**Objectives:** briefly review cardiac arrest and approach to ACLS, discuss ROSC and its goals, explain guidelines for hemodynamic support, discuss indications and contraindications for TTM and TH.

Cardiac arrest is responsible for nearly 0.5 million deaths per year annually. This does not include the thousands of patients who do not survive to hospital discharge. Guidelines have been shifting for several decades now. Advanced Cardiac Life Support (ACLS) is a set of clinical management guidelines that outcome the intervention of various life-threatening conditions. It is the standard of urgent healthcare delivery in North America. Despite its portrayal in Hollywood and the public perception of CPR, most studies do not favor the majority of patients reaching hospital discharge, especially with good neurologic outcome. As research continues, clinicians must focus on what has been shown to provide the best possible chance at neurologic recovery.

The most critical actions to perform during ACLS:

1. **Excellent CPR**
2. **Defibrillation as soon as possible** (if indicated for shockable rhythms)

When cardiac arrest happens

Patient enters cardiac arrest → unresponsive. They will lack a pulse and will not be breathing. **CPR needs to be started immediately.** Studies have repeatedly shown that the sooner CPR is begun, the chance of survival. If you are the one to feel no pulse, YOU start CPR. Do not call for others to come start CPR. YOU DO. It is the single most important intervention that can be offered. As each minute passes, the chance of surviving arrest drops by 10%. The above information is so incredibly high yield for boards and life it cannot be overstated.

If you are in a hospital, alert a healthcare provider and begin CPR. If you are outside a hospital, tell someone nearby to call 911 as you begin CPR. If you are alone, perform CPR for 2 minutes and then get help nearby and secure an AED. The AED should be turned on and pads placed. While charging the defibrillator, continue CPR. The time from pulse check to hitting “shock” on the defibrillator should not take more than 5 seconds.

Let’s proceed with the basic overall approach of “running a code”:

No pulse/no breathing → Begin CPR. **High Quality CPR means:** switching performers a MAX of q2 min or sooner, NO interruptions, >2 inches of depth, >100 compressions per min, allow complete chest recoil

**Pulseless Vent. Tachycardia & Vent. Fibrillation**

Begin CPR → Shock ASAP → CPR 2 min → Shock → CPR 2 min → Shock → CPR 2 min → Shock → CPR 2 min → Shock → CPR 2 min

-**Key points:** defibrillation is ONLY used in pulseless VT and VF. First dose of Epinephrine should be given during the 2nd round of CPR and given from then on every 3-5 minutes. After the third round of epinephrine, give a dose of 300 mg Amiodarone instead, followed by a 2nd dose of 150mg 2 rounds later. In summary, you should be alternating amiodarone and epinephrine every 3–5 minutes along with defibrillation when indicated.

The success rate of defibrillation is directly dependent on duration of the VF/VT and speed to initiate defibrillation. Pulse checks should NOT occur after shock delivery. Immediately resume CPR.

*Lidocaine has been found to be no different in outcomes than Amiodarone and can be given in place of it.*

**PEA/Asystole**

(Pulseless electric activity)

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-**Key points:** NO defibrillation should be performed in PEA/asystole. Think about it. Cardiac electrical activity is functioning, therefore more electricity is not going to help anything. The patient is suffering from some other pathology that is impairing cardiac muscle contractility. This is where we remember those dreaded H’s and T’s:

| Tension pneumothorax | H’s (acidosis/alkalosis) |
| Tamponade | Hypo/hyperkalemia |
| Toxicologic | Hypovolemia |
| Thromboembolic (PE) | Hypoxia |
| Thrombosis (MI) | Hypothermia |

To be clear, these “H’s and T’s” should be remembered for all causes of cardiac arrest, we just emphasize them during PEA/asystole due to inability to deliver defibrillation.
Talitha Cumi: ACLS and ROSC

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What’s the deal with ventilations? When should I do it?
- the ratio of 30:2 (CPR to ventilation) has long been preferred but it has been stated if no bag is available, straight CPR takes precedence until help arrives. If the patient has an advanced airway device, deliver ventilations at 6-8 per minute.
- in children however, it has been found 30:2 and 15:2 ratios are preferred since the #1 cause of “no pulse” in children is respiratory.
- End-tidal CO₂ should be utilized during CPR to reflect cardiac output and cerebral perfusion. Decreased EtCO₂ suggests poor compressions.

When do I intubate?
Despite popular belief, securing an advanced airway (supraglottic, LMA, endotracheal), is NOT a priority during CPR. In fact, it is argued (and studies support) NOT attempting any advanced airway as long as the patient can be given ventilations through bag-valvemask. Intubation, especially when using paralytics and sedatives, can drastically drop BP and create more problems.
If multiple providers are available and CPR is unhindered, a supraglottic airway or other airway adjuncts may be placed and may improve ventilatory effort.

Important note: since survival rates can be abysmal for resuscitation during cardiac arrest, solid evidence is difficult to come by for all these various therapies, the most controversial being medications. As of this writing, there is a growing body of evidence raising doubt over the effectiveness of epinephrine and its possibly deleterious effects during resuscitation. Some recent studies have suggested epinephrine does not improve survival to hospital discharge. Other studies now find that epinephrine can cause worse neurologic outcomes. Maybe the circulating catecholamines cause a negative effect on brain perfusion leading to worse neurologic outcomes. Maybe epinephrine needs to be at lower and less frequent dosing. Maybe we shouldn’t be using epinephrine at all? As of now, unless clinical gestalt dictates otherwise, you should follow published ACLS guidelines as defined above- THAT is what is being tested. Amiodarone has been found to provide little survival benefit as well and does not result in increased survival to hospital discharge.

When to terminate efforts: little data published to guide decision-making. The following factors strongly suggest halting efforts:
- duration of resuscitation >30 minutes, initial rhythm of asystole/PEA, unwitnessed arrest, prolonged time between arrest and initiation of ACLS, low EtCO₂ even after 20 minutes of high-quality resuscitation.
- bedside echo showing no cardiac wall motion. This finding alone should never terminate resuscitation, and it should never interfere with CPR.

Therapies that should not be done during a code: Atropine (no role in PEA/asystole), vasopressin, cardiac pacing for asystole or PEA.

Return of spontaneous circulation (ROSC):
Once pulses have been achieved, every possible attempt should be made at making sure you don’t lose them again. At the same time, there should be a vigorous investigation into what the cause of arrest was.

Critical objectives during ROSC:
- managing hemodynamics post-arrest
- minimizing brain injury
- securing a definitive, endotracheal airway if not done so already
- diagnose and treat suspected causes of arrest

Reassess the patient post-arrest. What did you miss while you were running the resuscitation? Rigid abdomen? Loud, harsh cardiac murmur? Decreased lung sounds unilaterally? Tachycardia is expected after ROSC, but bradycardia is ominous. Blood in the OG tube or rectum suggests GI bleeding, unilateral leg swelling suggests PE. Track marks on the skin suggest IVDA which in turn place the patient at risk for endocarditis or septic emboli.

Tests you should order: EKG, CXR, CT head. Bedside echocardiography should be strongly considered.
Other suggested studies: Blood gas, BMP, CBC, serial troponins, lactate

Any evidence of STEMI or new LBBB requires emergency reperfusion therapy. Taking it further, even in those without STEMI but had VF and pulseless VT and a concerning story of preceding chest pain/dyspnea/history of CAD also might benefit.
EKG immediately after ROSC may be unrevealing for STEMI or new LBBB. Cardiology should be consulted after ROSC in patients who suffered VF/VT arrest.

Benefits of bedside echocardiography:

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<tr>
<th>POCUS Finding</th>
<th>Possible extrapolations</th>
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<tr>
<td>Focal wall motion abnormalities</td>
<td>Myocardial infarction</td>
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<tr>
<td>Grossly dilated LV with global hypokinesis</td>
<td>Diminished EF suggesting grossly deficient LV function</td>
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<tr>
<td>RV strain, dilation (The D-Sign or McConnell’s Sign) with bowing of the septum into the LV</td>
<td>Pulmonary embolus</td>
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<tr>
<td>Apical ballooning during systole</td>
<td>Takotsubo cardiomyopathy</td>
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<tr>
<td>Interstitial wall edema (+/- wall hypokinesis and dilation)</td>
<td>Myocarditis</td>
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<td>Pericardial effusion</td>
<td>Tamponade</td>
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While this is occurring you should also conduct a neurologic evaluation. Patients who cannot perform any purposeful movements on basic command meet indication for therapeutic hypothermia or targeted temperature management (TTM).

### Hemodynamic support:

#### Cardiovascular

Maintain MAP goal >65 mmHg, preferably 80-100 to prevent cerebral vasoconstriction.

- IV fluids can assist in maintain a normal CVP ~8-12. Lactated Ringers should be the fluid of choice.

- There is no specific guideline or standardized research that clearly shows one vasopressor or inotropic agent that is superior to another in these patients.

  - Commonly used agents: Epinephrine, norepinephrine, dopamine.

  - There has been a gradual shift towards preferring norepinephrine in these patients, although the data is incomplete and as you can imagine changes often.

  - In terms of inotropic agents, dopamine or milrinone has been used. Both cause hypotension; dopamine predisposes patients to tachyarrhythmias.

#### Respiratory

- Endotracheal intubation with proof of placement

- Maintain normal ventilation (PaCO₂ 35-40), Normoxia >94% (not hyperoxia which can have worse outcomes).

- Monitor with end-tidal CO₂

### Neurologic

**Fever** = worse neurologic outcomes. It is the most common cause of death in patients with cardiac arrest outside the hospital.

32-36 C improves outcome. (89-93 F)

Targeted temperature management (TTM) refers to temperature control no higher than 36 Celsius. Therapeutic hypothermia (TH) refers to active range maintenance of 32-34 Celsius.

- Indications of TTM: any patient not following commands or having purposeful movements following cardiac arrest. Specifically, patients who have suffered from VF or pulseless VT (AKA “shockable rhythms”) seem to respond better and have greater chances of improved neurologic outcomes. Discussion of initiating TTM and TH needs to occur with the intensivist in the correct setting, as they will be the ones managing the patient’s care much longer than in the ED.

- Contraindications of TTM: no absolute contraindications. Relative contraindications include cardiogenic shock, uncontrolled bleeding.

- Duration: 24-48 hours

- Methods: infuse cold saline, >2 C per hour (1 L in 15 min can lower 1 C). Surface blankets work too. Do not do fluids in those with ESRD/CHF. Instead, prefer blankets.

- Side effects: shivering (sedate patient) poor coagulation, increased infection risk.

- Serial blood gases should be performed as ventilation requirements will decrease as the temperature decreases. The goal is still a PaCO₂ of 35-40.

### References


