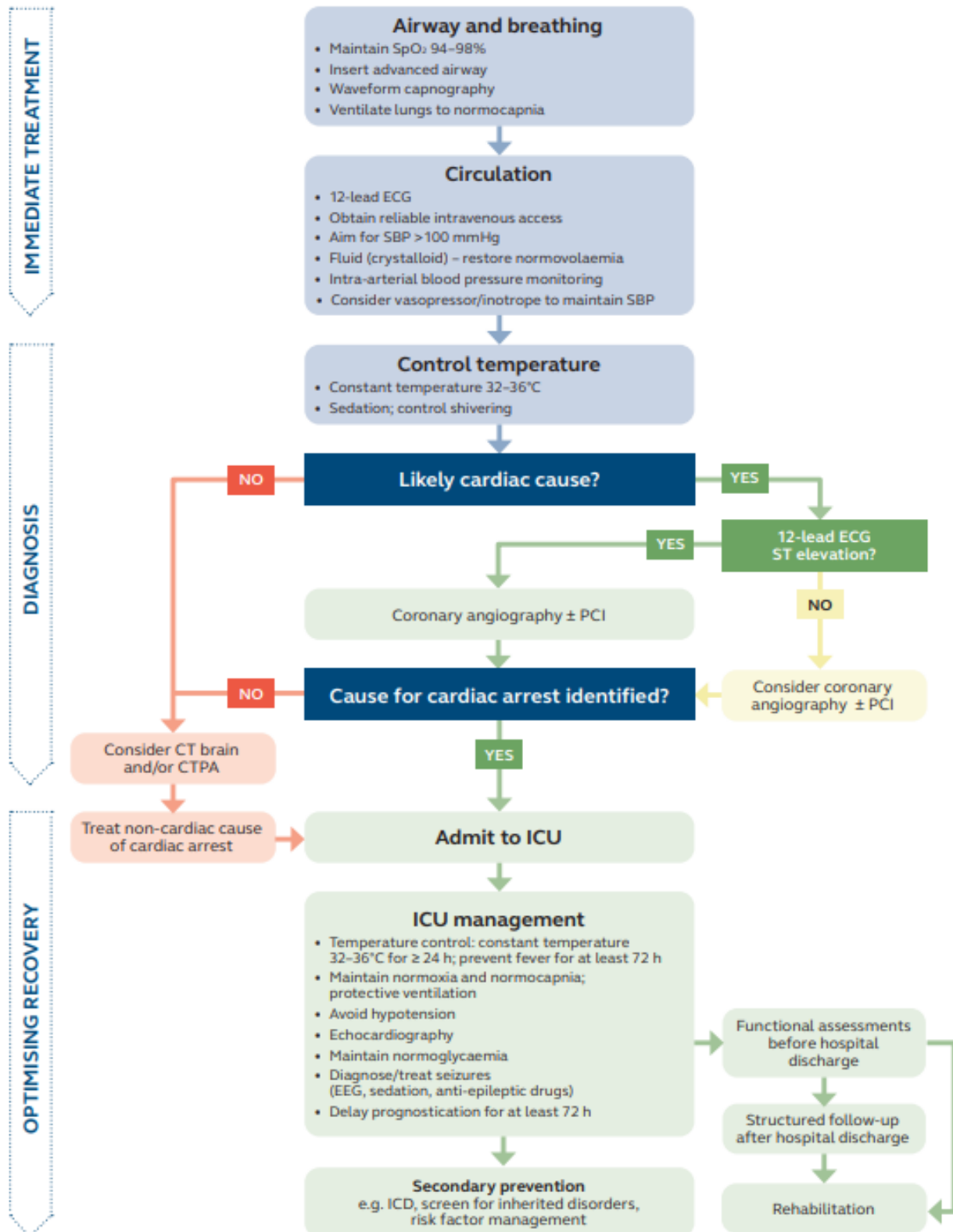


Post Cardiac Arrest Guidelines

AIM: To provide guidance on the management of comatose patients post cardiac arrest to minimise post cardiac arrest syndrome and secondary neurological insult
SCOPE: Adult general ICU patients in RSCH and PRH



1. INTRODUCTION

The aim of post cardiac arrest management is to minimise the effects of the post cardiac arrest syndrome and therefore maximise the chance of survival with a good neurological outcome. Post cardiac arrest syndrome is the pathophysiological process that follows the total body ischaemia during cardiac arrest and the subsequent reperfusion following return of spontaneous circulation (ROSC). It comprises of 4 factors:

- post-cardiac arrest brain injury
- post-cardiac arrest myocardial dysfunction
- systemic ischaemia and reperfusion response
- persistent precipitating pathology of the cardiac arrest

The post cardiac arrest syndrome manifests as vasodilation, endothelial injury, abnormalities of the microcirculation, activation of the coagulation pathways and contributes to the multi-organ failure often seen in these patients. The following ABCD approach rationalises the management of patients post cardiac arrest.

2. PROCESS

Airway, Breathing Circulation

Recommendation (Action)	Justification (Rationale)
Control Airway and Breathing	<p>Normal parameters should be maintained as hypoxia, hyperoxia and hypercarbia increase the likelihood of further cardiac arrest and secondary brain injury. Hypocarbia should also be avoided due to the subsequent cerebral vasoconstriction and decreased cerebral blood flow. Patients should be ventilated with protective lung strategies with tidal volumes of 6-8ml/kg as they develop a marked inflammatory response and are at risk of ARDS</p> <p>Suggested targets: PaO₂: 10-13 kPa SaO₂: 94-98 % PaCO₂: 4.5-6 kPa</p>

Recommendation (Action)	Justification (Rationale)
Coronary Reperfusion	<p>In patients with ST segment elevation (STE) or left bundle branch block (LBBB) on the post-ROSC electrocardiogram (ECG) more than 80% will have an acute coronary lesion. Immediate angiography and PCI when indicated should be performed in resuscitated out of hospital cardiac arrest (OHCA) patients whose initial ECG shows ST-elevation, even if they remain comatose and ventilated as part of an overall strategy to improve neurologically intact survival .The NICE Clinical Guideline 167 for the acute management of STEMI recommends:</p> <p>‘Do not use level of consciousness after cardiac arrest caused by suspected acute STEMI to determine whether a person is eligible for coronary angiography (with follow-on primary PCI if indicated)’.</p> <p>Acute coronary syndrome is a frequent cause OHCA. Therefore patients without an obvious cardiac cause on the ECG are worth discussing with Cardiology regarding the merits of angiography and PCI.</p>
Haemodynamics	<p>Post cardiac arrest myocardial dysfunction causes hypotension, low cardiac output and arrhythmias. Bedside echocardiography will help assess myocardial function. Due to vasodilation from the post cardiac arrest syndrome noradrenaline with or without an inotrope should be used following adequate fluid resuscitation. Avoid hypotension (MAP <65 mmHg). Treatment can be guided by adequate urine output and lactate measurements. Cardiac output monitoring should be considered in those patients requiring >0.2mcg/kg/min of noradrenaline. Intra aortic counter pulsation balloon pump (IABP) can be discussed with cardiology if there is resistant cardiogenic shock; other options include Impella™ or VA-ECMO for highly selected cases. Of note, IABP have been shown to have no mortality benefit at 30 days (IABP SHOCK II trial).</p>

Disability

Recommendation (Action)	Justification (Rationale)
Cerebral perfusion	Autoregulation of cerebral blood flow is impaired following cardiac arrest which means that cerebral perfusion varies with cerebral perfusion pressure. After ROSC mean arterial pressure should be maintained near to that of the patient's normal to maintain cerebral blood flow and minimise secondary brain damage.
Sedation	Patients need to be adequately sedation while they are being cooled and ventilated. Propofol and fentanyl should be used for sedation. See unit sedation guidelines. Patients undergoing TTM should be well sedated. If shivering occurs then counter warming the hands and feet can be attempted, but if it is persistent then neuromuscular blockade should be used in the form of a cisatracurium infusion.
Control of seizures	Generalised seizures and myoclonus are common following ROSC and seen in up to one third of patients. Clinical seizures and myoclonus may or may not be epileptic in origin and for persistent episodes EEG monitoring should be performed. Seizures will increase the metabolic rate and exacerbate the brain injury following cardiac arrest. Seizures should be managed with levetiracetam or sodium valproate in the first instance, in addition to sedative drugs. The choice of drug, doses and duration of treatment should be decided with the Neurologists where appropriate. Clonazepam and levetiracetam are useful for myoclonus. Routine seizure prophylaxis in post cardiac arrest patients is not recommended.

General ICU management

Recommendation (Action)	Justification (Rationale)
VTE Prophylaxis	Provide VTE prophylaxis according to usual practice.
Stress ulcer prophylaxis	Patients are at risk of peptic ulceration, and prophylactic agents are indicated. Enteral feeding is encouraged, although there may be benefit to limiting infusion rates in patients who are cooled to < 36 °C.
Control blood glucose	Target a blood glucose of between 7.8 and 10 mmol/L, using a variable rate insulin infusion. Hypoglycaemia may worsen neurological outcomes and should be avoided

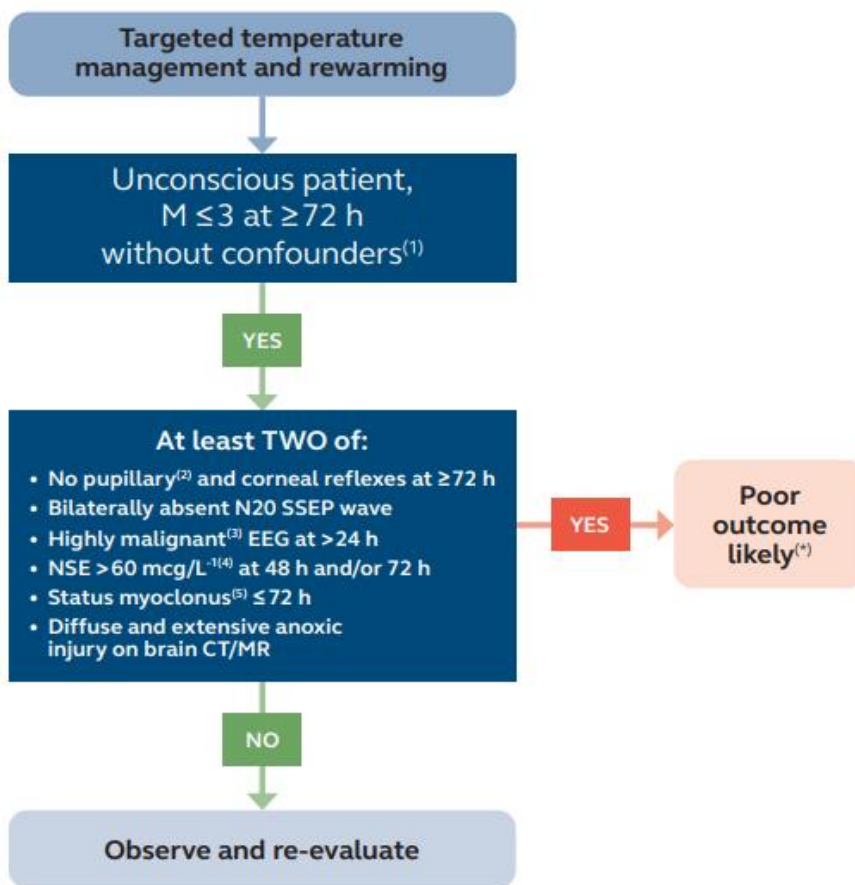
Temperature control

Recommendation (Action)	Justification (Rationale)
Rationale behind TTM	<p>Animal and human data indicate that mild induced hypothermia is neuroprotective and improves outcome after a period of global cerebral hypoxia. Hypothermia suppresses many of the pathways leading to delayed cell death and reduces the inflammatory response associated with the post-cardiac arrest syndrome.</p> <p>Randomised controlled trials demonstrated improved neurological outcome at hospital discharge and at six months in comatose patients after out-of-hospital VF cardiac arrest. Cooling was initiated within minutes to hours after ROSC and a temperature range of 32–34°C was maintained for 12–24 hours. In the TTM trial, 950 all-rhythm OHCA patients were randomised to 36 hours of temperature control at either 33°C or 36°C and were rewarmed slowly.</p> <p>There was no difference in mortality and detailed neurological outcome at 6 months was also similar. Importantly, patients in both arms of this trial had their temperature well controlled so that fever was prevented in both groups and provided strict normothermia (<37.5°C) after hypothermia until 72 h after ROSC.</p> <p>The optimal duration for TTM is unknown although it is currently most commonly used for 24 hours. Previous trials treated patients with 12–28 h of targeted temperature management. Observational trials found no difference in mortality or poor neurological outcome with 24 h compared with 72 h of hypothermia.</p>

Recommendation (Action)	Justification (Rationale)
<p>Temperature, duration and rhythm</p>	<p>The Advanced Life Support Task Force of the International Liaison Committee on Resuscitation (ILCOR) have made the following recommendations on TTM:</p> <ul style="list-style-type: none"> • Maintain a constant, target temperature between 32°C and 36°C for those patients in who remain unresponsive after ROSC • Maintain set temperature for at least 24 hours • Avoid fever (>37.7°C) for at least 72 hours after ROSC in patient who remain in coma / sedated
<p>How to control temperature</p>	<p>The practical application of TTM is divided into three phases: induction, maintenance and rewarming. External and/or internal cooling techniques can be used to initiate and maintain TTM.</p> <p>Temperature should be monitored using a rectal thermometer, confirmed with a second method.</p> <p>Methods of inducing and/or maintaining TTM include:</p> <ul style="list-style-type: none"> • Cooling blankets or pads (at PRH) • Thermoguard intravascular heat exchanger placed in the femoral vein (at RSCH). The Arctic Sun is also available at RSCH • If the above methods are unavailable it is possible to use simple ice packs, wet towels, cool bair hugger or cold fluids. These are inexpensive but may result in greater temperature fluctuations and do not enable controlled rewarming. <p>Rebound hyperthermia is associated with worse neurological outcome. Therefore, rewarming should be achieved slowly at 0.25–0.5°C of per hour.</p>

Recommendation (Action)	Justification (Rationale)
Physiological effects and complications of hypothermia	<ul style="list-style-type: none"> • Increases systemic vascular resistance and causes arrhythmias (usually bradycardia). The bradycardia caused by mild induced hypothermia may be beneficial as it reduces diastolic dysfunction. • Diuresis and electrolyte abnormalities such as hypophosphataemia, hypokalaemia, hypomagnesaemia and hypocalcaemia. • Decreases insulin sensitivity and insulin secretion causing hyperglycaemia. • Impairs coagulation and may increase bleeding. • Impairs the immune system and increases infection rates with an increased incidence of pneumonia. • Serum amylase concentration is commonly increased during hypothermia but the significance of this unclear. • Clearance of sedative drugs and neuromuscular blockers is reduced by up to 30% at a core temperature of 34°C.
Contraindications to hypothermia	<ul style="list-style-type: none"> • Severe systemic infection • Pre-existing medical coagulopathy • Severe haemodynamic instability

Prognostication



¹ Major confounders may include analgo-sedation, neuromuscular blockade, hypothermia, severe hypotension, hypoglycaemia, sepsis, and metabolic and respiratory derangements

² Use an automated pupillometer, when available, to assess pupillary light reflex

³ Suppressed background ± periodic discharges or burst-suppression, according to American Clinical Neurophysiology Society

⁴ Increasing NSE values between 24–48 h or 24/48 and 72 h further support a likely poor outcome

⁵ Defined as a continuous and generalised myoclonus persisting for 30 min or more

* Caution in case of discordant signs indicating a potentially good outcome

M = Motor component of GCS
NSE = Neuron Specific Enolase
SSEP = Somatosensory Evoked Potential

3. REFERENCES

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4 ONLINE RESOURCES

[Post-resuscitation care Guidelines | Resuscitation Council UK](#)

The use of this guideline is subject to professional judgement and accountability. This guideline has been prepared carefully and in good faith for use within the Department of Critical Care at Brighton and Sussex University Hospitals. The decision to implement this guideline is at the discretion of the on-call critical care consultant in conjunction with appropriate critical care medical/ nursing staff.