**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.

### Immediate Management

- Control airway, breathing and circulation
- Maintain normocapnia and normotension
- Control temperature to 32-36°C & prevent shivering

### Make diagnosis

**Likely cardiac cause**

- ST elevation on 12 lead, LBB or history suggestive of ACS
- Discuss with cardiologist on bleep 8850 need for angiography +/- PCI

**Likely non cardiac cause**

- Perform CT brain +/- CTPA

### Cardiac cause for arrest identified?

- Yes
- No

### Admit to ICU

- Temperature control 32-36°C for 24 hours as per ICU Consultant; prevent fever > 37.5°C for 72 hours. Use Thermoguard +/- external measures. Rewarm after 24 hours at 0.5°C/hr until 37°C
- Sedate and control shivering; cisatricurium infusion if required
- Maintain normoxia (PaO₂ > 8kPa) and normocapnia (PaCO₂ 4.5kPa), lung protection Vt 6-8mls/kg
- Optimise haemodynamics: urine output > 20mls/hr, lactate <2, consider echocardiography & CO monitoring
- Maintain blood glucose < 10 mmol/L
- Consider antibiotic if aspiration likely
- Treat seizures: levetiracetam, phenytoin, or benzodiazepines. Clonazepam for myoclonus
- Delay prognostication for at least 72 hours if cooled to <36°C
- Start Patient Diary

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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

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<tr>
<th>Recommendation (Action)</th>
<th>Justification (Rationale)</th>
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<tr>
<td>Control Airway and Breathing</td>
<td>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</td>
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| **Coronary Reperfusion** | 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:  

‘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)’.  

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. |
| **Haemodynamics** | 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 dobutamine should be used following adequate fluid resuscitation. Treatment can be guided by adequate urine output and lactate measurements. Cardiac output monitoring should be considered in those patients requiring >0.2mg/kg/min of noradrenaline. Intra aortic counter pulsation balloon pump (IABP) can be discussed with cardiology if there is resistant cardiogenic shock. However IABP have been shown to have no mortality benefit at 30 days (IABP SHOCK II trial). |
## Disability

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<td>Cerebral perfusion</td>
<td>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.</td>
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<td>Sedation</td>
<td>Patients need to be adequately sedated 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 cisatricurium infusion.</td>
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<tr>
<td>Control of seizures</td>
<td>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, phenytoin, sodium valproate or benzodiazepines. The 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.</td>
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| Rationale behind TTM | 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.

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.

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.

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. |
## Temperature, duration and rhythm

The Advanced Life Support Task Force of the International Liaison Committee on Resuscitation (ILCOR) have made the following recommendations on TTM:

- Maintain a constant, target temperature between 32°C and 36°C for those patients in whom temperature control is used.
- TTM is recommended for adults after OHCA with an initial shockable rhythm who remain unresponsive after ROSC.
- TTM is suggested for adults after OHCA with an initial nonshockable rhythm who remain unresponsive after ROSC.
- TTM is suggested for adults after IHCA with any initial rhythm who remain unresponsive after ROSC.
- If TTM is used, it is suggested that the duration is at least 24 h.

## How to control temperature

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.

Temperature should be monitored using a rectal thermometer, confirmed with a second method.

Methods of inducing and/or maintaining TTM include:

- Cooling blankets or pads (at PRH)
- Thermoguard intravascular heat exchanger placed in the femoral vein (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.

Rebound hyperthermia is associated with worse neurological outcome. Therefore, rewarming should be achieved slowly at 0.25–0.5°C of per hour.
| Physiological effects and complications of hypothermia | • 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, therefore use intravenous antibiotics for three days as part of SDD.  

• 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 | • Severe systemic infection  

• Pre-existing medical coagulopathy  

• Severe haemodynamic instability |
Prognostication

Cardiac arrest

Controlled temperature

Rewarming

Exclude confounders, particularly residual sedation

Unconscious patient, M=1-2 at ≥ 72 h after ROSC

One or both of the following:
- No pupillary and corneal reflexes
- Bilaterally absent N20 SSEP wave (1)

No

Wait at least 24 h

Yes

Two or more of the following:
- Status myoclonus ≤ 48 h after ROSC
- High NSE levels (2)
- Unreactive burst-suppression or status epileptics on EEG
- Diffuse anoxic injury on brain CT/MRI (2)

Indeterminate outcome
Observe and re-evaluate

Poor outcome likely

Use multimodal prognostication whenever possible

(1) At ≥ 24 h after ROSC in patients not treated with targeted temperature
(2) See text for details.
3. GLOSSARY

M = Motor component of GCS
NSE = Neuron Specific Enolase “Brain troponin”
SSEP = Somatosensory Evoked Potentials
FPR = False Positive Rate

4. REFERENCES


Lee BK, Lee SJ, Jeung KW, Lee HY, Heo T, Min YI. Outcome and adverse events with 72-hour cooling at 32 degrees C as compared to 24-hour cooling at 33 degrees C in comatose asphyxial arrest survivors. Am J Emerg Med 2014;32:297-301


4 ONLINE RESOURCES

https://www.resus.org.uk/resuscitation-guidelines/post-resuscitation-care