Pediatric Procedural Sedation

Essay

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Introduction

Children often present to physicians and other medical practitioners with painful conditions that require painful interventions.

Procedural sedation was defined by The American College of Emergency Physicians (ACEP) as "a technique of administering sedatives or dissociative agents with or without analgesics to induce a state that allows the patient to tolerate unpleasant procedures while maintaining cardio-respiratory function ***(American Society of Anesthesiologists, 2002).***

The Joint Commission on Accreditation of Healthcare Organizations (JCAHO) recognizes the risks involved with sedation and analgesia for these procedures and mandates that sedation practices throughout an institution be monitored and evaluated by the department of anesthesia ***(Coté and Wilson, 2006).***

Procedural sedation, as defined by the American Society of Anesthesiologists, occurs on a continuum, ranging from minimal sedation or anxiolysis to general anesthesia ***(Pitetti et al., 2003).***

The field of pediatric sedation has evolved significantly over the past two decades since the growing number of pediatric procedures requiring sedation outside of the traditional operating room setting ***(Coté and Wilson, 2006).***

Potential arenas for the sedation of children include the pediatric emergency department, intensive care unit, subspecialty procedure suites, ambulatory surgery centers, dental offices, and physician offices ***(Pitetti et al., 2003).***

Different medications and combinations of medications can be used to achieve the desired effect. Sedative drugs are medications that result in central nervous system depression. Use of these drugs may result in loss of protective reflexes, with subsequent respiratory and/or cardiac dysfunction.

Many of the clinical effects of medications administered to achieve sedation are dose-related and must be assessed individually for each child. Sedative drugs may be administered orally, intranasally, rectally, parenterally or by inhalation. Specific types of sedatives can be further defined by their characteristic or predominant clinical effect as: Sedatives, hypnotics, analgesics, anxiolytics, and Amnesic (antegrade) ***(Coté and Wilson, 2006).***

The most important guidelines for performing the sedation is the ability to manage all the potential complications, including hemodynamic instability, respiratory depression, and airway compromise ***(AAP Committee on Drugs, 2002).***

Aim of the Work

The aim of this work is to review the updates in pediatric procedural sedation for providing safe and effective sedation and analgesia in pediatric patients.

Chapter (1):
Pediatric Airway and Physiology

Pediatric Populations:

First of all, we should define pediatric subpopulations as shown below.

Table (1-1): Pediatric subpopulation, ***(Rudolph et al., 2002)***

Pediatric Subpopulation	Approximate Age Range
Newborn	birth to 1 month of age
Infant	1 month to 2 years of age
Child	2 to 12 years of age
Adolescent	12-21 years of age

Although the upper age limit used to define the pediatric population varies among experts, including adolescents up to the age of 21 is consistent with the definition found in several well-known sources ***(Rudolph et al., 2002).***

Through this essay we will be talking about procedural sedation for pediatrics with age group from 1 month to 12 years of age.

The Pediatric airway:

The most important feature of conducting safe pediatric sedation is the ability to assess and manage the pediatric airway. The upper airway is composed of three segments; the supraglottic, laryngeal and intrathoracic.

1. **Supraglottic**: the most poorly supported and collapsible segment of the upper airway that consists of the pharyngeal structures, and is the most impacted portion of the airway during sedation;
2. **Glottic (larynx)**: comprises the vocal cords, subglottic area, and cervical trachea;
3. **Intrathoracic:** consists of the thoracic trachea and bronchi.

(Pearson, 2002)

There are a number of developmental characteristics that distinguish the pediatric airway from the adult airway:

1. The pediatric airway is smaller in diameter and shorter in length.
2. The young child's tongue is relatively larger in the oropharynx.
3. The larynx in infants and young children is located more anteriorly.
4. The epiglottis in infants and young children is relatively long, floppy, and narrow.
5. In children younger than 10 years of age, the narrowest portion of the airway is below the glottis at the level of the cricoid cartilage.

(Steward, 2005)

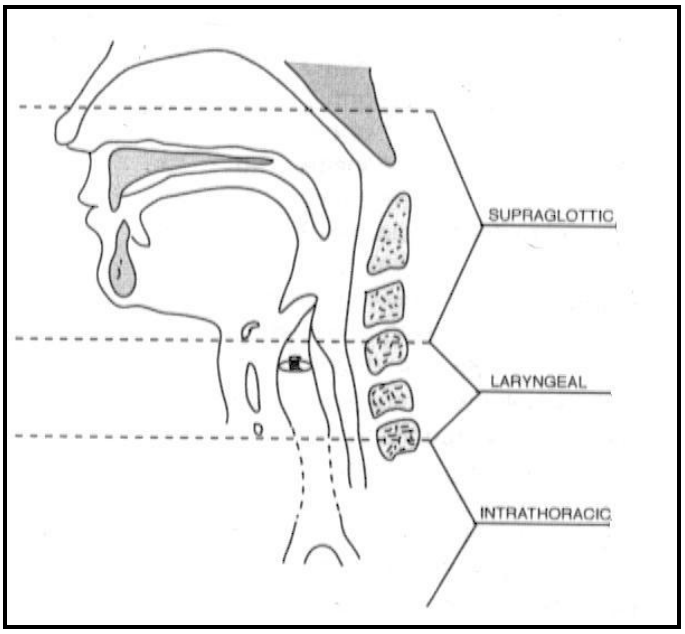

Fig. (1-1): Anatomy of the upper airway ***(Society for pediatric sedation, 2009)***

The small caliber of the pediatric upper airway, the relatively large tongue, and the "floppy" and relatively long epiglottis predispose young children to airway obstruction during sedation. In addition, the large occiput of the infant places the head and neck in the flexed position when the patient is placed recumbent, further exacerbating airway obstruction ***(Steward, 2005).***

During normal inspiration, negative intrapleural pressure generated in the thorax creates a pressure gradient from the mouth to the airways, resulting in airflow into the lungs. Extra thoracic airway caliber decreases during inhalation, whereas intrathoracic airway diameter tends to increase. Under normal

conditions, changes in airway caliber during respiration are clinically insignificant. However, significant narrowing of the upper airway increases airway resistance (R) and airflow velocity (Bernoulli effect), and a higher pressure gradient (ΔP) across the airway is required if minute ventilation is to be maintained as shown in (fig.1-2). Consequently, the greater negative pressure generated in the pharynx during inspiration tends to further collapse the upper airway ***(Litman, 2005).***

Neuromuscular control of the upper airway (cranial nerves IX, X and XII) is inhibited to a greater degree than diaphragmatic activity (phrenic nerve) during sedation/ anesthesia. Thus, the negative inspiratory pressure, that develop with diaphragmatic contraction, may reduce the diameter of the pharynx, a collapsible segment between two relatively well-supported structures, the nasal passage and the trachea. These areas typically involve narrowing of the anterior-posterior distance between the posterior pharynx and the soft palate, epiglottis, and, to a lesser degree, the base of the tongue. Consequently, the pharyngeal segment functions as a collapsible tube whose caliber is influenced by the pressure within the lumen of the airway ***(Hillman et al., 2003).***

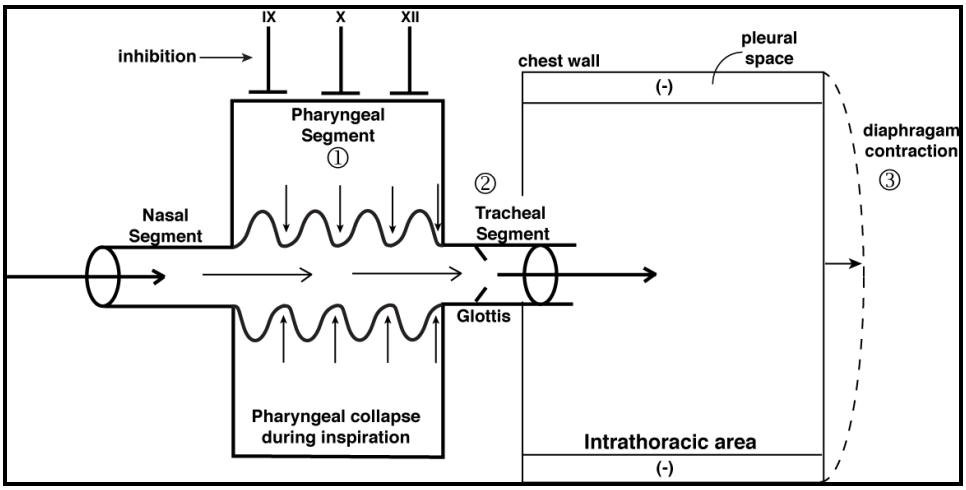

Fig. (1-2): The pharynx as a collapsible segment between two relatively well-supported structures ***(Society for pediatric sedation, 2009)***

Airway obstruction during moderate or deep sedation occurs in the supraglottic structures due primarily to the soft palate and epiglottis "falling back" to the posterior pharynx. While it was previously thought that the base of the tongue was the primary cause of upper airway obstruction during unconsciousness, MRI studies of the airway in sedated children demonstrate that the soft palate and epiglottis are the most likely structures causing pharyngeal obstruction ***(Steward, 2005).***

The other primary cause of upper airway obstruction during sedation is laryngospasm. Laryngospasm may be partial or complete and is defined as glottic musculature spasm. Risk factors for laryngospasm include secretions in the upper airway, airway manipulation, recent upper respiratory infection, gastro esophageal reflux disease, passive exposure to tobacco smoke, use of an airway device, young age and ASA classification

more than 2. Supraglottic obstruction and laryngospasm may be difficult to differentiate. One distinguishing feature of complete laryngospasm is the lack of response to simple airway maneuvers ***(Alalami et al., 2008).***

The keys to appropriately managing the pediatric airway during sedation are proper airway positioning and application of positive pressure ventilation when required. Routine management of the pediatric airway includes placement of the patient's neck in the sniffing position, and administration of "blow-by" oxygen. If obstruction persists despite these maneuvers, the patient's airway should be repositioned and a chin lift performed to move the supraglottic soft tissue structures, primarily soft palate and epiglottis, anteriorly and away from the posterior pharynx. If a simple chin lift fails to relieve the obstruction, this should be followed by a jaw thrust and application of positive pressure through a flow-inflating anesthesia bag and mask. Failure to relieve the obstruction following application of positive pressure (PEEP) suggests complete laryngospasm and requires positive pressure ventilation with cricoid pressure and endotracheal intubation when necessary ***(Hampson-Evans et al., 2008).***

Other anatomical considerations in pediatrics: Intra-vascular Access:

Children undergoing deep sedation should have an intravenous catheter in place. The availability of intravenous access allows the practitioner to administer medications that can

immediately treat airway obstruction, reverse bradycardia and administer specific reversal drugs for patients who become oversedated with benzodiazepines and opioids. Intravenous access also provides the practitioner with the ability to titrate sedative medications to a desired clinical effect. Cannulation of tiny pediatric veins can prove a challenge because of extensive subcutaneous fat. The saphenous vein has a constant location at the ankle and the practitioner can usually cannulate it even if it is not visible or palpable ***(Steward, 2005)***.

Pediatric Physiology:

Respiration: Respiratory Drive:

The basic drive to breath originates from within the central respiratory center located in the brainstem. Output from the respiratory center is modulated by a number of chemical (e.g., CO2, O2) and mechanical (e.g. lung mechanics) controllers. Changes in carbon dioxide concentration are among the most important determinants of respiratory drive from the medullary respiratory center. Minute ventilation (Tidal volume x Respiratory rate) typically increases linearly with rises in PCO2 ***(Roussos and Koutsoukou, 2003)***.

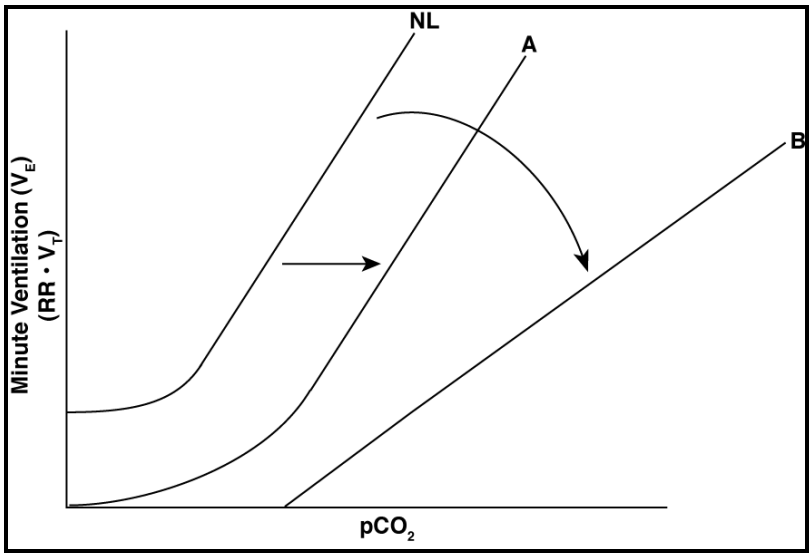

Fig. (1-3): Effect of sedative drugs on the central respiratory center's response to a given level of carbon dioxide where Line A represents light sedation and line B represents deeper sedation ***(Pattinson, 2008).***

Cardiovascular System:

Children differ from adults in their resting heart rate and blood pressure, Some sedative drugs can significantly affect vascular resistance and heart rate.

Heart rate:

A newborn routinely has heart rates up to the 150 beats/ min. with no cause for concern. With increasing the age, the heart rate slows considerably.

The heart rate may be higher during exercise or crying or in pain, and should be on the low end of the scale during sedation ***(Kliegman et al., 2007).***

Table (1-2): Normal heart rates (beats/min.) by age

Age	Normal Range Beats/ min.
Premature	120-170
0-3 months	100-150
3-6 months	90-120
6-12 months	80-120
1-3 years	70-110
3-6 years	65-110
6-12 years	60-95
Over age 12	55-85

(Kliegman et al., 2007)

Blood Pressure:

Blood pressure in children works in the opposite way as compared to the other parameters in that it tends to increase as children age. Blood pressure can be quite low in newborns, and remain on the low side until children reach toddlerhood.

Listed below are normal ranges for blood pressure in children ***(Kliegman et al., 2007).***

Table (1-3): Normal blood pressure rates (mmHg) by age ***(Kliegman et al., 2007)***

Age	Systolic mmHg	Diastolic mmHg
Premature	55-75	35-45
0-3 months	65-85	45-55
3-6 months	70-90	50-65
6-12 months	80-100	55-65
1-3 years	90-105	55-70
3-6 years	95-110	60-75
6-12 years	100-120	60-75
Over age 12	110-135	65-85

Central nervous system:

The following 3 myths regarding children and pain management are still commonly encountered: (Cravero and Havidich, 2011)

1. Children's immature central nervous system (CNS) cannot experience pain.
2. Children have no memory of pain.
3. Children easily become addicted to opioids.

The following 4 truths are still insufficiently appreciated: (Cravero and Havidich, 2011)

1. Even neonates demonstrate behavioral and hormonal changes in response to painful procedures

2. Children do not have to understand the meaning of pain to experience pain
3. Preemptive analgesia/anesthesia may decrease post injury opioid requirements
4. A child is likely to require deep sedation in many situations where an adult would require minimal or no sedation

At first it was presumed that infants exposed to the kind of stimuli they might experience in intensive care or surgery would either respond weakly because their pain system is too immature to function properly, or they would react in a similar way to adults but perhaps less efficiently. But to our surprise it was found that their reflex to pain or harm is greater and more prolonged than that of adults ***(Fitzgerald and Beggs, 2001).***

In addition to stimulation of nociceptors that send signals to the spinal cord and brain which prompt the body into awareness of pain, external factors also have as much, if not more, influence on a child's behavior. These include parental interactions with the child, preparation for the procedure, the clinician's skill, and the physical setting where the procedure is performed. Spending a few minutes preparing the anxious child for a procedure is always in the practitioner's best interest. Attempting to build trust and being honest about what will and will not hurt may go a long way toward ensuring cooperation ***(Wright et al., 2010).***