Optimizing Outcomes in Cardiac Arrest Care

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• American Heart Association - Volunteer
  • Member, ACLS Subcommittee of ECC
  • 2010 Guideline writing group “Special Situations” and “Post-arrest Care”
  • Member of Task Force - Experienced Provider Course
  • Member, Research Task Force and Clinical – GWTG
  • Liaison from AHA to ILCOR - ACLS
Objectives

• To review cardiac arrest pathophysiology and mechanisms to improve outcome

• To evaluate ACLS interventions to maximize outcome in cardiac arrest

• To briefly review post-cardiac arrest care and management strategies
The Chain of Survival
The *NEW* Chain of Survival
It’s as easy as: C.A.B.

• **Change:**
  - From A-B-C to C-A-B
  - Initiate chest compressions before ventilations

• **Why?**
  - Goal: To reduce delay to CPR, sequence begins with a skill that everyone can perform
  - Emphasize primary importance of chest compressions for professional rescuers
Mechanism of CPR

CPP = Aortic Pressure - Right Atrial Pressure
(CPP = Coronary Perfusion Pressure)
Fig 1.—Distribution of initial coronary perfusion pressures among patients without and with return of spontaneous circulation (ROSC). Each dot represents a patient. The bar is the mean ± SD.

Fig 2.—Distribution of maximal coronary perfusion pressures among patients without and with return of spontaneous circulation (ROSC). Each dot represents a patient. The bar is the mean ± SD.
Mechanism of CPR

CPP = Aortic Pressure - Right Atrial Pressure
(CPP = Coronary Perfusion Pressure)

CPP > 15 mmHg
What Increases CPP??

- Chest Compressions
  - More rapid
  - Deeper
  - Less Pauses

- Vasopressor Therapy
  - Epinephrine
  - Vasopressin
Compression Depth and ROSC

(Edelson, Resuscitation, 2006)
Compression Rate and ROSC

(Abella, Circulation, 2005)
Pre-Shock Pauses and ROSC

(Edelson, Resuscitation, 2006)
What Decreases CPP?

• Excessive Ventilations
Coronary Perfusion Pressure

CPP = AoD - RaM

CPP > 15 = adequate coronary perfusion
CPR Quality
- Push hard (≥2 inches [5 cm]) and fast (≥100/min) and allow complete chest recoil
- Minimize interruptions in compressions
- Avoid excessive ventilation
- Rotate compressor every 2 minutes
- If no advanced airway, 30:2 compression-ventilation ratio
- Quantitative waveform capnography
  - If PETCO$_2$ <10 mm Hg, attempt to improve CPR quality
- Intra-arterial pressure
  - If relaxation phase (diastolic) pressure <20 mm Hg, attempt to improve CPR quality

Return of Spontaneous Circulation (ROSC)
- Pulse and blood pressure
- Abrupt sustained increase in PETCO$_2$ (typically ≥40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

Shock Energy
- Biphasic: Manufacturer recommendation (120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered.
- Monophasic: 360 J

Drug Therapy
- Epinephrine IV/IO access
  - Epinephrine every 3-5 minutes
  - Amiodarone for refractory VF/VT
- Consider Advanced Airway
  - Quantitative waveform capnography
- Treat Reversible Causes

Continuous CPR
- Monitor CPR Quality
- Start CPR
  - Give oxygen
  - Attach monitor/defibrillator
- 2 minutes
- Check Rhythm
- If VF/VT
  - Shock
- Post-Cardiac Arrest Care

Shout for Help/Activate Emergency Response
• **Delayed Defibrillation**: 30% of patients
• **Lower probability of survival to discharge**: 22% versus 39%
• **Associated with**:
  • Non-cardiac admitting diagnosis
  • Hospital Beds < 250
  • Unmonitored unit
  • Nights/weekends
Time to Defibrillation and Survival

(Chan et al, NEJM, 2008)
ACLS Drugs – Summary

- Epinephrine: dose, interval unchanged
- Vasopressin: dose, use unchanged
- Amiodarone: dose, indications unchanged
- Lidocaine: dose, indications unchanged
- Atropine: Removed from pulseless arrest algorithm
- Sodium Bicarbonate: Routine use not recommended
- Calcium: Routine administration not recommended
- Magnesium: Routine administration not recommended.
Why Give Vasopressors in Cardiac Arrest?

- 1903/1906: Crile and Dolley
  - Intravenous epinephrine markedly improved resuscitation in dogs

- 1960s: Pearson and Redding
  - Performed series of experiments with a dog model of CPR employing 1 mg of epinephrine
# Epinephrine in Cardiac Arrest

<table>
<thead>
<tr>
<th>Method</th>
<th>Score</th>
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<tbody>
<tr>
<td>PPV</td>
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</tr>
<tr>
<td>PPV/Compressions</td>
<td>2/10</td>
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<tr>
<td>PPV/Epi</td>
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(Pearson and Redding Am Heart J 1963)
<table>
<thead>
<tr>
<th>Drug</th>
<th>Time of Arrest (min)</th>
<th>CPP</th>
<th>Result</th>
</tr>
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<tbody>
<tr>
<td>No Drug</td>
<td>10</td>
<td>18</td>
<td>3/10</td>
</tr>
<tr>
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<td>10</td>
<td>35</td>
<td>10/10</td>
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<td>12</td>
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<td>Phenyleph.</td>
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Vasopressor Mechanisms

Epinephrine: ALPHA + BETA

Isoproterenol: BETA

Phenylephrine: ALPHA
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“Patients receiving adrenaline during cardiac arrest had no statistically significant improvement in the primary outcome of survival to hospital discharge although there was a significantly improved likelihood of ROSC.”

Epinephrine Versus Placebo

- Pre-hospital ROSC: 8.4% (placebo) vs 23.5% (epinephrine)
- ED to hospital admission: 13% (placebo) vs 25.4% (epinephrine)
- Hospital discharge: 1.9% (placebo) vs 4% (epi) [NS] (50% relative reduction in mortality though not enough patients for statistical significance – thus, caution with interpretation of “negative” trial)

Time To Epinephrine - Adults

(Donnino et al. Circulation – Abstract)
Time To Epinephrine

(Donnino et al. *Circulation – Abstract*)
Time To Epinephrine

![Diagram showing survival with good neurologic function based on minute of first epinephrine administration.]

(Donnino et al. *Circulation* – Abstract)
Time To Epinephrine - pediatrics

(Unpublished Data - GWTG)
Amiodarone Vs. Lidocaine

Survival to Hospital Discharge??

No Difference but not powered for this

Amiodarone 5% vs. Lidocaine 3% (p = NS)

(Dorian et. al. NEJM)
Amiodarone vs. Lidocaine

- **Bottom Line:** Amiodarone currently has “the nod” but the study was small and had some flaws including provision of lipoprotein with deleterious effects to lidocaine group. Thus, giving lidocaine is acceptable alternative

- Currently, being reproduced with very large trial

- Remember, might drop CPP so epi should be on board
What happened to atropine?

- Absence of evidence...no trials
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ACLs: Waveform Capnography

• **Change:**
  • Quantitative waveform capnography is most reliable method to confirm and monitor correct ETT placement (Class I, LOE A)

• **Why:**
  • Unacceptably high incidence of unrecognized ET tube misplacement or displacement
  • Capnography has high sensitivity and specificity to identify correct endotracheal tube placement in cardiac arrest
ACLS: Waveform Capnography

- After intubation, exhaled carbon dioxide is detected, confirming tracheal tube placement
- Highest value at end-expiration
ACLs: Physiologic Monitoring During CPR

B. Capnography to monitor effectiveness of resuscitation efforts. This second capnography tracing displays the $P_{ETCO_2}$ in mm Hg on the vertical axis over time. This patient is intubated and receiving CPR. Note that the ventilation rate is approximately 8 to 10 breaths per minute. Chest compressions are given continuously at a rate of slightly faster than 100/min but are not visible with this tracing. The initial $P_{ETCO_2}$ is less than 12.5 mm Hg during the first minute, indicating very low blood flow. The $P_{ETCO_2}$ increases to between 12.5 and 25 mm Hg during the second and third minutes, consistent with the increase in blood flow with ongoing resuscitation. Return of spontaneous circulation (ROSC) occurs during the fourth minute. ROSC is recognized by the abrupt increase in the $P_{ETCO_2}$ (visible just after the fourth vertical line) to over 40 mm Hg, which is consistent with a substantial improvement in blood flow.
CPR Quality
• Push hard (≥2 inches [5 cm]) and fast (≥100/min) and allow complete chest recoil
• Minimize interruptions in compressions
• Avoid excessive ventilation
• Rotate compressor every 2 minutes
• If no advanced airway, 30:2 compression-ventilation ratio
• Quantitative waveform capnography
  – If PETCO₂ <10 mm Hg, attempt to improve CPR quality
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• Monophasic: 360 J

Drug Therapy
• IV/IO access
• Epinephrine: every 3-5 minutes
• Amiodarone for refractory VF/VT

Consider Advanced Airway
Quantitative waveform capnography

Treat Reversible Causes

Reversible Causes
• Hypovolemia
• Hypoxia
• Hydrogen ion (acidosis)
• Hypo-/hyperkalemia
• Hypothermia
• Tension pneumothorax
• Tamponade, cardiac
• Toxins
• Thrombosis, pulmonary
• Thrombosis, coronary

Shout for Help/Activate Emergency Response

Start CPR
• Give oxygen
• Attach monitor/defibrillator

2 minutes

Check Rhythm

If VF/VT

Shock Arrest Care

Return of Spontaneous Circulation (ROSC)

Post–Cardiac Arrest Care

Continous CPR

Monitor CPR Quality
Post–cardiac arrest care algorithm.

**Adult Immediate Post–Cardiac Arrest Care**

1. **Return of Spontaneous Circulation (ROSC)**
2. **Optimize ventilation and oxygenation**
   - Maintain oxygen saturation ≥94%
   - Consider advanced airway and waveform capnography
   - Do not hyperventilate
3. **Treat hypotension (SBP <90 mm Hg)**
   - IV/IO bolus
   - Vasopressor infusion
   - Consider treatable causes
   - 12-Lead ECG
4. **Follow commands?**
   - **No**
     - Consider induced hypothermia
   - **Yes**
     - STEMI or high suspicion of AMI
5. **Coronary reperfusion**
   - **Yes**
     - STEMI or high suspicion of AMI
   - **No**
     - Advanced critical care

**Doses/Details**

**Ventilation/Oxygenation**
- Avoid excessive ventilation. Start at 10-12 breaths/min and titrate to target \(\text{PETCO}_2\) of 35-40 mm Hg.
- When feasible, titrate \(\text{FIO}_2\) to minimum necessary to achieve \(\text{SpO}_2\) ≥94%.

**IV Bolus**
- 1-2 L normal saline or lactated Ringer's.
- If inducing hypothermia, may use 4°C fluid.

**Epinephrine IV Infusion**
- 0.1-0.5 mcg/kg per minute (in 70-kg adult: 7-35 mcg per minute)

**Dopamine IV Infusion**
- 5-10 mcg/kg per minute

**Norepinephrine IV Infusion**
- 0.1-0.5 mcg/kg per minute (in 70-kg adult: 7-35 mcg per minute)

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

Peberdy M A et al. Circulation 2010;122:S768-S786
The Cardiac Arrest Center Approach

**Treatment Recommendation:** A comprehensive, structured multidisciplinary system of care should be implemented in a consistent manner for the treatment of post-cardiac arrest patients (Class I, LOE B)"

Post Resuscitation Inflammatory Response

- IL-6
- TNF-\( \gamma \)
- Endotoxin
- Adhesion molecules
Post Resuscitation Organ Dysfunction

- Cerebral Edema
- Myocardial Dysfunction
- Acute Lung Injury
- Adrenal Dysfunction
- Shock Liver
- Renal Failure
- Bowel Ischemia
- Coagulopathy
Respiratory Dysfunction

• **Why**: Aspiration, Acute Lung Injury/ARDS

• Is there a different approach to management of mechanical ventilation than typical for pneumonia and ARDS?

  **Treatment Recommendation 1**: “in general it is appropriate to wean Fio$_2$ when saturation is 100%, provided the oxyhemoglobin saturation can be maintained $\geq$ 94% (Class I, LOE C)”
Respiratory Dysfunction

- **Why**: Aspiration, Acute Lung Injury/ARDS

- Is there a different approach to management of mechanical ventilation than typical for pneumonia and ARDS?

  **Treatment Recommendation 2**: “Ventilation rate and volume may be titrated to maintain high-normal PaCO2 (40 to 45 mmHg) (Class IIb, LOE C)”
33 degrees vs 36 degrees

Nielsen et al. Targeted Temperature Management at 33 v 36 degrees. NEJM (2013)
Survival Curve Differences

Figure 2. Cumulative Survival in the Normothermia and Hypothermia Groups. Censored data are indicated by tick marks.
Temperature Differences
Temperature Differences

[Graph showing temperature differences between 36°C and 33°C groups over time since randomization.]
Case 1

• 70 year old male s/p CABG in the PACU

• A nurse obtaining vital signs witnesses the patient become unresponsive and ashen and calls for help…
Case 1
What is the Rhythm?

1. Asystole
2. Pulseless Electrical Activity
3. Ventricular Tachycardia
4. Ventricular Fibrillation
Case 2

- 60 year old male who is post-op from a bowel resection ends up boarding in the PACU overnight because of a bed crunch...

- Patient remains intubated because of hypotension requiring fluids/pressors

- At 3 AM, while preparing for urgent surgery on another patient, the nurse calls for help and...
Case 3

- 78 year old female history of COPD is on the hospital ward recovering from pneumonia

- She is found by nursing staff to be unresponsive and breathing at a rate of less than 8 breaths per minute

- She is obtunded and hypoxic but has a good pulse; you are called to intubate the patient…
Acute Respiratory Failure $\rightarrow$ Cardiac Arrest

- Worst outcome as is oftentimes almost equal to death

- Incidence:
  - Anesthesia Operating Rooms: 1 in 10,000
  - Emergency Department: 4-5% (literature), may be more like 1-2% at BIDMC
  - Hospital Wards: 5-15%
  - ICU: unknown

- Possibly preventable

- Risk factors -
Acute Respiratory Failure → Cardiac Arrest
Hemodynamics not always considered and intervention was rarely given

• Only 10% of patients with ARF received a fluid bolus

• None of pre-intubation hypotensive patients received a fluid bolus
Pre-event hypotension and outcome

• AHA GWTG analysis from 2009-2011
  – After multivariable adjustment, we found that cases with pre-event hypotension/hypoperfusion were more likely to progress to cardiac arrest (OR 1.8 [95% CI: 1.4-2.3])

• BIDMC ICU data (2010-2012)
  – Case-control R-C cases and R’s
  – Prelim results: hypotension/decreased urine output

• International data from our group: Korean EM data registry indicating that pre-intubation hypotension is independently associated with increased risk of cardiac arrest (OR 3.7 [95% CI: 1.6-8.7], p = 0.01)
Post-intubation hypotension a common complication associated with poor outcome

- For patients overall, statistically significant drop in blood pressure pre- to post-intubation

p = 0.03
Time from RSI to Cardiac Arrest

Fig. 1. Histogram demonstrating timing of peri-intubation cardiac arrest for study subjects.

(Heffner et al Resuscitation 2013)
Table 1 Intubation care bundle management

Pre-intubation
1. Presence of two operators
2. Fluid loading (isotonic saline 500 ml or starch 250 ml) in absence of cardiogenic pulmonary edema
3. Preparation of long-term sedation
4. Preoxygenation for 3 min with NIPPV in case of acute respiratory failure (FiO₂ 100%, pressure support ventilation level between 5 and 15 cmH₂O to obtain an expiratory tidal volume between 6 and 8 ml/kg and PEEP of 5 cmH₂O)

During intubation
5. Rapid sequence induction: etomidate 0.2–0.3 mg/kg or ketamine 1.5–3 mg/kg combined with succinycholine 1–1.5 mg/kg in absence of allergy, hyperkalemia, severe acidosis, acute or chronic neuromuscular disease, burn patient for more than 48 h and medullar trauma
6. Sellick maneuver

Post-intubation
7. Immediate confirmation of tube placement by capnography
8. Norepinephrine if diastolic blood pressure remains <35 mmHg
9. Initiate long-term sedation
10. Initial “protective ventilation”: tidal volume 6–8 ml/kg of ideal body weight, PEEP <5 cmH₂O and respiratory rate between 10 and 20 cycles/min, FiO₂ 100% for a plateau pressure <30 cmH₂O

*NIPPV non-invasive positive pressure ventilation, PEEP positive end expiratory pressure, FiO₂ inspired oxygen fraction

(Jaber et al Intensive Care Medicine 2010)
Pre- Post- Intervention

(Jaber et al Intensive Care Medicine 2010)
Main Interventions

1) Optimal pre-oxygenation (and continued oxygenation) strategy

2) Optimize hemodynamics and monitoring

3) Approach to mechanisms of intubation/sedation strategy

4) Post-intubation hemodynamic optimization, monitoring, vent management
ACUTE RESPIRATORY FAILURE CHECKLIST

PREPARE PATIENT
- Apply monitoring devices (Defibrillator, spO2)
- Apply EtCO2 monitoring (With RT)
- Pre-oxygenate the patient with 100% oxygen per non-rebreather, bag-valve mask ventilation with high-flow oxygen, or NIV
- If unresponsive, establish oral airway
- Remove headboard from bed
- Check and document vital signs (BP, pulse, RR, spO2)
- Ensure IV access (Prepare for potential fluid bolus)
- Initiate fluid bolus and vasopressors if needed to optimize hemodynamics
  - Phenylephrine in pharmacy bag
  - Levo and Dopa in code cart
- Confirm K+

PREPARE EQUIPMENT
- Have bag-valve mask hooked up to oxygen and turn on to 15 L/minute or greater
- Prepare suction
- Place airway box and bougie at head of bed
- For known difficult airway, notify RT to access difficult airway cart
- Consider avoidance of propofol
  - Etomidate and Ketamine located in pharmacy bag
- Prepare post-intubation sedation plan
- Initiate transfer plan to ICU

PREPARE TEAM
- Identify:
  - Team Leader
  - Anesthesia/ED Attending
  - Respiratory Tech
  - CC Nurse
  - Pharmacy
- If needed request Emergent Surgical Airway: Call Operator Services at 2-1212 and identify that you are requesting an Emergent Surgical Airway. Be prepared to provide the following information:
  - Location (floor, room)
  - East or West Campus
  - Your Name
  - Your call back Number

POST-INTUBATION CARE
- Verify tube placement with BOTH auscultation and continuous EtCO2; document
- Document lip line
- Re-check and document vital signs (BP, pulse, RR, spO2)
- Optimize RR considering autpeep vs acidosis
- Continue fluid therapy and/or vasopressors as needed
- Continue to monitor and document vital signs
- Ensure expedient transfer to ICU

IN EVENT OF DIFFICULT INTUBATION CRICOTHYROIDOTOMY KIT IS LOCATED IN BOTTOM OF CODE CART
Case 4

- 66 year old female admitted with a NSTEMI is on the floor being monitored when an alarm is set off and while you are at the nursing station, you see the following…
Case 4
What is the most important intervention?

1. High quality CPR
2. Early epinephrine
3. Defibrillation
4. Amiodarone