# Pitfalls and Problems in Pediatric Anesthesia

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The etiology of cardiac arrest in the pediatric patient has changed as practice has changed in the care of these patients over the past 20 yr. The Pediatric Closed Claims Study in 1993 showed respiratory events were the most common category accounting for 43% of claims with inadequate ventilation seen in half of the respiratory events. The typical profiles in this category of inadequate ventilation were healthy, non-obese children breathing halothane spontaneously whose arrest was preceded by hypotension or bradycardia. These children were difficult to resuscitate successfully, 70% died and 30% had permanent central nervous system impairment. Pulse oximetry was used in 7% of the Closed Claim cases and capnometry was used in 5% (1). Recently the Pediatric Perioperative Cardiac Arrest (POCA) Registry has provided some new data. Out of 1,089,200 anesthetics, there were 150 cardiac arrests that were deemed anesthesia related (1.4/10,000) (2). Several points are relevant in analysis of this data.

First, an increased incidence of cardiovascular causes (32%) differed from previous studies including the Pediatric Closed Claims Study in 1993 in which only 13% were from cardiovascular causes. This may have some basis on the fact that using chest compression was necessary to be included in this new data or the fact that the use of pulse oximetry in 98% and capnography in 86% of cases may be more effective in preventing respiratory than cardiovascular events. Most of the cardiac arrests (82%) occurred during induction or maintenance of anesthesia. Bradycardia (54%), hypotension (49%), abnormality of $Spo_2$ (46%) or inability to measure blood pressure (25%) were the most common antecedent events. Twenty-one percent of arrests occurred during emergency surgery.

Second, infants are at increased risk. Infants <1 yr of age accounted for 55% of the anesthesia related cardiac arrests. Several pediatric studies have confirmed that infants <1 yr have the highest anesthetic risk (3,4) and that mortality is inversely proportional to age with the highest risk in the <1 mo of age group (5). This may be related not only to a higher ASA physical status classification with underlying patient disease (particularly congenital heart disease) but also to cardiovascular depression by inhalational agents. In infants <30 days of age the MAC of halothane in oxygen is 0.87 (6), compared with MAC of 1.08 in children 1–6 mo of age (7). With isoflurane, the MAC for preterm infants (<32 wk) is 1.28, 32–37 wk is 1.41 (8), and for term (0–1 mo) 1.60, with 1–6 mo being 1.87. Only sevoflurane appears to be different with the MAC being constant at 3.2–3.3 for neonates and infants less than 1 mo, decreasing to 3.0 at 1–6 mo, and 2.5–2.8 for 7 mo–12 yr (9). Studies have shown that sevoflurane may be less of a myocardial depressant and have less potential for producing bradycardia than halothane in infants (10,11). Sevoflurane may be beneficial in children with congenital heart disease, who were particularly at risk in the POCA data. A recent study showed that children with congenital heart disease who received halothane experienced twice as many episodes of severe hypotension as those who received sevoflurane. Recurrences of hypotension occurred despite increased vasopressor use in the halothane treated patients compared with the sevoflurane treated patients. An increased incidence of hypotension was seen in children less than 1 yr of age compared with older children and patients with preoperative cyanosis had a higher risk of developing severe desaturation. Thus, sevoflurane may have hemodynamic advantages over halothane in infants and children with congenital heart disease (12). In addition, if an infant is intubated without muscle relaxants twice the dose of inhalational agent is needed in 95% of children. This will cause more myocardial depression (13).

Third, 33% of all anesthesia-related cardiac arrests occurred in previously healthy ASA I–II patients. These were mostly medication-related errors (64%). Fifty percent of the arrests caused by halothane cardiovascular depression were seen at inspired concentrations of 2% or less, with the median age being 6 mo. Controlled ventilation may accelerate the increase in halothane concentration especially with prolonged exposure as a result of difficult IV access. Four cases of arrest occurred after probable intravascular injection of local anesthetics. These occurred during combined halothane and caudal anesthesia with injection of bupivacaine 0.25% with 1/200,000 epinephrine despite negative test dose and aspiration. All had ventricular

arrhythmias but were successfully resuscitated without injury.

Mortality rate in ASA III–V patients was 37% compared with 4% in ASA I–II patients. ASA III–V was the strongest predictor of mortality followed by emergency status. Overall mortality rate in all arrests was 26% (2).

## Classification of Cardiac Arrests

### *Cardiac Disease*

Although most patients who present with a previously undiagnosed heart murmur do not have significant pathology, some do have anatomic disease. Lesions that are implicated with problems during anesthesia are those that include pulmonary hypertension, hypoplastic pulmonary arteries and ventricles and left to right shunts. Murmurs should be characterized before surgery, especially in infants. A history of easy fatigability or poor feeding with failure to thrive should alert the anesthesiologist that this might be a pathologic murmur. A call should be placed to the pediatrician to see if the murmur has been characterized—if not, a pediatric cardiology consult possibly with ECHO may be necessary before surgery.

Sudden death does occur in pediatric patients and unfortunately can occur during a surgical procedure. In children <1 yr of age the most common cause is ductus dependent, complex cyanotic congenital heart disease, followed by myocarditis and prolonged QT syndrome. From the first year through the third decade the most common cardiac causes are myocarditis, hypertrophic cardiomyopathy, coronary artery disease and congenital anomalies, aortic dissection, and conduction system abnormalities. In older children, prodromal symptoms are seen in 50% of patients with the most common symptoms being syncope or chest pain. ECG abnormalities along with symptoms during high-risk behavior (strenuous exercise) such as palpitations or chest pain, need prompt cardiac evaluation, especially if symptoms are elicited in a preoperative interview (14).

### *Difficult Airway*

Loss of a patent airway has been a common cause of acute deterioration and cardiac arrest. In the patient with a difficult airway, mask ventilation or intubation may be impossible. It is important to maintain spontaneous respiration with an inhalational agent such as sevoflurane in these patients. It is also important to have a variety of airway equipment available to deal with these situations. This may include various size masks, airways, Laryngeal Mask Airways (LMAs), Bullard laryngoscope, and pediatric fiberoptic bronchoscopes. Also obstructed tubes, esophageal intubation, or dislodged tubes may precede arrest. In small children, sounds of air passage may be transmitted from the esophagus and are misinterpreted as being from the airway. Obstruction or kinking of the tube may cause progressive hypoxemia or hypercarbia, making resuscitation more difficult.

### *Intravascular Volume*

Intravascular fluid loss and current volume status are often underestimated in the pediatric patient especially in newborns. Poor vascular access may compound these problems. Assessment of intravascular volume depends more on clinical signs than invasive measurements that are used in the adult. By the time the pediatric patient becomes hypotensive they are severely behind in fluid and can be close to a cardiac arrest situation.

### *Inhalational Agents*

Anesthetic agent overdose, particularly halothane, in the face of decreased intravascular volume, is one of the most common causes of sudden hypotension, especially in infants. Bradycardia (<100 bpm) is an ominous sign in infants. In a study of causes of bradycardia in infants <1 yr of age, 1/3 was due to inhalational agent, 1/3 to hypoxemia and 1/3 to patient disease or surgical factors (15). Inhalational agent overdose responded to discontinuation of the agent and atropine in most cases but some needed epinephrine and chest compression. In addition, continuous auscultation of heart sounds is a clinically useful tool for the hemodynamic monitoring of anesthetized infants and children. In a recent study under halothane anesthesia a dramatic dose dependent decrease in amplitude of $S_1$ and $S_2$ heart sounds occurred in all 19 patients ages 6 mo–12 yr. Monitoring was accomplished by a precordial stethoscope. These changes were clearly audible, occurred rapidly, and were followed by corresponding decreases in heart rate and blood pressure (16). Thus, heart sound changes may be an early warning sign of decreased cardiac function and impending disaster. Dysrhythmias occurred when halothane was administered with "nasal masks" for dental surgery. A strong association between halothane and ventricular arrhythmias particularly ventricular tachycardia was seen; however, all episodes resolved spontaneously. No serious dysrhythmias were seen with sevoflurane. Unfortunately end-tidal $CO_2$ level was not reported in this study (17).

### *Succinylcholine-Induced Arrest*

In infants who have not been given atropine, especially in the presence of hypoxemia, the potential for bradycardia with succinylcholine is considerable. Administration of succinylcholine to a patient with unrecognized myopathy can result in massive potassium

release and sudden arrest. This is not malignant hyperthermia (MH), which has a slower onset. Also, rhabdomyolysis may present with bradycardia or cardiac arrest in contrast to tachycardia, tachypnea, PVCs, hypertension and hyperthermia, which are common with MH.

### *Topical Vasoconstrictor-Induced Cardiac Arrest*

Application of topical vasoconstrictors, such as phenylephrine, has led to arrests in pediatric patients undergoing general anesthesia. The scenario is severe hypertension in which baroreceptor-induced bradycardia does not follow because of pretreatment with atropine or glycopyrrolate. In response to these hemodynamic changes, $\beta$-blocking agents are sometimes administered. This has led to pulmonary edema, cardiac arrest, and death. Cardiac arrest has occurred with both esmolol and labetalol but only labetalol has been associated with death, perhaps because of the briefer duration of action of esmolol. Isoproterenol, epinephrine, and dopamine did not consistently reverse this myocardial dysfunction but glucagon (which has been effective in reversing the cardiovascular effects of $\beta$ blockers) may be beneficial. The dose is 5–10 mg in adults, 1 mg in children (infusion of 1–5 mg/h). Current recommendations for topical phenylephrine use are limiting the dose to 20 $\mu$g/kg in children up to 25 kg and, if hypertension ensues, waiting 10–15 min before antihypertensive agents are given, then using direct vasodilators or $\alpha$ receptor antagonists. Administration of $\beta$ blockers, calcium channel blockers and increasing inhalational agents should be avoided while glucagon should be considered to counteract the $\beta$-blocking effect if these drugs have been administered. Using a topical vasoconstrictor such as oxymetazoline 0.025–0.05% may be safer than phenylephrine 0.25–1% (18).

### *Intravascular Local Anesthetic Injection*

In the POCA Registry data intravascular injection of local anesthetic during caudal anesthesia occurred despite negative aspiration and lack of response to a test dose. This has occurred with both needle and catheter use. Incremental injection rather than bolus injection has been advised for earlier detection of an intravascular injection (19). Also use of agents with less myocardial toxicity such as ropivacaine or levobupivacaine may be indicated.

### *Allergic Reactions*

Allergic reactions may occur in pediatric patients in relationship to drug administration (e.g., antibiotics, muscle relaxants) or on repeated exposure to latex. Clinical signs of anaphylaxis include increased vascular permeability (urticaria and laryngeal edema), contraction of smooth muscle (bronchospasm), vasodilatation (flushing and hypotension), stimulation of sensory nerve endings (pruritus), and cardiac histamine receptor stimulation (tachycardia and arrhythmia). Allergy to muscle relaxants has been shown to be responsible for more than 80% of anaphylactic shock during anesthesia (20), but is less frequent in children than adults (21). Recently, an anaphylactic reaction to cisatracurium has been reported in a child (22). Latex allergy has been implicated in 17% of episodes of intraoperative anaphylaxis and is the most frequent cause in children (21). Although it has been associated in patients with neural tube defects it has also been reported in patients after multiple surgeries (23) and even in children receiving prolonged mechanical home ventilation (24). Total avoidance of latex products is essential in these patients. Latex allergy may present up to 1-1/2 h after starting the case. The onset is of sudden bronchospasm followed by profound hypotension seemingly unrelated to any other events. Although a delay in onset may be seen once the signs become evident, progression to hypotension and arrest may be rapid (25). Treatment of suspected intraoperative anaphylaxis should include 100% oxygen, discontinuance of anesthetics, blood, antibiotic and muscle relaxant infusions, and withdrawal of exposure to latex. Epinephrine should be administered along with volume resuscitation, $H_1$ and $H_2$ blockers, and steroids. Albuterol inhaler may be very effective in treating bronchospasm and sodium bicarbonate can be used for persistent acidosis and hypotension. Blood samples should be sent for allergy investigation including immunoglobulin E antibodies to latex (if suspected), tryptase, and histamine levels.

### *Investigation and Management of Intraoperative Cardiac Arrest*

1. Pulse oximetry is an early warning sign of developing hypoxemia or decreased perfusion and precedes clinical signs in anesthetized children (26). If the pulse oximeter stops working and the noninvasive blood pressure monitor keeps reading, something is wrong. Don't ignore the monitors.
2. An absent or poor capnograph tracing is indicative of loss of cardiac output or impaired ventilation. It may be the earliest warning of events with the greatest likelihood for serious morbidity, even before the onset of desaturation (27). The amount of expired $CO_2$ can correlate well with cardiac output during cardiopulmonary resuscitation (28). End-tidal $CO_2$ may also have some prognosticative ability, if the level is 10 mm Hg or less measured 20 min after initial

resuscitative efforts, death can be predicted with 100% sensitivity and specificity (29).

3. A stethoscope monitor is invaluable. Changes in intensity of heart sounds may alert you to problems before bradycardia and hypotension become apparent.
4. The airway must be rechecked when the cause of sudden deterioration is unclear. If the trachea is not intubated, intubate immediately. If this is not possible (because a difficult airway), use an LMA or bag and mask ventilation. Children in out-of-hospital arrests whose airway management was randomized to receive bag and mask ventilation until they reached the hospital had outcomes that were statistically identical to those in whom the trachea was intubated in the field (30). Look for common problems first: airway, volume status, and inhalational agent overdose. Discontinue anesthetic agents and administer 100% oxygen.
5. Start CPR early—it can be effective in maintaining adequate circulation. To be effective a peripheral pulse should be discernible.
6. Obtain a blood gas and electrolyte panel—this can be helpful in determining the cause of the arrest.
7. Epinephrine is the single most useful drug. Don't waste time with repeated atropine doses. The dose recommended by the American Heart Association is 10 $\mu$g/kg administered IV every 3–5 min or 100 $\mu$g/kg intratracheally. Large dose epinephrine (100–200 $\mu$g/kg) may cause post arrest myocardial dysfunction and necrosis but may be useful if the diastolic pressure is $<$20 mm Hg. There is inadequate pediatric data for routine use of vasopressin during cardiac arrest.
8. Routine calcium administration does not improve outcome but is indicated for hyperkalemia, hypermagnesemia, calcium channel blocker excess, and documented hypocalcemia. It is not indicated for electromechanical dissociation or asystole (31,32).
9. Magnesium should be used for hypomagnesemia and Torsades de pointes (polymorphic ventricular tachycardia) at a dose of 25–50 mg/kg with a maximum dose of 2 g.
10. Routine administration of sodium bicarbonate does not improve outcome and should be given only for severe metabolic acidosis, hyperkalemia and hypermagnesemia at a dose of 1 mEq/kg.
11. Obtain a chest radiograph to help rule in or out the cause of the arrest. Tension pneumothorax should be on your differential diagnosis list.
12. Have ready access to a defibrillator. Be sure it is working properly and that pediatric paddles are available. Defibrillation with 2–4 J/kg is the mainstay of therapy for pulseless ventricular fibrillation (VF) and ventricular tachycardia (VT). Amiodarone is an alternative to shock resistant VF and VT. The recommended loading dose is 5 mg/kg over several minutes with repeated doses up to 15 mg/kg. Side effects include hypotension, which is due to the antagonism of adrenergic receptors and calcium channels and may require calcium salts and vasopressors for treatment.

## Laryngospasm

Laryngospasm is a frequent occurrence during administration of anesthesia to pediatric patients. The incidence is 8.7/1000 patients in the total population and 17.4/1000 in those aged 0–9 yr (33), with the age group 1–3 mo having the greatest incidence (more than three times the rate of any other age group). Although laryngospasm has been seen as a self-limiting complication, 5/1000 laryngospasm patients have a cardiac arrest (33). Other complications include bronchospasm, hypoxia, gastric aspiration, arrhythmias, and delayed recovery. In addition, pulmonary edema has been reported as a serious complication (34).

### *Etiology*

Laryngospasm occurs as a response to glottic or subglottic mucosal stimulation and continues long after the stimulus is gone. Risk factors include younger age, nasogastric tube or oral airway placement, endoscopy or esophagoscopy, upper respiratory infections, or volatile anesthetics (induction with isoflurane and desflurane).

Two areas have recently come into focus as relevant to the risk of development of laryngospasm. First, of 15,183 children in a day surgery unit who had general anesthesia, those who developed laryngospasm were 2.05 times more likely to have active upper respiratory infection. They were also more likely to be younger and be undergoing airway surgery (35). Anesthetizing a child with URI may have a higher incidence of complications if intubation is required. Bronchospasm is more frequent in intubated patients (36,37). In children with a URI, use of an LMA instead of tracheal intubation has a similar incidence of laryngospasm, but the incidence of mild bronchospasm was higher in the intubated group. Therefore, the LMA may be a suitable alternative if the decision is made to proceed with anesthesia in a child with URI (38).

Secondly, in children exposed to environmental tobacco smoke, 9.4% developed laryngospasm compared with only 0.9% without domestic environmental tobacco smoke exposure. All episodes occurred on emergence from general anesthesia. The risk for laryngospasm is greater if the source of passive smoke is from the mother (39,40).

### *Management*

Laryngospasm may present as complete or incomplete airway obstruction. If incomplete airway obstruction is present, sounds should be present (e.g., grunts, expiratory squeaks). Complete or incomplete airway obstruction should have initial treatment with jaw thrust and chin lift. The middle finger of each hand should be placed in the "laryngospasm" notch, which is situated behind the lobule of the pinna of each ear. It is bounded anteriorly by the ascending ramus of the mandible adjacent to the condyle, posteriorly by the mastoid process of the temporal bone and cephalad by the base of the skull. Press firmly inward toward the base of the skull with both fingers at the same time lifting the mandible at the right angle to the plane of the body (jaw thrust or forward displacement of the mandible). This will convert laryngospasm within one or two breaths to laryngeal stridor and then to unobstructed respirations (41). This should include administration of 100% oxygen with gentle positive pressure. It must be remembered that incomplete airway obstruction may rapidly become complete.

Complete airway obstruction shares many of the signs of incomplete airway obstruction - tracheal tug, retractions of the chest wall and marked abdominal respiration, but there is no sound present. Positive pressure will not "break" laryngospasm in the presence of complete airway obstruction (42). It may worsen it by forcing supraglottic tissues downward into the glottic opening. High pressure generated by the flush valve may dilute anesthetic gases and lead to a lighter level of anesthesia. It may also force gas down the esophagus into the stomach, making ventilation more difficult. If IV access is present succinylcholine 1.5 mg/kg with atropine 0.02 mg/kg can be given. If no IV access is present IM succinylcholine 4 mg/kg is recommended to be given in the deltoid muscle. Intralingual atropine and succinylcholine can cause ventricular arrhythmias in patients anesthetized with halothane (43). If laryngospasm is sustained and the child is extremely hypoxic it may be necessary to intubate without muscle relaxants. If these measures have not proved successful in securing the airway, cricothyrotomy or emergency tracheostomy may be required.

### *Prevention*

Mucosal stimulation and tracheal suctioning should only be done when the patient is deeply anesthetized. The effect of lidocaine is controversial. IV lidocaine 2 mg/kg given 1 min before extubation may attenuate or prevent laryngospasm. However Leicht (44) has suggested that IV lidocaine does not prevent laryngospasm if the trachea is extubated after the patient begins to swallow. To benefit from lidocaine, patients must be extubated before swallowing begins.

## Aspiration

A previous study reported that the incidence of anesthesia related pulmonary aspiration in a university affiliated pediatric hospital was 0.10% (twice that reported in adults) (45). In a new retrospective report, the pediatric groups at risk were identified by Warner (46). In 56,138 infants, children and adolescents younger than 18 yr of age underwent 63,180 general anesthetics. Anesthesia for elective procedures comprised 93.5% of all cases. The overall frequency of perioperative pulmonary aspiration was 1:2,632 anesthetics, 24 patients (0.04%). Emergency procedures were associated with a greater frequency of aspiration than were elective procedures (1:373 vs 1:4,544 anesthetics, $P < 0.001$). There was no difference across ages or ASA physical status classifications. Seven of the 24 patients were ASA I–II children undergoing elective procedures (1 in 8,000 cases), the same ratio as the adult population. None of these aspirations resulted in serious pulmonary complications.

Fifteen of the 24 children did not develop respiratory symptoms within 2 h. Five of the other nine needed respiratory support, with three needing mechanical ventilation for >48 h. None died from the pulmonary aspiration. If patients were asymptomatic within 2 h of the aspiration they were sent home (if they were ambulatory patients) or to a regular nursing unit. If they were symptomatic and had a $Spo_2$ >90% on room air or supplemental oxygen they were sent to a regular nursing unit. All others were admitted to an intensive care area.

The majority of patients who aspirated had bowel obstruction or ileus especially those <3 yr of age. Possibly these children had decreased effectiveness of the lower esophageal sphincter (47–49). Young children have more air in the stomach because of swallowed air and higher intraabdominal pressure as a result of crying or gagging. Also, the ability to apply effective cricoid pressure may be more difficult in infants and toddlers especially when gagging occurs. The majority of patients who aspirated, gagged, or coughed during induction or airway manipulation in which paralysis with muscle relaxants was absent or insufficient to prevent a gag or cough. Unlike adults, in which 1/3 of patients aspirated at the end of anesthesia, only 1 in 8 aspirations occurred at this time in children, possibly because children are usually awake with good gag reflexes before extubation.

### *Gastroesophageal Reflux and Other Gastrointestinal Disorders*

Patients with gastroesophageal reflux have normal gastric emptying of liquids but delayed gastric emptying of solids (50), which is not related to the severity of reflux symptoms. It is true, particularly in children >3 yr of age, that gastric emptying may be delayed if

reflux is present (51). In patients presenting for upper endoscopy after a fast of 6 h (age <6 mo) or 8 h (age >6 mo) for solids and liquids, the gastric residual volume was similar to healthy patients for elective surgery. Thus a 6- to 8-h fast from solids and liquids seems adequate to protect these particular patients from the risk of aspiration (52).

## Conclusion

Many pitfalls and problems in pediatric anesthesia can be avoided by early recognition, quick intervention and strict attention to details of management.

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