MANAGEMENT OF THE POST-CARDIAC ARREST SYNDROME

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Abstract—Background: Recent advances in resuscitation science have revolutionized care of the cardiac arrest patient. Dramatic departures from time-honored advanced cardiac life support therapies, such as cardiocerebral resuscitation and bundled post-arrest care, have given rise to a new paradigm of resuscitation practices, which has boosted the rate of neurologically intact survival. Objectives: This article reviews the pathophysiology of the post-cardiac arrest syndrome, the collective pathophysiology after return of spontaneous circulation, and presents management pearls specifically for the emergency physician. This growing area of scientific inquiry must be managed appropriately to sustain improved outcomes. Discussion: The emergency physician must understand this pathophysiology, manage resuscitated patients according to the latest evidence, and coordinate with appropriate inpatient resources. Conclusion: The new approach to cardiac arrest care is predicated on a chain of survival that spans the spectrum of care from the prehospital arena through the emergency, intensive, and inpatient settings. The emergency physician is a crucial link in this chain. © 2012 Elsevier Inc.

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INTRODUCTION

Mastery of resuscitation is one of the few niches in medicine that falls squarely under the domain of the practicing emergency physician (EP). During the past decade, our understanding of best resuscitation practices has changed dramatically. A milestone in the evolution of cardiac arrest resuscitation was cardiocerebral resuscitation (CCR), a model of cardiopulmonary resuscitation (CPR) that de-emphasized ventilation and emphasized myocardial and cerebral perfusion (1–4). CCR represented a departure from familiar advanced cardiac life support practices (5). An explicit goal of CCR was to maximize meaningful neurologic survival after sudden cardiac arrest. This goal sparked renewed interest in post-cardiac arrest physiology, and a thorough, evidence-based understanding of the post-cardiac arrest period will hopefully lead to even more improvement in patient outcomes. To establish current understanding and identify knowledge gaps, the International Liaison Committee on Resuscitation (ILCOR) published its consensus statement regarding the post-cardiac arrest syndrome (PCAS), which was incorporated into the 2010 ILCOR guidelines on CPR and emergency cardiovascular care (6,7). These guidelines make specific recommendations for treatment and prognostication based on available evidence or expert consensus opinion. This article reviews the pathophysiology of the PCAS and presents management pearls specifically for the EP.

DISCUSSION

Pathophysiology

The pathophysiology of the PCAS is distinguished by brain injury, myocardial dysfunction, a systemic ischemia/reperfusion response, and any underlying pathophysiology that precipitated the arrest (6). Understanding
these processes can guide the clinician in formulating an appropriate management strategy. A comprehensive discussion of cardiac arrest therapy and management will follow in a later section.

**Brain injury.** Cerebral edema, ischemic degeneration, and impaired autoregulation characterize the brain injury pattern in the PCAS (8–12). Brain injury alone contributes greatly to overall morbidity and mortality in the resuscitated cardiac arrest patient. Neuronal tissue is especially vulnerable to ischemia, given its high metabolic demand and use of oxygen as a metabolic substrate. Ischemia induces cerebral edema and is further complicated by impaired vascular autoregulation (8,9). In the absence of functional hemodynamic regulation, neuronal injury can be exacerbated on either end of the spectrum – ongoing ischemia/microinfarctions or hyperemic reperfusion (13–19). Reperfusion itself also exacerbates neuronal injuries by activating apoptotic cellular pathways and subjecting the tissue to free radical formation and mitochondrial injury (20,21). The brain is uniquely sensitive to other systemic metabolic conditions such as pyrexia, hyperglycemia, and seizures in the post-arrest phase (22–24). Clinically, brain injury in the PCAS manifests as a range of neurologic deficits including neurocognitive dysfunction, seizures, myoclonus, coma, and brain death.

**Myocardial dysfunction.** Myocardial dysfunction in the PCAS seems to be reversible and is characterized largely by global hypokinesis (25,26). Certainly, underlying coronary artery disease or acute coronary syndrome can exacerbate myocardial dysfunction. Patients tend to be hemodynamically labile in the post-arrest phase, and a devastating combination of catecholamine excess/surge and myocardial stunning contributes to tenuous hemodynamics (27,28). Clinically, this manifests as tachycardia, hypotension, decreased ejection fraction, elevated left ventricular end-diastolic pressure, decreased cardiac output, and diastolic dysfunction (29).

**Systemic ischemia/reperfusion response.** The global systemic ischemia/reperfusion response in the PCAS is the ultimate representation of shock and is characterized by a systemic inflammatory immune response, impaired vasoregulation, increased coagulation, adrenal suppression, impaired oxygen delivery and utilization, and immunosuppression (30–36). CPR, though sustaining partial coronary and cerebral perfusion pressures, cannot compensate for the ongoing aerobic metabolism during cardiac arrest and can ultimately result in the multiple organ failure syndrome (37,38). Inflammatory cascades cause immunosuppression, endothelial dysfunction, and activation of coagulation pathways in the microcirculation. Systemic stress causes relative adrenal insufficiency, which may manifest as hypotension requiring vasopressor support. Similar to the patient suffering from septic shock, this ischemia/reperfusion response in the PCAS may be reversible and responsive to early goal-directed therapy.

**Persistent precipitating pathology.** Persistent precipitating pathology in the PCAS is related to any specific disease process that may be the underlying cause for the cardiac arrest. Examples include acute coronary syndromes, pulmonary disease, hemorrhage, sepsis, toxic exposures, and environmental insults. Therapies and management strategies are disease specific and must be coordinated with management of post-cardiac arrest neurologic, myocardial, and systemic disease processes. Clearly, the pathophysiology of PCAS patients is exceedingly complex, so early collaboration with critical care specialists is encouraged.

**Management**

PCAS management in the emergency department (ED) is truly a multidisciplinary process that begins in the prehospital phase, continues through the ED course, and must be coordinated with both admitting and consulting physicians. It requires leadership on the part of the EP to ensure adequate communication between involved parties and appropriate use of available resources. The EP may initiate certain therapies while performing others in consultation with other physicians. General principles of care for the critically ill patient apply, with some PCAS-specific principles being tailored to the needs of the individual patient. Much like early goal-directed therapy for sepsis, the EP should be prepared to deliver a bundle of directed care for the post-cardiac arrest patient (39).

**Advanced airway procedures.** Advanced airway management should be deferred until arrival at the ED or after several cycles of compressions (4). Ideally, prehospital care providers should focus sharply on delivering continuous, high quality, uninterrupted chest compressions. Passive oxygenation and ventilation are provided via facemask with 100% FiO₂. This is a dramatic departure from historic advanced cardiac life support guidelines, which emphasized more definitive airway measures such as endotracheal intubation (5). Theoretically, the blood of a patient who sustains a sudden cardiac arrest should be adequately oxygenated for several minutes. The greatest chance for achieving return of spontaneous circulation (ROSC) occurs through the seamless integration of bystander CPR, uninterrupted compressions by emergency medical services personnel, defibrillation, and transport to definitive care (1–4). Prehospital
intubation causes unnecessary interruptions in CPR and should be deferred until after several cycles of uninterrupted compressions or arrival at the receiving facility (40). However, if a primary pulmonary cause is suspected as the inciting event, the provider should naturally focus on achieving adequate oxygenation and ventilation. After ROSC, advanced airway procedures such as endotracheal intubation should be completed rapidly without hampering ongoing resuscitation. An inadequate airway can be temporized with any number of available supraglottic devices that can be inserted rapidly without hampering chest compressions.

**Best Clinical Practice:** Supraglottic airway devices are ideal for intra-resuscitation airway management. Advanced airway procedures such as endotracheal intubation should be reserved for after ROSC.

**Oxygenation**

Critically ill patients are typically initially oxygenated with 100% FiO₂, which is then titrated down while maintaining adequate PaO₂. The primary goal during and immediately after resuscitation of the cardiac arrest patient is avoiding hypoxemia. The clinical effects of hyperoxia, however, are still being explored. Some preclinical trials demonstrated excessive oxidative stress and free radical formation with hyperoxia during early reperfusion (20,21,41,42). A large multicenter cohort study demonstrated increased inpatient mortality of post-cardiac arrest patients who were hyperoxemic at Intensive Care Unit admission compared to those who were normoxemic and even hypoxemic (43).

**Best Clinical Practice:** Use sufficient FiO₂ after ROSC to avoid hypoxia. The EP may titrate down FiO₂ as long as peripheral oxygen saturation is maintained above 94% or PaO₂ above 60 mm Hg, or both.

**Ventilation**

Interestingly, the cerebrovascular response to CO₂ is preserved in the post-cardiac arrest patient, despite loss of cerebral autoregulation. Patients with traumatic brain injury have demonstrated potentially harmful cerebral ischemia from hyperventilation-induced cerebral vasoconstriction (44). The deleterious effects of aggressive ventilation on cerebral perfusion are well documented in the prehospital resuscitation literature. Hyperventilation raises intrathoracic pressure and decreases cardiac output (45,46). Conversely, hypoventilation contributes to hypoxia, hypercarbia, and associated metabolic acidosis. The strategy of permissive hypercapnia, often used in patients with adult respiratory distress syndrome, may not be applicable to post-cardiac arrest patients with underlying acidosis and labile hemodynamics (47). In patients receiving therapeutic hypothermia, lowering the body temperature decreases metabolic demand and may decrease required minute ventilation. Clinicians familiar with venous blood gas or end-tidal CO₂ interpretation may use these as surrogates for PaCO₂, as long as these surrogates correlate with PaCO₂ and normocarbia is maintained.

**Best Clinical Practice:** Ventilate to achieve normocarbia and monitor with arterial blood gas. Clinicians may elect to monitor venous blood gas or end-tidal CO₂ if these surrogates reliably correlate with PaCO₂.

**Monitoring**

Especially in this era of ED crowding and patient boarding, it is not uncommon for critical care patients to remain in the ED for an extended length of stay. Methods of routine and invasive monitoring are well within the EP’s scope of practice. Resuscitated patients should receive continuous electrocardiographic, pulse oximetry, core temperature (bladder, esophagus), and urine output monitoring. Central venous and arterial access should be obtained to monitor central venous pressure (CVP) and continuous arterial pressure. Routine imaging such as radiography and computed tomography should be performed as clinically indicated. Laboratory values such as complete blood count, chemistries, lactate, central venous O₂ (SvO₂), and arterial blood gas levels should be monitored carefully and frequently to guide ongoing resuscitation.

**Best Clinical Practice:** Every patient suffering from post-cardiac arrest syndrome should receive invasive monitoring. The EP may be involved as time and institutional resources allow.

**Hemodynamic Optimization**

PCAS myocardial dysfunction and ischemia/reperfusion response have much in common with sepsis, which responds to early hemodynamic optimization and early goal-directed therapy (48–51). The hallmark of this strategy is optimization of preload, afterload, contractility, and the balance between oxygen delivery and utilization. Hemodynamic optimization prevents the inflammatory cascade and organ dysfunction associated with sepsis and, by extension, the PCAS. Clinical data used to measure these hemodynamic parameters in the ED include CVP, mean arterial pressure (MAP), SvO₂, urine output, lactate level, and hemoglobin concentration. Although this constitutes an
excellent approach for initial resuscitation of patients after cardiac arrest, it has not been evaluated in the PCAS setting (52). Hypotension, however, should be avoided because it has been shown to result in higher inpatient mortality (53).

The PCAS presents unique challenges in hemodynamic management, different from those associated with sepsis. Several underlying causes of cardiac arrest such as cardiac tamponade, right-sided myocardial infarction, pulmonary embolism, and tension pneumothorax confound the target CVP by causing baseline elevations independent of volume status. The need to perfuse the post-ischemic brain is at odds with relieving strain on the post-ischemic heart. Loss of cerebral autoregulation exacerbates this challenge because cerebral perfusion pressure (CPP) becomes predominantly dependent on MAP (CPP = MAP − ICP). Elevated central venous oxygen saturations do not necessarily reflect adequate oxygen delivery in patients with the PCAS due to impaired oxygen use at the cellular level, a phenomenon known as venous hyperoxia (54). Urine output can be misleading in post-cardiac arrest patients treated with therapeutic hypothermia due to the cold-induced diuresis that often ensues. Lactate clearance is also unreliable in the setting of therapeutic hypothermia and seizures, both of which are common in the PCAS. The optimal hemoglobin concentration for post-cardiac arrest patients is not known, but one Norwegian protocol that demonstrated improved outcomes used a target hemoglobin of 9–10 g/dL (39).

Best Clinical Practice: There is little evidence that points to the best resuscitation practices in the PCAS; however, a focused, goal-directed approach can be modeled on the parameters used in sepsis. Hypotension should be avoided.

Circulatory Support

Despite appropriate hemodynamic optimization strategies, complications such as dysrhythmias and hypotension are common in the PCAS (29). Volume depletion, myocardial dysfunction, and impaired vasoregulation contribute to reduced cardiac output. The threats posed by ischemic myocardial foci underscore the importance of identifying underlying acute coronary syndrome and initiating reperfusion measures. There is no evidence to support the administration of prophylactic anti-dysrhythmic agents to patients with the PCAS. If the cardiac arrest was caused by a primary dysrhythmia, the patient should be evaluated for placement of a pacemaker/implantable cardioverter-defibrillator. Although myocardial stunning is generally temporary and reversible, an aggressive treatment strategy that wholeheartedly embraces reperfusion is warranted in patients who experience it. Hemodynamic optimization involves judicious administration of intravenous fluids to correct intravascular volume depletion and overcome impaired vasoregulation (55). Second-line therapies might include inotropes or vasopressors. Because vasoactive agents might exacerbate myocardial ischemia, it is imperative to reperfuse/revascularize the myocardium. Failure of these therapies may necessitate invasive mechanical measures such as an aortic balloon pump, a left ventricular assist device, or extracorporeal membranous oxygenation (56,57).

Best Clinical Practice: Circulatory support measures for patients with the PCAS include volume resuscitation, inotropes, and vasopressors. Identification and rapid treatment of underlying acute coronary syndrome (ACS) are of critical importance.

Management of ACS

Identification and management of ACS in the post-cardiac arrest patient is crucial to the delivery of definitive care. Typically, coronary angiography and revascularization after cardiac arrest are reserved for the patient with acute ischemic electrocardiographic changes and a superior neurologic status (58). However, the role of coronary angiography is constantly evolving, and there is mounting evidence that any patient who experiences cardiac arrest may benefit from urgent revascularization (59–69).

Most patients who experience cardiac arrest have coronary artery disease (70,71). Underlying coronary lesions are common in cardiac arrest patients; acute myocardial infarction remains the most common cause of sudden cardiac death (72,73). Even patients without clinical features of ACS have a high prevalence of coronary lesions. Traditional ischemic electrocardiographic changes and chest pain are poor predictors of acute coronary occlusion in the post-cardiac arrest period (61,69).

The recent improvements in strategies for cerebral resuscitation and the concomitant potential for neuronal recovery heighten the importance of recognizing ACS early. Cardiac arrest carries an extremely high mortality from neurologic dysfunction. Some clinicians argue that cardiac catheterization should be reserved for patients who demonstrate neurologic improvement (58). Because a patient’s neurologic trajectory does not become evident until several days after ROSC, the practice of restricting catheterization to those who exhibit neurologic improvement may deprive a select group of patients of survival (74,75). Catheterization defines coronary anatomy and identifies coronary vs. non-coronary causes of cardiac arrest. Early intervention may preempt and
therefore obviate the need for more invasive supportive measures.

Clinicians should consider coronary angiography in all post-cardiac arrest patients. If percutaneous coronary intervention (PCI) is not immediately available for patients with electrocardiographic changes indicative of ischemia, they should receive intravenous thrombolysis while awaiting transport to a PCI-capable facility (76,77).

Best Clinical Practice: Recognition and treatment of ACS are critical in the post-cardiac arrest patient. The EP should encourage coronary angiography in all post-arrest patients, especially when ACS is suspected.

**Therapeutic Hypothermia**

Therapeutic hypothermia is the only therapy in the post-cardiac arrest phase proven to increase survival. It is an integral component of bundled care for PCAS (39,59,78–85). Although the ideal timing, induction method, target temperature, duration, and re-warming rate have yet to be determined, consensus is to initiate cooling as soon as possible after ROSC in patients who do not follow commands, continue at 32–34°C for 24 h, and re-warm at 0.25–0.5°C per hour (81,84). If therapeutic hypothermia cannot be performed or is contraindicated, the clinician should at least prevent the occurrence of hyperthermia, because neurologic outcome worsens with increasing body temperature (86).

Hypothermia can be induced in the prehospital arena or in the ED. The benefits seem to be independent of the cooling method. A low-cost method that can be used in the prehospital phase is the application of ice packs (87). Induction is easier to perform early, because core body temperature typically decreases within 1 h after ROSC. Various methods have been developed to achieve effective and rapid cooling. The simplest is the intravenous administration of 30 mL/kg of ice-cold isotonic fluids (88–92). More costly modalities involve surface cooling and closed-loop systems that measure and regulate temperature simultaneously (85,93). Regardless of the method chosen, neuromuscular blockade is essential to suppress shivering. Maintenance is predicated on continuous core temperature monitoring via esophageal or bladder temperature probes. It is a multidisciplinary endeavor that mandates cooperation from prehospital care personnel, nurses, emergency physicians, and intensivists.

Therapeutic hypothermia is not without complications (94). It can increase systemic vascular resistance, thereby reducing cardiac output, and induce dysrhythmias, most commonly bradycardia (85). Cold-induced diuresis, which is common, can exacerbate hemodynamic instability and cause electrolyte imbalances (95,96). Hyperglycemia is also common, because hypothermia decreases insulin sensitivity and secretion (79). It can impair platelet and clotting function, reduce the clearance of sedative and paralytic agents, and cause immunosuppression (96).

The primary challenge for the EP is coordination of hypothermia induction with other PCAS therapies. Communication with consulting and admitting physicians is crucial to this process. Hypothermia can be induced before coronary angiography and safely maintained during the procedure (39,60,62,97–99).

Best Clinical Practice: Hyperthermia should be avoided at all costs in patients with the PCAS. A simple means of inducing hypothermia (32–34°C) in the ED is with intravenous administration of 30 mL/kg of ice-cold isotonic fluids. Cooling may be safely initiated before percutaneous coronary intervention.

**Sedation/Paralysis**

Effective ongoing sedation or paralysis is essential for most PCAS patients. Traditional indications apply, but the PCAS poses additional challenges. The cerebral insult alone may dictate the need for sedation, because it often manifests along a spectrum between confusion and coma. Furthermore, effective sedation/paralysis minimizes the metabolic demand of neuronal tissue. It is also an essential component of therapeutic hypothermia, because achieving and maintaining a temperature of 32–34°C is difficult without suppression of shivering (100). Neurologically intact patients without other clinical indications do not need sedation or paralysis.

Best Clinical Practice: Sedation or paralysis is often needed in the PCAS patient, especially when initiating therapeutic hypothermia.

**Seizure Control**

The cerebral insult of PCAS often manifests as seizures or myoclonus, both of which dramatically increase cerebral and systemic metabolic demand (24,74,101–103). Their suppression is crucial. Benzodiazepines or other antiepileptics such as phenytoin may be employed, but the clinician should be prepared to move quickly to second-line sedating agents such as propofol or a barbiturate (104). Clonazepam is the most effective agent for myoclonus (105). Electroencephalography should be performed on any PCAS patient demonstrating activity suspicious for seizure or myoclonus. The EP should be vigilant for non-convulsive status epilepticus in the PCAS patient who has a poor neurologic response.
Best Clinical Practice: Seizure activity should be treated promptly with acute antiepileptic therapy. The clinician should be vigilant for non-convulsive status epilepticus.

Glucose Control

Hyperglycemia is common after cardiac arrest due to the upregulated stress response. Tight blood glucose control remains controversial in the critical care setting; it has been shown to improve survival in a surgical intensive care setting, but this outcome did not convey to the medical intensive care setting (106,107). A meta-analysis of intensive insulin therapy demonstrated no statistically significant effect on mortality in critically ill patients; in fact, it was accompanied by a six-fold increase in the risk of severe hypoglycemic events (108). The blood glucose concentration should be monitored frequently in the PCAS patient, especially when instituting therapeutic hypothermia. Values up to 8 mmol/L (144 mg/dL) have not been associated with increased mortality (59,109,110). The strategy of extremely tight glycemic control coupled with intensive insulin therapy has been abandoned in favor of a more permissive approach.

Best Clinical Practice: Blood glucose should be monitored closely, especially during therapeutic hypothermia, and maintained at a normal range.

Antibiotics

Antibiotic therapy is tailored to the clinical setting and precipitating pathology that caused the cardiac arrest (i.e., sepsis or other overwhelming microbial infection). However, details about events preceding an out-of-hospital cardiac arrest are often scant. The clinician should be vigilant for incidental aspiration (59,78). The systemic ischemia/reperfusion response of PCAS causes immunosuppression, so these patients are at high risk for ventilator-associated pneumonia during their hospital course (111).

Best Clinical Practice: Be vigilant for aspiration or pneumonia during the events preceding cardiac arrest. Antibiotics should be tailored to the individual patient and clinical setting.

Steroids

PCAS patients can have a relative adrenal insufficiency (33,112). Steroid therapy in the intensive care setting remains controversial. However, a meta-analysis demonstrated no significant effect of corticosteroids on 28-day mortality, intensive care mortality, or hospital mortality (113). Unless there is a clinical history of known adrenal insufficiency, this therapy should be discussed and coordinated with the admitting critical care specialist.

Best Clinical Practice: Unless the patient has a specific history of adrenal insufficiency, corticosteroid therapy should not be employed routinely in the ED without consulting the admitting intensivist.

Renal Replacement Therapy

Renal failure is very common in the critically ill patient and is a common complication of PCAS (60). Certain electrolyte imbalances and other metabolic causes of cardiac arrest may necessitate ED initiation of hemodialysis or other versions of renal replacement therapy. Otherwise, the typical indications apply for a critically ill patient.

Best Clinical Practice: Certain metabolic causes of cardiac arrest necessitate early initiation of renal replacement therapy by the EP.

Case-specific Therapies

Persistent precipitating pathology in patients with the PCAS includes hypoxemia, hypovolemia, hypokalemia, hyperkalemia, hypothermia, acidosis, tension pneumothorax, cardiac tamponade, toxins, pulmonary embolism, ACS, and cerebrovascular catastrophes. Treatment and management strategies are disease specific and must be coordinated with management of the neurologic, myocardial, and systemic disease processes that can follow cardiac arrest. The EP is well poised to maintain a broad differential when considering causes of cardiac arrest and orchestrate the appropriate response.

Best Clinical Practice: Treatment of persistent conditions that caused the arrest must be disease specific and coordinated with other aspects of care.

CONCLUSION

Consensus guidelines not only delineate treatment algorithms but also identify knowledge gaps regarding the epidemiology and pathophysiology of the PCAS as well as the management and predicted outcome of patients with the syndrome (6). Emergency physicians are instrumental in initiating and coordinating appropriate care for this challenging patient population.

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REFERENCES


ARTICLE SUMMARY

1. Why is this topic important?
   Resuscitation from cardiac arrest is a core competency of emergency medicine, and emergency physicians should be current with the latest resuscitation practices. The International Liaison Committee on Resuscitation has identified post-resuscitation care after return of spontaneous circulation as a key link in the chain of survival.

2. What does this study attempt to show?
   This article reviews the pathophysiology of the post-cardiac arrest syndrome and provides management pearls for the emergency physician.

3. What are the key findings?
   The pathophysiology of the post-cardiac arrest syndrome comprises brain injury, myocardial dysfunction, a systemic ischemia/reperfusion response, and persistent precipitating pathology. The traditional mantra of airway, breathing, and circulation has been re-interpreted and applied to this specific patient population. Key interventions, such as management of acute coronary syndromes, therapeutic hypothermia, effective sedation/paralysis, and seizure control, can be initiated by the emergency physician.

4. How is patient care impacted?
   The emergency physician is in the prime position to initiate post-resuscitation care. Meaningful neurological recovery is predicated on an intact chain of survival that spans from the prehospital arena through the emergency department (ED) and into the intensive care or inpatient settings. With the additional challenges of ED overcrowding and boarding of critically ill patients, the emergency physician must be prepared to provide appropriate care for resuscitated cardiac arrest patients.