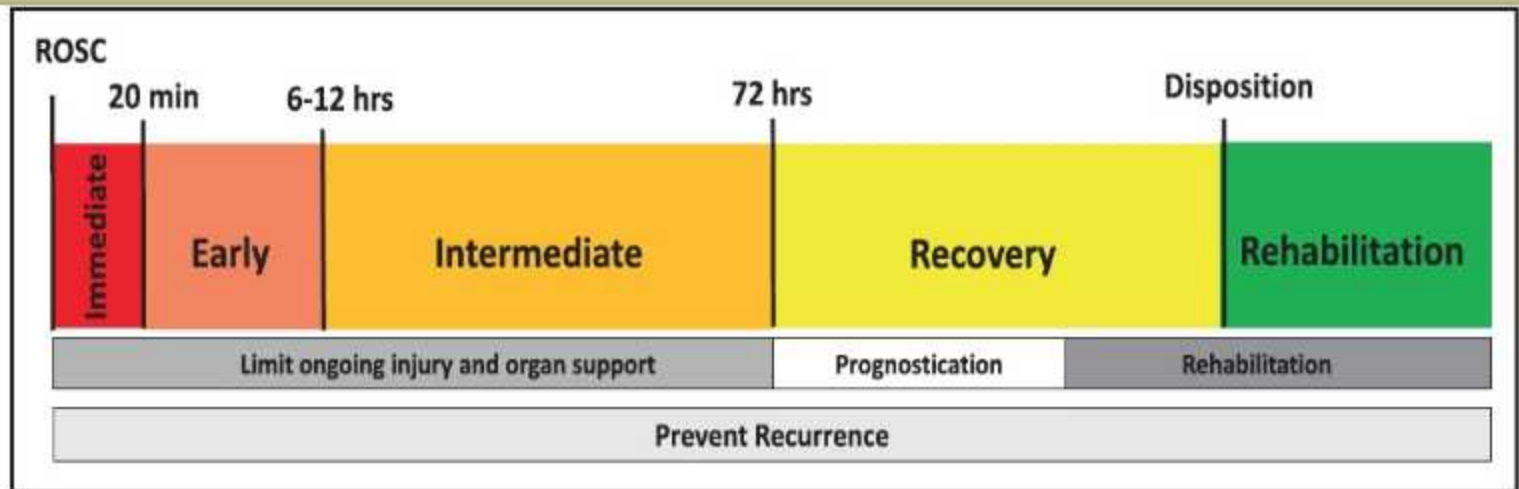




post-cardiac  
arrest care  
(PCAC)



**Figure 1.** Phases of post-cardiac arrest syndrome.

ROSC indicates return of sustained circulation. Adapted from Neumar et al.<sup>4</sup> Copyright © 2008, American Heart Association, Inc.

- The immediate phase: the first 0 to 20 min after ROSC
- The early phase: from 20 min up to 6 to 12 hours
- The intermediate phase: 12 to 72 hours
- The recovery phase: approximately 72 hours to day 7
- The rehabilitation phase

The goal of PCAC is to increase not only survival to hospital discharge but also survival with favorable neurological outcome.



- **ongoing assessment of the resuscitation,**
- **determining and managing the etiology of the arrest**
- **maintain and or minimize brain injury with TTM,**
- **consideration of vasoactive drugs,**
- **preventing decompensation**
- **managing the patient in the emergency department, setting, and/or while transporting to PICU.**

AHA SCIENTIFIC STATEMENT  
Pediatric Post-Cardiac Arrest Care

TTM: targeted T management

PCAS highlighting the pathophysiology and the need for continued multisystem support after ROSC. It was determined that all resuscitations from CA result in predictable sequelae in the days to weeks following the arrest now accepted as **4 key components of PCAS:**

- **post-cardiac arrest brain injury,**
- **post-cardiac arrest myocardial dysfunction,**
- **systemic ischemia/reperfusion response,**
- **persistent precipitating pathophysiology**

AHA SCIENTIFIC STATEMENT  
Pediatric Post-Cardiac Arrest Care

**PCAS: post-cardiac arrest syndrome**

Post-CA brain injury remains a leading cause of morbidity and mortality in adults and children due to **limited tolerance of ischemia, hyperemia, or edema.**

- The first 3 phases of PCAS involve **hypoxemic-hypotensive perfusion with energy deprivation.**
- With ROSC, there is a burst of reactive oxygen species, and oxidative stress may ensue in tissue that is depleted of antioxidants. As a result, reperfusion is associated with excitotoxicity, calcium accumulation, and free radical-mediated cell injury or death.



- As a result, **reperfusion** is associated with **excitotoxicity, calcium accumulation, and free radical-mediated cell injury or death.**
- Both **neuronal cellular necrosis** and **apoptosis** result from this cascading injury and can continue **in the days to weeks after ROSC.**
- A variety of post-CA clinical conditions, including hyperoxia, hypoxemia, and hypotension, can exacerbate the neuronal injury.

# Oxygenation and Ventilation

- ✧ All intubated children require continued assessment to ensure
  - **proper ETT positioning**, including continuous monitoring of oxygenation (pulse oximetry), and
  - **ongoing monitoring of ventilation (continuous EtCO<sub>2</sub> monitoring, &/or intermittent ABG assessment).**
- ✧ Insertion of a gastric tube helps to reduce gastric distension and may prevent vomiting.
  
- **D**: Dislodged or displaced ETT(right mainstem or esophageal location)
- **O**: Obstructed endotracheal tube (mucous plug, kinked ET)
- **P**: Pneumothorax
- **E**: Equipment failure (ventilator malfunction, O<sub>2</sub> disconnected or off)

## Avoid low and high arterial oxygen

- **Once ROSC has been achieved**, The lowest possible FiO<sub>2</sub> should be used to maintain an **O<sub>2</sub> sat of 94%-99%** to avoid hypo or hyperoxemia.
  - **Small observational studies have failed to show an association between arterial oxygenation and mortality in resuscitated children.**
  - ✧ However, in one large, retrospective, multicenter observational pediatric study of **1875 infants** and children who **survived to PICU admission**, analysis showed that:
    - ✧ **hypoxemia** (PaO<sub>2</sub> <60 mmHg) &
    - ✧ **hyperoxemia** (PaO<sub>2</sub> ≥300 mmHg)
- independently & significantly increased the ERD by 90 & 25 %, respectively.

**ERD: estimated risk of death**



# Monitor ventilation

- ✧ The 2015 international consensus recommendations suggest that PaCO<sub>2</sub> after ROSC targeted and severe **hypocapnia (PaCO<sub>2</sub> <30 mmHg)** or **hypercapnia (PaCO<sub>2</sub> >50 mmHg)** should be limited.
- ✧ In one study on **223 infants and children ROSC upon IHCA** was **associated with a mortality of 50 & 59 %, respectively**, compared with 33% mortality if PaCO<sub>2</sub> was 30-50 mmHg.

## **Arterial CO<sub>2</sub> tension influences cerebral perfusion in both children and adults.**

- ❑ Preclinical studies suggest that hyperventilation decreases coronary perfusion and survival after CA
- ❑ Hyperventilation causes **cerebral vasoconstriction** and can decrease cerebral blood flow (CBF), thereby potentially exacerbating cerebral ischemia.
- ❑ Hypercapnia causes **cerebral vasodilation** and **increases CBF**.

- Global myocardial dysfunction occurs even in the absence of a cardiac cause of the arrest, and the severity may be related to the duration of no-flow time during cardiac arrest.
- Myocardial dysfunction has been associated with early mortality despite successful initial resuscitation in children and adults.
- The onset of PCA myocardial dysfunction begins within hours of the arrest, peaks at  $\approx 8$  hours, begins to improve at 24 h, and typically resolves within 48 to 72 hours.
  - cardiovascular ischemia/ reperfusion injury
  - cytokine-mediated cardiovascular dysfunction,
  - induced myocardial injury secondary to catecholamines or electric shocks



# Clinical manifestations of myocardial dysfunction

- Hypotension,
- LV and RV systolic or diastolic dysfunction
  - reduced CO,
  - arrhythmias, and
  - pulmonary edema, leading to recurrent CA
- No support for routine administration of prophylactic antiarrhythmics after ROSC, but rhythm disturbances during this period may warrant therapy.
- Treatment depends on the cause and hemodynamic consequences of the arrhythmias. No therapy for PACs and PVCs other than maintenance of adequate perfusion and NL fluid and electrolyte balance.
- Ventricular arrhythmias may signify more serious myocardial dysfunction.

- Although there is insufficient evidence to suggest the optimal timing or frequency of PCA echo, it is a beneficial, noninvasive tool for identifying myocardial dysfunction and congenital and acquired cardiac abnormalities.
- A 12-lead ECG is helpful in establishing the cause of arrest



# Systemic Ischemia/Reperfusion

- The combination of systemic ischemia/reperfusion produces a state similar to the sepsis syndrome, with elevated cytokines, the presence of endotoxin in plasma, activation of coagulation pathways, and inhibition of anticoagulant pathways
- Clinical manifestations of systemic ischemia/reperfusion include capillary leak with intravascular hypovolemia, vasoplegia, coagulopathy, hyperglycemia, adrenal insufficiency, and impaired oxygen utilization and delivery, contributing to multisystem organ dysfunction.

# Persistent Precipitating Pathophysiology

- Management of the child after cardiac arrest includes diagnosis and treatment of the precipitating cause of cardiac arrest.
- Failure to identify and correct the original cause of cardiac arrest leaves the patient at risk for secondary injury and even recurrence of cardiac arrest.



# Hemodynamic Monitoring

- Approximately 95% of pediatric IHCA occurs in an ICU, and almost 50% of these patients will have arterial catheters in place before the cardiac arrest.
- If possible, an arterial catheter should be placed for continuous intra-arterial pressure monitoring to facilitate the identification and treatment of hypotension.
- Central venous catheters may be useful to monitor central venous o2 sat and to provide a route for the administration of fluids and medications.

# Hypoperfusion and Hypotension

- Perfusion is compromised after CA, and patients are often hypotensive. Of note, **cardiogenic shock** occurs frequently in survivors of CA.
- After ROSC in a child, circulatory instability may be the result of:
  - Ongoing fluid loss,
  - Decreased cardiac function, and/or
  - Harmful alterations in SVR
- Based on data poor perfusion is associated with increased morbidity and mortality. Thus, vasoactive drug therapy is recommended, and should be tailored to each patient.
- Parenteral fluids, inotropes, and vasoactive drugs are to be used as needed to maintain a systolic blood pressure  $>5^{\text{th}}$  percentile for age.



**Table 1. PCAS: Monitoring**

General monitoring
Oxygen saturation, continuously by pulse oximetry
Capnography (quantitative)
Arterial blood pressure (intra-arterial when possible or noninvasive)
Blood glucose (point of care)
Cardiac telemetry, continuous
ECG
Temperature, continuous core (esophageal, bladder, or rectal)
Urine output
Blood gas, arterial (pH, $P_{aO_2}$ , $P_{aCO_2}$ )
Serum lactate, arterial
Blood glucose, electrolytes, creatinine, complete blood count, coagulation profile
Venous oxygen saturation
Central venous pressure
Chest radiograph
Additional hemodynamic monitoring
Echocardiography
Neurological monitoring
Neurological clinical examination, serial
EEG, continuous
Imaging: brain CT or brain MRI

- Hypotension after ROSC is associated with:

- \* decreased survival to hospital discharge
- \* decreased survival W favorable neurologic outcome

- The 2015 international guidelines recommend that parenteral fluids and vasoactive medications be used to maintain the **systolic BP>5th percentile for age.**
- ✧ If hypovolemia is suspected in a patient with cardiogenic shock, the clinician should carefully infuse **5 to 10 mL/kg of isotonic fluids (N/S or Ringer's lactate) over 10 to 20 minutes** followed by reevaluation of endpoints.



## Septic Shock

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### Fluid Boluses

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**2020 (Updated):** In patients with septic shock, it is reasonable to administer fluid in 10 mL/kg or 20 mL/kg aliquots with frequent reassessment.

**2015 (Old):** Administration of an initial fluid bolus of 20 mL/kg to infants and children with shock is reasonable, including those with conditions such as severe sepsis, severe malaria, and dengue.

### Choice of Vasopressor

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**2020 (New):** In infants and children with fluid-refractory septic shock, it is reasonable to use either epinephrine or norepinephrine as an initial vasoactive infusion.

**2020 (New):** In infants and children with fluid-refractory septic shock, if epinephrine or norepinephrine are unavailable, dopamine may be considered.

## **Early and continuous epinephrine infusion for post-arrest hypotension is the preferred agent in pediatric patients.**

- One retrospective study suggested that early epinephrine (within 15 min of arrest):
  - decreased the time to ROSC,
  - higher survival rate and
  - better neurologic outcomes in non-shockable OHCA.
- Epinephrine a peripheral vasoconstrictor, **improves BP, also a potent inotropic and chronotropic agent.**

**Dopamine, norepinephrine, and dobutamine also improve BP but are recommended as 2<sup>nd</sup> line therapies, or in specific pre-existing conditions such as renal failure or cardiogenic shock.**



**Table 2. Vasoactive Infusions That May Be Used to Optimize Hemodynamics During PCAS**

Medication	Dose Range	Type of Drug	Side Effects
Dobutamine	2–20 µg/kg per 1 min IV/IO	Inotrope; vasodilator	Tachyarrhythmias; peripheral vascular injury
Dopamine	2–20 µg/kg per 1 min IV/IO	Inotrope; chronotrope; renal and splanchnic vasodilator in low doses; vasopressor in high doses	Tachyarrhythmias; peripheral vascular injury
Epinephrine	0.1–1 µg/kg per 1 min IV/IO	Inotrope; chronotrope; vasodilator in low doses; vasopressor in high doses	Tachyarrhythmias; peripheral vascular injury
Milrinone	0.25–0.75 µg/kg per 1 min IV/IO	Inotrope; lusitrope; vasodilator	Hypotension
Norepinephrine	0.1–2 µg/kg per 1 min	Vasopressor	Peripheral vascular injury

Inamrinone	0.75–1 mg/kg IV/IO over 5 min; may repeat 2x; then: 2–20 µg/kg/min	Inodilator
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Sodium nitroprusside	1–8 µg/kg/min	Vasodilator; prepare only in D5W
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1

### Optimize Ventilation and Oxygenation

- Titrate  $\text{FiO}_2$  to maintain oxyhemoglobin saturation 94%-99% (or as appropriate to the patient's condition); if possible, wean  $\text{FiO}_2$  if saturation is 100%.
- Consider advanced airway placement and waveform capnography.
- If possible, target a  $\text{Pco}_2$  that is appropriate for the patient's condition and limit exposure to severe hypercapnia or hypocapnia.

2

### Assess for and Treat Persistent Shock

- Identify and treat contributing factors.\*
- Consider 20 mL/kg IV/IO boluses of isotonic crystalloid. Consider smaller boluses (eg, 10 mL/kg) if poor cardiac function suspected.
- Consider the need for inotropic and/or vasopressor support for fluid-refractory shock.

3

### \*Possible Contributing Factors

**Hypovolemia**  
**Hypoxia**  
**Hydrogen ion (acidosis)**  
**Hypoglycemia**  
**Hypo-/hyperkalemia**  
**Hypothermia**  
**Tension pneumothorax**  
**Tamponade, cardiac**  
**Toxins**  
**Thrombosis, pulmonary**  
**Thrombosis, coronary**  
**Trauma**

4

### Hypotensive Shock

- Epinephrine
- Dopamine
- Norepinephrine

5

### Normotensive Shock

- Dobutamine
- Dopamine
- Epinephrine
- Milrinone

6

- Monitor for and treat agitation and seizures.
- Monitor for and treat hypoglycemia.
- Assess blood gas, serum electrolytes, and calcium.
- If patient remains comatose after resuscitation from cardiac arrest, maintain targeted temperature management, including aggressive treatment of fever.
- Consider consultation and patient transport to tertiary care center.

## Management of shock After ROSC



# Analgesia & sedation

- ❑ should be used to ensure comfort and prevent shivering.

shivering can occur at different goal temperatures during TTM, including both therapeutic hypothermia and therapeutic normothermia

- ❑ Combinations of **opioids and benzodiazepines** are commonly used in adults, although sedative-anesthetic agents such as **propofol & dexmedetomidine** are also options.
- ❑ must be balanced against the risk of complications:
  - infection and pneumonia,
  - hypotension, and
  - prolonged mechanical ventilation
- ❑ The use of NMB masks response during the clinical neurological examination and can potentially lead to oversedation, undersedation, or masking of worsening neurological examination findings. In addition, NMB will mask seizures.

## Sedation and Neuromuscular Blockade

- The 2010 AHA PALS guidelines recommended controlling pain and discomfort with analgesics (eg, morphine, fentanyl) and sedatives (eg, lorazepam or midazolam).
- Neuromuscular blocking agents (eg, vecuronium or pancuronium) with analgesia or sedation (or both) may improve oxygenation and ventilation in case of patient- ventilator dyssynchrony or severely compromised pulmonary function.
- The use of NMB masks response during the clinical neurological examination and can potentially lead to over or undersedation, or masking of worsening neurological examination findings.
- In addition, NMB will mask seizures.



# BS Monitoring

**Prevent, & treat hypoglycemia ( $\leq 45$  mg/dL in the newborn and  $\leq 60$  mg/dL in the child) to avoid further neurologic insult.**

- Severe hyperglycemia can also be problematic because it can lead to uncontrolled post CA **osmotic diuresis**, which can exacerbate volume depletion and hemodynamic instability.


Sustained hyperglycemia (2 consecutive measurements of serum glucose  $\geq 150$  mg/dL is associated with **higher mortality** in critically ill children and should be avoided.

## **Seizures occur in 10%-50% of children who remain encephalopathic after achieving ROSC.**

- about half of children with post-ROSC seizures experience exclusively nonconvulsive (subclinical, EEG only) seizures, which cannot be identified by clinical observation alone.
- Seizures could not be predicted from any clinical or resuscitation variables.
- Seizures were associated with unfavorable gross neurological outcomes at discharge but not with higher mortality.



American Clinical Neurophysiology Society Critical Care  
Continuous EEG Guidelines Committee recommends  
continuous EEG monitoring for adult and pediatric patients who  
remain encephalopathic after CA to identify electrographic seizures



The statement recommends initiating EEG monitoring as soon as feasible, continuing monitoring for 24 to 48 hours in most patients, but continuing until after 24 hours of normothermia in patients treated with hypothermia

**seizures increase metabolic demand,**

- worsen metabolic dysfunction,
- increase ICP
- secondary brain injury

For these reasons, many clinicians aim to treat seizures, although the approach is generally guided by the child's overall medical condition and other prognostic indicators.

- insufficient evidence to determine whether treatment of clinical or electrographic seizures results in improved patient outcomes and what the optimal methods are to manage seizures after CA.



**2015 (Old):** An electroencephalography for the diagnosis of seizure should be promptly performed and interpreted and then should be monitored frequently or continuously in comatose patients after ROSC.

**2015 (Old):** The same anticonvulsant regimens for the treatment of status epilepticus caused by other etiologies may be considered after cardiac arrest.

**Why:** For the first time, the Guidelines provide pediatric-specific recommendations for managing seizures after cardiac arrest. Nonconvulsive seizures, including nonconvulsive status epilepticus, are common and cannot be detected without electroencephalography. Although outcome data from the post-cardiac arrest population are lacking, both convulsive and nonconvulsive status epilepticus are associated with poor outcome, and

treatment of status epilepticus is beneficial in pediatric patients in general.

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- Acute, electrographic seizures are treated with benzodiazepines, levetiracetam, or phenytoin. myoclonic seizures may be refractory to treatment.
- Potential adverse effects of anticonvulsants:
  - cardiac arrhythmias,
  - hypotension, and
  - respiratory depression
- It must be taken into into consideration that any sedation induced by these medications may complicate the neurological examination.



- To treat the child who remains comatose after OHCA, the 2015 AHA PALS guidelines update recommended that it is reasonable either to maintain continuous normothermia (TTM to 36°C–37.5°C) for 5 days or
- to maintain 2 days of continuous hypothermia (TTM to 32°C–34°C) followed by 3 days of continuous normothermia (TTM to 36°C–37.5°C).
- Because increased mortality was associated with  $T_s < 32^\circ\text{C}$ , if TTM to 32–34°C is used, meticulous care must be provided to prevent  $T < 32^\circ\text{C}$


# T management

- TTM to 32°C to 34°C can be divided into 3 phases: induction, maintenance, and rewarming.

- **surface-cooling methods**

- positioning servo-controlled cooling blankets under or above the patient
- use of ice packs around the body,
- core-cooling methods (IV catheters circulating cold saline) or
- combination approach

core T should be continuously monitored

- Hypokalemia,
- hypophosphatemia,
- hypomagnesemia,  may precipitate arrhythmias
- Hypocalcemia
- decreases insulin sensitivity



- The maintenance phase of TTM requires careful monitoring to avoid fluctuations in T.

No optimal method or rate of rewarming after TTM. In children, rewarming is generally accomplished at a rate  $0.5^{\circ}\text{C}/2\text{h}$  to reduce the risk of:

- cerebral hyperperfusion,
  - vasogenic edema, and
  - acute systemic hypotension documented during rewarming in traumatic brain injury
- 
- For children who are comatose after OHCA and IHCA, TTM to  $32^{\circ}\text{C}$ - $34^{\circ}\text{C}$  for 24 to 48 hours is relatively safe.



- 2 recent multicenter multinational randomized controlled trials
- children comatose within 6 hours of ROSC were randomly assigned to TTM to 32°C-34°C or to 36°C-37.5°C.
- Those on lower T range were cooled to 32°C-34°C for 48 hours, rewarmed over 16 - 24 hours, and maintained at 36°C-37.5°C until 5 days after the initiation of TTM.
- Children receiving TTM to the higher T range were actively maintained at 36°C-37.5°C for 5 days.

the percentage of survivors with favorable neurological outcomes at 1 year did not significantly differ between 2 groups.

## **Fever is common in children after resuscitation from CA**

- During PCAC, fever ( $\geq 38^{\circ}\text{C}$ ) should be aggressively treated.
- **Prompt availability and anticipatory use of cooling blankets and anti-pyretics should be used as a routine practice.**

- Remember that once TTM is initiated, the TT should be maintained consistently for 12-24 h, W/O intermittent rewarming, as unintentional or early re-warming has been associated with poor neurologic outcomes compared to patients who did not undergo TTM at all.
- Despite contradicting evidence, current guidelines recommend the use of **TTM in both OHCA and IHCA**, as well as in CA due to **shockable or non-shockable rhythms**.



# Identification and Treatment of Adrenal Dysfunction

- Approximately 30% of critically ill children have relative adrenal insufficiency, but this has not been evaluated in children resuscitated from CA.
- a recent meta-analysis did not demonstrate a difference in outcomes between those who did and those who did not receive exogenous steroids.
- Based on guidelines for the management of pediatric and neonatal sepsis consider steroid administration if the patient is at risk for adrenal insufficiency with refractory shock.
- Insufficient evidence to support the routine use of corticosteroids after CA.

Phase of Injury	Pre-Event	Cardiopulmonary Arrest	Post-Cardiac Arrest Syndrome			
Injury Mechanisms			<b>Brain Injury</b> <ul style="list-style-type: none"><li>Cerebral hypoperfusion</li><li>Cerebral hyperemia and hyperoxia</li><li>Cerebral inflammation</li><li>Impaired cerebrovascular autoregulation</li><li>Oxidative stress</li><li>Free-radical-mediated injury</li><li>Cortical and white matter injury</li></ul>	<b>Myocardial Dysfunction</b> <ul style="list-style-type: none"><li>Hypoxemic-hypotensive perfusion</li><li>Myocardial stunning</li><li>Peak around 8 hours</li><li>Resolves 48-72 hr</li></ul>	<b>Systemic Ischemia/Reperfusion</b> <ul style="list-style-type: none"><li>Hypoxemic-hypotensive perfusion</li><li>Free-radical-mediated reperfusion injury</li><li>SIRS</li><li>Adrenal Suppression</li></ul>	<b>Persistence of Precipitating Pathology</b>
Clinical Symptoms			Coma, Cerebral edema, Seizures, Myoclonus, Encephalopathy	Hypotension, LV & RV diastolic and systolic dysfunction, Low cardiac output, Arrhythmias, Pulmonary edema, Recurrent arrest	Coagulopathy, Hypotension, Pyrexia, Hypovolemia, Hyperglycemia, Impaired tissue oxygen utilization, Infection, Multi-organ dysfunction	Cognitive impairment, Spasticity, Sympathetic hyperarousal
Monitoring				<ul style="list-style-type: none"><li>Pulse oximetry</li><li>Capnography</li><li>Cardiac telemetry</li><li>Blood pressure monitoring</li><li>Temperature</li><li>Urine output</li></ul>	<ul style="list-style-type: none"><li>Organ perfusion (electrolytes)</li><li>Ventilation (PaCO<sub>2</sub> or end-tidal CO<sub>2</sub>)</li><li>Acid-base status (blood gases; lactate)</li><li>Inflammation and infection (CXR, CBC)</li><li>Coagulation; Kidney function</li><li>Echocardiography; Arrhythmia monitoring (consider electrophysiology consultation)</li><li>CNS injury (cEEG)</li><li>CNS imaging (if CNS cause suspected)</li></ul>	<ul style="list-style-type: none"><li>Cognitive, emotional, and physical disability assessments</li></ul>
Treatment Interventions		<ul style="list-style-type: none"><li>CPR</li><li>Early transport</li><li>Transport to pediatric tertiary care center</li><li>Proactive monitoring and support of organ function</li></ul>	<ul style="list-style-type: none"><li>Administer oxygen</li><li>Vasopressors</li><li>Parenteral fluids</li><li>Treat proximal cause of arrest</li></ul>	<ul style="list-style-type: none"><li>Targeted temperature management (32°C-34°C or 36°C-37.5°C)</li><li>Normoxia (94%—98%)</li><li>Normocapnia (PaCO<sub>2</sub> 35-45 mm Hg)</li><li>Avoid hypoxemia, hyperoxia, hypocapnia and hypercapnia</li><li>Set hemodynamic goals; keep SBP &gt; 5th %ile</li><li>Maintain normoglycemia</li><li>Treat seizures (clinical and electrographic)</li><li>Screen for ECMO</li><li>Monitor for and treat AKI; sedation as needed</li></ul>	<ul style="list-style-type: none"><li>Early mobilization</li><li>Consult rehabilitation services</li><li>Treat sympathetic hyperarousal</li></ul>	
Prognostic Factors	<ul style="list-style-type: none"><li>Age &gt; 1 yr</li><li>Preexisting condition</li><li>Interventions in place</li><li>Cause of arrest</li><li>Night / weekends</li><li>Congenital heart disease</li><li>Pulmonary artery hypertension</li></ul>	<ul style="list-style-type: none"><li>CPR duration</li><li>Witnessed</li><li>Bystander CPR</li><li>EMS response time</li><li>Calcium &amp; Bicarbonate administration</li><li>Shorter time to epinephrine</li><li>Non-shockable rhythm</li><li>Intubation</li><li>CPR quality</li><li>ECPR</li></ul>	<ul style="list-style-type: none"><li>Lack of pupillary responsiveness</li><li>Abnormal motor response to pain</li><li>Seizures</li><li>Early hypotension</li><li>Substantially abnormal EEG background</li><li>Elevated blood glucose</li><li>Elevated blood lactate</li><li>Neuron-specific enolase, S100B</li></ul>			

**Figure 2. Phases of cardiac arrest with associated mechanisms, clinical symptoms, monitoring, treatment interventions, and prognostics factors.**

AKI indicates acute kidney injury; CBC, complete blood count; CNS, central nervous system; CPR, cardiopulmonary resuscitation; CXR, chest x-ray; cEEG, continuous electroencephalogram; ECMO, extracorporeal membrane oxygenation; ECPR, extracorporeal cardiopulmonary resuscitation; EEG, electroencephalogram; EMS, emergency medical service; LV, left ventricular; RV, right ventricular; SBP, systolic blood pressure; and SIRS, systemic inflammatory response syndrome.



# prognosis

- ❑ Overall survival rates from 2005 to 2013 data collection range from only 6.4% to 10.2%.
- ❑ survival to hospital discharge after OHCA has not significantly changed over the past 10 years.
- ❑ Risk-adjusted rates of ROSC in IHCA increased from 42.9% in 2000 to 81.2% in 2009, and risk-adjusted rates of survival to discharge improved from 14.3% in 2000 to 43.4% in 2009 W/O an increase in unfavorable neurological outcome.
- ❑ Factors for all ages with a better prognosis:
  - short duration of arrest,
  - early initiation of CPR,
  - hypothermia as the cause, and
  - IHCA



# prognosis

- ❑ Factors associated with unfavorable neurologic outcomes from OHCA:
  - decreased age,
  - sudden infant death syndrome, and
  - Blunt trauma
  
- ❑ Factors associated with decreased survival after IHCA:
  - older age,
  - pre-existing conditions,
  - Interventions (ETT, mechanical ventilation),
  - use of vasopressors at the time of arrest, &
  - arrests occurring during night and weekend shifts.

❑ **For both OHCA and IHCA:**

- pre-arrest rhythms of bradycardia and VF/VT were associated with the highest survival, and
  - PEA was associated with higher survival than asystole.
- 
- Post-cardiac arrest brain injury and myocardial dysfunction are the leading causes of morbidity and mortality in children.
  - Myocardial dysfunction develops in ~2/3 of patients after ROSC, & may subsequently improve.

**Phase of  
Injury**

**Pre-Event**

**Prognostic  
Factors**

- Age > 1 yr
- Preexisting condition
- Interventions in place
- Cause of arrest
- Night / weekends
- Congenital heart disease
- Pulmonary artery hypertension



# Cardiopulmonary Arrest

- CPR duration
- Witnessed
- Bystander CPR
- EMS response time
- Calcium & Bicarbonate administration
- Shorter time to epinephrine
- Non-shockable rhythm
- Intubation
- CPR quality
- ECPR

## Post-Cardiac Arrest Syndrome

- Lack of pupillary responsiveness
- Abnormal motor response to pain
- Seizures
- Early hypotension
- Substantially abnormal EEG background
- Elevated blood glucose
- Elevated blood lactate
- Neuron-specific enolase, S100B

**Table 4. Summary of Key Prearrest and Intra-Arrest Factors That Are Independently Associated With Outcomes**

Phase	Factor*	Outcome Type	Survival	Arrest Location	
Prearrest	Younger age:				
	Fink et al <sup>13</sup>	Survival to hospital discharge	Decreased	OHCA	
	Goto et al <sup>188</sup>	1-mo survival			
	Older age <sup>21,188</sup>	Survival to hospital discharge	Decreased	IHCA	
	Preexisting condition: Genetic/metabolic <sup>152</sup> Acute renal failure <sup>7,107,190</sup> Sepsis <sup>21,191</sup> Hepatic insufficiency <sup>21</sup> Hematologic/oncologic/immunologic <sup>152,191,192</sup> Baseline neurological abnormality <sup>21,192</sup> Congenital heart disease <sup>21</sup>	Survival to hospital discharge	Decreased	IHCA	
	Preexisting lung/airway disease <sup>18</sup>	Survival to hospital discharge	Increased	OHCA	
	Postoperative patient <sup>152</sup> Post-cardiac surgery <sup>193</sup>	Survival to hospital discharge	Increased	IHCA	
	Intervention in place: Endotracheal tube <sup>152,190</sup> Vasopressor infusion <sup>17,191,192</sup>	Survival to hospital discharge	Decreased	IHCA	
	Cause of arrest:				
	SIDS <sup>194</sup>	1-y survival	Decreased	OHCA	
	Trauma <sup>195</sup>	Survival to hospital discharge	Decreased	IHCA	
	Drowning <sup>12,18</sup>	Survival to hospital discharge	Increased	OHCA	
	Asthma <sup>21</sup>	Survival to hospital discharge	Increased	IHCA	
	Day and time of arrest:				
	Nights <sup>196</sup>	Survival to hospital discharge	Decreased	IHCA	
	Nights <sup>196</sup>	1-mo survival	Decreased	OHCA	
	Weekends:				
	Meert et al <sup>194</sup>	1-y survival	Decreased	OHCA	
	Kitamura et al <sup>196</sup>	1-mo survival			
	Public-access defibrillation <sup>197</sup>	1-mo survival	Increased	OHCA	
	Shorter EMS response time <sup>198</sup>	1-mo survival	Increased	OHCA	
Intra-arrest	Witnessed status:				
	Goto et al <sup>188</sup>	1-mo survival	Increased	OHCA	
	Fink et al <sup>13</sup>	Survival to hospital discharge			
	Meert et al <sup>194</sup>	1-y survival			
	Andersen et al <sup>198</sup>	Survival to hospital discharge			
	Arrest rhythm VF/pVT:				
	Tijssen et al <sup>12</sup>	Survival to hospital discharge	Increased	OHCA	
	Kitamura et al <sup>196</sup>	1-mo survival			
	Goto et al <sup>188</sup>				
	Initial VF/pVT vs initial non-VF/pVT <sup>199,201</sup> : PEA vs asystole <sup>198</sup> Bradycardia <sup>199</sup>	Survival to hospital discharge	Increased	IHCA	
	PEA vs asystole <sup>198</sup>	Survival to hospital discharge	Increased	OHCA	
	Asystole <sup>13</sup> PEA <sup>12</sup>	Survival to hospital discharge	Decreased	OHCA	
	Subsequent VF/pVT vs primary VF/pVT <sup>199</sup> Subsequent VF/pVT vs primary non-VF/pVT <sup>199</sup>	Survival to hospital discharge	Decreased	IHCA	
	Subsequent VF/pVT vs sustained non-VF/pVT <sup>198</sup>	1-mo favorable neurological survival	Increased	OHCA	



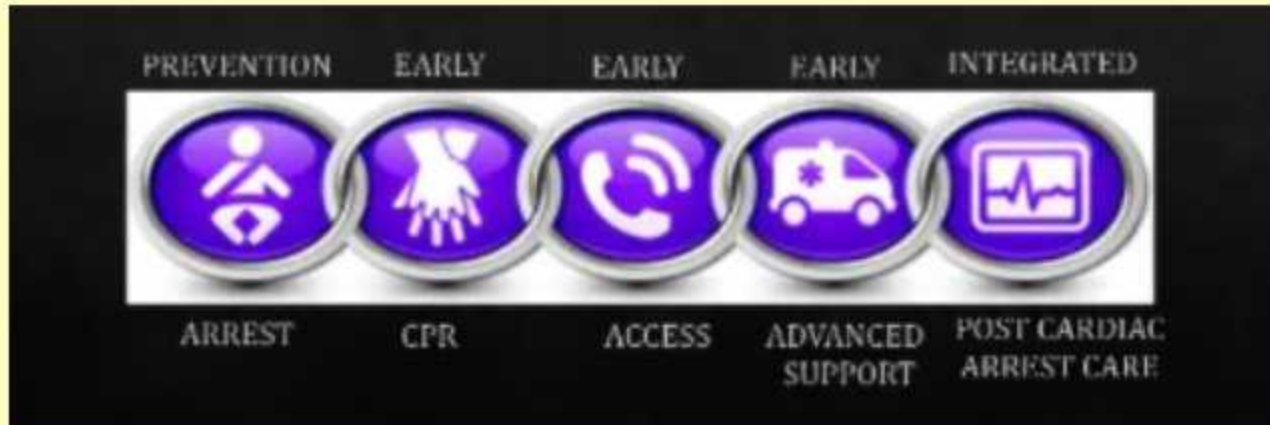
Table 4. Continued

Phase	Factor*	Outcome Type	Survival	Arrest Location
Intra-arrest (Continued)	Shorter time to shock for subsequent VF/VT <sup>188</sup>	1-mo favorable neurological survival	Increased	OHCA
	CPR with ventilation vs chest compression-only CPR:			
	Infants <sup>202</sup>	Survival to hospital discharge	Increased	OHCA
	>1 y of age <sup>202,203</sup>	1-mo favorable neurological survival or survival to hospital discharge	No difference	
	Bystander CPR <sup>200</sup>	1-mo survival	Increased	OHCA
	Dispatcher-assisted CPR <sup>200</sup>	1-mo survival	Increased	OHCA
	Less frequent epinephrine administration <sup>204</sup>	Survival to hospital discharge	Increased	IHCA
	Shorter time to epinephrine <sup>188</sup>	Survival to hospital discharge	Increased	IHCA
	Use of ECPR <sup>190</sup>	Survival to hospital discharge	Increased	IHCA
	Shorter EMS scene time <sup>12</sup>	Survival to hospital discharge	Increased	OHCA
	Diastolic blood pressure $\geq 25$ mmHg in infants, $\geq 30$ mmHg in children during CPR <sup>205</sup>	Survival to hospital discharge	Increased	IHCA
	AHA-compliant CPR depth (>1 y) $\geq 51$ mm <sup>206</sup>	Survival to hospital discharge	Increased	IHCA
	Drugs administered during CPR: Calcium <sup>87,152</sup> Sodium bicarbonate <sup>152,190</sup> Epinephrine <sup>190</sup> Atropine <sup>18</sup> Epinephrine <sup>18,188</sup>	Survival to hospital discharge	Decreased	IHCA
	Longer duration of CPR			
	Goto <sup>188</sup>	1-mo survival	Decreased	OHCA
	López-Herce et al <sup>207</sup>	1-y survival		OHCA
	Meert et al <sup>194</sup>			IHCA
	Del Castillo et al <sup>192</sup>	Survival to hospital discharge		
	Matos et al <sup>192</sup>			
	Endotracheal intubation during CPR <sup>208</sup>	Survival to hospital discharge	Decreased	IHCA

AHA indicates American Heart Association; CPR, cardiopulmonary resuscitation; ECPR, extracorporeal resuscitation (use of extracorporeal circulation during resuscitation); EMS, emergency medical services; IHCA, in-hospital cardiac arrest; OHCA, out-of-hospital cardiac arrest; PEA, pulseless electrical activity; SIDS, sudden infant death syndrome; VF/pVT, ventricular fibrillation/pulseless ventricular tachycardia; and VF/VT, ventricular fibrillation/ventricular tachycardia.

\*When a series of publications studying prognostic factors is derived from a single registry, the most recent publication from that registry is cited in this table.

- Survivors of cardiac arrest can have significant dysfunction, and parents of child survivors often report limitations in their daily activities.
- Children who survive cardiac arrest are often left with anoxic brain damage, and face numerous problems with daily living, so all outcomes should be explained in detail to family.



## Evaluation and Support for Cardiac Arrest Survivors

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**2020 (New):** It is recommended that pediatric cardiac arrest survivors be evaluated for rehabilitation services.

**2020 (New):** It is reasonable to refer pediatric cardiac arrest survivors for ongoing neurologic evaluation for at least the first year after cardiac arrest.

**Why:** There is growing recognition that recovery from cardiac arrest continues long after the initial hospitalization. Survivors may require ongoing integrated medical, rehabilitative, caregiver, and community support in the months to years after their cardiac arrest. A recent AHA scientific statement highlights the importance of supporting patients and families during this time to achieve the best possible long-term outcome.<sup>6</sup>





**Thanks for attention**



# Transfer to a pediatric center

- ❑ If the child is not being treated in a center with pediatric emergency and critical care expertise, should be stabilized and rapidly transferred for definitive care at a regional pediatric center.
- ❑ Critically ill or injured children typically benefit from transport by a team with pediatric expertise and advanced treatment capability.
- ❑ In some cases (expanding epidural hematoma) rapid transport by even a non-pediatric team may be advantageous.
- ❑ Prior to transfer, the clinician should speak directly to the clinician who will be taking charge of the patient at the receiving hospital.
- ❑ All **medical chart, medication record, lab results**, copies of ancillary studies [**radiographs, ECGs**]) should be sent.

# Family presence during resuscitation

- ❖ Observational studies indicate that caretakers should be given the option of being present during the in-hospital CPR of their child.

## **Key findings include:**

- ❖ Most parents believe it is their right, and reported that their presence was beneficial to the patient even if he or she was a family member.
- ❖ 2/3 of caretakers reported that their presence helped with their adjustment to the death and the grieving process.
- ❖ The presence of a family member, in most instances, was not stressful to staff and did not negatively impact staff performance, otherwise they should be respectfully asked to leave.



# Rapid response teams

- An **RRT**, also known as a **medical emergency team (MET)**, consists of personnel from medical, nursing and/or respiratory therapy who have critical care training and are available 24 hours /D, seven D/W for evaluation and treatment of patients who show signs of clinical deterioration and are located in non-critical care settings.
- ❑ A meta-analysis with a total of 347,618 patient admissions found that implementation of a **RRT** was associated with a **significant reduction in deaths from CA** when compared to historical control periods.
- ❑ However, **decreased mortality after implementation of a RRT** was not found in all studies.

# Rapid response teams

- A cohort study of 29,294 patient admissions (7257 admissions after institution of a RRT) compared hospital-wide mortality rates and rates of respiratory and CPAs outside of the ICU before and after implementation of an RRT in a children hospital. Major findings included:
- The mean monthly mortality rate decreased from 1.0 to 0.8 deaths/100 discharges
- The mean monthly code rate decreased from 2.5 to 0.7 codes/1000 patient admissions.

**The benefit of an RRT is not consistent across all settings.**



- Donation after circulatory determination of death are commonly encountered in emergency departments and include cases of SIDS, sepsis, abusive, or accidental trauma.
- It is preferred to defer any discussion involving the potential for organ donation to your local organ procurement organization.



- ❑ In 2017, the **Therapeutic Hypothermia After Pediatric CA** trial compared therapeutic hypothermia ( $33^{\circ}\text{C}$ ) with therapeutic normothermia ( $36.8^{\circ}\text{C}$ ) after OHCA, and did not show a statistically significant difference in **1-year neurologic outcomes or mortality**.
- ❑ One large trial including **950 patients** with OHCA compared a T target ( $36^{\circ}\text{C}$ ) to traditional TTM ( $33^{\circ}\text{C}$ ), & found **no difference in mortality** suggesting the importance of the intervention of TTM itself.

- **Therapeutic hypothermia** to maintain CBT at **32 - 34°C** has been evaluated in children based upon evidence for improved neurologic outcome in neonates and selected adults.
- However, for children resuscitated from OHCA **therapeutic hypothermia for 48 hours** has not shown improved outcomes.
- **No benefit of therapeutic hypothermia compared with therapeutic normothermia in 329 children resuscitated from IHCA.**

**OHCA: out of hospital cardiac arrest**  
**IHCA: in hospital cardiac arrest**

- ❑ Insufficient evidence to determine the optimal BS
  - ❑ ideal method of controlling BS
  - ❑ the ideal duration of any glucose control, and
  - ❑ the ideal frequency of glucose monitoring needed to reduce the risk of hypoglycemia.
- 
- Evidence indicates that **BS should be maintained below this threshold**, but the role of "tight control" that uses insulin to achieve a specified BS range is of uncertain value in children after CA.



# Drugs Used to Maintain CO

## ✧ Epinephrine

- ✧ 0.1-1  $\mu\text{g/kg/min}$  IV/IO inf.
- ✧ Low-dose infusions (0.1  $\mu\text{g/kg/min}$ ) generally produce  $\beta$ -adrenergic action (potent inotropy & decreased SVR).
- ✧ Higher-dose infusions (0.3  $\mu\text{g/kg/min}$ ) cause  $\alpha$ -adrenergic vasoconstriction.
- ✧ Because there is great inter-patient variability, titrate the drug to the desired effect.
- ✧ Epinephrine may be preferable to dopamine in patients (esp. infants) with marked circulatory instability & decompensated shock.

- If the etiology of the CA is suspected to be arrhythmia, pediatric cardiac consultation is strongly recommended then antiarrhythmic agents such as lidocaine or amiodarone should be considered.
- Remember that antiarrhythmic drugs like amiodarone, procainamide, and sotalol are contraindicated in patients with long-QT syndrome and Brugada syndrome.
- arrhythmias are commonly observed during TTM, particularly bradycardia, which usually do not require treatment.



# Drugs Used to Maintain CO

- Myocardial dysfunction is common after CA.
- SVR & PVR are increased except in some cases of septic shock.
- The potential adverse effects of catecholamines include:
  - local ischemia & ulceration,
  - tachycardia,
  - atrial & ventricular tachyarrhythmias,
  - HTN, &
  - metabolic changes (hyperglycemia, increased lactate concentration, & hypokalemia)



# Drugs Used to Maintain CO

## ➤ Norepinephrine

- 0.1-2  $\mu\text{g/kg/min}$  IV/IO inf.
- A potent inotropic & peripheral vasoconstrictor
- Titrate an infusion to Rx. shock with low SVR (septic, anaphylactic, spinal, or vasodilatory) unresponsive to fluid.

- ✧ If hypovolemia is suspected in a patient with cardiogenic shock, the clinician should carefully infuse **5 to 10 mL/kg of isotonic fluids** (N/S or Ringer's lactate) **over 10 to 20 minutes** followed by reevaluation of endpoints.

- Evaluate target endpoints:
  - Blood pressure (5th percentile minimum)
  - Quality of pulses (strong, central + distal)
  - Skin perfusion (warm, capillary refill <2 seconds)
  - Mental status (alert)
  - Urine output ( $\geq 1$  mL/kg per hour, once effective circulating volume is restored)

## Detecting and Treating Seizures After ROSC

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**2020 (Updated):** When resources are available, continuous electroencephalography monitoring is recommended for the detection of seizures following cardiac arrest in patients with persistent encephalopathy.

**2020 (Updated):** It is recommended to treat clinical seizures following cardiac arrest.

**2020 (Updated):** It is reasonable to treat nonconvulsive status epilepticus following cardiac arrest in consultation with experts.



**Table 81.7** Medications to Maintain Cardiac Output and for Postresuscitation Stabilization\*

MEDICATION	DOSE RANGE	COMMENT
Inamrinone	0.75-1 mg/kg IV/IO over 5 min; may repeat 2x; then: 2-20 µg/kg/min	Inodilator
Dobutamine	2-20 µg/kg/min IV/IO	Inotrope; vasodilator
Dopamine	2-20 µg/kg/min IV/IO in low doses; pressor in higher doses	Inotrope; chronotrope; renal and splanchnic vasodilator
Epinephrine	0.1-1 µg/kg/min IV/IO	Inotrope; chronotrope; vasodilator in low doses; vasopressor in higher doses
Milrinone	50-75 µg/kg IV/IO over 10-60 min then 0.5-0.75 µg/kg/min	Inodilator
Norepinephrine	0.1-2 µg/kg/min	Inotrope; vasopressor
Sodium nitroprusside	1-8 µg/kg/min	Vasodilator; prepare only in D5W

# Drugs Used to Maintain CO

## ✓ Dopamine

- ✓ 2-20  $\mu\text{g}/\text{kg}/\text{min}$  IV/IO inf.
- ✓ Titrate to Rx. shock that is unresponsive to fluid & when SVR is low.
- ✓ At higher doses ( $> 5 \mu\text{g}/\text{kg}/\text{min}$ ), dopamine stimulates cardiac  $\beta$ -adrenergic receptors, but this effect may be reduced in infants & in chronic CHF.
- ✓ Infusion rates  $> 20 \mu\text{g}/\text{kg}/\text{min}$  may result in excessive vasoconstriction.

# Drugs Used to Maintain CO

## ➤ Dobutamine Hydrochloride

- ✧ 2-20  $\mu\text{g/kg/min}$  IV/IO inf.
- ✧ A selective effect on  $\beta_1$ - &  $\beta_2$ -adrenergic receptors
- ✧ Increases myocardial contractility & usually decreases PVR.



# Drugs Used to Maintain CO

## ⌘ Sodium Nitroprusside

- ❑ 1-8  $\mu\text{g/kg/min}$  IV/IO inf
- ❑ Increases CO by decreasing vascular resistance (afterload)
- ❑ If hypotension is related to poor myocardial function, consider using a combination of sodium nitroprusside to reduce afterload & an inotrope to improve contractility.
- ❑ Prepare only in 5%DW.

# Drugs Used to Maintain CO

## ✧ Inodilators

- Inamrinone: 0.75-1 mg/kg IV/IO over 5 min; may repeat x 2; then: 2-20  $\mu\text{g/kg/min}$
- Milrinone: 50-75  $\mu\text{g/kg}$  IV/IO over 10-60 min; then 0.5-0.75  $\mu\text{g/kg/min}$
- Augment CO with little effect on myocardial O<sub>2</sub> demand
- For Rx. of myocardial dysfunction with increased systemic or pulmonary vascular resistance
- Administration of fluids may be required because of the vasodilatory effects.
- A long half-life with a long delay in reaching a new steady-state hemodynamic effect after changing the infusion rate (18 h with inamrinone & 4.5 h with milrinone).
- In case of toxicity, if you stop the infusion the adverse effects may persist for several hours.



- Clinical manifestations of brain injury after arrest include coma, cerebral edema, seizures, myoclonus, sympathetic hyperarousal, and long-term neurobehavioral deficits.

**Neuroimaging can help identify a cerebral cause of CA and assess the degree of severe brain injury.**

**The 2015 AHA guidelines recommend the time to prognosticate neurological outcomes in patients not treated with TTM is 72 hours after ROSC.**



**0 minutes**

- Recognition of shock:
  - Diminished peripheral pulses
  - Cool, pale, or mottled skin
  - Prolonged capillary refill time
  - Altered mental status
  - Tachycardia or bradycardia

**5 to 15 minutes**

- Identify and treat life-threatening conditions
- Administer 100 percent oxygen
- Perform endotracheal intubation in patients with airway compromise or impending respiratory failure
- Establish vascular access

- Infuse isotonic crystalloid (eg, normal saline):
  - 20 mL/kg over 5 to 10 minutes in patients with uncompensated shock\*
  - 20 mL/kg over 5 to 20 minutes in patients with compensated shock
- Identify need for time-sensitive treatments based upon underlying condition (eg, blood transfusion for hemorrhage, epinephrine for anaphylaxis, or prostaglandin E<sub>1</sub> for infants with ductal-dependent congenital heart disease)
- Initiate continuous monitoring of heart rate, blood pressure, and pulse oximetry
- Obtain diagnostic studies (including bedside glucose)

- Evaluate target endpoints:
  - Blood pressure (5th percentile minimum)
  - Quality of pulses (strong, central + distal)
  - Skin perfusion (warm, capillary refill <2 seconds)
  - Mental status (alert)
  - Urine output ( $\geq 1$  mL/kg per hour, once effective circulating volume is restored)

## Major New and Updated Recommendations

### Changes to the Assisted Ventilation Rate: Rescue Breathing

**2020 (Updated):** (PBLIS) For infants and children with a pulse but absent or inadequate respiratory effort, it is reasonable to give 1 breath every 2 to 3 seconds (20-30 breaths/min).

**2010 (Old):** (PBLIS) If there is a palpable pulse 60/min or greater but there is inadequate breathing, give rescue breaths at a rate of about 12 to 20/min (1 breath every 3-5 seconds) until spontaneous breathing resumes.

### Changes to the Assisted Ventilation Rate: Ventilation Rate During CPR With an Advanced Airway

**2020 (Updated):** (PALS) When performing CPR in infants and children with an advanced airway, it may be reasonable to target a respiratory rate range of 1 breath every 2 to 3 seconds (20-30/min), accounting for age and clinical condition. Rates exceeding these recommendations may compromise hemodynamics.

**2010 (Old):** (PALS) If the infant or child is intubated, ventilate at a rate of about 1 breath every 6 seconds (10/min) without interrupting chest compressions.

**Why:** New data show that higher ventilation rates (at least 30/min in infants [younger than 1 year] and at least 25/min in children) are associated with improved rates of ROSC and survival in pediatric IHCA. Although there are no data about the ideal ventilation rate during CPR without an advanced airway, or for children in respiratory arrest with or without an advanced airway, for simplicity of training, the respiratory arrest recommendation was standardized for both situations.

### Cuffed ETTs

**2020 (Updated):** It is reasonable to choose cuffed ETTs over uncuffed ETTs for intubating infants and children. When a cuffed ETT is used, attention should be paid to ETT size, position, and cuff inflation pressure (usually <20-25 cm H<sub>2</sub>O).

**2010 (Old):** Both cuffed and uncuffed ETTs are acceptable for intubating infants and children. In certain circumstances (eg, poor lung compliance, high airway resistance, or a large glottic air leak) a cuffed ETT may be preferable to an uncuffed tube, provided that attention is paid to [ensuring appropriate] ETT size, position, and cuff inflation pressure.

**Why:** Several studies and systematic reviews support the safety of cuffed ETTs and demonstrate decreased need for tube changes and reintubation. Cuffed tubes may decrease the risk of aspiration. Subglottic stenosis is rare when cuffed ETTs are used in children and careful technique is followed.

### Cricoid Pressure During Intubation

**2020 (Updated):** Routine use of cricoid pressure is not recommended during endotracheal intubation of pediatric patients.

**2010 (Old):** There is insufficient evidence to recommend routine application of cricoid pressure to prevent aspiration during endotracheal intubation in children.

**Why:** New studies have shown that routine use of cricoid pressure reduces intubation success rates and does not reduce the rate of regurgitation. The writing group has reaffirmed previous recommendations to discontinue cricoid pressure if it interferes with ventilation or the speed or ease of intubation.

### Emphasis on Early Epinephrine Administration

**2020 (Updated):** For pediatric patients in any setting, it is reasonable to administer the initial dose of epinephrine within 5 minutes from the start of chest compressions.

**2015 (Old):** It is reasonable to administer epinephrine in pediatric cardiac arrest.

**Why:** A study of children with IHCA who received epinephrine for an initial nonshockable rhythm (asystole and pulseless electrical activity) demonstrated that, for every minute of delay in administration of epinephrine, there was a significant decrease in ROSC, survival at 24 hours, survival to discharge, and survival with favorable neurological outcome.

Patients who received epinephrine within 5 minutes of CPR initiation compared with those who received epinephrine more than 5 minutes after CPR initiation were more likely to survive to discharge. Studies of pediatric OHCA demonstrated that earlier epinephrine administration increases rates of ROSC, survival to intensive care unit admission, survival to discharge, and 30-day survival.

In the 2018 version of the Pediatric Cardiac Arrest Algorithm, patients with nonshockable rhythms received epinephrine every 3 to 5 minutes, but early administration of epinephrine was not emphasized. Although the sequence of resuscitation has not changed, the algorithm and recommendation language have been updated to emphasize the importance of giving epinephrine as early as possible, particularly when the rhythm is nonshockable.

### Invasive Blood Pressure Monitoring to Assess CPR Quality

**2020 (Updated):** For patients with continuous invasive arterial blood pressure monitoring in place at the time of cardiac arrest, it is reasonable for providers to use diastolic blood pressure to assess CPR quality.



## Opioid Overdose

**2020 (Updated):** For patients in respiratory arrest, rescue breathing or bag-mask ventilation should be maintained until spontaneous breathing returns, and standard PBLs or PALS measures should continue if return of spontaneous breathing does not occur.

**2020 (Updated):** For a patient with suspected opioid overdose who has a definite pulse but no normal breathing or only gasping (ie, a respiratory arrest), in addition to providing standard PBLs or PALS, it is reasonable for responders to administer intramuscular or intranasal naloxone.

**2020 (Updated):** For patients known or suspected to be in cardiac arrest, in the absence of a proven benefit from the use of naloxone, standard resuscitative measures should take priority over naloxone administration, with a focus on high-quality CPR (compressions plus ventilation).

**2015 (Old):** Empiric administration of intramuscular or intranasal naloxone to all unresponsive opioid-associated life-threatening emergency patients may be reasonable as an adjunct to standard first aid and non-healthcare provider BLS protocols.

**2015 (Old):** ACLS providers should support ventilation and administer naloxone to patients with a perfusing cardiac rhythm and opioid-associated respiratory arrest or severe respiratory depression. Bag-mask ventilation should be maintained until spontaneous breathing returns, and standard ACLS measures should continue if return of spontaneous breathing does not occur.

**2015 (Old):** We can make no recommendation regarding the administration of naloxone in confirmed opioid-associated cardiac arrest.

**Why:** The opioid epidemic has not spared children. In the United States in 2018, opioid overdose caused 65 deaths in children younger than 15 years and 3618 deaths in people 15 to 24 years old,<sup>9</sup> and many more children required resuscitation. The 2020 Guidelines contain new recommendations

for managing children with respiratory arrest or cardiac arrest from opioid overdose.

These recommendations are identical for adults and children, except that compression-ventilation CPR is recommended for all pediatric victims of suspected cardiac arrest. Naloxone can be administered by trained providers, laypersons with focused training, and untrained laypersons. Separate treatment algorithms are provided for managing opioid-associated resuscitation emergencies by laypersons, who cannot reliably check for a pulse (Figure 5), and by trained rescuers (Figure 6). Opioid-associated OHCA is the subject of a 2020 AHA scientific statement.<sup>10</sup>

## Myocarditis

**2020 (New):** Given the high risk of cardiac arrest in children with acute myocarditis who demonstrate arrhythmias, heart block, ST-segment changes, and/or low cardiac output, early consideration of transfer to ICU monitoring and therapy is recommended.

**2020 (New):** For children with myocarditis or cardiomyopathy and refractory low cardiac output, prearrest use of ECLS or mechanical circulatory support can be beneficial to provide end-organ support and prevent cardiac arrest.

**2020 (New):** Given the challenges to successful resuscitation of children with myocarditis and cardiomyopathy, once cardiac arrest occurs, early consideration of extracorporeal CPR may be beneficial.

**Why:** Although myocarditis accounts for about 2% of sudden cardiovascular deaths in infants,<sup>11</sup> 5% of sudden cardiovascular deaths in children,<sup>11</sup> and 6% to 20% of sudden cardiac death in athletes, previous<sup>12,13</sup> PALS guidelines did not contain specific recommendations for management. These recommendations are consistent with the 2018 AHA scientific statement on CPR in infants and children with cardiac disease.<sup>14</sup>

## Single Ventricle: Recommendations for the Treatment of Preoperative and Postoperative Stage I Palliation (Norwood/Blalock-Tausig Shunt) Patients

**2020 (New):** Direct (superior vena cava catheter) and/or indirect (near infrared spectroscopy) oxygen saturation monitoring can be beneficial to trend and direct management in the critically ill neonate after stage I Norwood palliation or shunt placement.

**2020 (New):** In the patient with an appropriately restrictive shunt, manipulation of pulmonary vascular resistance may have little effect, whereas lowering systemic vascular resistance with the use of systemic vasodilators (alpha-adrenergic antagonists and/or phosphodiesterase type III inhibitors), with or without the use of oxygen, can be useful to increase systemic delivery of oxygen ( $DO_2$ ).

**2020 (New):** ECLS after stage I Norwood palliation can be useful to treat low systemic  $DO_2$ .

**2020 (New):** In the situation of known or suspected shunt obstruction, it is reasonable to administer oxygen, vasoactive agents to increase shunt perfusion pressure, and heparin (50–100 units/kg bolus) while preparing for catheter-based or surgical intervention.

**2020 (Updated):** For neonates prior to stage I repair with pulmonary over-circulation and symptomatic low systemic cardiac output and  $DO_2$ , it is reasonable to target a  $PaCO_2$  of 50 to 60 mm Hg. This can be achieved during mechanical ventilation by reducing minute ventilation or by administering analgesia/sedation with or without neuromuscular blockade.

**2010 (Old):** Neonates in a prearrest state due to elevated pulmonary-to-systemic flow ratio prior to Stage I repair might benefit from a  $PaCO_2$  of 50 to 60 mm Hg, which can be achieved during mechanical ventilation by reducing minute ventilation, increasing the inspired fraction of  $CO_2$ , or administering opioids with or without chemical paralysis.



**2015 (Old):** For patients with invasive hemodynamic monitoring in place at the time of cardiac arrest, it may be reasonable for rescuers to use blood pressure to guide CPR quality.

**Why:** Providing high-quality chest compressions is critical to successful resuscitation. A new study shows that, among pediatric patients receiving CPR with an arterial line in place, rates of survival with favorable neurologic outcome were improved if the diastolic blood pressure was at least 25 mm Hg in infants and at least 30 mm Hg in children.<sup>2</sup>

### Detecting and Treating Seizures After ROSC

**2020 (Updated):** When resources are available, continuous electroencephalography monitoring is recommended for the detection of seizures following cardiac arrest in patients with persistent encephalopathy.

**2020 (Updated):** It is recommended to treat clinical seizures following cardiac arrest.

**2020 (Updated):** It is reasonable to treat nonconvulsive status epilepticus following cardiac arrest in consultation with experts.

**2015 (Old):** An electroencephalography for the diagnosis of seizure should be promptly performed and interpreted and then should be monitored frequently or continuously in comatose patients after ROSC.

**2015 (Old):** The same anticonvulsant regimens for the treatment of status epilepticus caused by other etiologies may be considered after cardiac arrest.

**Why:** For the first time, the Guidelines provide pediatric-specific recommendations for managing seizures after cardiac arrest. Nonconvulsive seizures, including nonconvulsive status epilepticus, are common and cannot be detected without electroencephalography. Although outcome data from the post-cardiac arrest population are lacking, both convulsive and nonconvulsive status epilepticus are associated with poor outcome, and

treatment of status epilepticus is beneficial in pediatric patients in general.

### Evaluation and Support for Cardiac Arrest Survivors

**2020 (New):** It is recommended that pediatric cardiac arrest survivors be evaluated for rehabilitation services.

**2020 (New):** It is reasonable to refer pediatric cardiac arrest survivors for ongoing neurologic evaluation for at least the first year after cardiac arrest.

**Why:** There is growing recognition that recovery from cardiac arrest continues long after the initial hospitalization. Survivors may require ongoing integrated medical, rehabilitative, caregiver, and community support in the months to years after their cardiac arrest. A recent AHA scientific statement highlights the importance of supporting patients and families during this time to achieve the best possible long-term outcome.<sup>3</sup>

### Septic Shock

#### Fluid Boluses

**2020 (Updated):** In patients with septic shock, it is reasonable to administer fluid in 10 mL/kg or 20 mL/kg aliquots with frequent reassessment.

**2015 (Old):** Administration of an initial fluid bolus of 20 mL/kg to infants and children with shock is reasonable, including those with conditions such as severe sepsis, severe malaria, and dengue.

#### Choice of Vasopressor

**2020 (New):** In infants and children with fluid-refractory septic shock, it is reasonable to use either epinephrine or norepinephrine as an initial vasoactive infusion.

**2020 (New):** In infants and children with fluid-refractory septic shock, if epinephrine or norepinephrine are unavailable, dopamine may be considered.

### Corticosteroid Administration

**2020 (New):** For infants and children with septic shock unresponsive to fluids and requiring vasoactive support, it may be reasonable to consider stress-dose corticosteroids.

**Why:** Although fluids remain the mainstay of initial therapy for infants and children in shock, especially in hypovolemic and septic shock, fluid overload can lead to increased morbidity. In recent trials of patients with septic shock, those who received higher fluid volumes or faster fluid resuscitation were more likely to develop clinically significant fluid overload and require mechanical ventilation. The writing group reaffirmed previous recommendations to reassess patients after each fluid bolus and to use either crystalloid or colloid fluids for septic shock resuscitation.

Previous versions of the Guidelines did not provide recommendations about choice of vasopressor or the use of corticosteroids in septic shock. Two RCTs suggest that epinephrine is superior to dopamine as the initial vasopressor in pediatric septic shock, and norepinephrine is also appropriate. Recent clinical trials suggest a benefit from corticosteroid administration in some pediatric patients with refractory septic shock.

### Hemorrhagic Shock

**2020 (New):** Among infants and children with hypotensive hemorrhagic shock following trauma, it is reasonable to administer blood products, when available, instead of crystalloid for ongoing volume resuscitation.

**Why:** Previous versions of the Guidelines did not differentiate the treatment of hemorrhagic shock from other causes of hypovolemic shock. A growing body of evidence (largely from adults but with some pediatric data) suggests a benefit to early, balanced resuscitation using packed red blood cells, fresh frozen plasma, and platelets. Balanced resuscitation is supported by recommendations from the several US and international trauma societies.

Inadequate response

Targets achieved

- Continue monitoring and treatment of shock
- Treat underlying condition
- Admit to hospital

15 to 30 minutes

- Begin treatment of glucose, electrolyte, and calcium abnormalities
- For possible cardiogenic shock, begin vasoactive drug therapy ¶
- For possible sepsis, give antibiotics
- Repeat isotonic crystalloid infusion in 20 mL/kg boluses as needed for persistence of decreased perfusion to a total of 60 mL/kg Δ
- Evaluate target endpoints after each bolus

Inadequate response

Targets achieved

- Continue monitoring and treatment of shock
- Treat underlying condition
- Admit to hospital

30 to 60 minutes

- Re-evaluate presumed cause of shock
- For possible hypovolemic shock, re-evaluate estimate of fluid losses, continue fluid replacement, consider colloid
- For possible sepsis, unresponsive to fluid, begin vasoactive drug therapy ◇
- For hemorrhagic shock, give blood products

**0 minutes**

- Recognition of shock:
  - Diminished peripheral pulses
  - Cool, pale, or mottled skin
  - Prolonged capillary refill time
  - Altered mental status
  - Tachycardia or bradycardia

**5 to 15 minutes**

- Identify and treat life-threatening conditions
- Administer 100 percent oxygen
- Perform endotracheal intubation in patients with airway compromise or impending respiratory failure
- Establish vascular access

- Infuse isotonic crystalloid (eg, normal saline):
  - 20 mL/kg over 5 to 10 minutes in patients with uncompensated shock\*
  - 20 mL/kg over 5 to 20 minutes in patients with compensated shock
- Identify need for time-sensitive treatments based upon underlying condition (eg, blood transfusion for hemorrhage, epinephrine for anaphylaxis, or prostaglandin E<sub>1</sub> for infants with ductal-dependent congenital heart disease)
- Initiate continuous monitoring of heart rate, blood pressure, and pulse oximetry
- Obtain diagnostic studies (including bedside glucose)

- Evaluate target endpoints:
  - Blood pressure (5th percentile minimum)
  - Quality of pulses (strong, central + distal)
  - Skin perfusion (warm, capillary refill <2 seconds)
  - Mental status (alert)
  - Urine output ( $\geq 1$  mL/kg per hour, once effective circulating volume is restored)

Inadequate response

Targets achieved

- Continue monitoring and treatment of shock
- Treat underlying condition
- Admit to hospital

**15 to 30 minutes**

- Begin treatment of glucose, electrolyte, and calcium abnormalities
- For possible cardiogenic shock, begin vasoactive drug therapy<sup>¶</sup>
- For possible sepsis, give antibiotics
- Repeat isotonic crystalloid infusion in 20 mL/kg boluses as needed for persistence of decreased perfusion to a total of 60 mL/kg<sup>Δ</sup>
- Evaluate target endpoints after each bolus

Inadequate response

Targets achieved

- Continue monitoring and treatment of shock
- Treat underlying condition
- Admit to hospital

**30 to 60 minutes**

- Re-evaluate presumed cause of shock
- For possible hypovolemic shock, re-evaluate estimate of fluid losses, continue fluid replacement, consider colloid
- For possible sepsis, unresponsive to fluid, begin vasoactive drug therapy<sup>◇</sup>
- For hemorrhagic shock, give blood products