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REVIEW ARTICLE

Cardiology

State-of-the-art considerations in post-arrest care

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Abstract

Cardiac arrest has a high rate of morbidity and mortality. Several advances in post-cardiac arrest management can improve outcome, but are time-dependent, placing the emergency physician in a critical role to both recognize the need for and initiate therapy. We present a novel perspective of both the workup and therapeutic interventions geared toward the emergency physician during the first few hours of care. We describe how the immediate care of a post-cardiac arrest patient is resource intensive and requires simultaneous evaluation for the underlying cause and intensive management to prevent further end organ damage, particularly of the central nervous system. The goal of the initial focused assessment is to rapidly determine if any reversible causes of cardiac arrest are present and to intervene when possible. Interventions performed in this acute period are aimed at preventing additional brain injury through optimizing hemodynamics, providing ventilatory support, and by using therapeutic hypothermia when indicated. After the initial phase of care, disposition is guided by available resources and the clinician's judgment. Transfer to a specialized cardiac arrest center is prudent in centers that do not have significant support or experience in the care of these patients.

KEYWORDS

coma, critical care, emergency treatment, heart arrest, hypothermia, outcomes, resuscitation

1 | INTRODUCTION

Cardiac arrest accounts for over 500,000 deaths per year in the United States and leads to significant functional disability in those who survive.[1] Advances in cardiac resuscitation and the care of post-cardiac arrest patients have decreased mortality.[2–6] These developments include targeted temperature management (TTM), therapeutic hypothermia, and a focus on preventing secondary organ injury.

The emergency physician is ideally positioned to initiate these time-dependent therapies; thus, recognizing the necessary steps in caring for the post-cardiac arrest patient is critical. This review will explore the key actions that need to take place in the first few hours of care to assess and manage the post-cardiac arrest patient.

2 | GENERAL GOALS AND CONSIDERATIONS

The immediate care after achieving return of spontaneous circulation is complex, requiring focused assessments to determine the etiology of arrest while providing intensive care to mitigate end-organ damage. Along with these physiologic considerations, disposition planning

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should begin when the patient arrives. This ensures timely definitive care (eg, catheter lab is activated if STEMI is evident or outside facility is contacted early for initiation of transfer if required).

Rapid recognition and management of these factors is crucial in the first minutes to hours of care. Failure to awaken due to brain injury is the most common cause of death following resuscitation from cardiac arrest.[7] Thus, preventing additional brain injury directs many of the interventions emergency physicians will institute in the first few hours. The post-return of spontaneous circulation patient is at high risk of hemodynamic instability and may require hemodynamic support to prevent secondary end-organ damage from hypoperfusion. Preventing hypotension is a critical first step in the post-return of spontaneous circulation care bundle. Similarly, preventing hypoxemia and maintaining normocarbia is required to prevent secondary injury. Post-return of spontaneous circulation patients who remain comatose after resuscitation should receive targeted temperature management to prevent additional neuronal injury.[8] Although the ideal temperature is debated, either cooling to between 32°C and 34°C or 36°C is recommended by both US and international guidelines.

As these above interventions are started, the emergency physician should consider potential etiologies of the arrest and initiating any applicable therapies. The following sections will discuss each of these in more detail.

3 | DETERMINING THE ETIOLOGY

During and after resuscitation, it is critical to begin evaluating the patient for the cause of the cardiac arrest and to start treatment of any actionable causes. Although acute coronary syndrome is a common etiology, it is wise to adapt a wide differential.[9] Most cardiac arrest victims have a medical etiology for the arrest. However, even in patients who have sustained trauma along with cardiac arrest, medical causes should still be considered as cardiac arrest may be antecedent to the trauma. Reversible causes of cardiac arrest should be rapidly investigated to prevent relapse. Ensure the patient's vital signs are being accurately monitored and that reliable vascular access has been obtained. Table 1 lists key reversible conditions to consider and their possible etiologies.

3.1 | History and examination

A direct history from the patient of the events leading up to the cardiac arrest is generally not possible given most are comatose after resuscitation. Even if conscious and alert, patients often experience a degree of retrograde amnesia after resuscitation. If the patient is unable to participate, a history should be obtained from the emergency responders, family, or any witnesses. Important factors to try to elucidate are past medical history, social history, medications, recent changes in health, and the circumstances and events leading up to the cardiac arrest. Also inquire about any workup and/or treatment started en route to the emergency department. This information will help to organize the differential and provide a foundation for further workup and treatment.

TABLE 1 Potential etiologies of cardiac arrest

Cardiac	Acute coronary syndrome; primary arrhythmia; congenital/structural heart disease; secondary arrhythmia or cardiomyopathy; cardiogenic shock; pulmonary hypertension; right ventricular failure
Pulmonary	Large airway obstruction; pulmonary failure (ie, chronic obstructive pulmonary disease, asthma)
Infectious	Distributive shock from sepsis
Central nervous system	Intracranial hemorrhage; seizure
Bleeding	Trauma or non-traumatic exsanguination
Metabolic	Diabetic ketoacidosis
Electrolyte	Hypokalemia; hyperkalemia; hypomagnesemia
Mechanical	Pericardial tamponade; pulmonary embolus; tension pneumothorax
Environmental	Electrocution injury; hypothermia; anaphylaxis
Toxicological	Overdose or poisoning

As with all critically ill patients, the clinical examination begins with evaluation of the airway, breathing, and circulation. Compromise of any of these can rapidly lead to re-arrest. Table 2 provides an overview of physical exam findings and potential etiologies associated with the finding.

3.2 | Neurologic examination

A thorough neurologic exam is crucial. It rarely identifies the etiology of arrest, but can be used to provide a baseline of illness severity, which informs not only prognosis but also informs intervention strategy.[11,12] This examination should be performed in the absence of sedative or paralytic agents. Acidosis and hypotension are common in the post-arrest patient, and should be corrected. Assessing both motor findings (ie, Glasgow Coma Scale or Full Outline of UnResponsiveness [FOUR]) and brainstem reflexes (eg, pupillary, corneal, oculocephalic, gag, and cough) is required to adequately describe the severity of brain injury in the post-arrest patient.[13–15] A patient with no motor response and absent brainstem reflexes has a worse prognosis than a patient with no motor response but intact brainstem responses.[11,12]

Assessment of other organ system dysfunction can be accomplished using the Sequential Organ Failure Assessment (SOFA) score. This can be assessed serially during the hospital course and can be obtained rapidly during the initial ED evaluation. Layering the neurologic and cardiopulmonary subscores of the SOFA allows the clinician to determine the Pittsburgh Cardiac Arrest Category of the patient. This validated scale is associated with survival, development of multi-organ failure, and neurologic outcome.[11,12] Moreover, it can be used to guide other interventions including neuroimaging, coronary angiography,

TABLE 2 Examination findings and potential cardiac arrest etiology

Organ system	Clinical finding	Potential etiology
Pulmonary	Diminished breath sounds	Unilateral: Pneumothorax; right mainstem intubation Bilateral: Pulmonary edema
Cardiovascular	New murmur	Papillary muscle rupture or other anatomic abnormality
	Bradycardia	Toxidrome; hypoxia; metabolic/electrolyte abnormalities
	Unequal pulses or blood pressure[10]	Aortic dissection
Extremity	Unilateral swelling/erythema	Pulmonary embolism
	Hemodialysis fistula	Hyperkalemia
Abdomen	Distention/rigidity	Hemorrhage; inflammatory process
	Pulsatile mass	Aortic aneurism rupture
Skin	Cyanosis	Hypoxia; methemoglobinemia; sulfhemoglobinemia
	Mottling; slow capillary refill	Septic shock; hemorrhagic shock
	IV injection sites	Opioid overdose
	Open wounds/cellulitis	Septic shock
	Diffuse urticaria	Anaphylactic shock
Neurologic	Focal motor deficits	Cerebrovascular accident
	Global motor deficits	Toxidrome; hypoxic/anoxic brain injury

and guide the temperature set point for the patient.[16,17] Potential benefits of deeper cooling include a decrease in metabolic demand, decreased intracranial pressure, and suppression of seizure activity.[18] For example, patients with devastating neurologic injury have not been shown to benefit from early coronary angiography.[16] However, those with mild to moderate coma derive benefit from early coronary angiography even if STEMI is not present on the initial EKG. Recent data suggest that depth of coma may be used to determine optimal goal temperature (described below).[17]

3.3 | Initial phase of care

As with other critically ill patients, a point-of-care blood glucose can provide a reversible cause (Figure 1). An ECG can diagnose STEMI—a common and reversible etiology of arrest.[19] Coronary angiography can lead to additional cardiovascular interventions such as the placement of an intra-aortic balloon pump, Impella device, or transition to extra-corporeal membrane oxygenation. There should be a low threshold to activate the cardiac catheterization lab in the post-arrest setting, because evidence shows that early coronary angiography and percutaneous coronary intervention, if indicated, increases survival and decreases morbidity.[20–23] A recent randomized controlled trial demonstrated no difference in outcome between patients receiving early or late percutaneous coronary intervention, but was confounded by a low rate of coronary artery disease and unclear inclusion criteria.[24] Some evidence suggests that patients without ST-elevation benefit from cardiac catherization.[25,26] Thus, guidelines recommend that coma is not a reason to withhold percutaneous coronary intervention in this population.[27] QTc prolongation should prompt investigation into the patient's medications and possible toxidromes. Less common but likely to recur causes of cardiac arrest include Brugada syndrome, Wolf-Parkinson-White Syndrome, and hypertrophic obstructive cardiomyopathy.[28]

The general laboratory workup includes electrolytes, renal function, liver function, arterial blood gas with co-oximetry, toxicology (eg, illicit drugs, ETOH, salicylates, acetaminophen), and CBC. Opiate overdose is a common etiology of arrest.[28,29] Electrolyte abnormalities should be corrected, especially potassium and magnesium derangements. These are common following resuscitation from cardiac arrest and predispose to arrhythmia. If hypokalemia is found on testing, empiric magnesium should be given as well.[9] Blood gas will reveal metabolic abnormalities as well as possible hemoglobin abnormalities (eg, carboxyhemoglobinemia, methemoglobinemia). Renal and liver function tests help with determining the extent of end-organ damage and involvement. A CBC is to mainly assess for severe anemia requiring transfusion. Other labs should be obtained based on the history and presentation of the patient.

Chest radiography is useful to evaluate both clinical interventions (central line placement, endotracheal tube placement) and potential etiologies and sequelae of the resuscitation (pneumonia or pneumothorax). If there is suspicion of a pulmonary embolism, a computed tomography (CT) angiogram of the chest is the best modality to assess for this. D-dimer elevation is common following resuscitation from cardiac arrest, and elevated levels have been associated with poor outcome.[30] It has not been used to risk stratify for pulmonary embolism in this high-risk cohort. Intracranial hemorrhage is found in up to 5% of CT imaging in this cohort, and CT of the brain should be obtained in all comatose post-arrest patients.[31] CT of the brain can also diagnose cerebral edema, which has been associated with poor outcome.[31] Bedside ultrasonography, including the eFAST and rapid ultrasound for shock and hypotension exams, can also be used to evaluate for intraabdominal bleeding, cardiac tamponade, or pneumothorax. General cardiac function can also be assessed. Increase in right heart

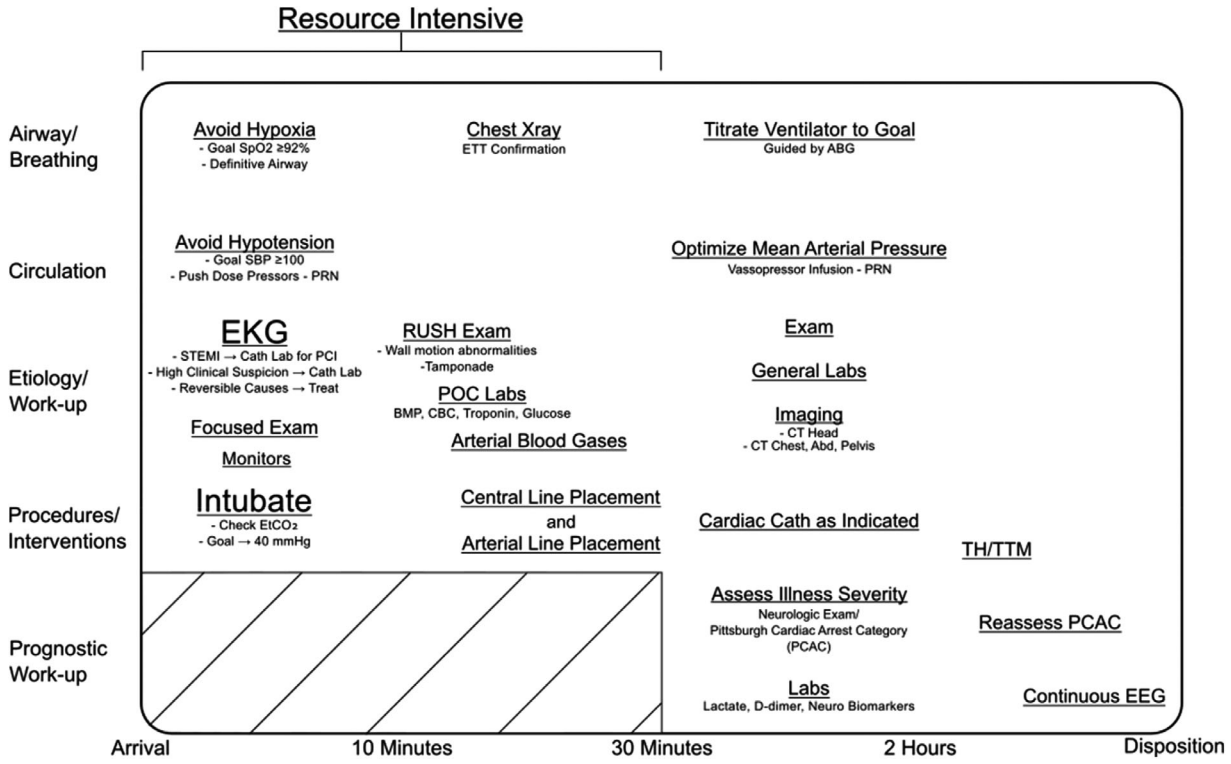

FIGURE 1 Temporal diagram of initial post resuscitation care. The initial phase is resource-intensive and requires several team members to provide care similar to a trauma activation. The goal in this phase is stabilization. Once the patient is stabilized, intensive care with fewer team members at the bedside may be accomplished

volume or right ventricular hypokinesis can be signs of increased pulmonary pressure secondary to a pulmonary embolism. Significant fluid in the pericardium may be a sign of cardiac tamponade.

3.3.1 | Neuroprotective strategies and considerations

Neurologic damage is the leading cause of mortality in patients that experienced out-of-hospital cardiac arrest and is associated with increased mortality in the setting of in-hospital cardiac arrest as well.[7] Hyperthermia after cardiac arrest is associated with poor neurologic outcomes, and thus, is to be avoided. Elevated core temperature (temperature >37°C) during the initial 48 hours after cardiac arrest is associated with increased mortality.[32]

The current data demonstrate improved neurologic outcomes with the use of therapeutic hypothermia and targeted temperature management.[6,33-35] We define therapeutic hypothermia as a core body temperature of 32°C to 34°C and targeted temperature management a core body temperature of <36°C. One meta-analysis of 11 studies, 3 of which were randomized trials, demonstrated lower mortality (odds ratio [OR], 0.51; 95% confidence interval [CI], 0.41–0.64) as well as improved neurologic outcome (OR, 2.48; 95% CI, 1.91–3.22) in patients who underwent either therapeutic hypothermia or targeted temperature management after cardiac arrest compared to those who did not.[36] The optimal core temperature is not known; thus, guidelines target a range between 32°C and 36°C.[2,37] The majority of these trials predominantly enrolled patients with shockable primary rhythms, witnessed status, and bystander cardiopulmonary resuscitation (CPR). These cohorts reflect the minority of patients resuscitated from cardiac arrest. The recently published HYPERION trial demonstrated improved neurologic outcomes at 90 days for patients with a non-shockable primary rhythm of arrest receiving 33°C as their target temperature.[34] Recent work has also demonstrated improved outcomes with a lower targeted temperature in patients with more severe illness severity on hospital arrival.[17]

In one small retrospective study, the goal temperature in post-cardiac arrest patients was changed from 33°C (n = 24) to 36°C (n = 52). In the 36°C group, there was significantly less utilization of active cooling (100% in the 33°C group vs 70% in the 36°C group), less time spent at the target temperature (87% in the 33°C group vs 50% in the 36°C group), and an increase in rates of fever (0% in the 33°C group vs 19% in the 36°C group).[38] The data showed a trend toward worse neurologic outcomes in the 36°C group. Regardless of the goal temperature selected, devices with active temperature control and a feedback loop should be used to minimize deviations from goal temperature.

3.4 | Populations

Contraindications for temperature control after cardiac arrest are advanced medical directives stating the patient does not want

aggressive measures or if the clinical situation does not merit such intervention. Otherwise, patients who are unable to follow commands should have temperature management initiated immediately. Choice of goal temperature should be chosen based on the clinical presentation. For example, therapeutic hypothermia may not be optimal in a patient with active noncompressable bleeding, but targeted temperature management is safe and reasonable in this scenario. Both therapeutic hypothermia and targeted temperature management can be used in pregnant patients, hemodynamically unstable, and/or undergoing coronary catheterization or receiving thrombolytics, though therapeutic hypothermia may increase risk of bleeding in the latter 2 groups.[23,39–43] Most importantly, preventing fever is critical in all post-cardiac arrest patients. It is unacceptable to neither monitor nor maintain temperature in the post-arrest patient.

3.5 | Initiation, goals, and duration of therapy

Active management of the post-cardiac arrest patient's core body temperature should be initiated as soon as possible and maintained for at least 48 hours.[6,32,37,44,45] The optimal timing of initiation of therapeutic hypothermia or targeted temperature management is unclear. Randomized trials have not shown improvement of outcomes with prehospital initiation of therapeutic hypothermia.[46,47] One trial demonstrated worse outcomes in those arriving at goal temperature rapidly while others have demonstrated improved outcomes.[48–50] Unfortunately, none of these trials have stratified enrollment based on initial neurologic injury, limiting the use of their results.

One approach would be to maintain 36°C for 24 hours in patients with moderate coma (intact brainstem responses) or shockable primary rhythm.[17,37] In patients with non-shockable primary rhythm or more severe coma (loss of brainstem reflexes), consider 33°C for 24 hours.[17,34] Potential benefits of deeper cooling include a decrease in metabolic demand, decreased intracranial pressure, and suppression of seizure activity.[51–54]

In one study of 355 patients randomized to either 24 or 48 hours of therapeutic hypothermia, survival in the 48-hour group was 69%, whereas survival in the 24-hour group was 64% (absolute difference 4.9%; 95% CI, −5% to 14.8%). This did not meet their specified goal of a survival difference of 15%.[55] The recently funded Influence of Cooling Duration on Efficacy in Cardiac Arrest Patients (ICECAP trial) will randomize subjects to different duration of therapeutic hypothermia and may provide additional data on optimal cooling duration.

3.6 | Induction of therapeutic hypothermia

The predominant methods used to induce therapeutic hypothermia or targeted temperature management are intravascular and surface cooling. The former is in the form of cold intravenous fluids and venous catheters, whereas the latter involves direct application of a cooling blanket, gel adhesive, ice, or cold fluid to facilitate cooling. Although the rate of cooling may differ, there have not been differences in neurologic outcome when comparing the methods.[56–59] Importantly, patients are often mildly hypothermic post-cardiac arrest because of the cooled peripheral circulation returning to the core.[33] Monitoring temperature is critical before induction of therapeutic hypothermia or targeted temperature management. These data are fed back to the cooling device, facilitating a consistent temperature.[34] Recent work in traumatic brain injury cohorts has shown that variation in temperature regulation has been associated with worse neurologic outcome.[33]

Intravenous administration of 30 mL/kg of cold (4°C [39°F]) isotonic saline with the assistance of a pressure bag is shown to lower core temperature by >2°C per hour.[46,60,61] A single liter of cold saline administered over 15 minutes can result in a 1°C reduction in core temperature. These rates are similar to those seen with commercially available cooling devices. However, this approach may not be optimal in patients with known heart failure, advanced renal disease, or evidence of acute pulmonary edema.

Surface cooling strategies and/or endovascular cooling devices should be implemented. Surface cooling uses items such as cooling blankets, ice packs, and cooling vests to decrease the core body temperature via conduction. If ice packs are used, they should be applied to the neck, groin, and axillae next to major vascular bundles. Adjuvant measures include cool water baths or cool water with fans to increase evaporative cooling. Alternatively, intravascular cooling catheters circulate coolant fluid inside of a catheter in the superior or inferior vena cava, resulting in a cooling rate of up to 2°C/h.[56]

3.7 | Temperature monitoring

It is crucial to continuously monitor core body temperature during therapeutic hypothermia and targeted temperature management as it closely correlates with brain temperature.[69] Measurement of the central venous temperature is considered the gold standard. Esophageal or bladder temperature correlates closely with central venous temperature and follows changes in temperature.[70,71] Rectal temperature may trail behind rapid core temperature changes by up to 1.5°C.[70] Axillary, oral, and tympanic temperature are poor surrogates during therapeutic hypothermia and should be avoided.

3.8 | Rewarming

It is unlikely that rewarming will be done in the ED. However, if rewarming is required, most clinicians choose a rate of 0.25°C to 0.5°C per hour.[4,72] Preclinical studies involving traumatic brain injury demonstrated loss of the benefits of therapeutic hypothermia when the rate of rewarming surpassed 0.5°C per hour.[73–75]

Devices that automatically monitor and regulate temperature during the entire therapeutic hypothermia process (induction, maintenance, and rewarming phases) reduce the risk of excessive rates of temperature change during rewarming.[76,77] If these devices are

not readily accessible, manual rewarming can be used with close monitoring of the patient's temperature.

3.9 | Potential complications

Although therapeutic hypothermia and targeted temperature management improve outcomes after resuscitation from cardiac arrest, side effects are common. Existing data suggest that these sequelae represent the critical illness of the patient rather than a sequelae of therapeutic hypothermia or targeted temperature management therapy. Clotting is an enzymatically mediated process. In vitro studies show core temperatures below 35°C impede the clotting cascade and platelet function, though this has not been demonstrated by in vivo studies.[78–81] Studies specific to therapeutic hypothermia have not shown a significant difference in the incidence of bleeding in post-cardiac arrest patients receiving therapeutic hypothermia versus those who remain normothermic.[6,37,44,82] If a therapeutic hypothermia patient develops significant non-compressible bleeding, the patient should be actively rewarmed to a core temperature of 36°C. Therapeutic hypothermia has been shown to disrupt leukocyte function, but data do not demonstrate more infections in those receiving therapeutic hypothermia or targeted temperature management or increased mortality in those infected.[37,39,44,83]

Shivering is a natural response to hypothermia designed to increase core body temperature. Suppression of this is crucial during therapeutic hypothermia or targeted temperature management.[60,62,63] Multiple strategies have been explored. A continuous infusion of propofol at 20 μg/kg/min with titration up to 50 μg/kg/min may be used. If the response is inadequate with propofol alone, a bolus of fentanyl at 0.5–1.0 μg/kg can be added or a continuous infusion of fentanyl starting at 25–100 μg/h may be used. Addition of intermittent administration of a benzodiazepine such as midazolam is reasonable as well. Continuous infusion of midazolam at 2–10 mg/h instead of propofol is reasonable in hypotensive patients but may confound the neurologic examination. Although midazolam infusion can result in deposition in adipose tissue, therapeutic hypothermia also reduces clearance and excretion of midazolam.[64] Dexmedetomidine is also effective in the reduction of shivering, but its side effects of hypotension and bradycardia can limit its desirability in this patient population.[65] Short-acting medications such as remifentanil have also been shown to effectively suppress shivering and have been associated with shorter ICU length of stay.[66]

Neuromuscular blocking agents are highly effective at preventing shivering but confound the neurologic examination and may mask seizure movement. Electroencephalogram monitoring is recommended because of the high risk of seizures post-cardiac arrest.[67,68]

Hypothermia slows cardiac conduction leading to bradycardia and QT prolongation.[84,85] Bradycardia is common in patients undergoing therapeutic hypothermia and does not generally necessitate intervention. If ventricular tachyarrhythmias occur such as ventricular fibrillation, studies on animal models report comparable results with defibrillation during hypothermia as normothermia.[86,87] The use of therapeutic hypothermia has not correlated with increased need for vasopressor support regardless of whether the patient required vasopressor support prior to induction of therapeutic hypothermia.[88,89]

Induction of therapeutic hypothermia results in diuresis, potentially leading to electrolyte abnormalities, including hypokalemia, hypomagnesaemia, and hypophosphatemia.[90] Potassium shifts into the intracellular space during induction of therapeutic hypothermia, also decreasing serum levels.[90–92] Consequently, serum electrolytes should be frequently monitored and replaced during therapeutic hypothermia and targeted temperature management. Total body potassium may not be low; thus, conservative replacement is recommended.

Patients undergoing therapeutic hypothermia can become insulin resistant.[84,93] Therapeutic hypothermia decreases CYP3A4 metabolism, potentially altering serum levels of other medications.[94–96] To our knowledge, there are no studies illustrating how to guide titration of medication based on serum levels during therapeutic hypothermia or targeted temperature management.

4 | RESPIRATORY AND HEMODYNAMICS

4.1 | Respiratory management and considerations

Airway compromise can lead to re-arrest. Immediately following return of spontaneous circulation, it is critical to confirm that the airway is secure with a definitive airway (ie, endotracheal intubation), and the patient is adequately ventilated to prevent recurrent arrest. Gastric insufflation is common during CPR; thus, a gastric tube should be placed to decompress the stomach.

Based on the current data, the targets for patients undergoing mechanical ventilation include a $PaCO_2$ of 40 mm Hg (or an end-tidal CO_2 of 35 mm Hg) and an oxygen saturation (SpO_2) >94%. A normal $PaCO_2$ (35–45 mm Hg) is shown to correlate with improved outcomes when compared to hypercapnia or hypocapnia.[97–100] Hypocapnia may lead to hypocapnia-induced cerebral vasoconstriction and, in turn, cerebral hypoperfusion. Thus, hyperventilation should be avoided when possible. Hyperventilation can also reduce preload and compromise cardiac output and coronary perfusion pressure.[101] Hypoventilation can lead to hypercapnia, potentially increasing cerebral blood flow, cerebral edema, and exacerbation of neuronal injury.

The goal when titrating oxygen concentration in the post arrest patient is to avoid any episode of hypoxia while avoiding prolonged hyperoxia (defined as a PaO_2 >300). Prolonged periods of hyperoxia have been linked to worse outcomes in multiple studies.[102,103] Titrating FiO_2 to the lowest rate that supports an SpO_2 ≥94% (or a PaO_2 of ≈100 mm Hg) is reasonable. Most arterial blood gas measurements do not account for the effect of therapeutic hypothermia or targeted temperature management on PaO_2 concentrations. The reported PaO_2 may be artificially higher than the actual value, meaning the patient's actual PaO_2 may be lower than reported. Recent data suggest a slightly higher PaO_2 (around 100–120 mm Hg) is reasonable in patients undergoing therapeutic hypothermia or targeted temperature management.[100]

4.2 | Hemodynamic management and considerations

Ensuring adequate blood pressure post-cardiac arrest is crucial in minimizing secondary damage. In the comatose post-arrest patient, a mean arterial pressure (MAP) of ≥65 mm Hg may be inadequate. Cerebral autoregulation is impaired after return of spontaneous circulation and an MAP goal of 80–100 mm Hg may be targeted.[104–106] Additional goals that indicate adequate perfusion are maintaining central venous pressure at 8–12 mm Hg and urinary output of >0.5 mL/kg/h. Intravenous fluids, either normal saline or lactated ringers, are to be used to achieve and/or maintain the central venous pressure goal of 8–12 mm Hg. If large volumes are required, lactated ringers may prevent the hyperchloremic metabolic acidosis that can develop in patients given large amounts of normal saline. Continuous blood pressure monitoring with an arterial line is helpful when titrating vasopressors and inotropes.

Vasopressors and/or inotropes are often required in the 24–48-hour period after resuscitation because of depressed myocardial function secondary to the cardiac arrest.[107,108] Frequently used vasopressors include dopamine, norepinephrine, and epinephrine. However, norepinephrine may be safer to use in the setting of cardiogenic shock given the increased risk of arrhythmias when using dopamine.[109] In cardiogenic shock, dobutamine and milrinone are potentially useful for inotropic support. However, both agents are vasodilators that may cause hypotension. Dobutamine can precipitate tachyarrhythmias as well, likely because of its chronotropic effects.

Although coronary angiography provides anatomic information and may yield the etiology of arrest, additional interventions to provide cardiovascular support such as ventricular assist devices or intra-aortic balloon pumps may also be placed. Several sites are exploring the use of extra corporeal membrane oxygenation placement in the cardiac catheterization lab for post arrest patients with good neurologic outcomes.[110]

5 | GENERAL SUPPORTIVE MEASURES

Standard critical care measures should be implemented in the post-cardiac arrest patient population. This includes deep vein thrombosis prophylaxis, stress ulcer prophylaxis, and early physical and occupational therapy. Elevation of the head of the bed to 30° can help reduce intracranial pressure and reduce the chance of aspiration.

5.1 | Role of antibiotics

Pneumonia is common, occurring in 50% to 70% of patients.[111,112] However, existing data have not demonstrated a mortality difference between those with and without pneumonia. A recent randomized, controlled trial demonstrated a lower incidence of pneumonia in patients treated with 2 days of amoxicillin-clavulanate rather than placebo. There was no difference in mortality, length of stay, or ventilator-free days between the groups.[113]

6 | DISPOSITION

Care for post-cardiac arrest patients is resource intensive and requires coordination from multiple specialties. Facilities that manage over 50 post-cardiac arrest patients per year have better outcomes when compared to those that care for fewer.[114–117] Centers that have cardiac catheterization capabilities demonstrate better outcomes in this population.[114,115] Regional cardiac arrest centers may concentrate these resources and improve long-term outcomes.[118] Thus, it is reasonable to transfer a post-cardiac arrest patient to a tertiary care facility that has access to advanced resources and is experienced in managing these patients.

7 | CONCLUSIONS

Bundles of care that include aggressive resuscitation of cardiopulmonary dysfunction, neuroprotective care such as therapeutic hypothermia and targeted temperature management, and delayed neuroprognostication have improved outcomes in the patient successfully resuscitated from cardiac arrest. The emergency physician plays a critical role in evaluating, stabilizing, and implementing these therapies to optimize outcomes. In facilities that do not routinely provide comprehensive care to the post arrest patient, consideration of transfer to a cardiac arrest center should be considered.

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CONFLICT OF INTEREST

The authors have no conflict of interest to disclose.

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