep as possible. This is to be accomplished without any airway protective or opening devices. The exam is to occur in a procedure room distant from the main operating rooms.

Questions:
How do you assess adequate depth of sedation that will mimic “sleep like” conditions during a flexible airway endoscopy? How might the agents chosen impact your patient with OSA?

Intraoperative Care:
The patient was induced with 100% FiO₂ and 8% sevoflurane. He experienced multiple obstructive episodes with desaturation during induction, which was improved with jaw thrust and an oral airway. Intravenous access was obtained. A ketamine and propofol infusion was started and titrated during the case. The oral airway was removed prior to the onset of the endoscopic exam. The patient maintained adequate spontaneous ventilatory efforts and had occasional obstruction during endoscopy exam.

Questions:
What anatomic and physiologic changes occur at the varying depths of anesthesia? What are the effects of various anesthetic agents in regards to airway tone and degree of respiratory depression? Review the difficult airway algorithm and the airway maneuvers can be used to decreased upper airway obstruction in children.

Postoperative Care:
The patient again has multiple episodes of airway obstructions with desaturation after removal of the flexible scope and during emergence from anesthesia. His respiratory status improves after complete arousal from anesthesia and he is taken to the PACU for recovery. His O₂ saturation is 98% on 2L nasal cannula, but he drops to the low 90s on room air. The PACU nurses are asking for a sign out because his parents are anxious to get home to beat traffic.

Questions:
Is this patient ready to be discharged home? What criteria do you/your institution use to determine appropriateness of discharge to home? Are these criteria the same for post general anesthesia and post sedation? Are the criteria the same when there is documented OSA?

Comment [WBB1]: You had that the airway was “improved” by the endoscopy?? Do we want it left that way or should we just say that the kid had some obstruction during the exam.
Discussion:

Pediatric obstructive sleep apnea (OSA) has many implications for the anesthesiologist in the perioperative period. Like adults, children typically present with mild symptoms including snoring, observed nocturnal apneas or choking episodes with frequent arousals, enuresis, and daytime sleepiness or behavior issues associated with sleep deprivation. Cor pulmonale may be a later finding on EKG (1). Normal ventilation during sleep requires intact upper airway anatomy and coordinated airway reflexes, as well as proper ventilatory responses to hypoxemia and hypercapnia. Children with OSA may have alterations in one or more of these protective mechanisms. Anatomical alterations including soft tissue hypertrophy, craniofacial dysmorphism, neuromuscular weakness, or obesity may also predispose a child to have OSA (2). Children with OSA are at increased risk of obstructive events upon induction and emergence from general anesthesia. Airway management of these patients may be more difficult due to craniofacial abnormalities and redundant soft tissue. Children with OSA may have diminished ventilatory response to CO2; therefore drugs known to cause ventilatory depression must be used judiciously (3). Intraoperatively and post operatively, these children often display reduced opioid requirements due to possible upregulation of the mu opioid receptor; patients are also more prone to hypoventilation and hypoxia from respiratory depression (4).

The gold standard for diagnosis of OSA is a sleep study, or polysomnogram. This is a comprehensive evaluation of biophysical changes that occur during sleep. This study is most often performed at night, to mimic normal sleep patterns. Here, eye movement (EOM), brain activity (EEG), skeletal muscle activity (EMG) and cardiac function (EKG) are monitored. In addition, respiratory patterns, and interruptions in respiratory effort that result in hypoxia or hypercapnia are measured via respiratory airflow indicators, capnometry, and pulse oximetry. Sleep studies can be used to diagnose or rule out a variety of sleep disorders, including obstructive sleep apnea. Obstructive breathing irregularities identified during the study can be classified as apneas or hypopneas. Apnea is defined as a >90% reduction in airflow despite continued respiratory effort for at least two breaths. Hypopnea is a >50% reduction in airflow with associated respiratory effort for at least two breaths followed by an arousal and/or 3% oxygen desaturation. An apnea-hypopnea index (AHI) is then calculated as the average number of apneas or hypopneas per hour sleep. The RDI is calculated similarly to the AHI; however it includes respiratory disruptions not defined as either an apnea or hypopnea. Micro arousals seen on polysomnography typically result from respiratory disruptions and are defined as awakenings 1.5-3s in duration. For diagnostic purposes, an AHI >1 with a minimum oxygen saturation <92% satisfies criteria for OSA in children 1-12 years of age. The severity of OSA can be reflected by the AHI or RDI, as well as the frequency and severity of hypoxemia and hypercapnia (2).

Pediatric sedation, particularly in patients with OSA poses a risk for unintentional progression along the continuum of sedation. The ASA guidelines delineate four levels of sedation/anesthesia based on the physiologic changes present at each stage. These terms are as follows: (5)
1. Minimal Sedation “Anxiolysis” - Patients will respond normally to verbal commands, and no interventions are needed to maintain a patent airway. Cardiovascular functions are unaffected. Coordination may be impaired. This level of sedation is rarely adequate for diagnostic/therapeutic procedures in children.

2. Moderate Sedation/Analgesia – A drug-induced depression of consciousness during which patients respond purposefully to verbal commands and/or to light tactile stimulation. No interventions are required to maintain a patent airway. Cardiovascular function is usually maintained.

3. Deep Sedation/Analgesia – A drug-induced depression of consciousness during which patients cannot be easily aroused but will respond purposefully to repeated or painful stimulation. Reflex withdrawal is NOT considered a purposeful response. The ability to independently maintain a patent airway and ventilatory function may be impaired. Cardiovascular function is usually maintained.

4. General Anesthesia – A drug-induced loss of consciousness during which patients are not arousable. Airway reflexes are lost. The ability to independently maintain a patent airway and ventilatory function is often impaired. Positive Pressure ventilation may be required because of depressed spontaneous ventilation or drug-induced depression of neuromuscular function. Cardiovascular function may be impaired.

These terms were not specifically designed for children and furthermore, in children, deep levels of sedation are usually required to tolerate both painful and nonpainful procedures. There is also a tendency to misinterpret any response to stimulation as purposeful, and then underestimate the level of sedation. The levels of sedation, as defined by the ASA, make the assumption that there is a consistent correlation between depth of sedation and the ability to maintain a patent airway. This correlation has not been carefully studied in children, especially when different drugs are used for sedation, i.e. propofol, fentanyl, dexmedetomidine, ketamine, benzodiazepines etc. (6)

There are numerous case reports and clinical studies attempting to document the risks of sedation in children. The essential conclusions are that ALL sedatives and narcotics have caused problems even in “recommended doses” and ALL clinical areas using sedation have reported adverse events. Children 1-5 years old are at most risk. Respiratory depression and obstruction are the most frequent causes of adverse events. Adverse events occur because of multiple drugs, drug errors/overdose, inadequate evaluation, monitoring or practitioner skills, and premature discharge. (7)

Often pediatric procedural sedation will require advanced airway management, and the specific considerations of the pediatric patient need to be recognized. “What you see is what you get” is the maxim that describes the pediatric airway. It is unusual, in pediatric anesthesia, to stumble upon an unanticipated difficult airway, but this presumes that the airway is properly examined. Micrognathia, short neck, microstomia, and immobility of the neck all predict a difficult laryngoscopy. Syndromes with altered facial structure might also present with a difficult airway. (8) However, there are many anatomical and physiologic differences between the pediatric and the adult airway. The pediatric airway has: a small compliant trachea, an anterior/cephalad
glottis, small nares and jaw, a larger head (mainly the occiput), abundant soft tissue, a long narrow epiglottis, cords that slant anteriorly, a cricoid ring that is the narrowest point, horizontal ribs, a short trachea, and the larynx is cone-shaped. The physiology is also different from the adults with: increased O₂ consumption and CO₂ production, increased respiratory rate, alveolar ventilation twice that of adults, smaller FRC, ventilation is diaphragm dependent with easy fatigue of the diaphragm and intercostals, and decreased mechanical advantage of the chest wall.

The pediatric airway is most commonly lost immediately after induction. Loss of a patent upper airway when the child is breathing spontaneously may result from upper air obstruction, or laryngospasm. Airway obstruction is progressive and the dimensions of the small upper airway decrease further as the level of anesthesia is deepened. The airway dimensions can also be further compromised by the presence of large tonsils and adenoids, and with the relaxation of the genioglossus muscle with inhalation induction. As the airway begins to obstruct, as evidenced by suprasternal/supraclavicular retractions and paradoxical breathing, inspiratory noises due to a partially closed glottis may be heard. With complete airway obstruction and/or laryngospasm, the noisy airway suddenly becomes silent. Factors that predispose to laryngospasm in children include young age, greater ASA physical status, upper respiratory tract infection, airway anomaly, passive smoking, and foreign fluids/material in the airway. (9)

The pediatric anesthesiologist is often called upon to provide procedural sedation or general anesthesia for diagnostic or therapeutic procedures performed outside of the operating room theater. Clinicians, who are not familiar with pediatric sedation, and might be unaware of the physiologic responses when deep sedation is combined with medical conditions such as OSA, are the practitioners usually requested to perform these procedures. As a consulting physician, it is the role of the anesthesiologist to have a thorough discussion with the proceduralist prior to initiation of sedation or general anesthesia. It is important to understand the nature, duration, and indication for the procedure prior to providing anesthesia care. The anesthesiologist must reserve the right to delay or relocate a procedure if it is in the best interest of the patient. Effective, professional, communication between practitioners prior to and during pediatric sedation is essential for the safety of the patient and for good procedural outcomes.

Pediatric sedation, particularly in patients with anatomic or physiologic airway pathology such as OSA, presents a challenge for the pediatric anesthesiologist. Here, a thorough understanding of the patients' pathology, careful preparation and planning, and open discussion between practitioners must all occur for successful outcomes.
References:


