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Post-Arrest Interventions That Save Lives

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Synopsis

Patients resuscitated from cardiac arrest require complex management. An organized approach to early post-arrest care can improve patient outcomes. Priorities include completing a focused diagnostic workup to identify and reverse the inciting cause of arrest, stabilizing cardiorespiratory instability to prevent rearrest, minimizing secondary brain injury, evaluating the risk and benefits of transfer specialty care center and avoiding early neurological prognostication.

Keywords

percutaneous coronary intervention; temperature targeted management; seizure; mechanical circulatory support

Introduction

Care of patients resuscitated from cardiac arrest is challenging. Mortality and morbidity is common even after return of spontaneous circulation (ROSC). In managing patients these patients, clinicians must simultaneously address multiple problems. Goals in the early post-arrest period include cardiopulmonary stabilization and prevention of secondary brain injury. Given the complexity and importance of the minutes immediately after ROSC, an organized approach is crucial and is associated with demonstrable outcome benefits.¹

Identifying causes of arrest that require immediate intervention

Autopsy series demonstrate cardiac etiologies, particularly coronary artery disease, are the most common cause of out-of-hospital cardiac arrest (OHCA).² However, only a minority of OHCA victims are successfully resuscitated and survive to hospital care. The demographics of the subgroup seen and evaluated in the emergency department (ED) are distinct from the broader group of OHCA victims.³ A recent large cohort study classified the arrest etiology

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after a full inpatient diagnostic workup, finding only a minority of cases were due to acute coronary syndrome (ACS) or other cardiac causes.⁴ A rapid, directed work-up is needed to evaluate for treatable arrest etiologies (Table 1). A brief history should be obtained from EMS or family regarding the circumstances of the arrest. In some cases, history may suggest the underlying cause (e.g. antecedent chest pain followed by ventricular fibrillation may suggest ACS, while presence of drug paraphernalia at the scene may suggest overdose). A focused physical exam can evaluate for signs of trauma, gastrointestinal hemorrhage, primary neurological catastrophes such as intracerebral hemorrhage, etc.

Because cardiovascular etiologies are common, an electrocardiogram (ECG) should be obtained on all post-arrest patients. Although the data on emergent cardiac catheterization post-arrest are mixed (see below), patients with acute coronary syndromes and survivable neurological injury likely benefit from early revascularization.⁵ Laboratory testing including a comprehensive metabolic panel, troponin, lactate, arterial blood gas, glucose, complete blood count, and coagulation studies should be obtained. Severe metabolic disarray, such as hyperkalemia, diabetic emergencies, and hypoglycemia, can result in arrest. Point-of-care ultrasonography may inform both initial resuscitation efforts and narrow the differential diagnosis.^{6,7}

Chest x-ray can help with endotracheal tube placement and rule-out a pneumothorax. In patients with coma or abnormal neurological findings, early post-arrest computerized tomographic (CT) imaging of the brain obtained in the ED is abnormal in 5–10% of patients.⁸ Abnormalities include early cerebral edema, an ominous prognostic sign, and acute cerebrovascular pathology responsible for the initial arrest. Chest compressions themselves commonly result in rib or sternal fractures and associated pneumothorax or solid organ injury.⁹ For this reason, many centers obtain comprehensive cross-sectional imaging to screen for both etiologies and consequences of OHCA. Chest imaging is also sensitive and specific for diagnosis of pulmonary embolism (PE)¹⁰. Arrest from PE defines the embolism as “high-risk,” and these patients should be considered for thrombolytic therapy.¹¹

Preventing early rearrest

One in 5 cases of OHCA that regains pulses will rearrest, and rearrest worsens survival.^{12–14} The timing is biphasic, occurring both minutes after ROSC and then hours later. Early rearrest can occur when patients shocked out of ventricular fibrillation (VF) re-fibrillate, particularly in the setting of ongoing myocardial ischemia. These patients may benefit from antidysrhythmic agents such as amiodarone or lidocaine. Refractory ventricular arrhythmias can be managed with mechanical circulatory support and coronary revascularization.¹⁵ Recurrence of pulseless electrical activity (PEA) is also common as bolus dose vasopressors given during CPR are cleared. Administration of vasoactive agents by continuous infusion is commonly needed in the minutes after initial resuscitation. Patients with cardiogenic shock may require inotropic support.^{16–18} The median time to delayed rearrest is about 5 hours.¹⁴ Delayed rearrest often results from persistent myocardial dysfunction,¹⁹ systemic inflammation with associated vasoplegia and multisystem organ failure.^{19,20}

Percutaneous coronary intervention (PCI)

Patients with ST-elevations on ECG post-arrest require immediate coronary angiography.^{5,21,22,23} High doses of vasopressors, particularly bolus administration during CPR, can cause transient coronary vasospasm and result in a variety of ECG abnormalities including ST elevation. It is reasonable to obtain a repeat ECG a few minutes after ROSC as many abnormalities rapidly resolve, but cardiac catheterization should typically not be delayed to obtain serial ECGs over time. Centers capable of performing early neurological risk stratification may consider deferring PCI in patients with early objective evidence of severe brain injury, as these patients are unlikely to benefit from revascularization.²⁴ However, given the limited specificity of many early neuroprognostic signs,²⁵ these decisions should be made with the utmost of caution and only by clinicians with special expertise. Public reporting of post-procedural mortality may create a perverse disincentive to offer PCI to critically ill patients including those resuscitated from OHCA.²⁶ Medical decision-making should optimize care for the individual patient rather than reflecting fear of publicly reported outcomes.

Timing of PCI for OHCA patients without ST-elevations is controversial. Post-arrest ECG is neither sensitive nor specific for diagnosis of acute coronary syndrome (ACS).^{27–29} For patients with ACS, revascularization can improve myocardial function and prevent recurrent arrhythmias. Nevertheless, a recent randomized controlled trial failed to find benefit from emergent versus delayed PCI in OHCA patients without ST-elevations.³⁰ The overall incidence of culprit lesions was only 15% in this study, reflecting the low incidence of cardiac etiologies of OHCA patients who survive to the hospital. Other limitations to the study include delayed initiation of targeted temperature management (TTM) and hemodynamic resuscitation, and lack of baseline risk stratification of neurologic illness.

Overall, if pretest probability for ACS is high based on available data and there are neither contraindications to anticoagulation nor evidence of devastating primary brain injury, early PCI should be considered in discussion with interventional cardiology.

Mechanical Circulatory Support

Use of mechanically circulatory support (MCS) during and after cardiac arrest is steadily increasing.^{31,32} MCS can preserve brain and coronary perfusion, buying time for definitive treatment in the case of refractory cardiac arrest. After initial ROSC, MCS can augment or replace inadequate cardiac output and may offer myocardial protection in some settings.³³

Initiating MCS during CPR (termed extracorporeal CPR (E-CPR) requires venoarterial (VA)-extracorporeal membrane oxygenation (ECMO). In the case of refractory ventricular arrhythmias from ACS, VA-ECMO can maintain cerebral perfusion and act as a bridge to coronary revascularization.³⁴ In the case of massive pulmonary embolism or other causes of acute right ventricular failure, VA-ECMO bypasses the failing right ventricle and pulmonary circulation to maintain systemic perfusion. Use of ECPR is growing worldwide.³⁵ Institutional protocols, inclusion criteria and outcomes of E-CPR vary widely across centers and regions.^{34,36–40} E-CPR is an expensive and complex intervention with little high-level

evidence supporting its use. Nevertheless, it has reasonable face validity and future randomized trials may demonstrate benefit.⁴¹

ECMO may also be useful in the early post-arrest period. VA-ECMO provides biventricular and pulmonary support, so may have a role in both left and right ventricular failure or refractory hypoxemia. Compared to other MCS devices, VA-ECMO is also the most invasive and has the highest associated procedural risks.³⁵ Intra-aortic balloon pumps (IABP) and Impella™ catheters can also be used in the post-arrest period to support patients with significant cardiogenic shock. Despite physiological rationale for IABP, among patients with cardiogenic shock from acute myocardial infarction, IABP does not improve outcomes compared to conventional care.⁴² Additionally, it unclear if Impella™ confers significant outcome benefit compared to IABP or conventional care for patients with cardiogenic shock.^{43,44}

Preventing Secondary Brain Injury

Anoxic brain injury is most common proximate cause of death and disability for patients admitted after OHCA.^{45–48} The brain is highly susceptible to ischemia after cardiac arrest. Hypoxic ischemic injury can lead to impaired cerebral autoregulation, cerebral edema and delayed neurodegeneration.¹⁹ Secondary brain injury can occur in the hours to days following ROSC and worsens outcomes.

Optimal Perfusion Pressure—Adequate cerebral perfusion is necessary after cardiac arrest to prevent further neuronal damage in the already-injured brain. Hours to days after ROSC, cerebral hypoperfusion can develop.⁴⁹ During this time, cerebral autoregulation is often impaired, and a smaller decrease in mean arterial pressure (MAP) can cause drastic changes to cerebral blood flow, with critical opening pressures for the cerebral vasculature exceeding 110mmHg.⁵⁰ Thus, even in the absence of systemic hypotension cerebral perfusion may be inadequate.^{51,52} The ideal MAP after cardiac arrest is unknown and likely needs to be tailored to patient specific parameters. Multiple observation studies have demonstrated improved neurologic outcome with maintaining a higher MAP (>80 mmHg), even if vasopressors are required.^{53–55} Two recent randomized control trials comparing a MAP of 65–75mmHg to 80–100mmHg, demonstrated that higher MAPs was feasible but not associated with secondary outcomes of favorable neurologic status.^{56,57} One concern about augmenting post-arrest MAP is the potential to decrease cardiac output if there is concomitant myocardial dysfunction. Our local practice is to maintain MAPs greater than 80mmHg, if other organ systems are not harmed. In any event, hypotension should be avoided.

The best method used to maintain adequate MAP is also unknown. Fluid resuscitation may be required initially and is dependent on the clinical scenario and patient's current volume status. Vasopressor or inotropic medications are also often necessary, but the selection of vasoactive agents depends on the type of underlying shock. There is growing literature suggesting potential for harm when epinephrine infusions are used in patients with cardiogenic shock.^{58,59}

Ventilator Management – Oxygenation and Ventilation—Hypoxia is consistently associated with worse outcomes after cardiac arrest.⁶⁰ Hyperoxia may also cause secondary brain injury, likely mediated by oxidative stress during reperfusion.^{61,62} The optimal range for partial pressure of arterial oxygen (PaO₂) is unknown but 80–200mmHg is reasonable and a PaO₂ greater than 300mmHg should be avoided. If cooling is ongoing, blood gas results must be temperature corrected.

Cerebral blood flow can be altered through ventilation.²¹ Hyperventilation-induced hypocapnia causes cerebral vasoconstriction and can cause cerebral ischemia and secondary brain injury.⁶³ Mild therapeutic hypercapnia can improve cerebral blood flow and may reduce biomarkers of brain injury⁶⁴ although early phase trial data are mixed.⁵⁶ Cerebral vasodilation also increases cerebral blood volume which can increase intracranial pressure and worsen ischemia.⁶⁵ Current guidelines recommend normocapnia (PaCO₂ 35–45mmHg) in the post-arrest period.^{5,21}

Targeted Temperature Management—Targeted temperature management (TTM) likely offers neuroprotection after cardiac arrest patients.^{5,21,66} The optimal target temperature is unknown. Earlier trials demonstrated benefit from cooling to 32 to 34°C compared to either normothermia or no temperature management.^{67,68} Subsequently, the large TTM trial showed no difference between 33°C and 36°C.⁶⁹ These studies enrolled only or mostly patients with initial shockable rhythms. The TTM study was also limited by a lack of standardization of cooling protocols and used a noninferiority trial design.⁶⁹ More recently, a smaller RCT showed benefit of cooling to 33°C compared to 37°C for patients with an initial nonshockable rhythm.⁷⁰ Fever after cardiac arrest must be avoided; each degree above 37°C is associated with worse outcome.⁷¹

TTM should be started immediately, as delays can reduce the benefit of cooling. Timing of TTM initiation must be balanced with need for any life-saving diagnostic work-up or interventions (e.g., PCI) and any contraindications (e.g., uncontrolled hemorrhage). The best method for induction is unknown.

Shivering secondary to cooling can result in inability to meet temperature goals, so pharmacological (sedation with or without neuromuscular blockade) and nonpharmacological (skin counter-warming) interventions may be necessary. If sedation is required, short acting medications (fentanyl, propofol or dexmedetomidine) are preferable, and benzodiazepines should be avoided if possible.⁷²

Seizures—Many patients who remain comatose after ROSC have abnormal electroencephalogram (EEG) patterns on the spectrum of seizure activity.^{73,74} Seizures may worsen secondary brain injury. However, currently there is little evidence that treating these patterns improve neurologic outcomes.⁷⁵ It is reasonable to treat post-cardiac arrest patients who have generalized tonic-clonic seizures with antiepileptic drugs, although generalized tonic-clonic seizures are an uncommon manifestation of global anoxic injury.⁷⁶ Myoclonus can be observed clinically in the first hours following ROSC, and was historically thought to be ominous. More recent research shows that its presence does not invariably predict poor outcome.^{77,78} EEG can differentiate myoclonus subtypes associated with poor outcome from

those that are not ominous.^{79,80} Transfer to a center with EEG monitoring should be considered to evaluate for seizures and for prognostication purposes.

Transfer to Specialty Care Center

For resuscitated patients who remain comatose, early transfer to a high-volume specialty care center should be considered.¹ Transport to a cardiac resuscitation center has been associated with increased survival and improved short- and long- term outcomes.^{81–85} These specialty care centers can offer PCI, cardiac and neurologic critical care and TTM. For unstable patients, active engagement of a critical care transport team is crucial when arranging interfacility transport. Using a critical care transport team has been shown to be safe and feasible.⁶⁰ Both secondary prevention (arrhythmia work-up with defibrillator implantation, medication assisted treatment for opioid addiction) and rehabilitation (neurologic and cardiac) have contributed to better outcomes for cardiac arrest survivors,⁸⁶ supporting the importance of transfer to specialty care center.

Delayed Neuroprognostication

Clinical nihilism and early limitations in care may result in avoidable mortality.^{47,48} Overall, quality in post-cardiac arrest neuroprognostication studies is low and no single sign, symptom or diagnostic test can accurately predict poor neurologic outcome in the first 24 hours.²⁵ Validated risk stratification tools exist but are not intended to rule out recoverable disease.⁸⁷ Early brain imaging can aid with risk stratification and prognostication but should not be used as a single modality of prognosis in the immediate scenario.^{8,88,89} More advanced neuroprognostication tools, such as EEG, MRI, and blood biomarkers, are less useful in the acute post-resuscitation period. When the prognosis is unclear, as is common in the acute phase of post-arrest care, resuscitation should continue and early withdrawal of life-sustaining therapy should be avoided.

Summary

Post-arrest care that improves survival and functional status, starts in the prehospital setting and continues to discharge planning. In the emergency department, parallel post-resuscitation efforts should focus on work-up of underlying etiologies, preventing rearrest and minimizing secondary brain injury.

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Key Points

- A focused diagnostic work-up to identify and reverse the inciting cause of arrest is essential to prevent rearrest and improve outcomes
- Percutaneous coronary intervention should be considered for all post-arrest patients
- Preventing secondary brain injury by initiating targeted temperature management, maintaining optimal perfusion pressure, optimizing ventilator management and controlling seizures can further improve outcomes
- Transfer to specialty care should be considered during initial resuscitation efforts
- Neurologic prognostication and withdrawal of life-sustaining therapy for perceived poor neurological prognosis should be delayed in all cases

Table 1.

Diagnostic evaluation of the post-cardiac arrest patient

Underlying etiology	Work-up
Cardiac <ul style="list-style-type: none"> • Acute coronary syndrome • Structural heart disease • Cardiomyopathies • RV failure (e.g., pulmonary hypertension, pulmonary embolus) • Arrhythmia 	ECG Troponin BNP Point-of-care Ultrasonography CXR Cardiac catheterization
Pulmonary <ul style="list-style-type: none"> • Primary respiratory failure (e.g., COPD, asthma) • Large airway obstruction 	CXR Blood gas CT angiography of chest Peak and plateau pressures Bronchoscopy
Trauma <ul style="list-style-type: none"> • Exsanguination • Pneumothorax (rib/sternal fractures) • Cardiac tamponade • Solid organ laceration 	Point-of-care Ultrasonography Comprehensive cross-sectional imaging (CT chest/abdomen/pelvis) CXR
Neurologic <ul style="list-style-type: none"> • Stroke • Subarachnoid hemorrhage 	CT head CT angiography head/neck CT perfusion
Septic shock	Cultures (blood, urine, +/- sputum) CXR Complete Blood Count Lactate
Metabolic derangements <ul style="list-style-type: none"> • Diabetic ketoacidosis • Hypoglycemia • Hyperkalemia 	Comprehensive metabolic panel
Exposures <ul style="list-style-type: none"> • Toxicological • Environmental (e.g., electrocution, hypothermia) 	Detailed history Urine Drug Screen (ethanol level, acetaminophen etc.) ECG for intervals