

Anesthetizing children: little people, big problems!

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At the time of graduation from residency, we possess the most knowledge of anesthesia than at any other time in our lives. Dr. S. Burwell, Dean of the Harvard Medical School, said in 1956: "Half of what you are taught as medical students will in 10 years have been shown to be wrong. And the trouble is none of your teachers know which half". Our understanding of the etiology and pathogenesis of many of the "big problems" that children present may lead to better outcomes and better quality of care, and it is the quintessential proposition for the delivery of excellence in clinical care to these "little people". In this lecture we review a number of contentious and controversial issues that face pediatric anesthesiologists on a daily basis and present practical, evidence based approaches to addressing them.

Key words: anesthetizing children, understanding of etiology and pathogenesis, excellence in clinical care

EPIDEMIOLOGY

Cardiac arrest is a widely used metric to gauge anesthetic risk. The incidence of anesthesia-related cardiac arrest and mortality in non-cardiac surgery in children has been reported to be between ~1 : 10,000–40,000 (1–3). The incidence of cardiac arrest is greater in children than in adults, and in infants <1 year of age compared with older children (1–4). Children with congenital heart disease and those undergoing cardiac surgery, those with hypovolemia and hyperkalemia from transfusion with old blood and those with respiratory disease pre-

sent additional risks of cardiac arrest. Other variables associated with an increased risk of cardiac arrest include ASA P/S \geq and emergency surgery.

PREOPERATIVE PREPARATION

Every child who is scheduled for anesthesia requires a complete preoperative history, physical examination and appropriate laboratory testing. Few laboratory tests are routinely performed in healthy children undergoing elective surgery, with the exception of pregnancy testing in menarchal women. The fasting guidelines, which are quite liberal and well enshrined in the ASA guidelines, are quite clear and unambiguous (5). However, how to handle a child who is chewing gum preoperatively was not addressed in the ASA guidelines and is not uni-

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formly handled by our colleagues. In a randomized study, children who chewed sugared or sugarless gum presented with twice the gastric fluid volumes than children without gum, but this was offset, in part, by a 10–15% greater pH in the children who chewed (6). A reasonable approach to the child who is chewing gum is to have him or her expectorate the gum preoperatively; if the gum is swallowed, the anesthetic is canceled. The time interval from expectorating until induction is usually one hour, giving adequate time for any excess gastric volume to return to normal.

Medication and material allergies present serious and complex problems for anesthesiologists. Allergies can present a confusing and complex picture that is not easily diagnosed in the preoperative period, particularly in children. Epidemiologic studies in children have determined that multiple (>4) surgeries, spina bifida and congenital urological problems (requiring repeated urethral catheterizations) predispose to latex sensitivity (and anaphylaxis) at an incidence of up to 70% (7–9). These children should be skin tested for latex allergy. Alternately, a clinical diagnosis of latex allergy testing may confirm their sensitivity by asking the parents whether their lips/tongue swell when the child contacts a toy balloon to their lips or the dentist inserts a latex rubber dam into the mouth. If latex anaphylaxis is suspected, avoid exposure to latex gloves and products that contact internal body structures. If a latex anaphylactic reaction occurs, then treat immediately with adrenaline using a sliding scale: 1–2 µg/kg for very mild bronchospasm up to >10 µg/kg for cardiac arrest. Evidence has demonstrated that removing latex products from the operating room and from mucosal contact (i. e., non-latex catheters) has all but eliminated this problem (8).

Although propofol has been in clinical use for more than 2 decades and deeply entrenched in our clinical practice, allergy and anaphylaxis to propofol has been poorly explained. The membrane stabilizing component of propofol, ovalecithin, is derived from egg yolk. Most allergies to egg are gastrointestinal allergies and they are caused by the egg albumin (egg white). There have been no allergies reported to lecithin in the literature. In a recent review of children with egg allergy who received propofol, only one child with a history of anaphylaxis to eggs experienced an anaphylactoid reaction

(10). Therefore, propofol may be given to children with egg allergy, but best avoided in those with egg anaphylaxis.

The most common presenting illness that can complicate anesthesia in infants and children is the URI. Although no prospective randomized studies have been conducted in children with URIs, one recent study has summarized the criteria for canceling anesthesia in a child with a URI (11):

1. Purulent oropharyngeal secretions.
2. Lower respiratory tract signs.
3. Fever >38.5 °C.
4. Change in sensorium, behavior, eating habits.

The presence of any one of these criteria is sufficient to cancel anesthesia for 4–6 weeks. I have used these criteria for more than 15 years and have not been disappointed. Children with lesser signs can be anesthetized safely: chronic rhinorrhea can be temporarily controlled with oxymetazoline or neosynephrine nose drops. Children who are younger than 1 year of age, premature, who had recent bronchiolitis, and who live in a second-hand smoking environment are at increased risk of perioperative airway problems (12).

Obstructive sleep apnea is the most severe form of sleep-disordered breathing, that may complicate T & A surgery as well as ANY OTHER surgery for which a child is scheduled (13). In the absence of a sleep study, clinical criteria do not reliably predict the presence of OSA in children. However, it is crucial to recognize those children with manifestations of OSA including morning somnolence, nocturnal enuresis, weight gain or loss, poor eating habits, poor school performance, inability to concentrate, attention deficit disorder and behavioral problems as possibly being associated with OSA. Snoring is an insensitive metric for diagnosing OSA. Caution must be exercised when administering opioids to children whose nocturnal saturation nadir is <85%; these children may express an exaggerated response to the usual doses of opioids (13). It would be preferable to avoid opioids in these children (use ketamine or tramadol), titrate the dose of opioids very carefully (beginning with very small doses) or, where possible, use regional blocks. Although the severity of OSA improves in most children after T & A surgery, as many as one-third of those with severe OSA do not improve by 6 months post-surgery and require further investigation and therapy.

Assessing the pediatric airway

“What you see is what you get” is the maxim that describes the pediatric airway. It is quite unusual to stumble upon a difficult airway that could not be anticipated a priori, in children. But this presumes that the airway was examined preoperatively. The child should be asked to open his/her mouth, stick out the tongue and/or extend the neck to assess whether a difficult airway exists. Micrognathia, short neck, microstomia, and immobility of the neck all portend a difficult laryngoscopy. It is crucial to have a resource text of syndromes immediately available to identify unfamiliar facies/disorders and determine whether the syndrome is likely to present a difficult airway (14).

Getting the child to the OR

Children of all ages have fears (14). Children aged 1–6 years present the greatest challenge as they are often quite resistant to separation from their parents. Consequently, it is in this age group of children where we expend most of our efforts during the preoperative period. Many strategies have been developed to minimize the emotional upheaval and anxiety during this stressful preoperative period. Education, distraction, and familiarity with the environment (including Child Life Instructors) are all effective to varying degrees (14–16). Parental presence has been a contentious practice in which parents have held that their presence is helpful in defusing the child’s anxiety although the literature presents a rather different interpretation (17).

Defusing the induction

Many healthy children are frightened when they first arrive in the operating room despite preoperative teaching and premedication. Before leaving the parents, we offer the child a choice of flavors for the mask (flavored lip balms) and permit the child to apply it to the mask. In the OR, the child (2–6 years old) sits on the OR table with the child’s back resting against your chest as we apply the pulse oximeter probe. We bring the mask towards the child’s face slowly with only 70% nitrous oxide in oxygen flowing while reminding the child of the flavor he/she picked. We avoid sevoflurane at the outset because of its pungency. If the child is concerned about the odor of sevoflurane, we rotate the mask 90° so the cuff occludes the nose. As soon as the child ceases to interact with you while breath-

ing nitrous oxide, we administer a single step-wise increase in sevoflurane concentration to 8%.

Refusing the mask

Some children have been stigmatized by previous bad experiences during induction of anesthesia and refuse to breathe the dreaded “mask”. One solution is to remove the mask from the circuit, place the elbow of the circuit between your fingers and cup your hands under the child’s chin (nitrous oxide is heavier than air). Gradually bring your hands closer and closer to the nose/mouth while distracting him/her with conversation/song until there is a virtual mask seal. At this point, 8% sevoflurane is introduced and/or the mask is reattached.

INDUCING ANESTHESIA

We administer 70% nitrous oxide for 1–2 minutes and then introduce 8% sevoflurane in one step. For children >6 years, we can perform a single breath induction with sevoflurane (18). In this case, the anesthetic circuit is primed with 8% sevoflurane ± nitrous oxide before the child arrives in the OR. We practice taking vital capacity breaths with the child. At end-expiration, we apply the mask instructing the child to take a vital capacity breath through the mouth and hold it while you count aloud. A single breath induction will be demonstrated with a video recording.

Once consciousness is lost during a sevoflurane induction, even while administering 8% sevoflurane, the depth of anesthesia is not as deep as it is with halothane. When intravenous access is attempted within 30 seconds of loss of eyelash reflex, the child is more likely to withdraw the extremity and develop laryngospasm (>5%), whereas when the attempt is delayed for 120 seconds, movement is diminished and laryngospasm does not occur (19). It is important not to decrease the inspired concentration of sevoflurane or eliminate nitrous oxide unless respiration diminishes because the children are lightly anesthetized. Respiratory depression and apnea are much more likely to occur if the child was premedicated with midazolam, and far less likely to occur if no premedication was used. When respiration diminishes, simply assist ventilation manually while maintaining a normal carbon dioxide tension. To facilitate a return of spontaneous ventilation, gradually decrease

the concentration of sevoflurane or nitrous oxide. Some recommend that the inspired concentration of sevoflurane should be decreased to 1.5 MAC as soon as the eyelash reflex is lost, to avoid epileptiform EEG activity in children (20). The risk of epileptiform EEG activity appears to increase in the presence of large concentrations of sevoflurane (i. e. 8%) and hyperventilation (20, 21), although frank seizures are exceedingly rare. The risk from decreasing the concentration of anesthetics as soon as consciousness is lost or during periods of stimulation is awareness (22, 23).

The airway

The airway is most commonly lost during or immediately after induction of anesthesia or after extubation of the trachea. Loss of a patent upper airway when spontaneously breathing through a mask may result from upper airway obstruction, often relieved by manually adjusting how the facemask is applied to the face. Application of a proper jaw thrust (24) will be demonstrated using a video (described below). If the jaw thrust does not establish an airway, laryngospasm may be the cause. Factors associated with laryngospasm include young age (infants), greater ASA physical status, upper respiratory tract infection, airway anomaly, passive smoking, foreign fluids/material in the airway and laryngeal mask airway (25). To prevent the development of laryngospasm or to stop it from progressing, it is important to review a management algorithm (26). A properly fitting facemask is essential. If the airway begins to obstruct (as evidenced by suprasternal and supraclavicular retractions, chest wall in-drawing and extreme diaphragmatic excursions), inspiratory noises (crowing) due to a partially closed glottis may be heard. With airway closure (i. e. laryngospasm), the noisy airway becomes silent. To remedy this rapidly deteriorating situation, a tight-fitting facemask should be held to the face with 100% oxygen flowing at an appropriate rate. The pop-off valve should be closed to pressurize the breathing circuit to maintain 5–10 cm H₂O pressure during expiration. Rather than use oral airways, I apply the “jaw thrust” maneuver to the **condyles** (i. e. not to the angle of the mandible) of the mandible as will be demonstrated (24). This will be demonstrated with videos during the lecture. No fingers other than the single digit applied to each condyle need touch the mandible.

Whatever you do, DO NOT FORCE GAS into the “lungs” against a closed glottis because the gas preferentially inflates the stomach. If the child is making small respiratory efforts (i. e. faint but audible noises are present), then augment the respiratory efforts in synchrony with the child’s efforts. If the laryngospasm does not abate, respiratory chest movement continues without vocalization and the capnogram is undetectable, administer atropine 20 µg/kg and propofol 1–2 mg/kg IV (27, 28). It is prudent not wait until hypoxia relaxes the vocal cords. Rather, intervention before the saturation is too low or bradycardia is detected by administering intravenous succinylcholine. If complete laryngospasm has occurred, then promptly administer atropine followed by succinylcholine, 0.5–1.0 mg/kg intravenously or 3–4 mg/kg intramuscularly, if no intravenous access is available (14).

EMERGENCY

Emergence from inhalational anesthesia in children generally follows three stages: 1) early arousal with bucking and coughing (at which time the trachea of adults but not children may be extubated); 2) a quiet period of straining, apnea or shallow breathing; and 3) bucking, coughing and purposeful movements. During the second phase of emergence, oxygen saturation often transiently decreases and decreases dramatically, even with the tracheal tube in place. This often occurs after the child’s cough, breath-holding or strains, all of which reduce the FRC and increase V/Q mismatch. To prevent or reverse the desaturation, ventilate the lungs manually with 100% oxygen, use positive end-expiratory pressure as needed to recruit alveoli and, above all, be patient. If the tracheal tube is still in the larynx, oxygen saturation will return to baseline with adequate ventilation. Extubation of the trachea in children should occur only during the third phase of emergence.

Laryngospasm may also occur immediately after extubation in children. It occurs when the tracheal tube is removed during a light plane of anesthesia or if a foreign body (blood, gastric juice or mucous) irritates the larynx. To avoid laryngospasm, remove the tube during the third stage only. If you are not sure it is the right time to extubate the trachea, leave the tube in for an extra few minutes! Removing the tube prematurely prolongs the anesthetic

by about 3 times the minutes “saved”. If laryngospasm occurs at this time, apply the jaw thrust as described above, releasing the pressure every two or so minutes. Pressure at the head of the condyles of the mandible is exceedingly painful and prompts the child to try crying or vocalizing. This forces the child’s vocal cords open breaking laryngospasm. If this maneuver fails to relieve the laryngospasm, follow the algorithm and add 100% oxygen with positive pressure, and prepare the laryngoscope and tracheal tube, atropine and succinylcholine as needed.

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VAIKŲ ANESTEZIJA: MAŽI ŽMONĖS, DIDELĖS PROBLEMOS!

Santrauka

Baigdami rezidentūrą mes turime daugiausia anestezijos žinių nei bet kuriuo kitu gyvenimo etapu. 1956 m. Harvardo Medicinos mokyklos dekanas dr. S. Burwellas sakė: „Po 10 metų paaiškės, kad pusė tų žinių, kurias jūs įgijote būdami medicinos studentais, nėra teisingos. Problema ta, kad nė vienas iš jūsų dėstytojų nežino – kuri pusė.“ Daugelio „didžiųjų problemų“, kurios pasitaiko dirbant su vaikais, etiologijos ir patogenezės suvokimas yra esminis uždavinys siekiant nepriekaištingos šių „mažų žmonių“ klinikinės priežiūros. Šiame straipsnyje apžvelgiame daugybę ginčytinų ir kontroversiškų problemų, su kuriomis dažnai susiduria vaikų anesteziologai, ir pateikiame praktinius, įrodymais pagrįstus jų sprendimo metodus.

Raktažodžiai: vaikų anestezija, etiologijos ir patogenezės suvokimas, nepriekaištinga klinikinė priežiūra