The new guidelines for cardiopulmonary resuscitation: a critical analysis

Liliane Zorzela,[1] Daniel Garros,[2] Allan R. de Caen[3]

Abstract

Objective: To describe the new American Heart Association (AHA) guidelines for pediatric life support, based on the scientific evidence evaluated by the International Liaison Committee on Resuscitation, and endorsed and disseminated by North American resuscitation councils.

Sources: The guidelines for basic and advanced life support published in *Circulation* in November 2005 were reviewed together with subsequent publications on the same topics, identified in PubMed and MEDLINE using the keywords cardiac arrest, basic life support, advanced life support, cardiopulmonary resuscitation and pediatric resuscitation.

Summary of the findings: The greatest guideline changes are in the area of basic life support. The new guidelines emphasize the new chest compression/ventilation ratio for trained health professionals, which is now 15:2 for all children except neonates. Also emphasized is the need for harder and faster chest compressions, and the need to avoid hyperventilation during and after cardiorespiratory arrest. The use of high-dose epinephrine has been removed, as have some other previous recommendations.

Conclusions: The most recent AHA guidelines for pediatric resuscitation are focused primarily on basic life support care. They are based on the best available scientific evidence, although further research is required to validate these changes and provide new evidence for future guidelines.

J Pediatr (Rio J). 2007;83(2 Suppl):S64-70: Cardiorespiratory arrest, resuscitation, intubation, death, epinephrine.

Introduction

With the objectives of standardizing the care of critically ill children and increasing their likelihood of survival, the International Liaison Committee on Resuscitation (ILCOR) analyzes the resuscitation literature, and every 5 years publishes an evidence-based evaluation of resuscitation science. Many of the world's resuscitation councils then translate the evidence into treatment recommendations known as guidelines. In November 2005 the new American Heart Association (AHA) guidelines for cardiopulmonary resuscitation (CPR) were published in *Circulation*,[1] and the new training manuals published by the AHA are currently available for use in training courses for health professionals.[2]

Adequate basic CPR, rapid access to emergency medical services, provision of advanced life support and prevention of secondary sequelae are necessary to achieve good outcomes in critically ill children. The most significant changes to the pediatric guidelines have been extrapolated from studies performed on manikins, adult humans or animals. The objective of this brief review is to point out the changes that were made in the latest resuscitation guidelines and to relate them to the Brazilian context.

1. Fellow clínico e intensivista, UTI Pediátrica, Stollery Children's Hospital, Edmonton, AB, Canadá.
2. Staff e professor adjunto, UTI Pediátrica, Departamento de Pediatria, Stollery Children's Hospital, Universidade de Alberta, Edmonton, AB, Canadá.
3. Staff e professor adjunto, UTI Pediátrica, Departamento de Pediatria, Stollery Children's Hospital, Universidade de Alberta, Edmonton, AB, Canadá. Representante canadense, International Liaison Committee on Resuscitation (ILCOR).

The authors reviewed the 2005 AHA guidelines for basic and advanced pediatric life support,[1] in addition to the many worksheet reviews performed as part of the ILCOR process. Studies published on these topics since December 2005 were also reviewed, using the PubMED and MEDLINE databases. The keywords searched were *cardiac arrest*, *basic life support*, *advanced life support*, *cardiopulmonary resuscitation* and *pediatric resuscitation*.

Airway and ventilation

The first step necessary in caring for the critically ill child is the rapid recognition of respiratory failure, since this is the most common cause of cardiorespiratory arrest (CRA) in children. Signs such as tachypnea, erratic respiration, nasal flaring, intercostal retractions, moaning, cyanosis and altered level of consciousness are important markers of respiratory distress.

Pre-hospital support of ventilation and oxygenation by means of bag-mask ventilation is safer than and as effective as tracheal intubation, providing it is performed correctly.[3-5] An adequate seal between face and mask is crucial! Opening the airway with mild extension of the neck is necessary for adequate chest expansion, which is the best indicator of good ventilation. If chest expansion is insufficient, it is necessary to reassess the position of the patient, the size of mask and adequacy of seal between mask and face.

During ventilation with bag and mask, gastric distention can occur, which can compromise ventilation[6] and lead to aspiration of gastric contents. In order to avoid this, care should be taken to use the lowest inspiratory pressure needed to move the chest. Cricoid pressure can be applied to minimize the risk of aspiration in the unconscious patient.[3,6] After intubation, a nasogastric or orogastric tube can be used to reduce intraabdominal pressure.

An important guideline change: laryngeal masks are as effective as tracheal intubation, but should only be used by professionals experienced in their use.[7]

In the past, cuffed tracheal tubes were only recommended for children over 8 years of age, as the natural subglottic "narrowing" in young children can function as a physiological cuff. In the 2005 AHA guidelines, cuffed tracheal tubes can be used for children of all ages except neonates.[8-10] Advantages to the use of cuffed tracheal tubes include that the cuff offers a certain level of protection against gastric aspiration, in addition to optimizing ventilation by minimizing air leakage. This is especially important in situations where there is poor lung compliance or increased airway resistance. Another relevant consideration is the reduced number of reintubations necessary to achieve the appropriate sized endotracheal tube (ETT).[8] The following formulas can be used in choosing ETT size:

- Tracheal tube with cuff (mm) = (age in years/4) + 3;

- Tracheal tube without cuff (mm) = (age in years/4) + 4.

There have been long-standing concerns that the use of cuffed tubes in small children could damage the tracheal mucosa, increasing the risk of subsequent subglottic stenosis. Subglottic stenosis is generally associated with the use of excessively sized endotracheal tubes, and with prolonged duration of intubation. As the size of cuffed tracheal tube used for a child should be smaller than the chosen size of uncuffed tube (see the formula above), this risk should be reduced. It is, however, extremely important that cuff pressure be routinely checked. Pressure should be kept below 20-25 cm H_2O, in order to allow sufficient perfusion of the tracheal mucosa and to avoid ischemia.[8-11] In North America, the presence of respiratory technicians in intensive care units (ICU) facilitates rigorous monitoring of ETT cuff pressures. In Brazil, this careful monitoring may become diluted among the many responsibilities of ICU physicians and nurses, but must be strived for as the appropriate standard of care to be provided to critically ill patients.

There is no single method that is 100% accurate for verifying correct ETT position after tracheal intubation, despite the common practices of relying on auscultation, the presence of water vapor in the tracheal tube, and chest expansion. The confirmation of exhaled CO_2 using a colorimetric detector(low cost) or by capnography to confirm ETT position is simple to apply and reliable in children with a perfusing rhythm weighing more than 2 kg. The correct ETT position must be verified soon after intubation and immediately after any movement of the patient.[11-14] There is not enough data to recommend for or against the measurement of exhaled CO_2 during a pediatric cardiac arrest.

What is perhaps the greatest change in the new guidelines relates to hyperventilation! This should be avoided at all costs during resuscitation.[15,16] With hyperventilation, intrathoracic pressure increases, reducing cardiac output and coronary perfusion. The reduction in CO_2 levels during hyperventilation also reduces cerebral blood flow and increases the risk of cerebral ischemia. The resulting respiratory alkalosis leads to a displacement of the oxygen-hemoglobin dissociation curve to the left and, consequently, reduces the delivery of oxygen to the tissues. The recommended ventilatory rate during CRA is from 8 to 10 breaths per minute. If the patient's airway has been intubated, it is not necessary to stop chest compressions while providing ventilations. If the patient has only suffered a respiratory arrest, the ventilated rate should be between 12 and 20 breaths per minute.[3,17]

Hyperventilation is a useful/recommended strategy only when caring for patients with increased intracranial pressure, and an immediate risk of transtentorial herniation.

Compression/ventilation ratio

The ideal ratio for compressions and ventilation for children in cardiac arrest is unknown. The 2000 AHA guidelines recommended different ratios for different ages: 3:1 in newborn infants, 5:1 for up to 8 years of age, and 15:2 in children over 8 years of age. This was difficult to remember!

In children, CRA is generally the result of hypoxemia, justifying the choice of a greater ventilation/compression ratio (more ventilations per cycle). Evidence does exist, however,[16-18] for the relatively greater importance of chest compressions:

- in victims of cardiac arrest secondary to hypoxia, the amount of ventilation required to maintain the ventilation-perfusion relationship is reduced, as cardiac output is also reduced during CPR;

- in patients where arrhythmia is the cause of cardiac arrest, ventilation is relatively less important than chest compressions;

- there is a significant reduction in coronary perfusion during the time that chest compressions are suspended in order to provide ventilation during CRA;

- when there is only one resuscitator, using a compression: ventilation ratio of 5:1, the time taken to change positions between ventilation and compressions can occupy up to 30% of the total time spent performing CPR;

- using the 5:1 compression/ventilation ratio, a higher PO2 is achieved than with 15:2, but, since cardiac output is reduced, there is still less oxygen supplied to tissues;

- the number and duration of interruptions in chest compressions relates directly to a lower chance of restoring a spontaneous circulation.

For health professionals, the compression/ventilation ratio should be 15:2 for children of all ages (except neonates). For educational reasons and for the reasons cited above, the decision was made to recommend for a single chest compression/ventilation ratio for CPR when provided by lay resuscitators, or single rescuers (generally in a pre-hospital setting), with a ratio of 30:2 being recommended for adults and children (beyond the newly born).

The guidelines emphasize the importance of minimizing interruptions to chest compressions, and of stressing the quality of compressions – harder and faster – in order to restore a spontaneous circulation. Once the patient's airway has been secured with tracheal intubation or a laryngeal mask, it is no longer necessary to interrupt chest compressions to provide ventilations; in that setting, compressions should continue at 100/minute, and ventilations be provided at 8-10 breaths/minute.

Animal studies have proved that good CPR technique is an important factor in optimizing neurological outcome. If it is not possible to perform both chest compressions and ventilations, the simple provision of either is better than no resuscitation efforts at all.[19,20]

Circulation

Faced with a child in suspected cardiac arrest, the time taken to check for a pulse must be no more than 10 seconds. If after this time it is not possible to detect the brachial pulse (infant) or the femoral pulse (older child) in a child who is not responding to stimuli (does not move or breathe normally), then chest compressions are begun. Studies have shown that even health professionals have problems confirming the presence or absence of a pulse.[21,22] If a heart rate of less than 60 beats per minute is confirmed, coupled with signs of low cardiac output, chest compressions should be initiated. Compressions should be applied to the lower half of the sternum for children of all ages, taking care not to compress over the xiphoid process. After each compression, the chest is allowed to recoil completely to allow blood to return to the heart.[23] In infants and younger children, the two thumb technique, with the professional's hands encircling the patient's chest, is the favored technique. Greater compressive force and increased intrathoracic pressure allows for increased coronary perfusion when compared to the two finger technique.[24] For older children, one or both hands should be placed over the lower sternum using the heel of the hand, as with adults, in order to achieve sufficient force to adequately compress the chest, i.e. to 1/3 to 1/2 its diameter. The rescuer needs to avoid flexion of the elbows, and must keep his arms completely extended. Compressions should be at a rate of 100/minute, with minimal interruptions. Adequate chest compressions are essential for the return of spontaneous circulation.[25]

Sudden cardiac arrest in children is generally due to ventricular fibrillation, or ventricular tachycardia (VT) with no pulse. In this situation a defibrillator should be used as quickly as possible. While awaiting the arrival of a defibrillator, chest compressions should be provided.[25] Automated external defibrillators can be used for children over 1 year of age.[26] They have become commonplace in public areas such as airplanes and shopping centers. The use of biphasic defibrillators is at least as effective as monophasic ones, and causes less myocardial damage. Pediatric pads are used for children less than 10 kg, and adult sized ones otherwise. The first shock should be 2 J/kg. Three sequential ('stacked') shocks are no longer recommended.

After a single defibrillation, it is necessary to restart compressions immediately and continue for 2 minutes before

checking for pulse and heart rhythm. If necessary, the next shock delivered should be 4 J/kg. If the rhythm remains unchanged after two defibrillations, the use of drugs is indicated. The first choice is epinephrine, but after that, antiarrhythmic agents such as amiodarone can be used. The new sequence is now: shock (2 J/kg) → CPR for 2 minutes → check rhythm/pulse → shock (4 J/kg) → CPR for 2 minutes + drug - check rhythm/pulse → shock (4 J/kg) → CPR + drug.

Medications used during cardiorespiratory resuscitation

The preferred routes for administration of medication during cardiac arrest are intravenous or intraosseous. In patients where there is difficulty obtaining intravenous access, an intraosseous needle should be immediately placed.

The delivery of medication via ETT is being discouraged. The absorption of drugs given by this route is inconsistent. Post-resuscitation hypertension can occur as a result of the slow absorption of intrapulmonary epinephrine.[27] Diastolic hypotension resulting from the β-agonist effect of the epinephrine can potentially occur as well, causing coronary hypoperfusion.[28] Another important negative feature of giving drugs via the ETT is that chest compressions must be suspended while the medication is administered. Nevertheless, in patients with no other vascular access who have already been intubated, the administration of lipid soluble drugs (atropine, naloxone, epinephrine and lidocaine – ANEL) via ETT is still an accepted option in the most recent AHA guidelines. Ideal drug dosages for endotracheal administration are unknown. Animal studies suggest that larger drug doses are needed, always followed by a 5 mL saline flush, and five ventilations to improve drug absorption. Drugs administered via ETT should have dosages adjusted as follows: epinephrine (0.1 mg/kg), atropine (0.03 mg/kg) and lidocaine (2-3 mg/kg).

The 2005 AHA guidelines offer some other recommendations on medication use during CPR:

- Epinephrine: this is the most commonly given drug during cardiac arrest, at a dosage of 0.01 mg/kg (0.1 mL/kg using a drug concentration of 1:10,000). The use of high doses – 0.1 mg/kg (0.1 mL/kg at a concentration of 1:1,000) – is discouraged, as the majority of studies do not demonstrate a survival benefit over the standard dose, and its use is possibly associated with worsened neurological outcomes.[29] The exception to this recommendation is in cases of intoxication by β-blockers, when high dose epinephrine may be necessary.

- Amiodarone: reduces atrium-ventricular (AV) conduction, prolongs the AV node refractory period and slows ventricular conduction. It should be given in boluses of 5 mg/kg up to a maximum total dose of 15 mg/kg; slow infusion is recommended (20 to 60 minutes) in order to reduce side effects, such as hypotension, bradycardia and cardiac conduction blockade. Amiodarone can be used by continuous infusion at a dosage of 5-15 mcg/kg/min after the bolus dose. Amiodarone is being used with increased frequency for the treatment of supraventricular arrhythmias, especially in cardiac ICU for the treatment of junctional tachycardia, VT and ventricular fibrillation, as it suppresses premature ventricular depolarization. Unfortunately, the majority of data on this drug's use has been extrapolated from adult studies.[30]

- Glucose: should not be administered during CPR, except when hypoglycemia is documented. Hyperglycemia is associated with poor prognosis in CPR.[31]

- Vasopressin: this is still a controversial drug in CPR. Some adult studies have demonstrated that vasopressin has similar efficacy to epinephrine during cardiac arrest. However, there is only one pediatric study,[32] with four patients, in which it was used as a rescue drug after prolonged cardiac arrest, with spontaneous circulation returning in three patients. Animal studies have shown that vasopressin when used with epinephrine, improves outcome.[33] The role of vasopressin in pediatric CPR is still inconclusive, and further studies need to be performed before it can be definitively recommended.

Rhythm disturbances

For the treatment of supraventricular tachycardia in the hemodynamically stable patient (palpable pulse and normal arterial pressure), vagal maneuvers may be used initially (for example: ice on the face), but, in the event of treatment failure, the drug of choice is adenosine at a dose of 0.1 mg/kg, or 0.2 mg/kg in cases refractory to the initial dose. Adenosine has a short half-life and should be injected rapidly. The most effective means of administration is to use two syringes at the same time, one containing the drug and the other with a the saline solution push.

Amiodarone is also useful in treating pediatric supraventricular tachycardia, and can be used (with the doses cited above) when adenosine has failed to convert to, or to maintain a normal sinus rhythm, in hemodynamically stable patients. Procainamide, in doses of 15 mg/kg by infusion over 30-60 minutes, is another treatment option if other interventions fail. Significant hypotension and heart block may occur during the infusion of procainamide, especially if given after amiodarone.

Hemodynamically unstable patients with supraventricular tachycardia should receive electrical cardioversion as quickly as possible.[34] Remember to set the defibrillator to synchronized mode! The dose is 0.5-1 J/kg on the first attempt and 2 J/kg in subsequent attempts.

Hemodynamically stable patients with VT can be treated with amiodarone or procainamide (at the doses already cited). In VT patients without a pulse or VF victims, CPR

should be started immediately while awaiting a defibrillator, as defibrillation is the treatment of choice.

Post-cardiorespiratory arrest management

As soon as a spontaneous circulation has returned, the objective of further treatment is to maintain the patient's hemodynamics and to minimize possible neurologic sequelae.

Hyperventilation after CRA is detrimental and should be avoided. Hyperventilation causes reduced venous return to the heart and subsequent cardiac output, and reduces cerebral blood flow. Acceptable levels of pCO2 depend on the clinical circumstances. When caring for patients with asthma or severe lung disease, elevated levels of pCO2 may be tolerable (permissive hypercapnia), while in neurological patients, normal levels of pCO2 should be maintained.

Studies of some adults post-CRA suggest that periods of post-resuscitation moderate hypothermia (32-34 ºC) offers protection to the central nervous system (CNS) with subsequently improved neurologic outcome. After CRA, hyperthermia is a common occurrence and is associated with worse prognosis. In patients who remain comatose after CPR, the new guide suggests maintaining body temperature between 32 and 34 °C for 12 to 24 hours.[35-37]

After CRA there is significant myocardial dysfunction and attention should be given to maintaining patient hemodynamics, if necessary with the use of inotropic and vasoactive drugs.

Hyperglycemia and hypoglycemia in critically ill children are associated with a lower chance of survival and worsened neurologic progress. It is unclear whether this is the cause or merely an association with worsened outcome. In critically ill adults in surgical ICU, strict glycemic control may lead to improved outcome.[31] Later studies aimed at maintaining normal glucose levels in patients in an adult medical ICU did not demonstrate the same protective effect.[32] The unnecessary administration of glucose during or soon after CPR may impact negatively on prognosis.[31] Therefore, the objective is to maintain normoglycemia.

Post-cardiorespiratory arrest prognosis

There is unfortunately no clear indicator suggesting at what point continued efforts of CPR are futile. There are certain indicators that suggest a likelihood of better prognosis, including: short duration of CRA, early initiation of CPR, hypothermia as the cause of CRA (rare in Brazil), CRA in a hospital environment and prompt extracorporeal circulation after CRA.[38,39] Duration of resuscitation is directly related to neurological prognosis, i.e. the longer CPR lasts the greater the risk of neurological damage. There are isolated reports of prolonged CRA with minimal sequelae, in particular

when good CPR is performed. For these reasons there is no consistent guideline as to when to stop resuscitation.[40,41]

Presence of the family during cardiopulmonary resuscitation

Recent studies[42] analyzed a practice that is in vogue in North America, which is to allow families to be present during CPR on their loved ones. The reactions of family members and the attitudes of health professionals have been positive. This practice is not a recommendation of the new guidelines, merely an observation/option. Nevertheless, to allow parents or guardians to be present, with the help of appropriate support (social worker, cleric or nurse) to explain in real time what is taking place is worthy of consideration by those responsible for local emergency rooms and ICU.

Conclusions

The goal of resuscitation guidelines is to improve the care provided to critically ill children. Every 5 years, ILCOR performs reviews of the relevant literature and publishes its recommendations so that national/regional resuscitation councils can re-align their protocols, striving for optimal care in cases of CRA. Unfortunately, the majority of the guidelines changes for pediatrics are extrapolated from studies with manikins, adult humans and animals. The emphasis on good basic life support in these latest guidelines, such as the pre-hospital universal compression/ventilation ratio of 30:2, justifies redoubling the efforts in training society's first-responders.

Only time and further study will reveal whether the outlined changes in the 2005 AHA guidelines will have a positive impact on the survival of children who are victims of CRA. At the very least, these are the best guidelines that can be currently made, based on the little pediatric evidence that exists.

References

1. 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation. 2005;112(Suppl I):IV1-203.

2. Ralston M, Hazinski MF, Zaritsky AL, Schexnayder SM, Kleinman ME. Pediatric Advanced Life Support Provider Manual. Dallas: American Heart Association; 2006.

3. Gausche M, Lewis RJ, Stratton SJ, Haynes BE, Gunter CS, Goodrich SM, et al. Effect of out-of-hospital pediatric endotracheal intubation on survival and neurological outcome: a controlled clinical trial. JAMA. 2000;283:783-90.

4. Stockinger ZT, McSwain NE Jr. Prehospital endotracheal intubation for trauma does not improve survival over bag-valve-mask ventilation. J Trauma. 2004;56:531-6.

5. Pitetti R, Glustein JZ, Bhende MS. Prehospital care and outcome of pediatric out-of-hospital cardiac arrest. Prehosp Emerg Care. 2002;6:283-90.

6. Berg MD, Idris AH, Berg RA. Severe ventilatory compromise due to gastric distention during pediatric cardiopulmonary resuscitation. Resuscitation. 1998;36:71-3.

7. Park C, Bahk JH, Ahn WS, Do SH, Lee KH. The laryngeal mask airway in infants and children. Can J Anaesth. 2001;48:413-7.

8. Khine HH, Corddry DH, Kettrick RG, Martin TM, McCloskey JJ, Rose JB, et al. Comparison of cuffed and uncuffed endotracheal tubes in young children during general anesthesia. Anesthesiology. 1997;86:627-31.

9. Newth CJ, Rachman B, Patel N, Hammer J. The use of cuffed versus uncuffed endotracheal tubes in pediatric intensive care. J Pediatr. 2004;144:333-7.

10. Deakers TW, Reynolds G, Stretton M, Newth CJ. Cuffed endotracheal tubes in pediatric intensive care. J Pediatr. 1994;125:57-62.

11. Bordet F, Allaouchiche B, Lansiaux S, Combet S, Pouyau A, Taylor C, et al. Risk factors for airway complications during general anaesthesia in paediatric patients. Paediatr Anaesth. 2002;12:762-9.

12. Campbell RC, Boyd CR, Shields RO, Odom JW, Corse KM. Evaluation of an end-tidal carbon dioxide detector in the aeromedical setting. J Air Med Transp. 1990;9:13-5.

13. Bhende MS, Thompson AE. Evaluation of an end-tidal CO2 detector during pediatric cardiopulmonary resuscitation. Pediatrics. 1995;95:395-9.

14. Cardoso MM, Banner MJ, Melker RJ, Bjoraker DG. Portable devices used to detect endotracheal intubation during emergency situations: a review. Crit Care Med. 1998;26:957-64.

15. Aufderheide TP, Sigurdsson G, Pirrallo RG, Yannopoulos D, McKnite S, von Briesen C, et al. Hyperventilation-induced hypotension during cardiopulmonary resuscitation. Circulation. 2004;109:1960-5.

16. Abella BS, Alvarado JP, Myklebust H, Edelson DP, Barry A, O'Hearn N, et al. Quality of cardiopulmonary resuscitation during in-hospital cardiac arrest. JAMA. 2005;293:305-10.

17. Berg RA, Sanders AB, Kern KB, Hilwig RW, Heidenreich JW, Porter ME, et al. Adverse hemodynamic effects of interrupting chest compressions for rescue breathing during cardiopulmonary resuscitation for ventricular fibrillation cardiac arrest. Circulation. 2001;104:2465-70.

18. Wik L, Kramer-Johansen J, Myklebust H, Sorebo H, Svensson L, Fellows B, et al. Quality of cardiopulmonary resuscitation during out-of-hospital cardiac arrest. JAMA. 2005;293:299-304.

19. Herlitz J, Engdahl J, Svensson L, Young M, Angquist KA, Holmberg S. Characteristics and outcome among children suffering from out of hospital cardiac arrest in Sweden. Resuscitation. 2005;64:37-40.

20. Berg RA, Hilwig RW, Kern KB, Ewy GA. "Bystander" chest compressions and assisted ventilation independently improve outcome from piglet asphyxial pulseless "cardiac arrest". Circulation. 2000;101:1743-8.

21. Eberle B, Dick WF, Schneider T, Wisser G, Doetsch S, Tzanova I. Checking the carotid pulse check: diagnostic accuracy of first responders in patients with and without a pulse. Resuscitation. 1996;33:107-16.

22. Owen CJ, Wyllie JP. Determination of heart rate in the baby at birth. Resuscitation. 2004;60:213-7.

23. Aufderheide TP, Pirrallo RG, Yannopoulos D, Klein JP, von Briesen C, Sparks CW, et al. Incomplete chest wall decompression: a clinical evaluation of CPR performance by EMS personnel and assessment of alternative manual chest compression-decompression techniques. Resuscitation. 2005;64:353-62.

24. Whitelaw CC, Slywka B, Goldsmith LJ. Comparison of a two-finger versus two-thumb method for chest compressions by healthcare providers in an infant mechanical model. Resuscitation. 2000;43:213-6.

25. Cobb LA, Fahrenbruch CE, Walsh TR, Copass MK, Olsufka M, Breskin M, et al. Influence of cardiopulmonary resuscitation prior to defibrillation in patients with out-of-hospital ventricular fibrillation. JAMA. 1999;281:1182-8.

26. Berg RA, Chapman FW, Berg MD, Hilwig RW, Banville I, Walker RG, et al. Attenuated adult biphasic shocks compared with weight-based monophasic shocks in a swine model of prolonged pediatric ventricular fibrillation. Resuscitation. 2004;61:189-97.

27. Efrati O, Ben-Abraham R, Barak A. Endobronchial adrenaline: should it be reconsidered? Dose response and haemodynamic effect in dogs. Resuscitation. 2003;59:117-22.

28. Mielke LL, Lanzinger MJ, AschkeC, Entholzner EK, Wilhelm MG, Henke J, et al. Plasma epinephrine levels after epinephrine administration using different tracheal administration techniques in adult CPR porcine model. Resuscitation. 2001;50:103-8.

29. Perondi MB, Reis AG, Paiva EF, Nadkarni VN, Berg RA. A comparison of high-dose and standard-dose epinephrine in children with cardiac arrest. N Engl J Med. 2004;350:1722-30.

30. Dorian P, Cass D, Schwartz B, Cooper R, Gelaznikas R, Barr A. Amiodarone as compared with lidocaine for shock-resistant ventricular fibrillation. N Engl J Med. 2002;346:884-90.

31. Van den Berghe G, Wouters P, Weekers F, Verwaest C, Bruyninckx F, Schetz M, et al. Intensive insulin therapy in the critically ill patients. N Engl J Med. 2001;345:1359-67.

32. Van den Berghe G, Wilmer A, Hermans G, Meersseman W, Wouters PJ, Milants I, et al. Intensive insulin therapy in the medical ICU. N Engl J Med. 2006;354:449-61.

33. Mann K, Berg RA, Nadkarni V. Beneficial effects of vasopressin in prolonged pediatric cardiac arrest: a case series. Resuscitation. 2002;52:149-56.

34. Guyette FX, Guimond GE, Hostler D, Callaway CW. Vasopressin administered with epinephrine is associated with a return of a pulse in out-of-hospital cardiac arrest. Resuscitation. 2004;63:277-82.

35. Wik L, Hansen TB, Fylling F, Steen T, Vaagenes P, Auestad BH, et al. Delaying defibrillation to give basic cardiopulmonary resuscitation to patients with out-of-hospital ventricular fibrillation: a randomized trial. JAMA. 2003;289:1389-95.

36. Hypothermia After Cardiac Arrest Study Group. Mild therapeutic hypothermia to improve the neurologic outcome after cardiac arrest. N Engl J Med. 2002;346:549-56.

37. Shafi NI, Mariscalco MM. Considering the use of induced hypothermia in a pediatric patient with traumatic brain injury: A critical appraisal of two meta-analyses. Pediatr Crit Care Med. 2006; 7:468-72.

38. Lopez-Herce J, Garcia C, Rodriguez-Nunez A, Dominguez P, Carrillo A, Calvo C, et al. Long-term outcome of paediatric cardiorespiratory arrest in Spain. Resuscitation. 2005;64:79-85.

39. Thiagarajan RR, Bratton SL. Extracorporeal membrane oxygenation for cardiac arrest: when to use it, and what are the outcomes? Crit Care Med. 2006;34:1285-6.

40. Takasu A, Saitoh D, Kaneko N, Sakamoto T, Okada Y. Hyperthermia: is it an ominous sign after cardiac arrest? Resuscitation. 2001;49:273-7.

41. Lopez-Herce J, Garcia C, Dominguez P, Carrillo A, Rodriguez-Nunez A, Calvo C, et al. Characteristics and outcome of cardiorespiratory arrest in children. Resuscitation. 2004;63:311-20.

42. Gold KJ, Gorenflo DW, Schwenk TL, Bratton SL. Physician experience with family presence during cardiopulmonary resuscitation in children. Pediatr Crit Care Med. 2006;7:428-32.

Correspondence:
Daniel Garros
Room 3A3.03 WMC, PICU
8440-112 street
Edmonton, AB T6G 2B7 – Canada
E-mail: dgarros@capitalhealth.ca