Pediatric Cardiopulmonary Resuscitation: Cases

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Background

- Epidemiology
  - 8-20/100,000 US children suffer cardiac arrest annually.
- Factors that influence survival outcomes
  - environment in which arrest occurs
  - pre-existing condition of child
  - duration of “no flow” state
  - initial EKG rhythm detected
  - quality of interventions provided
- High quality CPR may double survival rate in out-of-hospital arrest
- Favorable neurologic survival in hospital arrest 8-33%
- Favorable neurologic survival to discharge for out-of-hospital arrest 0-16%

- Four phases of Cardiac arrest
  - Pre-arrest
    - Early recognition is best
    - Children more likely to have respiratory compromise prior to pulseless arrest
    - In-hospital rapid-response teams
    - Pediatric Early Warning Scores (PEWS)
  - No flow (untreated cardiac arrest)
    - Early Recognition and initiation of compressions
  - Low flow (active CPR)
    - Effective CPR—coronary and cerebral perfusion
  - Post resuscitation
    - Secondary reperfusion injury
    - Minimize cellular dysfunction and death

Animal models of sudden VF arrest suggest that acceptable PaO2 and PaCO2 persist for up to 8-10 minutes during active CPR without rescue breathing.

- Decreased pulmonary blood flow during CPR
- PaO2 and PaCO2 do not vary significantly from pre-arrest state
- Outcomes for bystander-initiated CPR without rescue breathing are similar in adults with primary cardiac arrest

Asphyxia models show that rescue breathing is critical during CPR.

- Progressive decrease in PaO2 and PaCO2 from baseline
- Continued pulmonary blood flow before cardiac arrest depletes O2 reservoir
- Pediatric patients with a respiratory etiology prior to their cardiac arrest must get an airway and breathing!

First PALS guidelines established 1988

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**General Considerations**

### BLS/PALS 2015 Update

- [BLS Healthcare Provider Pediatric Cardiac Arrest Algorithm for 2 or More Rescues—2015 Update](http://cpr.heart.org/AHAEC/CCP/HealthcareTraining/HealthcareProfessional/BasicLifeSupportBLS/UCM_473189_Basic-Life-Support-BLS.jsp)

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Case 1

A 6 month old premature (33 week) male has developed worsening cough and congestion over the past 4 days. His parents took him to the pediatrician earlier today. He was diagnosed with viral bronchiolitis and prescribed bulb suction and albuterol. Over the past several hours his work of breathing has worsened, including tachypnea, intercostal, subcostal and supraclavicular retractions. EMS was called and could not obtain pulse ox reading, but noted perioral cyanosis and placed him on oxygen.

Vitals on arrival to Rural ED:  T 100.6 HR 180, RR 18, BP 80/40, SpO2 76% on non-rebreather mask
IV access is placed in triage for which he has minimal response to stick.

Does this patient concern you?
While waiting to be seen by ED he becomes mottled, blue and unresponsive in the presence of an ED nurse. Next steps?

Color is blue, extremities cold, no respiratory effort, no femoral or brachial pulse palpated after 10 seconds. What now?

CPR initiated at a rate of 100 compressions/min, and a ratio of 30 compressions: 2 breaths. Now another RN, an RT and an MD enter the room? What should you change?

Case 1 (cont.)

Next Steps

1. Assign Roles
2. Convert to 2 person CPR (Ratio of 15:2)
3. Place on monitors (or AED)

Patient placed on monitors and noted to be in PEA rhythm. MD takes over lead role while RN1 administers high quality chest compressions, RN2 draws medications, RT manages airway with head-tilt chin lift and bag mask ventilation. CPR continues for 2 cycles at a ratio of 15:2 with administration of IV epinephrine (.01mg/kg of 1:10,000 dilution) x2 without ROSC.

What are your next steps?
Hs and Ts? What was cause of patient’s respiratory arrest? We are now 5 minutes into resuscitation.

Options?

Airway options?

LMA vs Endotracheal intubation depending on available expertise

Case 1 (cont.)

RT places size appropriate LMA at bedside. After placement, chest rise improves and chest compressions continue for 2 more minutes. Breaths given at rate of every 3 seconds after placement of advanced airway.

On repeat check at 7 minute mark he has palpable axillary and femoral pulses. He is now pink. HR 180s, RR 20 (via bag ventilation through LMA), BP 98/55, SpO2 95%. He is slowly starting to arouse and respond to touch and fight against the LMA.

Next steps?
- Intubation
- control temperature
- transfer to higher level of care
Case 1 Review

• Respiratory compromise preceding cardiac arrest
  • Accounts for the majority of pediatric cases
  • Pre-arrest management is critical
  • Nasal suctioning
  • Oxygen
  • Respiratory support

• Consider advanced airway management
  • May only reverse cardiac arrest with reversal of hypoxia
  • Infants have very limited oxygen reservoir in lungs

Case 2

A 16 yo healthy, athletic male collapses suddenly during a soccer match. A bystander (the referee) trained in BLS immediately notes the boy to be unresponsive, not breathing and pulseless. The bystander begins high quality CPR with rescue breathing at a rate of 100 compressions/min and ratio of 30 compressions: 2 breaths.

EMS arrives and continues CPR. AED demonstrates a shockable rhythm. 2 defibrillations are attempted on the pitch then 4 more en route to the nearest pediatric ED. IO was placed. CPR ongoing throughout process.

You are in that pediatric ED and have taken the call regarding this patient. What are you thinking?
Considerations of this case
- Out of hospital arrest
- Immediately recognized (very brief no flow state)
- Good quality CPR throughout
- Defibrillation per AE

- What are your next steps?
  - Organize resuscitation team and assign roles
  - IV access
  - Call PICU and Pediatric Surgery teams (so they can come assist if available)
  - eCPR

Patient arrives with active chest compressions in progress. Immediately placed on monitors with the following rhythm:

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Case 2 (cont.)

- CPR is continued via PALS algorithm including defibrillation at 2J/kg and administration of IO Epinephrine (.01mg/kg of 1:10,000) dilution

- IV Access obtained, LMA placed with change of breaths to every 6 seconds. Compressions continue at rate of 100/min. PICU

- He receives another 4 defibrillations and 3 doses of Epi before ROSC. Total time from arrest to ROSC is 40 minutes. He is subsequently intubated and transferred to the PICU on a epinephrine drip.

- 3 days later he is awoken from pharmacologic sedation and responds appropriately to commands.
Considerations—SCD in Children/Adolescents

- Very rare
- 25% of events are associated with sports
  - Athletic, seemingly healthy males
  - African-American predominance
  - Often not discovered on screening sports physical
- Causes: Arrhythmic v non-arrhythmic
  - Hypertrophic cardiomyopathy
  - Coronary artery anomalies
  - Long QT syndrome
- Push for screening

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Case 3

A 2 month old female with known hypoplastic left heart syndrome (HLHS) presents with hypoxia and poor perfusion. She underwent the Norwood procedure with atrial septectomy, construction of a neo-aorta and placement of a Blalock-Thomas-Taussig shunt from the right subclavian artery to right pulmonary artery. Her mother states that she had developed diarrhea and vomiting 2 days ago and has not been able to keep down any formula or pedialyte throughout today.

On arrival to the ED she has capillary refill ~5 seconds and visible cyanosis. Her peripheral pulses are 1+ axillary and femoral.

Vitals: T 98, HR 180 (ST), RR 70, BP 55/32 SpO2 55%

What do you think of this patient? What should her normal SpO2 be?
Case 3 (cont.)

What are your next steps?
• Placed on monitors
• Gentle administration of oxygen with improvement in SpO2 to 58%.
• Multiple attempts at IV access failed.
• Cardiology and PICU called. PICU alerts CT surgeon who is driving in.
• During all of this, patient suddenly becomes unresponsive, loss of pulse with following rhythm:

Now What?

Case 3 (cont.)

• 2 person CPR started by ED staff at ratio of 15 compressions : 2 breaths
• IO access emergently obtained, Epi given at 2 minute mark
• Intubated by ED MD at 2 minutes
• At 4 minute mark 2nd dose of Epi (.01mg/kg IO 1:10,000 dilution) given, with ROSC confirmed at 6 minutes. SpO2 of note now 80%. With post arrest NIBP of 85/55
• Over the next several minutes SpO2 drops back down to 55% with drop in NIBP to 50/28. Luckily PICU shows up at this time.
• Both parameters improve again with administration of .01mg/kg epinephrine IO at 1:10,000 dilution and 10ml/kg fluid bolus
• Patient continues to require epi dosing every 5-6 minutes until CT surgeon arrives
Considerations with HLHS

Stage 1 Palliation for HLHS
- Common atrium (allows mixing of oxygenated and deoxygenated blood)
- Use of MPA to reconstruct systemic outflow tract (Neo-Aorta)
- Placement of BT shunt for pulmonary blood flow
- Circuit in parallel physiology
  - Pulmonary blood flow entirely dependent on BT shunt flow
  - SpO2 75-85%
  - Prone to clot with low flow states (dehydration, narrowing etc.)
  - Blood flows down path of least resistance (PVR vs SVR).
  - Oxygen is a pulmonary vasodilator
    - ↓PVR and can ↓SBF

Case 3 (cont.)

• Patient emergently taken to cardiac catheterization lab where obstructive thrombus in BT shunt confirmed. After shunt was recannalized in cath, patient’s SpO2 improved to 78% and BP normalized to 80/48 on low dose epi drip.

• Patient was successfully extubated 2 days later and underwent stage 2 palliation 3 months later.
Conclusions

- Cardiac arrest in children is associated poor outcomes…
  - …but early recognition, management of Hs and Ts and quality CPR can drastically improve outcomes
- Most causes in young children are respiratory
- Sudden cardiac death is rare in children, but can occur
  - Push for better cardiac screening for adolescent athletes
- With the initiation of the joint heart program with Cincinnati Children’s Hospital, we will see more children with congenital heart disease in our community