Overview

Resuscitation science is undergoing a renaissance, with a rapid expansion of our understanding of the physiology of resuscitation and a dramatic increase in outcomes studies guiding therapeutic interventions. Hospital-based resuscitation has unique features related to the etiology of arrest, the timing of various arrest phases, and the resources and personnel available to respond. Our discussion here will be structured around the initial assessment and optimal treatment of the cardiopulmonary arrest patient, post-arrest care, and evaluation and treatment of the perfusing patient including rapid response team concepts.

Core Concepts

I. There has been an exponential increase in our knowledge regarding the resuscitation physiology and optimal therapy, requiring more frequent updates and a flexible curriculum.

II. Resuscitation is a rare and stressful event, even for healthcare providers, underscoring the importance of simplicity, consistency, and role-specific training.

III. Resuscitation requires increasingly complex teamwork and choreography, which mandates regular simulation-based training.

IV. Hospital resuscitation is different than prehospital resuscitation, with most arrests representing the final consequence of hypoxemia and hypotension. This requires a different approach to resuscitation and affords the opportunity to intervene during the pre-arrest period.

V. Continuous quality improvement efforts and performance evaluations should be closely integrated with resuscitation training.
**BASIC RESUSCITATION ALGORITHM**

**Unresponsive**
- **Open Airway** (head tilt/chin lift)
  - Agonal breathing/apneic
  - No definite pulse

**Responsive**
- **Breathing**
  - Definite pulse

**Pulse Check (optional)**
- **Supportive Measures**
  - Support airway (head tilt/chin lift/jaw thrust, NPA)
  - Initiate RRT or Code Blue (x36111)
  - Obtain vital signs
  - Monitor (consider placing defib pads)
  - Ensure vascular access

**Cardiopulmonary Arrest**
- Yell for help
- Initiate Code Blue (x36111)
- Start chest compressions

**Defibrillator Available**
- **Place Defibrillator**
  - Defibrillator mode (1)
  - Analyze (2)
  - Shock (3) if indicated

**BVM Available**
- **BVM Ventilation**
  - 2-person BVM as soon as possible
  - Optimal mask position (jaw lift, head tilt, tight seal, “two thumbs up” mask hold)
  - 1 breath per 10 compressions (continuous compressions with no pauses)

- **Circulation Issue**
  - Supine (watch airway)
  - Fluid bolus
  - Pressors
  - Blood

- **Ventilation Issue**
  - Oxygen
  - Upright position
  - Consider Narcan
  - Assist BVM

- **Dysrhythmia**
  - Supine (watch airway)
  - Place defib pads
  - Prep defibrillator for pacing/cardioversion

- **Neurological Issue**
  - Oxygen, fluids
  - Check glucose
  - Fluid bolus
  - Consider Narcan

- **Resume compressions immediately**
- **Analyze for possible shock every 2 min**
- **Await Code Blue Team**
Initial Assessment

The goal in the initial assessment of the potential arrest victim is to rapidly identify cardiopulmonary arrest, initiate compressions, and call for help. The secondary goals include evaluation for possible defibrillation, and initiation of ventilations.

Responsiveness
In most cases, the arrest victim will be identified by a lack of responsiveness. Use of verbal and tactile stimuli to determine responsiveness should be immediately employed. A victim in cardiopulmonary arrest will be completely unresponsive. Clearly, this approach would not be appropriate for a paralyzed, intubated patient.

Spontaneous ventilation
The cessation of spontaneous breathing is one of the first manifestations of loss of cerebral perfusion. Thus, the lack of spontaneous breaths can be used as an indicator of cardiopulmonary arrest. The “gulping” or “gasping” respirations in the immediate post-arrest period do not count as spontaneous breathing. Again, this approach cannot be used for a patient undergoing mechanical ventilation or following administration of paralytics.

Pulse check
The absence of a palpable pulse has long been considered the gold standard for determining cardiopulmonary arrest. However, even the most experienced providers routinely err in making this determination. Thus, unresponsiveness and the absence of spontaneous breathing can be considered adequate to initiate chest compressions. It is worth noting that spontaneous breathing may not return immediately upon successful resuscitation, making palpation of a pulse important in determining perfusion status after resuscitation efforts have been initiated. In addition, palpation of pulses may be necessary with mechanical ventilation, since responsiveness and the presence of spontaneous breathing may be difficult to assess in these patients.

Other
Several additional sources of data may be used to help determine cardiopulmonary arrest. A monitor showing asystole, ventricular fibrillation, or a slow ventricular rhythm (<20 beats/min) can be assumed to represent cardiopulmonary arrest. The pulse oximetry or arterial line waveform will also disappear with cardiopulmonary arrest. A sudden drop in heart rate in a hypotensive or hypoxemic patient is an indication of impending arrest.
Chest Compressions

Compressions have become the foundation of resuscitation from cardiopulmonary arrest, regardless of etiology. The primary focus of the resuscitation should be the performance of continuous quality compressions.

**Continuous compressions**
The goal of chest compressions is to raise aortic pressure and improve perfusion. Unfortunately, it takes some time to reach a plateau pressure, even with good compressions, while interrupting compressions leads to an immediate drop in pressure. Thus, chest compressions should be performed continuously throughout the resuscitation, without pause for ventilation, rhythm analysis, intubation, or vascular access. In addition, compressions should be maintained during defibrillation charge and immediately following each shock.

**Compression depth**
Deeper compressions with full recoil produce better perfusion. This may limit the rate of compressions for larger patients. The compressor should be directly above the patient, with hands on the lower sternum, for maximum depth.

**Compression rate**
Compressions should be performed at the fastest rate possible to allow maximum depth and recoil, generally between 80/min (larger patients) and 120/min (smaller patients).

**Switching compressors**
Chest compressions produce significant fatigue when performed properly. Each individual should perform compressions no longer than 2 minutes at a time and may be relieved sooner if compression depth/recoil deteriorate or rate slows. Switching compressors should occur quickly with minimal pause in compressions. In addition, compressors should not be switched at the time of defibrillation to ensure immediate resumption of compressions following each shock.
Defibrillation

Ventricular fibrillation (VF) and pulseless ventricular tachycardia (PVT), which are “shockable” rhythms, require defibrillation with an automated external defibrillator (AED). The importance of compressions in defibrillation must be appreciated.

Compressions and defibrillation
The heart in a shockable rhythm must be “primed” with chest compressions prior to defibrillation once arrest times exceed 4-5 minutes. This priming effect decays very quickly, requiring compressions to be performed as close to the moment of defibrillation as possible. In addition, the heart requires perfusion immediately following defibrillation to encourage a viable rhythm. This requires the resumption of compressions immediately after each shock. If a shock is unsuccessful in converting from a shockable rhythm, several minutes of chest compressions are the most effective way to improve the chances of successful defibrillation with the next shock.

Witnessed arrest
Rapid defibrillation is the top priority for a “witnessed” (including monitored) arrest. The closest provider should initiate compressions while yelling for help. This should prompt rapid delivery of the defibrillator for immediate evaluation for a shockable rhythm while “priming” is maintained.

Unwitnessed arrest
Once an arrest exceeds 4-5 minutes, which is common in non-monitored settings, the fibrillating heart must be “primed” prior to a shock. Again, compressions should be initiated once cardiopulmonary arrest is confirmed. A “code blue” should be activated (or 9-1-1 called), and the closest defibrillator accessed. A shock may be delivered immediately if compressions have been performed for several minutes. Otherwise, at least 2-3 minutes of compressions should be performed prior to the first defibrillation attempt.
Ventilation

The role of ventilation in resuscitation from cardiopulmonary arrest has been de-emphasized due to the relatively lower oxygen requirements in an arrest state and the potential for positive-pressure ventilation to impede cardiac output. The initial rescuer should perform compressions alone, with ventilations initiated by subsequent responders once a bag-valve-mask becomes available.

Compression-to-ventilation ratio
The consequence of stopping chest compressions to provide ventilations appears to be too great to justify “interrupted” CPR. Instead, chest compressions should be continuous, with interposed ventilations delivered every 10\textsuperscript{th} compression.

Bag-valve-mask ventilation
The initial approach to ventilation should include the use of a bag-valve-mask by two rescuers. The first rescuer holds the mask tightly to the patient’s face using the “two thumbs up” approach, with head tilt and jaw thrust. The second rescuer squeezes the bag to deliver interposed ventilations, one every ten compressions. There should be no pause in compressions to deliver ventilations. Nasopharyngeal and oropharyngeal airways should be used whenever possible to maintain airway patency. Cricoid pressure can be applied by a third rescuer to minimize gastric insufflation.

Post-intubation ventilation
Once endotracheal tube confirmation has been performed, the ventilator continues to squeeze the bag to deliver interposed ventilations every 10\textsuperscript{th} compression. There should be no pause in compressions to deliver ventilations.
**All arrest patients**

*Causes of cardiopulmonary arrest*

The vast majority of inpatient cardiopulmonary arrest comes as the end result of hypoxemia or hypotension, which are addressed through high quality compressions, early pressor administration, and optimal ventilation. In addition, the underlying etiology of arrest should be considered and attempts made to reverse these whenever possible.

- Hypovolemic/shock – IV fluid bolus and/or blood product administration
- Suspected hyperkalemia (patient with renal disease or receiving potassium/digoxin/spironolactone) – sodium bicarbonate and calcium chloride administration
- Suspected hypoglycemia – D50
- Suspected acidosis – sodium bicarbonate
- Suspected hypomagnesemia (torsade de pointes or malnourishment) – magnesium sulfate
- Suspected pneumothorax (trauma or COPD patient) – needle decompression or chest tube insertion
- Suspected tamponade – pericardiocentesis
- Suspected vagal event - atropine
- Coronary thrombosis (recurrent VF/VT) – expedited revascularization

*Rhythm and pulse checks*

The general approach to resuscitation from cardiopulmonary arrest should emphasize continuous chest compressions, with minimal interruptions. Determining return of spontaneous circulation (ROSC) following resuscitation attempts is problematic, as extended periods of time may be spent (inappropriately) determining whether a pulse is present. In the majority of cases, CPR will be performed until arrival of the code team or paramedics.

If an AED is not available, then CPR should be performed continuously until arrival of the code team or paramedics. If an AED is accessed, then periodic brief pauses in CPR (every 2 minutes) are indicated to determine whether the rhythm is shockable and allow for defibrillation attempts. If the rhythm is determined to be “non-shockable”, then the patient may be assessed for ROSC. Since breathing may not return immediately, a pulse check must be performed. Unless there is absolute certainty about the presence of a pulse, compressions should be restarted as soon as possible.

*Post-arrest care*

Maintaining perfusion and oxygenation are the most important factors in preventing deterioration into cardiopulmonary arrest. Ventilations should be continued at a slow-to-moderate rate to avoid the hemodynamic effects of overaggressive ventilation. Fluids and early pressor infusions should be considered with hemodynamic instability, especially with systolic blood pressure values <70 mmHg.
Rapid Response Teams

The majority of hospital arrests result from hypoperfusion or hypoxemia. The majority of these patients manifest vital sign abnormalities for hours prior to arrest. Finally, much of the morbidity that accompanies diseases such as sepsis and acute coronary syndrome can be prevented with early intervention. Triggers for rapid response team activation fall into the following categories: circulation, ventilation, neurological, and infectious.

Circulatory issue
Indications of hypoperfusion include hypotension, tachycardia, altered mental status, and serum indicators of acidosis (low pH, elevated lactate, low bicarbonate, wide anion gap, elevated base deficit). Acute coronary syndrome and stroke represent focal circulatory issues. Dysrhythmias can cause circulatory problems by creating a heart rate that is too fast or slow, although the majority of tachycardias/bradycardias are caused by hypoxemia or hypoperfusion from other causes. Circulatory triggers include:

- 90>SBP>170
- 55>HR>120
- Acute chest discomfort
- Acute blood loss

While a diagnostic workup may be indicated, therapeutic interventions should occur in parallel to prevent further deterioration. These include:

- Supine positioning, which can create problems with airway patency in patients with altered mental status
- Fluid boluses
- Blood transfusion
- Pressor agents
**Ventilatory issue**

Hypoxemia is one of the most common etiologies of deterioration and cardiopulmonary arrest in the hospital environment. This may occur in patients with known pulmonary disease (pneumonia, COPD, asthma) or with the airway obstruction that accompanies a decrease in mental status (hypotension, hypoxemia, hypoglycemia, analgesia/sedation, sleep apnea). Respiratory triggers include:

- Increased work of breathing
- Stridor/noisy breathing
- 12>RR>28
- SpO2 <92% with increased FiO2
- ABG for respiratory concerns

While a diagnostic workup may be indicated, therapeutic interventions should occur in parallel to prevent further deterioration. These include:

- Maintaining airway patency (head tilt, chin lift, jaw thrust, nasopharyngeal airway, reversal of altered mental status)
- Supplemental oxygen with any hypoxemia (SpO2 <92%)
- Upright positioning
- Assisted ventilation using bag-valve-mask or bilevel ventilation (BiPAP)
- Intubation may be required and should be performed prior to arrest
**Neurological issue**
The majority of acute neurological deficits within the inpatient population, especially global alterations in mental status, are secondary to hypoperfusion, hypoxemia, hypoglycemia, or seizures. A stroke code should be activated with any concern about acute stroke, especially with focal neurological deficits. The rapid response or code blue teams should be activated with altered mental status or other signs of hypoperfusion/hypoxemia, including restlessness/agitation or confusion.

**Infectious issue**
Hyperthermia (T >39.5°C), hypothermia (T <35°C), or clinical suspicion of systemic infection should prompt a workup as well as an aggressive approach to therapy to avoid decompensated sepsis. Early sepsis criteria include indications of hypoperfusion along with clinical suspicion of infectious etiology. Interventions include appropriate hemodynamic monitoring, early administration of antibiotics, aggressive volume replacement, transfusion, use of pressors to promote perfusion (dobutamine), and consideration of steroids with suspected adrenal insufficiency.
# BART SKILLS CHECKLIST

Name: ___________________________ Date: ____________

Unit: ___________ Email: ________________ Instructor: _______________

## Initial Assessment
- Responsiveness
- Open airway
- Check for spontaneous breathing
- Pulse check (optional)
- Call for help (Code Blue or 9-1-1)
- Start compressions

## Compressions
- Appropriate depth & recoil
- Rate 80-120/min (guided by depth/recoil)
- Switch every 2 min (or less) with minimal delays

## Defibrillation
- Proper use of AED
- Clear & shock if indicated
- Restart compressions immediately

## Ventilations
- No pause in compressions
- One breath every 10 compressions
- Proper mask hold ("two thumbs up" with head tilt/jaw thrust/NPA/OPA)

## All Patients
- Continuous compressions

## Perfusion Check
- Appropriate interval (2 min) since last perfusion check or shock
- Hold compressions and perform AED check
- Pulse check if rhythm not shockable

## Perfusing Patients
- Oxygen administration
- Upright positioning (ventilatory issue)
- Assisted ventilations
- Supine positioning (circulatory issue)
- Transition to full arrest

## Pediatrics & Choking
- Appropriate pediatric CPR
- Infant choking (back blows & chest squeeze)
- Pediatric/adult choking (Heimlich & abdominal thrusts)

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