Cardiac arrest in the pediatric OR

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A number of studies have identified the cardiac arrest rate in children undergoing anesthesia.\textsuperscript{1-4} The rate of cardiac arrest that is attributable to anesthesia has decreased over the past 25 years to its current value of 1-10:10,000. However, the mortality rate in children after cardiac arrest remains a staggering 26%. Factors that increase the risk of cardiac arrest include: age less than 1 year, ASA III or greater and severe underlying disease. From the POCA registry,\textsuperscript{1} cardiac factors contribute to 36% of the arrests, medications to 20%, respiratory to 27% and equipment to 5%. Based on studies from abroad,\textsuperscript{2,3} factors that contributed to cardiac arrest differed from those in the POCA registry: respiratory factors contributed to 50-80% of the arrests, medications to 28% and cardiac to 11%.

Specific examples of these factors include:
1. hypoxia
2. cardiovascular causes: hypovolemia, hyperkalemia (from blood transfusions) and prolonged QT interval
3. medication (bupivacaine toxicity, side effects of medications (succinylcholine, clonidine) and medication errors)
4. misc.: equipment, failure to properly evaluate the child, rare medical problems (undiagnosed DMD, anterior mediastinal masses)

In this lecture we shall address a number of the above problems as they might occur in the clinical setting together with the appropriate interventions.

PEDIATRIC BCLS:
In 2005, the American Heart Association published new pediatric basic life support guidelines\textsuperscript{5} that dramatically changed resuscitation measures:
1. Caution is expressed regarding the use of tracheal tubes during resuscitation; LMA\textregistered s were advocated.
2. Cuffed tubes for all infants and children except neonates, provided the cuff pressure is <20 cmH\textsubscript{2}O
3. Verification of proper tube placement must include a CO\textsubscript{2} detection device
4. The compression/ventilation strategy has changed to 100 compressions/minute with 8-10 breaths/minute. No pauses in compressions.
5. Timing of 1 shock, CPR and drug administration during pulseless CPR is identical to ACLS.
6. Routine use of high dose epinephrine (in excess of 10 -30 mcg/kg) is not recommended.
7. Lidocaine is de-emphasized, it may be used for pulseless VT and VF if amiodarone is unavailable
8. Induced hypothermia (32-34C) should be considered for comatose children for 12-24h
9. Indications for the use of inolators
10. Termination of resuscitation should be considered.

CARDIAC ARREST:
Hypoxia.

The number one cause of cardiac arrest in infants and children is hypoxia until proven otherwise. This may arise as a result of respiratory problems (loss of the airway) or as a secondary effect of another problem (such as a cardiac problem). Managing the pediatric airway is one of the greatest challenges that face pediatric anesthesiologists. Understanding how to maintain the pediatric airway, retrieve the airway that is slipping away and bringing the child back from the brink of complete laryngospasm and bradycardia will be emphasized in my presentation. Strategies include administering 100% oxygen, maintaining CPAP, application of pressure AT THE CONDYLES of the ascending ramus of the mandible (not the angle), AVOIDING IPPV, then using atropine, propofol and succinylcholine before arrest occurs.

Cardiovascular causes.1,7

Hypovolemia is a completely avoidable clinical problem that occurs from failure to establish an adequately sized intravenous access and to maintain fluid homeostasis. Establishing inadequate size access is common when non-anesthesiologists begin to resuscitate children in the ER. Rarely do anesthesiologists rely on intraosseous needles for fluid resuscitation although these needles may be effective for large volume resuscitation. Underestimating blood loss commonly occurs when inexperienced anesthesiologists manage complex cases such as craniosynostosis, craniofacial surgery and neurosurgery where massive blood loss can occur, can occur rapidly and can occur occultly. Systolic blood pressure is an excellent and reliable sign of fluid volume in infancy; heart rate increases, respiratory variability in heart rate or pulse oximetry, urine output and less commonly CVP measurements all assist in estimating fluid status. Initial volume resuscitation requires large volume crystalloid administration (20 ml/kg repeated) and colloids. If cardiovascular instability or anemia occurs, blood should be used immediately to resuscitate the child.

Hyperkalemia is responsible for an unusually large number of cardiac arrests, particularly in children, according to the POCA registry. From a cardiac perspective, the cause of hyperkalemia is rapid transfusion of old cold blood into a centrally placed catheter in a small infant (right atrial volume may be as small as 5-10 ml). This is not a new concept, but inadequate teaching and understanding of the risks of rapid blood transfusion in such a manner is causing hyperkalemia-associated cardiac arrest each and every year.8,9 The message just is not being heard! To prevent this problem, one must consider several strategies. First, avoid falling behind in fluid management in the small infant (easy to do during craniofacial surgery). Second, do NOT use centrally placed lines for rapid blood transfusions. Use peripherally placed catheters and these often offer less resistance than the central lines. Third, rewarm the blood, especially if it is old, to
drive the potassium back into the red cells. Small volumes of plasma present in packed
cells likely contain a very high concentration of potassium that is transfused into a small-
sized right atrium could lead to life-threatening arrhythmias. Potassium induces
arrhythmias by increasing the resting membrane potential such that it approximates the
threshold potential, thus triggering action potentials throughout the myocardium, which
lead to erratic and irregular wide complex premature contractions. The treatment for this
cardiac conduction problem is definitive and unambiguous: intravenous calcium chloride
(10 mg/kg) or calcium gluconate (30 mg/kg). The dose should be repeated until the
rhythm returns to normal sinus rhythm. The response to calcium is exceedingly rapid but
if hyperkalemia continues, the effect of a single bolus of intravenous calcium could wane
with a recrudescence of the arrhythmias. In such a case, the dose of calcium must be
repeated yet again. There is no limit on how much calcium should be administered to
restore the cardiac rhythm. Cardiac output must be restored using CPR and/or
intravenous epinephrine in order to clear the potassium.

The mechanism by which calcium restores narrow complexes is not by decreasing
the serum potassium concentration. Rather, calcium has direct effects on the action
potential to raise the threshold potential, restoring the gap between the resting membrane
potential and the threshold potential. The wide complexes stop almost immediately.
Calcium also prolongs the repolarization time of the action potential that prevents a new
beat from beginning. In order to decrease the serum potassium, CPR and intravenous
epinephrine should be used to maintain cardiac output and remove potassium from the
myocardium.

**Medications.**

Cardiac arrest follows two possible mechanisms after succinylcholine: a direct
SA node effect and an indirect effect of hyperkalemia.

That the heart rate slows after a single dose of succinylcholine is not surprising if
one considers that succinylcholine is two fused acetylcholine molecules and acetylcholine
triggers the parasympathetic receptor in the SA node of the atrium. With the rich
innervation of parasympathetic receptors in the atrium in children, a single dose of
succinylcholine can precipitate asystole. This side effect of succinylcholine can be
precluded by the pre-emptive administration of intravenous atropine 20 mcg/kg.

Less predictably and more surprisingly, sudden cardiac arrest can occur after
succinylcholine (in spite of pretreatment with atropine) when it triggers acute
hyperkalemia. A number of clinical scenarios can lead to a hyperkalemic response to
succinylcholine including burn patients (after the first 24-48h), myopathies (NOT MH),
chronic intra-abdominal sepsis (ischemic bowel), upper and lower motor neuron lesions
and immobility. Within 30 seconds of administering any dose of succinylcholine, the
serum potassium increases dramatically reaching double digits. Heart rate rapidly slows,
QRS complexes widen and asystole may ensue. See above for a complete discussion of
hyperkalemia.

Inadvertent intravascular injection of local anesthetics may be a serious and
potentially fatal complication of regional anesthesia. The most dangerous local
anesthetic is racemic bupivacaine. When racemic bupivacaine reaches the myocardium,
its lipophilicity and affinity for the sodium-potassium pump, heralds ventricular
tachycardia that quickly progresses to asystole. Resuscitation is almost impossible. No
antiarrhythmics are effective. One of the most (or only) exciting developments in the
treatment of bupivacaine toxicity has been the recognition that a dose of intralipid can
reverse bupivacaine (and ropivacaine) toxicity.\textsuperscript{10,11} The mechanism by which Intralipid
reverses bupivacaine-induced myocardial toxicity is unclear. Several hypotheses have
been postulated.\textsuperscript{12} Bupivacaine has a greater affinity for Intralipid than the myocardium
creating a concentration gradient. Bupivacaine then moves along its concentration
gradient, out of the myocardium. Intralipid may also affect the activity of carnitine
acylcarnitine transferase helping to maintain triglyceride availability within myocardial
cells and therefore the availability of ATP. Intralipid is the definitive
treatment for this potentially fatal complication of local anesthetics. Although this is an unapproved use of
Intralipid, I strongly recommend that each OB and regional unit have an adequate supply
of intralipid (2-3 ml/kg) immediately available for resuscitation. This is NOT
Propofol…propofol should not be used for this purpose. Intralipid preparations, 10-20%.
comprised of medium chain triglycerides are preferred although this too is not firmly
established.

The remainder of the causes of cardiac arrest fall into miscellaneous categories
that include equipment difficulties. Examples of such problems will be cited time
permitting.

Diagnosing the cause of a cardiac arrest is essential if the resuscitation is to be
successful. This is a rare event that should be reversible in most children provided a
timely and definitive treatment is administered.

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