2010 AHA guidelines
Part 6：Electrical Therapies
Part 9：Post-Cardiac Arrest Care

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Defibrillation Plus CPR: A Critical Combination

- For every minute that passes between collapse and defibrillation, survival rates from witnessed VF SCA decrease 7% to 10% if no CPR is provided.
- If bystanders provide immediate CPR, many adults in VF can survive with intact neurologic function, especially if defibrillation is performed within 5 to 10 minutes after SCA.

New Recommendations to Integrate CPR and AED Use

- ***Shock First Versus CPR First：***
  - When VF is present for more than a few minutes, the myocardium is depleted of oxygen and metabolic substrates.
  - A brief period of chest compressions can deliver oxygen and energy substrates, increasing the likelihood that a shock may terminate VF.

New Recommendations to Integrate CPR and AED Use

- When an out-of-hospital cardiac arrest is not witnessed by EMS personnel, EMS may initiate CPR while checking the ECG rhythm and preparing for defibrillation.
- CPR should be performed while a defibrillator is being readied.

New Recommendations to Integrate CPR and AED Use

- With in-hospital SCA, there is insufficient evidence to support or refute CPR before defibrillation.
- However, in monitored patients, the time from VF to defibrillation should be under 3 minutes.

New Recommendations to Integrate CPR and AED Use

- ***1-Shock Protocol Versus 3-Shock Sequence***
  - If 1 shock fails to eliminate VF, the incremental benefit of another shock is low, and resumption of CPR is likely to confer a greater value than another shock.
  - The consensus was that rescuers using monophasic defibrillators should give an initial shock of 360 J.

New Recommendations to Integrate CPR and AED Use

- The rhythm analysis for a 3-shock sequence performed by commercially available AEDs can result in delays of up to 37 seconds between delivery of the first shock and delivery of the first postshock compression.

  → Shortening the interval between the last compression and the shock by even a few seconds can improve shock success.

Defibrillation Waveforms and Energy Levels

- *defibrillation* (shock success) ： typically defined as termination of VF for at least 5 seconds following the shock.
- lower-energy biphasic waveform shocks have equivalent or higher success for termination of VF than monophasic waveform shocks.

Defibrillation Waveforms and Energy Levels

- for biphasic defibrillators, providers should use the manufacturer's recommended energy dose (120 to 200 J).
- If the manufacturer's recommended dose is not known, defibrillation at the maximal dose may be considered.

Electrodes

- **Defibrillation With Implanted Cardioverter Defibrillator (ICD) ：**
  - Positioning the pads at least 8 cm away did not produce changes in pacing thresholds or sensing measurements.

※Do not place AED electrode pads directly on top of a transdermal medication patch → it may cause small burns of the skin.

Automated External Defibrillators (AED)

- **AED Use in Children ：**
  - 1 to 8 years of age：use a pediatric dose-attenuator if not available → use standard AED
  - 1 year of age： prefer manual defibrillator

    If not available → AED with pediatric attenuation

    If neither is available → standard AED

Synchronized Cardioversion

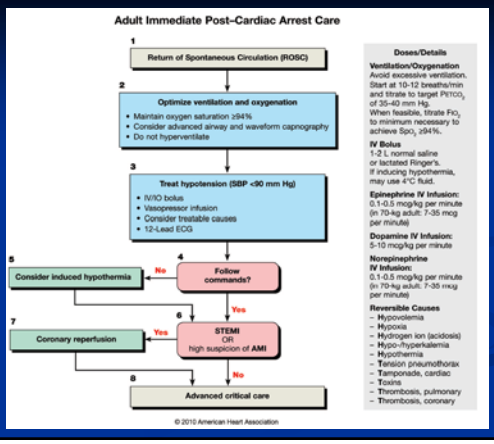
- **Supraventricular Tachycardias (Reentry Rhythms)**
  - atrial fibrillation：120 to 200 J (biphasic)
  - atrial flutter and other supraventricular tachycardias ：50 J to 100 J
- **Ventricular Tachycardia**
  - monomorphic VT with a pulse：100 J
  - monomorphic or polymorphic VT in the *unstable* patient：defibrillation doses

Summary

- rescuers must coordinate high-quality CPR with defibrillation to minimize interruptions in chest compressions and to ensure immediate resumption of chest compressions after shock delivery.
- The high first-shock efficacy of newer biphasic defibrillators led to the recommendation of single shocks plus immediate CPR instead of 3-shock sequences.

Post-Cardiac Arrest Care

- Subsequent objectives of post– cardiac arrest care are to
  - Control body temperature to optimize survival and neurological recovery
  - Identify and treat acute coronary syndromes (ACS)
  - Optimize mechanical ventilation to minimize lung injury
  - Reduce the risk of multiorgan injury and support organ function if required
  - Objectively assess prognosis for recovery
  - Assist survivors with rehabilitation services when required



Overview of Post–Cardiac Arrest Care

- *Airway Management*：
  - They should also elevate the head of the bed 30° if tolerated to reduce the incidence of cerebral edema, aspiration, and ventilatory-associated pneumonia.
  - providers should titrate inspired oxygen to the lowest level required to achieve an arterial oxygen saturation of 94%, so as to avoid potential oxygen toxicity.

- Pulse Oximetry/ABG
  - Rationale: Maintain adequate oxygenation and minimize Fio_2
  - Spo_2 ≥94%
  - Pao_2~100 mm Hg
  - Reduce Fio_2 as tolerated
  - Pao_2/Fio_2 ratio to follow acute lung injury

Overview of Post–Cardiac Arrest Care

- *Ventilation*：
  - Hyperventilation increases intrathoracic pressure and inversely lowers cardiac output.
  - The decrease in PaCO2 seen with hyperventilation can also potentially decrease cerebral blood flow directly.

- Mechanical Ventilation
  - Rationale: Minimize acute lung injury, potential oxygen toxicity
  - Tidal Volume 6–8 mL/kg
  - Titrate minute ventilation to $Petco_2$~35–40 mm Hg $Paco_2$~40–45 mm Hg
  - Reduce Fio_2 as tolerated to keep Spo_2 or Sao_2 ≥94%

Overview of Post–Cardiac Arrest Care

- *Hemodynamics*：
  - If the patient is hypotensive, fluid boluses can be considered.
  - Titrated to achieve a minimum systolic blood pressure of 90 mm Hg or a mean arterial pressure of 65 mm Hg.

- Treat Hypotension
  - Rationale: Maintain perfusion
  - Fluid bolus if tolerated
  - Dopamine 5–10 mcg/kg per min
  - Norepinephrine 0.1–0.5 mcg/kg per min
  - Epinephrine 0.1–0.5 mcg/kg per min

Overview of Post–Cardiac Arrest Care

- *Cardiovascular*：

- Continuous Cardiac Monitoring
  - Rationale: Detect recurrent arrhythmia
  - No prophylactic antiarrhythmics
  - Treat arrhythmias as required
  - Remove reversible causes
- 12-lead ECG/Troponin
  - Rationale: Detect Acute Coronary Syndrome/ST-Elevation Myocardial Infarction; Assess QT interval

- Treat Acute Coronary Syndrome
  - Aspirin/heparin
  - Transfer to acute coronary treatment center
  - Consider emergent PCI or fibrinolysis

- Treat Myocardial Stunning
  - Fluids to optimize volume status (requires clinical judgment)
  - Dobutamine 5–10 mcg/kg per min
  - Mechanical augmentation (IABP)

Overview of Post–Cardiac Arrest Care

- *Neurological*：
  - Therapeutic hypothermia is the only intervention demonstrated to improve neurological recovery.
  - It should be considered for **any** patient who is **unable to follow verbal commands** after ROSC.
  - Concurrent PCI and hypothermia are safe, with good outcomes reported for some comatose patients who undergo PCI.

- Core Temperature Measurement If Comatose
  - Rationale: Minimize brain injury and improve outcome
  - Prevent hyperpyrexia >37.7°C
  - Induce therapeutic hypothermia if no contraindications
  - Cold IV fluid bolus 30 mL/kg if no contraindication
  - Surface or endovascular cooling for 32°C–34°C×24 hours
  - After 24 hours, slow rewarming 0.25°C/hr

Overview of Post–Cardiac Arrest Care

- *Metabolic*：

- Serum Potassium
  - Rationale: Avoid hypokalemia which promotes arrhythmias
  - Replace to maintain K >3.5 mEq/L

- Urine Output, Serum Creatinine
  - Rationale: Detect acute kidney injury
  - Maintain euvolemia
  - Renal replacement therapy if indicated

- Serum Glucose
  - Rationale: Detect hyperglycemia and hypoglycemia
  - Treat hypoglycemia (<80 mg/dL) with dextrose
  - Treat hyperglycemia to target glucose 144–180 mg/dL
  - Local insulin protocols

- Avoid Hypotonic Fluids
  - Rationale: May increase edema, including cerebral edema

Targeted Temperature Management

- **Induced Hypothermia**
  - For patients with out-of-hospital ventricular fibrillation (VF) cardiac arrest. (Class I , LOEB)
  - cooled to 32˚C to 34˚C for 12 or 24 hours beginning minutes to hours after ROSC.
  - No randomized controlled trials have compared outcome between hypothermia and normothermia for non-VF arrest. (for PEA or asystole→ Class IIb, LOEB)

Targeted Temperature Management

- Timing of initiating hypothermia：
  - Current studies showed time to target temperature was not an independent predictor of neurological outcome.
- Duration of induced hypothermia：
  - At least 12 hours and may be 24 hours.
  - The effect of a longer duration of cooling on outcome has not been studied in adults.

Targeted Temperature Management

- Method of inducing hypothermia：
  - Feedback-controlled endovascular catheters
  - surface cooling devices
  - Cooling blankets and frequent application of ice bags

Targeted Temperature Management

- monitor core temperature：
  - using an esophageal thermometer, bladder catheter in nonanuric patients, or pulmonary artery catheter if other indicated.
  - Axillary and oral temperatures are **inadequate** for measurement of core temperature changes.
  - Bladder temperatures in anuric patients and rectal temperatures may differ from brain or core temperature

Targeted Temperature Management

- Possible Complication of induced hypothermia：
  - Coagulopathy
  - Arrhythmias
  - Hyperglycemia
  - Sepsis and pneumonia

Organ-Specific Evaluation and Support

- **Pulmonary System**
  - The beneficial effect of **high FIO2** on systemic oxygen delivery should be balanced with the deleterious effect of generating oxygen-derived free radicals during the reperfusion phase.
  - adjust the FIO2 to the minimum concentration needed to achieve arterial oxyhemoglobin saturation 94%, with the goal of avoiding hyperoxia wh-ile ensuring adequate oxygen delivery. **(Class I, LOEC)**

Organ-Specific Evaluation and Support

- **Cardiovascular System**
  - A 12-lead ECG should be obtained as soon as possible after ROSC to determine whether acute ST elevation is present **(Class I, LOE B).**
  - Therapeutic hypothermia can be safely combined with primary PCI after cardiac arrest caused by AMI.

Vasoactive Drugs for Use in Post–Cardiac Arrest

- **Vasopressors**
- Specific drug infusion rates cannot be recommended because of variations in pharmacokinetics and pharmacodynamics in critically ill patients.

Table 2. Common Vasoactive Drugs

Drug	Typical Starting Dose (Then Titrate to Effect)
Epinephrine	0.1–0.5 mcg/kg/min (In 70-kg adult, 7–35 mcg/min) • Useful for symptomatic bradycardia if atropine and transcutaneous pacing fail or if pacing is not available • Used to treat severe hypotension (eg, systolic blood pressure <70 mm Hg) • Useful for anaphylaxis associated with hemodynamic instability or respiratory distress[158]
Norepinephrine	0.1–0.5 mcg/kg/min (In 70-kg adult, 7–35 mcg/min) • Used to treat severe hypotension (eg, systolic blood pressure <70 mm Hg) and a low total peripheral resistance • Relatively contraindicated in patients with hypovolemia. It may increase myocardial oxygen requirements, mandating cautious use in patients with ischemic heart disease • Usually induces renal and mesenteric vasoconstriction; in sepsis, however, norepinephrine improves renal blood flow and urine output[159,160]

Vasoactive Drugs for Use in Post–Cardiac Arrest

- adrenergic drugs should **not** be mixed with sodium bicarbonate or other alkaline solutions in the IV line because there is evidence that adrenergic agents are inactivated in alkaline solutions.

Phenylephrine	0.5–2.0 mcg/kg/min (In 70-kg adult, 35–140 mcg/min) • Used to treat severe hypotension (eg, systolic blood pressure <70 mm Hg) and a low total peripheral resistance
Dopamine	5–10 mcg/kg/min • Used to treat hypotension, especially if it is associated with symptomatic bradycardia • Although low-dose dopamine infusion has frequently been recommended to maintain renal blood flow or improve renal function, more recent data have failed to show a beneficial effect from such therapy[161,162]
Dobutamine	5–10 mcg/kg/min • The (+) isomer is a potent beta-adrenergic agonist, whereas the (–) isomer is a potent alpha-1-agonist[163] • The vasodilating beta$_2$-adrenergic effects of the (+) isomer counterbalance the vasoconstricting alpha-adrenergic effects, often leading to little change or a reduction in systemic vascular resistance
Milrinone	Load 50 mcg/kg over 10 minutes then infuse at 0.375 mcg/kg/min • Used to treat low cardiac output • May cause less tachycardia than dobutamine

Modifying Outcomes From Critical Illness

- **Glucose Control**
  - Strategies to target moderate glycemic control (144 to 180 mg/dL) may be considered in adult patients with ROSC after cardiac arrest (Class IIb, LOE B).
  - Attemptsto control glucose concentration within a lower range (80 to110 mg/dL) should not be implemented after cardiac arrest due to the increased risk of hypoglycemia (Class III, LOE B).

Modifying Outcomes From Critical Illness

- Steroids
  - The post– cardiac arrest syndrome has similarities to septic shock, but the efficacy of corticosteroids remains controversial in patients with sepsis as well.
  - The value of the routine use of corticosteroids for patients with ROSC following cardiac arrest is uncertain.

Central Nervous System

- **Seizure Management**
  - An **EEG** should be performed with prompt interpretation as soon as possible and should be monitored frequently or continuously in comatose patients after ROSC (Class I , LOE C).
  - The anticonvulsant regimens for the treatment of seizures used for status epilepticus caused by other etiologies may be considered after cardiac arrest. (Class IIb, LOE C).

Prognostication of Neurological Outcome in Comatose Cardiac Arrest Survivors

- **Poor outcome** is defined as death, persistent unresponsiveness, or the inability to undertake independent activities after 6 months.
- Among adult patients who have *not* been treated with hypothermia, the absence of both pupillary light and corneal reflexes at 72 hours after cardiac arrest predicted poor outcome with high reliability.

Changes in Prognostication With Hypothermia

- Physical examination (motor response, pupillary light and corneal reflexes), EEG, SSEP, and imaging studies are less reliable for predicting poor outcome in patients **treated with hypothermia**.
- Durations of observation **greater than 72 hours** after ROSC should be considered before predicting poor outcome in patients treated with hypothermia (Class I, Level C).

Summary

- The post– cardiac arrest period is often marked by hemodynamic instability as well as metabolic abnormalities.
- Support and treatment of acute myocardial dysfunction and acute myocardial ischemia can increase the probability of survival.
- Interventions to reduce secondary brain injury, such as therapeutic hypothermia, can improve survival and neurological recovery.

Thanks for your attention !