North Wales Critical Care Network

POST CARDIAC ARREST CARE BUNDLE (for Critical Care Patients)
Introduction
Outcome in patients who present to the Emergency Department (ED) with return of spontaneous circulation after cardiac arrest remains poor. Overall survival to discharge after out-of-hospital cardiac arrest is low. Of those admitted to ICU approximately 25–40% will survive to discharge with conventional therapy. ICNARC data suggests that under a third (28.6%) ICU admissions for post resuscitation care survived to hospital discharge.

In recent years, it has become evident that the pathological events occurring at the time of cerebral hypoxia are delayed into the post-arrest period, exacerbating cell damage and neuronal death. There is growing evidence that interventional post-cardiac arrest care can lower the death rate and improve functional outcome for such patients.

Post Cardiac Arrest Care Bundle
Background Information
Interventions aimed at improving outcome after cardiac arrest have shown that mild induced therapeutic hypothermia (TH) is beneficial in adults. Recently, in March 2011 The National Institute for Clinical Excellence issued an Interventional Procedure guidance ‘Therapeutic hypothermia following cardiac arrest’ stating that current evidence on the safety and efficacy of TH following cardiac arrest is adequate to support its use.

In 2008 the Intensive Care Society set out ‘Standards for the Management of Patients after Cardiac Arrest’. Whilst this guidance includes therapeutic hypothermia it also sets out treatment recommendations in relation to airway and ventilation, circulation as well as disability i.e. optimising neurological recovery.

In order to guide for Post Cardiac Arrest care for patients in the North Wales Critical Care Units a care bundle has been developed. Post resuscitation commences at the location where return of spontaneous circulation is achieved, therefore this post cardiac arrest bundle should be commenced for all critical care patients at this point beit in the ED, catheter lab or Critical Care unit etc.

Post Cardiac Arrest Care Bundle
The Post Cardiac Arrest care bundle aims to optimise care for the individual patient by addressing the interventions known to potentially improve patient outcomes. Use of therapeutic hypothermia now includes...
comatose survivors of cardiac arrest associated initially with non-shockable rhythms as well as shockable rhythms\textsuperscript{6,7} and may also be of benefit for patients following cardiac arrest in hospital as well as out of hospital\textsuperscript{8}.

The bundle elements are:
\begin{itemize}
  \item Controlling ventilation
  \item Early coronary reperfusion (if appropriate)
  \item Haemodynamic optimisation
  \item Controlling temperature
  \item Controlling blood glucose
  \item Treatment of seizures
\end{itemize}

Compliant with care bundle philosophy, the aim is to implement all elements where indicated, appropriate and safe to do so.

**Bundle Element 1**

**Controlling ventilation**

There is no data supporting precise indications for intubation, ventilation and sedation after cardiac arrest. For patients who do not regain consciousness quickly and who are cerebrally obtunded they will normally require intubation, ventilation and sedation\textsuperscript{6}. The duration of sedation and ventilation may well be influenced by the use of therapeutic hypothermia (see Element 4); hypothermia will prolong the clearance of sedatives and neuromuscular blockers\textsuperscript{8} but short acting drugs will enable early neurological assessment\textsuperscript{6}.

Whilst there is also no data to support targeting a specific arterial Pa\textsubscript{CO\textsubscript{2}} after resuscitation hyperventilation may produce potentially harmful cerebral ischaemia\textsuperscript{9} Adequate oxygen delivery is essential\textsuperscript{6} however hyperoxia may exacerbate neuronal damage\textsuperscript{10,11}; the aim therefore is for normal oxygenation and normocapnia\textsuperscript{4,6}.

**Bundle Element 2**

**Early coronary reperfusion (if appropriate)**

Acute coronary occlusion with myocardial infarction is a common cause of sudden cardiac death and coronary artery disease accounts for two thirds of sudden cardiac deaths\textsuperscript{12-14}. Strategies aimed at restoring myocardial perfusion are an important part of the management of these patients. Restoring coronary blood flow and myocardial perfusion either by thrombolysis or percutaneous coronary intervention (PCI) has been demonstrated to improve outcomes in patients. The benefits of early post-cardiac arrest coronary angiography with subsequent PCI are well documented\textsuperscript{15-17}.

Several studies indicate that the combination of therapeutic hypothermia and PCI is feasible and safe after cardiac arrest caused by acute myocardial infarction\textsuperscript{3,21-24}. The patient can be brought to the angiography/catheter laboratory while cooling is commenced or continued.
Shorter intervals to reperfusion increase myocardial salvage, whereas delays to reperfusion increase morbidity and mortality\textsuperscript{25}. If thrombolysis is chosen as the reperfusion strategy, it should be started as soon as possible.

**Bundle Element 3**  
**Haemodynamic optimisation**

Post-cardiac arrest myocardial dysfunction causes haemodynamic instability, which manifests as hypotension, low cardiac index and arrhythmias\textsuperscript{26}. This post resuscitation myocardial dysfunction is usually transient and often reverses within 24-48 hours\textsuperscript{27}. If treatment with appropriate fluids and vasoactive drugs is insufficient to support the circulation, consider insertion of an intra-aortic balloon pump,\textsuperscript{3,21} where available, until myocardial function has recovered.

In the absence of definitive data, target the mean arterial blood pressure to achieve an adequate urine output (1ml/kg/hr)\textsuperscript{4,30} and normal or decreasing plasma lactate values, taking into consideration the patient’s usual blood pressure (if known), the cause of the arrest and the severity of any myocardial dysfunction\textsuperscript{30}.

**Bundle Element 4**  
**Controlling temperature**

A period of hyperthermia is common in the first 48 hours after cardiac arrest\textsuperscript{31-33}. Increased brain temperature contributes to brain ischaemia; poor neurological outcomes are associated with pyrexia\textsuperscript{34,35} and a temperature >39\degree C in the first 72 hours has been linked to the development of brain death\textsuperscript{32}.

Evidence indicates that, in the comatosed patient, lowering brain temperature as in therapeutic hypothermia can be neuroprotective and improves outcomes of patients who have suffered hypoxic ischaemia\textsuperscript{21,22,36-43}. Data indicates that commencing cooling as soon as possible produces better outcomes\textsuperscript{44} and that this should be maintained for adequate duration i.e. \~24 hours\textsuperscript{44-51} (See Appendix 1 for guidance).

Shivering associated with therapeutic hypothermia may require the use of neuromuscular blocking drugs.

**Bundle Element 5**  
**Controlling blood glucose**

Hyperglycaemia is common after cardiac arrest and is associated with a worse outcome\textsuperscript{2,4,36,52-57}.

Evidence for tight glycaemic control using insulin has not been sustained and in fact, because of the increased risk of hypoglycaemia, tight glycaemic control may worsen outcomes\textsuperscript{58-61}.

The consensus of evidence at present is that blood sugar after cardiac arrest should \textless{}10mmol/L\textsuperscript{62}. 

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North Wales  
Critical Care Network  
[Approved Sept 2011]
**Bundle Element 6**  
**Treatment of seizures**
Seizures or myoclonus or both occur in 5% to 15% of adult patients post resuscitation and 10% to 40% of those who remain comatose. Seizures increase cerebral metabolism by up to three-fold and may cause cerebral injury. There are no studies that address directly the use of prophylactic anticonvulsant drugs after cardiac arrest in adults.

**Summary**
As well as optimising care of the Post Cardiac Arrest patient this care bundle should facilitate audit of process. Although compliance is expected, especially where deviations from this agreed practice cannot be justified, the bundle is not designed to replace clinician judgment.
## Quick Guide for Post Cardiac Arrest Care Bundle – For Adults in Critical Care

<table>
<thead>
<tr>
<th>Bundle Element</th>
<th>Aims</th>
<th>Rationale</th>
<th>Exclusion</th>
<th>Compliance Audit Point</th>
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<tbody>
<tr>
<td><strong>Element 1: Control of ventilation</strong></td>
<td>Adequate ventilation  - Intubation, sedation and controlled ventilation where obtunded cerebral function  - Normal oxygenation (SaO$_2$ 94-98%)  - Normocapnia (PaCO$_2$ 4.5-5.0kPa) using ETCO$_2$ monitoring  - Short acting sedatives (to enable neuro assessment)  - Avoid paralytics if possible (boluses may be given for shivering where necessary)</td>
<td>Not all post cardiac arrest patients require tracheal intubation and ventilation but should be given oxygen via a facemask.  Too much oxygen during the initial stages of reperfusion can exacerbate neuronal damage.  Cerebral vasoconstriction caused by hyperventilation may produce potentially harmful cerebral ischaemia.  Therapeutic hypothermia (TH) prolongs the clearance of sedatives and neuromuscular blockers</td>
<td>Terminal care</td>
<td>Is the patient adequately ventilated? Or an attempt to achieve?</td>
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<td><strong>Element 2: Early coronary reperfusion (where appropriate)</strong></td>
<td>Restore myocardial perfusion  - Early Percutaneous Coronary Intervention (PCI) or Early Thrombolysis  - (Hypothermia (where appropriate) should be used in conjunction with these interventions – see element 4)</td>
<td>Restoration of coronary perfusion is a priority. If there is evidence of coronary occlusion, consider immediate revascularisation by thrombolysis or percutaneous coronary intervention. If the facilities are available, primary PCI is the preferred technique for revascularisation.</td>
<td>No evidence of coronary occlusion</td>
<td>Has there been early coronary reperfusion (where appropriate)?</td>
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<td><strong>Element 3: Haemodynamic optimisation</strong></td>
<td>Haemodynamic support and optimisation  - Maintain MAP at patient’s normal level and aim to achieve an adequate urine output (1ml/kg/hr)  - This may require fluids, diuretics, vasodilators, inotropes, IABP (where available)  - Administer noradrenaline to maintain MAP in the presence of a significant inflammatory response.  - Normalise electrolytes (K$^+$, Mg, PO$_4^{3-}$)  - Maintain serum potassium between 4.0 and 4.5 mmol/L.</td>
<td>Good outcomes have been achieved after out-of-hospital cardiac arrest using a MAP target from as low as 65-75 mm Hg to as high as 90-100 mm Hg.  Loss of normal cerebral autoregulation leaves cerebral perfusion dependent on MAP. Under these circumstances, hypotension will compromise cerebral blood flow severely and will compound any neurological injury.  Hypokalaemia may predispose to ventricular arrhythmias.</td>
<td>Terminal care</td>
<td>Has there been haemodynamic optimisation? Or an attempt to achieve?</td>
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<td><strong>Element 4: Controlling temperature</strong></td>
<td>Control temperature  - Unless contraindicated, commence Therapeutic Hypothermia (TH); see Appendix 1 for guidance  - Do not delay, preferably commence pre-ICU  - If contraindicated, maintain temperature below 37°C for 72hours post arrest using antipyretics and active cooling as required.</td>
<td>The risk of a poor neurological outcome increases for each degree of body temperature &gt;37°C.  In the comatose patient, lowering brain temperature as in TH can be neuroprotective and improves outcome of patients who have suffered hypoxic ischaemia.  Commencing cooling as soon as possible produces better outcomes; this should be maintained for adequate duration (~24hours)</td>
<td>Non sedated patients and TH Terminal care</td>
<td>Has temperature been controlled?</td>
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<td><strong>Element 5: Controlling glucose</strong></td>
<td><strong>Element 6: Treatment of seizures</strong></td>
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<td>Normalised blood glucose</td>
<td>Prompt treatment of seizures</td>
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<td>• Maintain blood sugar &lt;10mmol/L</td>
<td>• Maintenance therapy should be</td>
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<td>o Avoid hypo/hyperglycaemia at</td>
<td>started after the first event</td>
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<td>all costs</td>
<td>once potential precipitating causes</td>
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<td>• Treat hyperglycaemia with</td>
<td>(e.g. intracranial haemorrhage,</td>
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<td>insulin as per protocol</td>
<td>electrolyte imbalance, etc) are</td>
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<td>• Monitor blood glucose</td>
<td>excluded</td>
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<td>frequently especially during</td>
<td>• If continuous infusions of</td>
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<td>cooling and rewarming in TH</td>
<td>neuromuscular blocking drugs are</td>
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<td>necessary to control shivering,</td>
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<td>consider the use of continuous</td>
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<td>EEG monitoring (where available).</td>
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<td>There is a strong association</td>
<td>Seizures and/or myoclonus occur in</td>
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<td>between high blood glucose after</td>
<td>5%-15% of adult patients post</td>
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<td>resuscitation from cardiac arrest</td>
<td>arrest, and in 10-40% of those who</td>
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<td>and poor neurological outcome.</td>
<td>remain comatose.</td>
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<td>A recent large, international,</td>
<td>Seizures increase cerebral</td>
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<td>randomized trial found that</td>
<td>metabolism by up to threefold.</td>
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<td>intensive glucose control</td>
<td>Prolonged seizure activity may</td>
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<td>increased mortality among adults</td>
<td>cause cerebral injury, and should</td>
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<td>in the ICU: a blood glucose</td>
<td>be treated promptly and effectively</td>
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<td>target of 10 mmol/L or less</td>
<td>with benzodiazepines, phenytoin,</td>
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<td>resulted in lower mortality than</td>
<td>sodium valproate, propofol or a</td>
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<td>did a target of 4.5 – 6 mmol/L.</td>
<td>barbiturate. Clonazepam is the</td>
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<td>drug of choice for the treatment of</td>
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<td>myoclonus.</td>
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<td>Terminal care</td>
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<td>Has blood glucose been controlled?</td>
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<td>Possibly Terminal care</td>
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<td>Have seizures been treated</td>
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<td>promptly?</td>
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Appendix 1  Therapeutic Hypothermia for ICU patient following cardiac arrest
Dr Peyrasse, Dr Evans.

**Inclusion criteria**
Successfully resuscitated from cardiac arrest (spontaneous circulation)
AND GCS <10 (with no other cause for coma)
AND is suitable for ITU admission (as decided by ITU consultant)

**Exclusion criteria**
GCS >10 or responding to command
Patient unsuitable for ITU treatment
Other cause for coma

**Relative contraindications of TH**
Primary Coagulopathy.
Life threatening arrhythmias, severe cardiogenic shock, sepsis, trauma, pregnancy, haemorrhage.

**Initial management:**
Active cooling to 32 – 34°C in 4 hours
Induction with infusion of NS or Hartmann’s solution at 4°C followed by Cooling Machine
Surface cooling at 32°C, Consider more fluid infusion at 4°C (slowly) Paralyse if shivering (check sedation first), using boluses.

**Maintenance:**
Maintain temperature between 32°C and 34°C for 24 hours
PaO2 >11 kPa, PaCO2 4.5 to 5.0 Systolic to maintain U.O. BM control <10 mmol/l Apply Post Cardiac Arrest and Ventilator Bundles Check U/E and coagulation 12 hourly at least. Continue CVS and temperature monitoring

**If temp < 32°C**
Allow passive re-warming to 32-34°C CVS monitoring for arrhythmia

**Consider early re-warming if**
Significant arrhythmias (uncontrolled AF, VF or VT) CVS instability Coagulopathy or bleeding; Significant Coagulopathy

**After 24 hours**
Stop surface cooling Allow slow passive re-warming (Max 0.5°C per hour) Stop paralysis/sedation when temp >36°C Check U&E in particular K Monitor BMs (very) closely.

**General considerations**
- Don’t let core temperature drop below 30°C
- Thrombolysis can be considered
- Lactate tend to rise to around 5 – 6 during cooling (as well as liver enzymes and amylase)
- Clearance of most drugs will be reduced
- During cooling, Mg, K, Phosphate levels will drop and may need to be replaced (particularly Mg)
- During re-warming, K may rise: hence slow warming to allow kidney to excrete it
- Cooling induces Insulin resistance (risk of hyperglycaemia), warming may cause hypo glycaemia
- Avoid skin damage/bed sore (increased risk)
- Monitor / treat infection (increased risk and sign will be masked)
- Reduce enteral feeding (by up to 40%)
- Avoid hypovolaemia and hypotension (cooling causes cold diuresis)
- Patients tend to be bradycardic
- Adjust ventilation: cooling causes reduced O₂ consumption and CO₂ production
References


   http://guidance.nice.org.uk/IPG386

   http://www.ics.ac.uk/intensive_care_professional/standards_and_guidelines/standards_for_the_management_of_patients_after_cardiac_arrest_2008


critically ill adults: a meta-analysis. *JAMA* 300:933-44.


