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Haemodynamic and ventilator management in patients following cardiac arrest

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Abstract

Purpose of review—The purpose of this study is to review the recent literature describing how to assess and treat postcardiac arrest syndrome associated haemodynamics and manage oxygenation and ventilation derangements.

Recent findings—Postcardiac arrest syndrome is a well described entity that includes systemic ischemia-reperfusion response, myocardial dysfunction and neurologic dysfunction. Continued resuscitation in the hours to days following return of spontaneous circulation (ROSC) is important to increase the likelihood of long-term survival and neurological recovery. Post-ROSC hypotension is common and associated with worse outcome. Myocardial dysfunction peaks in the first 24 h following ROSC and in survivors resolves over the next few days. Hyperoxemia ($paO_2 > 300$ mmHg) and hypoxemia ($paO_2 < 60$ mmHg) are associated with worse outcomes and hyperventilation may exacerbate cerebral ischemic injury by decreasing cerebral oxygenation.

Summary—Patients who are successfully resuscitated from cardiac arrest often have hypotension and myocardial dysfunction. Careful attention to haemodynamic and ventilator management targeting normal blood pressure, normoxemia and normocapnia may help to avoid secondary organ injury and potentially improve outcomes.

Keywords

children; heart arrest; hyperoxemia; hyperventilation; hypotension; myocardial dysfunction; postcardiac arrest syndrome

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Conflicts of interest

There are no conflicts of interest.

INTRODUCTION

Outcomes from cardiac arrest have improved over the last decade [1,2], presumably due to an increased focus on early recognition of the arrest and high-quality cardiopulmonary resuscitation (CPR). After return of spontaneous circulation (ROSC), persistent pathophysiologic alterations, which have been identified as the ‘postcardiac arrest syndrome’ (PCAS), can contribute to poor outcomes. PCAS has four major components: brain injury, myocardial dysfunction, systemic ischemia/reperfusion response [3] and persistent precipitating disease [4]. Recognition and treatment of these key components may decrease the risk of ongoing secondary injuries and can thereby improve survival. In this review, a particular focus on haemodynamic and ventilatory management in the setting of PCAS and the impact of such strategies on patients’ outcome will be discussed.

HAEMODYNAMICS FOLLOWING RETURN OF SPONTANEOUS CIRCULATION

Hypotension and end-organ hypoperfusion due to a combination of myocardial dysfunction and a systemic ischemia reperfusion response occur commonly after successful resuscitation from cardiac arrest, yet resolve over the ensuing few days in survivors [5]. Some patients will have isolated myocardial dysfunction, others will have normal myocardial function with severe vascular instability and many will have a combination of both. In 2010, the American Heart Association (AHA) postarrest care guidelines recommended treating hypotension (SBP <90 mmHg in adults) with intravenous fluids and vasopressor infusions, targeting an SBP of more than 90 mmHg [6]. Nevertheless, the optimal postarrest blood pressure is unclear.

Postarrest hypotension

Hypotension following cardiac arrest is common. Trzeciak *et al.* [7] used the Project Impact database to show that 47% of 8736 adult patients admitted to an ICU following resuscitation from cardiac arrest had hypotension (SBP <90 mmHg). Although 50% of all patients died, patients who had systolic hypotension within 1h of ICU arrival were less likely to survive to discharge and less likely to survive with a favourable neurologic outcome.

Although Trzeciak *et al.* [7] addressed the impact of hypotension on outcome at a single point in time, the cumulative impact of hypotension had not been well described. Recently, Kilgannon *et al.* [8] prospectively measured noninvasive blood pressures every 15 min during the first 6 h after resuscitation from cardiac arrest and calculated a time-weighted average in 151 individuals without implementation of a standardized treatment protocol. When comparing survivors and nonsurvivors, patients with a favourable neurologic outcome had a significantly higher threshold time weight average mean arterial pressure (TWA-MAP) of 83 mmHg (± 13) than those with an unfavourable neurologic outcome: TWA-MAP of 77 mmHg (± 18), $P = 0.042$. The authors concluded that a TWA-MAP of more than 70 mmHg was associated with a favourable neurologic outcome at discharge. After adjusting for CPR duration or ‘downtime’ of more than 10 min, the odds of survival was 5.25 [95% confidence interval (95% CI), 1.73–15.98, $P = 0.003$] for patients who had a TWA-MAP of

more than 70 mmHg as compared with those with a TWA-MAP of 70 mmHg or less. Their data revealed a strong association between hypotension and poor neurologic outcome.

The impact of hypotension on outcome was corroborated in a large and systematic posthoc secondary analysis of the Targeted Temperature Management (TTM) trial [9■■■] to evaluate the impact of temperature [hypothermia (33°C) vs. normothermia (36°C)] on blood pressure [10■■]. Among 920 patients, lower blood pressures were associated with higher mortality rate. In addition, Bro-Jeppesen *et al.* [11] studied 310 comatose OHCA patients treated with therapeutic hypothermia on a standard protocol of volume resuscitation, targeted MAP of more than 65 mmHg and first vasopressor selection of dopamine with epinephrine/norepinephrine as secondary agents. Not surprisingly, patients who received dopamine with additional norepinephrine/epinephrine had a significantly higher mortality rate (49 vs. 23%, $P < 0.0001$, hazard ratio 2.0, 95% CI 1.3–3).

Topjian *et al.* [12■■] extended findings in adults to children postarrest. They found that children who had hypotension, defined as SBP less than 5 percentile for age and sex, were less likely to survive to discharge and less likely to survive with a favourable neurologic outcome. Interestingly, among 41% of children who received post-ROSC vasopressors, there was no difference in outcome between those with and without hypotension, suggesting that vasopressor use mitigated the adverse effects of postarrest hypotension. Perhaps vasopressor use allowed the clinicians to promptly treat hypotensive episodes and allowed a superior TWA-ABP.

Optimal mean arterial blood pressure targets

Because the data associating postarrest hypotension and/or vasopressor therapy with poor outcomes were all from observational studies, the study designs preclude determination of causality. For example, hypotension may simply be a marker of the severity of the hypoxic-ischemic and reperfusion injuries. Consequently, it remains unclear whether targeting a higher MAP improves outcomes. In addition, treatment with inotropes and vasopressors may improve brain and other organ blood flow, yet increase oxygen demand and consumption in hearts and brains with limited reserve.

In the study by Kilgannon *et al.* [8■■] correlating TWA-MAP with outcomes, the TWA-MAP thresholds greater than 70 mmHg were not associated with improved outcomes. When they evaluated the subset of patients who maintained a TWA-MAP of more than 70 mmHg, they found attaining TWA-MAP more than 70 mmHg without vasopressors was associated with a better neurologic outcome than attaining it with vasopressors (48 vs. 24%, $P = 0.01$). Nevertheless, attaining adequate TWA-MAP with vasopressors may improve outcomes among patients who cannot attain adequate TWA-MAP without vasopressors. Although one study found that a higher MAP was associated with better outcomes [13], another found that a MAP of more than 80 mmHg was not associated with better outcomes [14]; both showed that hypotension was associated with worse outcomes among adults treated with therapeutic hypothermia after resuscitation from cardiac arrests [14].

Post-ROSC hypotension and/or vasopressor/inotrope therapy are associated with worse outcomes following resuscitation from cardiac arrest. However, accurate assessment of the

cause of hypotension, including myocardial function, relative fluid status and vascular tone should inform optimal therapy. The systemic inflammatory response syndrome can result in vasodilation and capillary leak, which may respond to vasopressors and fluid resuscitation, respectively. The myocardial dysfunction may respond to inotropic support. Presumably, clinicians should determine the appropriate need for fluid resuscitation and inotropic or vasopressor support to optimally maintain adequate MAPs.

Postcardiac arrest myocardial dysfunction

Reversible postcardiac arrest myocardial dysfunction including depressed left ventricular ejection fraction (LVEF) and increased left ventricular enddiastolic pressure (LVEDP) was first described in animal models of ventricular fibrillation cardiac arrest [15–17]. These same findings have been described in clinical studies and shown to occur in the hours following successful resuscitation with recovery of function over the ensuing days [5]. The left ventricular dysfunction is significantly worse in patients with haemodynamic instability. Of note, longer duration of cardiac arrest and more intraarrest doses of epinephrine are also associated with more haemodynamic instability. Patients who survive to hospital discharge have higher LVEF in the first 24 h post-ROSC than nonsurvivors [18]. Similar to these findings in adults, children resuscitated from OHCA also showed that depressed myocardial function is associated with a lower rate of survival to discharge [19]. Importantly, cardiac arrests in many adults are associated with acute coronary syndromes and ST elevation. These patients may have ongoing acute myocardial ischemia as a contributor to the postarrest myocardial dysfunction, and they may benefit from a percutaneous coronary intervention (PCI).

Haemodynamic management

Hypotension is common after resuscitation from cardiac arrest and is multifactorial in cause. Although ideal MAPs are not known, it is clear that hypotension is associated with worse outcomes. Clinicians should aggressively monitor and treat hypotension targeting a MAP of more than 65 mmHg for adults and an SBP more than fifth percentile [20] for children (Table 1 and Fig. 1). Appropriate fluid resuscitation and/or vasopressor support and/or inotropic support should be provided in relation to individualized needs. In cases of persistent hypotension, clinicians should monitor cardiac output utilizing ScvO₂ and echocardiogram and monitor end-organ oxygen delivery by following lactate levels. The need for PCI should be considered in adults with postarrest myocardial dysfunction, especially those with ST elevation or focal wall motion abnormalities.

VENTILATOR MANAGEMENT

Ventilator management of the resuscitated patient must account for the underlying precipitating physiology causing the arrest, the impact of positive pressure ventilation on haemodynamics and the impact of oxygenation and ventilation on the acutely injured brain.

Pathophysiology of oxygenation and ventilation and impact on postanoxic brain

In patients suffering from postanoxic brain injury, lungs may be damaged from trauma due to chest compressions, aspiration of blood/gastric content or development of pneumonia

[21]. Post-ROSC ventilator-induced lung injury may also occur [22]. In experimental studies, mechanical ventilation can also directly injure the brain through aberrant vagal nerve inputs, which activate dopamine D₂ receptors in the hippocampus and induce local apoptosis without causing hypoxia, oxidative or inflammatory stress [23]. Moreover, the use of high tidal volume (V_T) can be harmful to the brain because it is associated with cerebral blood flow (CBF) instability, white matter injury and vascular leakage with oedema formation [24].

Postcardiac arrest ischemia-reperfusion injury activates various intracellular pathways and promotes mitochondrial dysfunction and eventually leads to cell death [25]. When CBF is restored, one proposed mechanism for brain damage involves generation of reactive oxygen species (ROS), including superoxide (O_2^-), peroxynitrite (NO_2^-), hydrogen peroxide (H_2O_2) and hydroxyl radicals (OH^-) [26]. Therefore, supra-normal oxygen concentrations may have detrimental effects on patients with postanoxic cerebral damage [27,28].

Carbon dioxide (CO_2) is a potent regulator of cerebrovascular tone and may induce profound and reversible effects on CBF, such that hypercapnia causes marked vasodilation and increased CBF, whereas hypocapnia causes vasoconstriction and decreased blood flow [29]. Thus, tight control of $paCO_2$ might be necessary in patients suffering from postanoxic brain injury. This may be very important in patients treated with TTM, which affects not only respiratory mechanics but also gas exchange (i.e. decreased CO_2 and increased O_2 during cooling procedures at unchanged minute ventilation) [30,31].

Clinical studies on hyperoxemia

Kilgannon *et al.* [32] demonstrated a higher hospital mortality among 6326 ICU patients post-ROSC who had hypoxemia (defined as $paO_2 < 60$ mmHg or altered gas exchange with $paO_2/FiO_2 < 300$) or hyperoxemia (defined as $paO_2 > 300$ mmHg) than those with normoxemia on the first arterial blood gas available within 24 h after admission. Bellomo *et al.* [33] also reported a higher mortality rate for 12 108 patients with hyperoxemia and hypoxemia than normoxemic patients. However, in a Cox proportional hazards regression model including adjustment for other relevant confounders, oxygen levels were no longer statistically associated with poor outcome. Limitations of these two studies include no data about the initial resuscitation, such as duration of no-flow, quality of CPR and initial rhythm; few patients received TTM; only the first or the worst arterial blood gas analysis was considered, which was probably not representative of overall oxygen exposure; and absence of neurological outcomes.

Several relatively small paediatric studies have failed to reveal an association of postarrest hyperoxemia and outcome. van Zelle *et al.* [34] found no relationship between paO_2 values and in-hospital mortality in a cohort of 200 patients. Moreover, mortality was even significantly lower in those children treated with TTM and exposed to high paO_2 levels. In a smaller retrospective cohort ($n = 74$), hyperoxemia occurred in half of patients within the first 24 h after cardiac arrest but was not associated with 6-month mortality [35]. In another study, neither oxygenation nor ventilation status was associated with patients' outcome, even after correction for potential confounders [36].

Several small studies in adult cardiac arrest patients have also challenged the potential association between high paO_2 levels and poor outcome [37■,38■,39,40]. A recent meta-analysis indicated that hyperoxemia (i.e. $paO_2 > 300$ mmHg) appeared to be associated with increased in-hospital mortality but not worsened neurological outcome; however, the results were inconsistent in subgroup analyses and biased by a large heterogeneity [41■]. A large prospective investigation adjusting for factors associated with the cause of the arrest, duration of arrest, arrest rhythm, comorbidities and other postarrest care will be necessary to determine whether post-ROSC hyperemia is indeed associated with worse outcomes.

Clinical studies on carbon dioxide

In adults, two cohort studies have initially evaluated the effects of $paCO_2$ abnormalities on the outcome of cardiac arrest patients. Roberts *et al.* [42] showed that the occurrence of hypocapnia (defined as $paCO_2 < 30$ mmHg) and hypercapnia (defined as $paCO_2 > 50$ mmHg) during the first 24 h after arrest was 27 and 33%, respectively (n = 193). In a multivariable logistic regression analysis, the presence of hypocapnia or hypercapnia was an independent predictor of poor neurological outcome. A larger retrospective study of 16 542 patients revealed that [43] hypocapnia ($paCO_2 < 35$ mmHg) was associated with higher mortality than normocapnia, while hypercapnia ($paCO_2 > 45$ mmHg) was associated with a higher rate of patients discharged home. More recently, a Finnish prospective study (n = 409) showed that mean 24-h $paCO_2$ of more than 45 mmHg was associated with favourable neurological outcomes. The latter two studies suggest that hypercapnia may be neuro-protective [38■].

Similarly, Del Castillo *et al.* [44] demonstrated hypocapnia and hypercapnia among 14 and 28% of 223 children postarrest, respectively. Hypocapnia and hypercapnia were associated with poor outcome, especially if occurring immediately after ROSC. In contrast, another paediatric study demonstrated that 38% of 195 patients had hypocapnia and 46% hypercapnia, yet $paCO_2$ abnormalities were not associated with outcome [36].

Ventilator management

The International Liaison Committee on Resuscitation (ILCOR) guidelines recommend titration of oxygen administration to obtain an oxygen saturation of 94–96% after ROSC [6]. Although this can be problematic in the prehospital [45■] and the data supporting such titration are not strong, it seems reasonable to attempt titration of oxygen administration with close monitoring (Fig. 2).

Hypocapnia and hypercapnia are very common in patients resuscitated from cardiac arrest. Because hypocapnia has been associated with a worse outcome in some studies and may decrease CBF to a potentially acutely injured brain, ‘normal’ $paCO_2$ targets (i.e. 35–45 mmHg) seem reasonable. Ongoing studies may elucidate whether mild hypercapnia can be neuroprotective (e.g. ACTRN12612000690853).

CONCLUSION

The PCAS consists of haemodynamic derangements and reperfusion injuries that may be ameliorated with careful attention to monitoring blood pressure, cardiac function, oxygenation and ventilation. Simple interventions such as monitoring and treating

hypotension, identifying and addressing myocardial dysfunction, preventing over and underventilation and preventing hypoxia and hyperoxia may impact survival and neurologic outcomes.

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KEY POINTS

- Postarrest hypotension is common and associated with lower survival rate.
- Postarrest hypotension may be due to a ‘sepsis’-like syndrome and/or myocardial dysfunction, which clinicians should diagnose and treat with the goals of decreasing secondary brain injury.
- Hyperoxia and hypoxia may be harmful to the brain, so clinicians should target normoxia.
- Overventilation and underventilation impact cerebral blood flow, so clinicians should target normocapnia.

FIGURE 1.

An algorithm to evaluate and treat hypotension in the patient resuscitated from cardiac arrest after hospital admission. CVP, central venous pressure; ScvO₂, central venous saturation.

FIGURE 2.

A step-by-step algorithm to adjust oxygen and carbon dioxide levels in patients resuscitated from cardiac arrest after hospital admission. FiO_2 , fraction of inspired oxygen; MV_{ent} , minute ventilation; SpO_2 , peripheral capillary oxygen saturation.

Table 1

Fifth percentile blood pressures by age and sex for the 50% height percentile

Age (years)	5 percentile male	5 percentile female
1	71	72
2	73	74
3	75	75
4	77	77
5	78	78
6	80	80
7	82	81
8	84	82
9	85	83
10	86	85
11	88	88
12	89	90
13	92	91
14	94	92
15	96	92
16	97	93
17	99	94

<https://sites.google.com/a/channing.harvard.edu/bernardrosner/pediatric-blood-press>.