



كلية الطب  
قسم التخدير والرعاية المركزة

# **Cardiac Arrest In Pediatric Anesthesia**

An Essay

Submitted For Partial Fulfillment Of Master Degree In  
Anesthesia

BY

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## Cardiac Arrest In Pediatric Anesthesia

توقف القلب المصاحب للتخدير في الأطفال

رسالة مقدمة للحصول على درجة الماجستير في التخدير

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work and his guidance and meticulous revision for the whole work.

## **LIST OF ABBREVIATION**

**POCA** = Pediatric perioperative cardiac arrest.

**ASA** = American society of anesthesiologist.

**MAC** = Minimum alveolar concentration.

**LMA** = Laryngeal mask airway.

**MH** = Malignant hyperthermia.

**URI** = Upper respiratory infection.

**ETS** = Environmental tobacco smoke.

**LEAN** = Lidocaine, Epinephrine, Atropine, Naloxone.

**VF** = Ventricular fibrillation.

**VT** = Ventricular tachycardia.

**SVT** = Supraventricular tachycardia.

**CPR** = Cardiopulmonary resuscitation.

**CCM** = Closed cardiac massage.

**OCM** = Open cardiac massage.

**ECMO** = Extracorporeal membrane oxygenation.

**PALS** = Pediatric advanced life support.

**AV** = Atrio-ventricular.

**PEA** = Pulseless electrical activity.

**EMD** = Electromechanical dissociation.

**AEDs** = Automatic external defibrillators.

**CNS** = Central nervous system.

**ROSC** = Return of spontaneous circulation.

**SIRS** = Systemic inflammatory response syndrome.

**LV** = Left ventricle.

**ATP** = Adenosine tri-phosphate.

**ABC** = Airway, Breathing & Circulation.

**ARDS** = Acute respiratory distress syndrome.

**AMP** = Adenosine mono-phosphate.

**MODS** = Multiple organ dysfunction syndrome.



## **Introduction**

Pediatric anesthesia has progressed rapidly throughout the years. From the first recorded case of pediatric anesthesia in 1842 to the latest advancements in training, technology, medicine and equipment in the last decades of this century.

Today, pediatric anesthesia has grown to include new anesthetic agents, more advanced technology and sophisticated equipment, and special training that provides education in all aspects of pediatric anesthesia (*Steward, 1992*)

Advances in pediatric anesthesia practice such as subspecialization, introduction of new drugs and better monitoring may have changed the liability profile of pediatric anesthesia practice. Pediatric malpractice claims from the 1970s and early 1980s showed a high proportion related to respiratory complications (inadequate ventilation) with 40% of complications thought to be preventable (*Jimenez et al., 2005*)

Despite advances in pediatric anesthesia, unexpected cardiac arrests still occur. The risk of anesthesia related cardiac arrest appears to be inversely proportional to age, with our youngest patients at the highest risk. Of all cases of cardiac arrest submitted to the Perioperative Cardiac Arrest (POCA) Registry, 80% were less than one of age. Any relation between age and risk results in large measure from the impact of underlying patient disease (*Murray et al., 2000*)

Outcomes for anesthetized children have improved over the past 50 years. As reflected in the decrease in anesthesia-related mortality rate from 14 deaths per 10,000 anesthetics in the 1904 study of Beecher and Todd to 0.2, 0.36, and 0.36 deaths per 10,000 anesthetics in recent series from France, Canada and the United States. Several series of outpatient anesthetics in healthy children have shown mortality of zero (*Keenan and Boyan, 1991*).

## **Etiology of Cardiac Arrest in Pediatric Anesthesia**

Along with a decline in the incidence of anesthesia-related cardiac arrest has come a change in the profile of causes of arrest. Forty years ago, airway obstruction and aspiration were more frequent, often from the lack of use or inappropriate use of endotracheal tubes. With increased use of muscle relaxants, inadequate ventilation became a relatively more common respiratory complication than airway obstruction or aspiration. In the last decade, respiratory complications have become relatively less common and cardiovascular complications more common (*Cheney, 1992*).

Cardiovascular and respiratory factors are the major causes of cardiopulmonary arrest in the pediatric population during anesthesia. In (1970) *Salem et al.*, reported that hypovolemia, preoperative anemia, pharmacologic toxicity (succinylcholine, potassium), hypoventilation and airway obstruction were the major cardiovascular and respiratory causes of anesthesia-related cardiac arrest in the pediatric population. 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,

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 70% of the Closed Claim cases and capnometry in 0% (*Murray et al., 1993*)

Recently the Pediatric Perioperative Cardiac Arrest (POCA) Registry has provided some new data that out of 1,089,200 anesthetics, there were 100 cardiac arrests which were deemed anesthesia related (1.4/100,000). Several points are relevant in analysis of this data (*Murray et al., 2000*)

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 (04%), hypotension (49%), abnormality of SpO<sub>2</sub> (46%) or inability to measure blood pressure (20%) were the most common antecedent events (*Olsson and Hallen, 1998*)

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 American Society of Anesthesiologists (ASA) Physical Status (PS) Classification with underlying patient disease (particularly congenital heart disease) but also to cardiovascular depression by inhalational agents (*Cohen et al., 1990*)

Infants <30 days of age the MAC of halothane is 8%, 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 less than one month, decreasing to 3% at 1-6 months, and 2.5-2.8% for 7 months - 12 years (*Lerman et al., 1994*).

Table (1): Comparison between MAC of different inhalational agents at different age of child (*Lerman et al., 1994*)

| Age of child       | MAC of halothane | MAC of isoflurane | MAC of sevoflurane |
|--------------------|------------------|-------------------|--------------------|
| <30 days           | 87%              | 1.60%             | 3.2-3.3%           |
| 1- 6 months        | 1.08%            | 1.87%             | 3%                 |
| 7 months- 12 years | 1.30%            | 2.2%              | 2.5-2.8%           |

Recent studies showed that sevoflurane has less myocardial depressant action and less potential for producing bradycardia than halothane in infants. Sevoflurane may also be safer for use in children with congenital heart disease, another high risk area. 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 less than one year of age compared with older children. Patients with preoperative cyanosis had a higher incidence of developing severe desaturation with halothane (*Russell et al., 2001*)

Third, 33% of all anesthesia related cardiac arrests occurred in previously healthy ASA grading I and II patients

mostly medication-related errors (64%). Fifty percent of the arrests caused by halothane cardiovascular depression which seen at inspired concentrations of 2% or less with the median age being 6 months. Controlled ventilation may accelerate the rise in halothane concentration associated with prolonged exposure to higher concentrations due to difficult intravenous access. Four cases of arrest occurred following probable intravascular injection of local anesthetics. These occurred during combined halothane and caudal anesthesia with injection of 0.2% 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 (*Linda and Loma, 2003*).