Problems and Pitfalls in Pediatric Anesthesia

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ANESTHESIA-RELATED CARDIAC ARREST IN THE PEDIATRIC PATIENT

The etiology of cardiac arrest in the pediatric patient has changed over the past 20 years as practice has evolved in the care of these patients. 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 profile in this category of inadequate ventilation were healthy, nonobese 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 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 which 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%) have differed from the Pediatric Closed Claims Study in 1993 where only 13% were from cardiovascular causes. This may have some basis in the fact that using chest compression was necessary as entry criteria for the POCA Registry 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 incidents before arrests occur. 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-year accounted for 55% of the anesthesia related cardiac arrests. Several pediatric studies have confirmed that infants <1-year have the highest anesthetic risk and that mortality is inversely proportional to age with the highest risk in the <1 month of age group. This may be notably related to a higher ASA Physical Status (PS) 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 is 0.87%, as compared with children 1–6 months of age - MAC of 1.08%. With isoflurane, the MAC for preterm infants (<32 weeks) is 1.28%, 32–37 weeks is 1.41%, and for term (0–1 month) 1.60%, with 1–6 months being 1.87%. Only sevoflurane appears to be different with the MAC being constant at 3.2%–3.3% for neonates and infants <1 month, decreasing to 3% at 1–6 months, and 2.5&-2.8% for 7 months–12 years.[3]

Recent studies show sevoflurane may be less of a myocardial depressant and have less potential for producing bradycardia than halothane in infants.[4] Sevoflurane may also be safer for use in children with congenital heart disease. In comparison with children receiving halothane, the halothane treated patients experienced twice as many episodes of severe hypotension as those who received sevoflurane. Recurrences of hypotension occurred despite increased vasopressor use in the halothane as compared to the sevoflurane treated patients. Risk of hypotension was increased in children <1 year of age compared with older children and patients with preoperative cyanosis had a higher incidence of developing severe desaturation with halothane. Thus sevoflurane may have hemodynamic advantages over halothane in infants and children with congenital heart disease.[5]

Third, 33% of all anesthesia related cardiac arrests occurred in previously healthy ASA PS 1 and 2 patients – 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 months. Controlled ventilation may accelerate the rise in halothane concentration compounded by prolonged exposure due to difficult IV access. Four cases of arrest occurred following probable intravascular injection of local anesthetics. These occurred during combined halothane and caudal anesthesia with injection of 0.25% bupivacaine with 1/200,000 epinephrine despite negative test dose and aspiration. They occurred when both needles and catheters were used to deliver the medication. All had ventricular arrhythmias but were successfully resuscitated without injury.

Mortality rate in ASA PS 3–5 patients was 37% compared to 4% in ASA PS 1–2 patients. ASA PS 3–5 was the strongest predictor of mortality followed by emergency status. Overall the mortality rate in all arrests was 26%.[2]

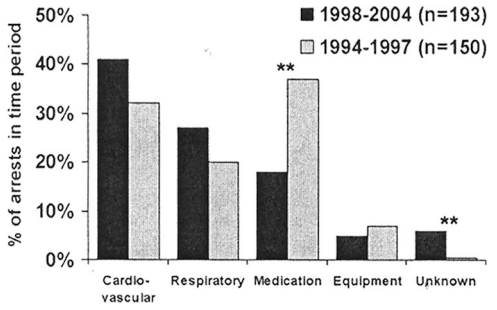

Figure 1. Cause of arrest: Causes of anesthesia-related cardiac arrest in 1998–2004 compared to 1994–1997. Data from 1994 to 1997 previously published and used with permission (Morray, et al. Anesthesiology 2000;93:6–14, © Lippincott Williams & Wilkins). Multiple and miscellaneous other causes (3% 1998–2004 vs 4% 1994–1997) not shown. $^{**}P < 0.01$, 1998–2004 vs 1994–1997 by Z test.

Since publication of the initial series 397 additional cases have been submitted to the POCA Registry and 49% of these arrests were related to anesthetic causes. In the data from 1998–2004, the profile has changed again (Fig. 1). Medication related causes have declined from 37% to 18% of the total due to the decline of cases of cardiovascular depression from inhaled agents, possibly due to the change from halothane to sevoflurane use. Respiratory causes have increased from 20% to 27% the most frequent etiology being laryngospasm. Cardiovascular causes of arrest increased from 32 to 41%. Hypovolemia (often from hemorrhage in spine fusion or craniectomy/craniotomy), the metabolic consequences of massive transfusion (usually hyperkalemia) or hyperkalemia from succinylcholine use were the most frequent known cause of arrest in this category. The exact cause of arrest could not be determined in some cases in the cardiovascular category – frequently these were children with congenital heart disease and an ASA physical status 3–5. Equipment problems (mainly complications from central venous catheter placement) have stayed fairly constant as a cause of arrest in pediatric patients being 7% in 1994–97 and 5% in the 1998–2004 data.

The demographic profile since 1998 has also changed, the percentage of ASA physical status 1 and 2 decreased from 33% to 25% and the percentage of patients <1 year of age decreased from 56 to 38% (Table 1). This may be due to a decreased incidence in the number of arrests reported due to inhalational agents. These arrests were more likely to occur in ASA physical status one or two patients who were <1 year of age. The mortality rate in the two time periods hasn't changed, being 26 and 28%, respectively.[6]

Another study evaluating the data in 92,881 patients from a tertiary care referral center between 1998–2005 indicated the incidence of anesthesia-related cardiac arrest was 0.65/10,000 anesthetics (less than the original POCA data). Both cardiac arrest incidence and mortality were highest among neonates (0–30 days of life) undergoing cardiac procedures. Most patients who

Table 1. Patient Characteristics in Anesthesia-Related Arrests

	1998–2004 $n = 93$	1994–1997[a] $n = 150$
ASA physical status[b]		
1	13 (7)*	23 (15)*
2	34 (18)	27 (18)
3	79 (42)	56 (37)
4	53 (28)	41 (27)
5	11 (6)	3 (2)
Emergency age	40 (21)	31 (21)
<1 mo	21 (11)	22 (15)
1–5 mo	41 (21)	42 (28)
6–11 mo	12 (6)*	19 (13)*
12 mo–5 yr	58 (30)	47 (31)
6–18 yr	60 (31)+	20 (13)+

Percentages in parentheses may not sum to 100% due to rounding.

ASA = American Society of Anesthesiologists.

[a] Previously published data used with permission (Morray et al. Anesthesiology 2000;93:6–14, © Lippincott Williams & Wilkins).

[b] Cases with missing data excluded.

* $P < 0.05$ 1998–2004 vs 1994–1997 by Z-test.

+ $P < 0.01$ 1998–2004 vs 1994–1997 by Z-test.

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experienced perioperative cardiac arrest (88%) had underlying congenital heart disease.[7]

In another report, the frequency of anesthesia-related cardiac arrests in patients with congenital heart disease undergoing cardiac surgery was 27.1/10,000 anesthetics with no mortality. Cardiac arrest was highest in the neonates.[8] In a Brazilian tertiary care hospital with 53,718 anesthetics over a 9-year period the incidence of anesthesia-related cardiac arrest was 3.35/10,000 and anesthesia-related deaths 0.56/10,000. Major causes of cardiac arrest were airway management and medication administration errors. Major risk factors were neonates and children <1 year (prematurity and congenital heart disease were also factors) and emergency surgery.[9] A final study from an academic pediatric medical center looking at 105,436 procedures (except cardiac catheterization) over a 5-year period reported an incidence of 2.67/10,000 cases. Risk factors included ASA physical status ≥3 and children <1 year of age. Those providers that spent ≤40% of time in the OR also indicated a risk factor.[10]

CLASSIFICATIONS 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 the diagnosis of pulmonary hypertension, hypoplastic arteries and ventricles and left to right shunts. Murmurs should be characterized prior to surgery – especially in infants. A history of easy fatigability or poor feeding with failure to thrive should alert the anesthesiologist that

this may 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 prior to surgery.

Respiratory Causes

Loss of airway in the pediatric patient has been a common cause of acute deterioration and cardiac arrest. In the POCA data from 1998–2004, the most common causes of respiratory arrest were laryngospasm, airway obstruction, inadequate oxygenation, inadvertent extubation, difficult intubation, and bronchospasm, in decreasing order.[6] Laryngospasm occurred more commonly in children <2 years of age but equally in ASA physical status 1–2 and 3–5 patients. One third of the patients had an upper respiratory tract infection (URI) or copious secretions. The outcomes of arrest following laryngospasm were 80% of patients had no complications, but 20% had negative pressure pulmonary edema requiring intubation. Most laryngospasm (two thirds) occurred during induction and the majority had no IV present at that time requiring IM succinylcholine. One third occurred during emergence or transport. An IV can be very helpful in managing these patients plus intubating as soon as possible if laryngospasm occurs can avoid negative outcomes.

In the patient with a difficult airway, mask ventilation or intubation may be impossible. It is important to maintain spontaneous ventilation with an inhalational agent without the use of muscle relaxants in these patients. A variety of airway equipment is necessary to deal with these situations. This may include various size masks, airways, LMAs, Bullard laryngoscope and pediatric fiberoptic bronchoscopes. Also obstructed tubes, esophageal intubation, or dislodged tubes may precede an 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 and Hyperkalemia

Intravascular fluid loss and current volume status are often underestimated in the pediatric patient especially in newborns. Lack of good vascular access can compound these problems. Assessment of intravascular volume depends more on clinical signs than invasive measures 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 an arrest situation.

Failure to secure adequate venous access and to keep up with the intraoperative blood loss make these causes of arrest anesthesia related.

Some arrests occur also from not only hypovolemia or hemorrhage, but also from massive transfusion resulting in hyperkalemia. Hyperkalemia from massive transfusion is also potentially preventable by awareness of the problem and using a few steps to reduce the amount of potassium in the transfused blood. As blood ages potassium leaks from the intracellar space into the plasma. This leakage is accelerated in irradiated blood. The anticoagulant used also influences how the blood ages. Packed cells, because of the reduced amounts of plasma have a lower potassium load than whole blood. To decrease the risk of hyperkalemic cardiac arrest the following recommendations will reduce the amount of potassium administered.

1. Use the freshest packed red blood cells available and avoid using whole blood.
2. Don't irradiate the blood except when absolutely necessary (e.g., a premature baby or immunocompromised child). When radiation is required, the time between irradiation and blood administration should be minimized.
3. In high risk situations (e.g., newborn or infant requiring >1 blood volume or with irradiated blood) measure the potassium in the blood to be transfused. If the potassium level is high, consider washing the cells in the cell saver and resuspending the cells in plasma prior to administration.[6]

Inhalational Agents

Anesthetic agent overdose in the face of decreased intravascular volume, is one of the most common causes of sudden hypotension, especially in infants. Bradycardia (<100 beats per minute) is an ominous sign. In a study of causes of bradycardia in infants <1 year of age, 1/3 was due to inhalational agent, 1/3 due to hypoxemia and 1/3 due to patient disease or surgical factors.[11] Inhalational agent overdose responded to a discontinuation of the inhalational agent and atropine in most cases but some needed epinephrine and chest compression. Continuous auscultation of heart sounds is a clinically useful tool for the hemodynamic monitoring of anesthetized infants and children. In a recent study during induction of anesthesia with halothane a dramatic dose dependent decrease in amplitude of S_1 and S_2 heart sounds occurred in all 19 patients ages 6 months–12 years. 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.[12] Thus heart sound changes may be an early warning sign of decreased cardiac function and impending disaster. Although sevoflurane has many advantages as to its safety profile it is not the "ideal" inhalational anesthetic agent with there still being concerns about airway fires, emergence delirium, Compound A, and electrical or clinical seizures.

Succinylcholin-Induced Arrest

In infants who have not been given atropine, especially in the presence of hypoxemia, the potential for bradycardia is significant with succinylcholine. 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 is likely to have bradycardia or arrest as its presenting sign in contrast to tachycardia, tachypnea, arrhythmias, hypertension, and hyperthermia that are common with MH.

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. Incremental rather than bolus injection has been advised for earlier detection of an intravascular injection.[13] Also use of agents with less myocardial toxicity such as ropivacaine may be safer.

Central Venous Catheter Complications

Placement of central venous catheters was the most common equipment-related cause for arrest in the POCA data.[6] Complications included injuries related to needle guidewire or catheter insertion (i.e., pneumothorax, hemothorax, and hemopericadium). Central catheters provide useful information however, maybe inserted more safely with techniques such as ultrasound guidance.[14,15]

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.[16] If your pulse oximeter stops working and your noninvasive BP 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 significant morbidity, even prior to the onset of desaturation.[17]
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 patient is not intubated, intubate immediately – if this is not possible (due to 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 that were intubated in the field.[18] Look for common problems first - airway, volume status, inhalational agent overdose, etc. Discontinue the anesthetic agents and administer 100% O_2.
5. Start CPR early – to be effective in maintaining adequate circulation a peripheral pulse should be discernible.
6. Vascular access that is reliable can make the difference as to the success of the resuscitation. A free flowing peripheral IV line may be all that is necessary since studies have shown that onset time and peak levels of resuscitation drugs (epinephrine, calcium, sodium bicarbonate, glucose) are similar whether given centrally or peripherally.[19] It is important that peripheral lines are flushed well with 5–10 mL of saline to ensure entry of the resuscitative drugs into the central circulation. If no IV access is present at the time of the arrest, the safest and easiest site to cannulate is the femoral vein, whose measured pressures accurately reflect central venous pressure. If no other access can be obtained, a styleted intraosseous needle can be inserted into the anterior tibia, distal femur, medial malleolus or anterior iliac spine. Any resuscitation drug that can be given IV can be given into the intraosseous space with similar onset times.[19] Drugs that can be administered via the trachea are described by the pneumonic LEAN: lidocaine, epinephrine, atropine, and naloxone. Onset and peak levels of epinephrine administered by this route is delayed as compared to the IV route.[20,21]
7. Epinephrine is the single most useful drug – don't waste time with repeated atropine doses. Although bradycardia is the most frequent rhythm preceding cardiac arrest in children, atropine alone is frequently not sufficient to produce return of circulation. Atropine is the drug of choice only for vagally mediated bradycardia, 0.02 mg/kg IV with a minimum dose of 0.1 mg. After adequate ventilation and oxygenation have been ensured, epinephrine is the drug of choice. The dose recommended by the American Heart Association is 10 μg/kg administered IV every 3–5 minutes or 100 μg/kg intratracheally diluted to 5 mL and followed by five manual ventilations.[20] High-dose epinephrine (100–200 μg/kg) may cause post arrest myocardial dysfunction and necrosis but may be useful if the diastolic pressure is $<$20 mm Hg. Vasopressin (0.4 μg/kg) after two doses of IV epinephrine 10 μg/kg may be effective as a "rescue" medication in prolonged hospital resuscitation.[22]
8. For initial fluid resuscitation current recommendations are to avoid glucose-containing solutions in children unless hypoglycemia is suspected or confirmed. Animal studies have reported that when hyperglycemia is produced prior to a cerebral ischemic event neurologic outcome is worse.[23,24]

This may be because increased lactic acid production in the brain aggravates neurologic injury.

9. Obtain a blood gas and electrolytes early – this can be helpful in determining the cause of the arrest.
10. Routine calcium does not improve outcomes but is indicated for hyperkalemia, hypermagnesiumia, calcium channel blocker excess and documented hypocalcemia. It is not indicated for electromechanical dissociation or asystole.[25,26]
11. 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.
12. Routine administration of sodium bicarbonate doesn't improve outcomes and should be given only for severe metabolic acidosis, hyperkalemia and hypermagnesemia at a dose of 1 mEq/kg.
13. Obtain a chest radiograph to help rule in or out the cause of the arrest. Tension pneumothorax should be on your differential diagnosis list.
14. Have ready access to a defibrillator. Be sure it is working properly and that pediatric paddles are available. Defibrillation with 2–4 joules/kg is the mainstay of therapy for pulseless ventricular fibrillation (VF) and ventricular tachycardia (VT).
15. Amiodarone can be used for "shock resistant" VF and VT. Amiodarone is a competitive inhibitor of both α and β adrenergic receptors[27] causing both vasodilatation and AV node suppression and is an alternative to lidocaine use an antiarrhythmic agent. The recommended loading dose is 5 mg/kg over several minutes to 1 hour with repeated doses up to 15 mg/kg.

STRATEGIES TO PREVENT CARDIAC ARREST IN THE PEDIATRIC PATIENT

1. Newer inhalational agents and improved monitoring may have already made a difference
2. Use of local anesthetics such as ropivacaine with less potential for toxicity
3. Regional techniques that include aspiration for blood, test dose and incremental not bolus injection
4. Limiting succinylcholine use to rapid securing of the airway and treatment of laryngospasm
5. Adequate IV lines and keeping up with intraoperative blood loss
6. Prevention of hyperkalemia with limited succinylcholine use and during transfusions (beware of old irradiated blood)
7. Early treatment of laryngospasm with the understanding that having an IV in place can be helpful
8. Safer techniques for CVP placement – such as use of 2D US/Doppler
9. Put high-risk children in experienced hands

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Age	Halothane (MAC)	Isoflurane (MAC)	Sevoflurane (MAC)
< 32 weeks		1.28	
32 – 37 weeks		1.41	
0 – 1 month	0.87	1.60	3.2 – 3.3%
1 – 6 months	1.08	1.87	3.2 – 3.3%
6 mo – 12 yrs			2.5%

Less Myocardial Depression with Sevoflurane than Halothane

- 20 children 2-12 years - showed less decrease in myocardial contractility with sevoflurane vs halothane
  - *Holzman RS et al Anesthesiology 1996;85:1260-7*
- 30 infants (average age 5-6 months)
  - Heart rate decreased 18-30% with halothane, heart rate was stable with sevoflurane
  - Blood pressure decreased less, less decrease in cardiac output as measured by ECHO with sevoflurane
    - *Wodey E Anesthesiology 1997;87:795-80*

POCA Data 1998-2004

- 397 new cases from 80 institutions- 193 (49%) are anesthesia-related
- Medication-related causes decreased from 37% to 18%
  - Near disappearance of cardiovascular depression of inhalational agents
- Cardiovascular causes increased from 32% to 41%
  - Hypovolemia (often from hemorrhage-spine fusion or craniotomy/craniectomy) or metabolic consequences of massive transfusion(usually hyperkalemia) or hyperkalemia from succinylcholine use

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POCA Data 1998-2004

- Respiratory-related causes increased from 20% to 27% -most frequent cause is laryngospasm
- Equipment-related causes increased from 7% to 5%, mostly in ASA PS III-V patients related to complications from CVP placement

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POCA Registry Cases 1994-1997 Versus 1998-2004

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		1994 - 1997	1998- 2004
ASA Physical Status			
	1	15% (33%)	7% (25%)
	2	18%	18%
	3	37%	42%
	4	27%	28%
		2%	6%
Age	< 1 Month	15%	11%
	1 - 5 Months	28% (55%)	21% (33%)
	6 - 11 Months	13%	6%
	12 mo - 5 yrs	31%	30%
	6 - 18 yrs	13%	31%
Emergency Surgery		21%	21%
Mortality		26%	28%

Cardiac Disease

- Murmurs in infants must be characterized prior to surgery (consult and ECHO)
- History of poor feeding, easy fatigability, failure to thrive, cyanosis - pathologic murmur

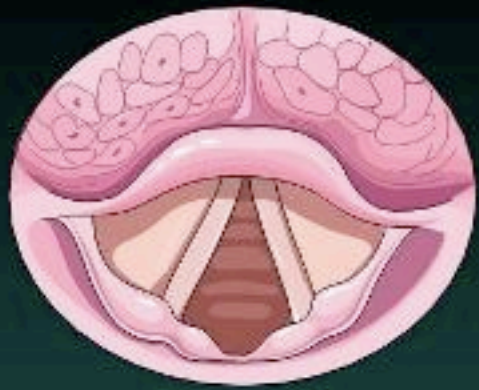
"Innocent Murmur" - Older Child

- Systolic Ejection Murmur
- Medium pitch
- Vibratory
- Lowest in L sternal border
- No radiation

Six Bad Words in a Cardiology Consult

- Severe
- Failure to thrive
- Hypertension (pulmonary)
- Shunt
- Complex
- Hypoplastic

Laryngospasm



- Most common is children less than 2 years of age
- Equal in ASA PS 1-2 and 3-5
- One third had URI or copious secretions
- 20% had negative pressure pulmonary edema
- One third occurred during induction, majority no IV present requiring IM succinylcholine
- Two thirds occurred during emergence or transport
- An IV can be helpful, intubate as soon as possible

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Treatment of Laryngospasm

- Apply CPAP
- Specialized airway maneuvers
  Pressure in layngospasm notch
  Pull mandible forward
- IV access-succinylcholine 1-2 mg/kg(?0.1-0.5 mg/kg) and atropine 0.02 mg/kg or consider propofol 0.5-0.8 mg/kg(incomplete airway obstruction)
- No IV access- succinylcholine 3-4 mg/kg IM with atropine 0.02 mg/kg IM
- Intubate as necessary

Investigation and Management - 3

- Vascular access that is reliable can make a difference as to success of the resuscitation
  - Peripheral IV administration of drugs give same peak level as central administration if line is flushed well
- No IV present

  Femoral

  Intraosseous - anterior tibia, distal femur, ASIS, medial malleolus - drugs will have same onset time

  Trachea - LEAN (lidocaine, epinephrine, atropine, naloxone) - onset delayed as compared to IV

Investigation and Management - 4

- Epinephrine is the single most useful drug - don't waste time with repeated atropine doses

  -AHA recommends 10 µg/kg - 1 to 10,000 epinephrine q 3-5 minutes or 100 µg/kg intratracheally

  -High dose (100 -200 µg/kg) IV may be useful if diastolic pressure is less than 20 mm Hg

Prevention of Cardiac Arrest-2

- Adequate intravenous lines and keeping up with intraoperative blood loss
- Prevention of hyperkalemia 1) during transfusions -(beware old irradiated blood) and 2) succinylcholine use
- Laryngospasm- earlier treatment and having an IV in place can be helpful
- Safer techniques for CVP placement- Use of 2D US/Doppler
- Put high risk children in experienced hands-the role of the pediatric anesthesiologist