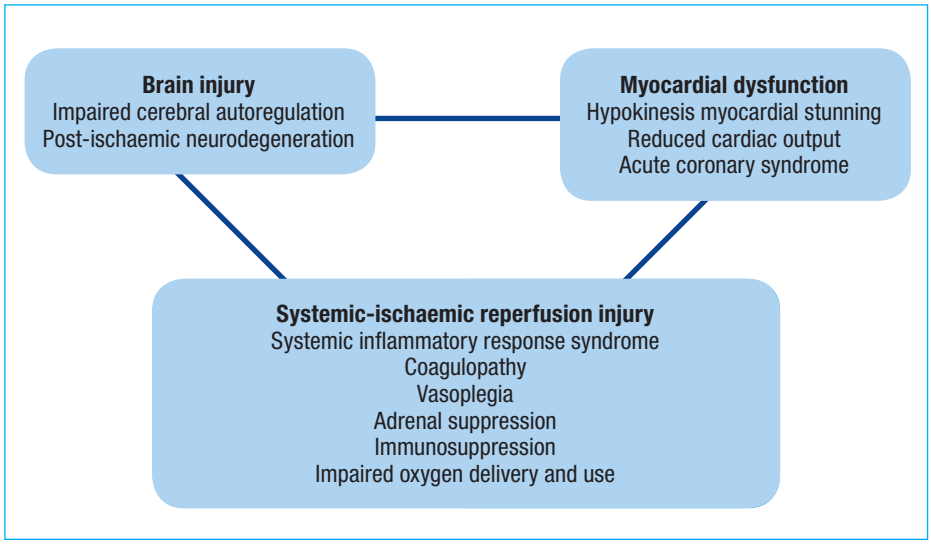
Hospital management of the post-cardiac arrest patient: priorities and challenges

The ambulance services treat approximately 30 000 patients with out of hospital cardiac arrests each year (Out of Hospital Cardiac Arrest Outcomes, 2016). Of these patients, return of spontaneous circulation by the point of arrival to hospital is achieved in 25%, but fewer than 10% of all patients survive to hospital discharge. In-hospital cardiac arrest occurs in over 15 000 patients per year and has a more favourable outcome (National Cardiac Arrest Audit, 2017). Return of spontaneous circulation is achieved in up to 50% of these patients, but survival to hospital discharge remains at around 20%. Survivors to hospital discharge therefore represent a minority, but individuals report a good quality of life with most returning to work (Elliott et al, 2011; Lilja et al, 2018).

During a cardiac arrest a global hypoxic injury occurs, to which the brain and myocardium are most sensitive. With return of spontaneous circulation may also come secondary injury as a result of generalized systemic ischaemic–reperfusion injury, delivering further insult to the most sensitive tissues (Nolan et al, 2008). This secondary injury is the post-cardiac arrest syndrome and is usually complete within the first 72 hours (*Figure 1*). Furthermore, the underlying precipitant of the cardiac arrest, which can include acute coronary syndrome, respiratory failure, stroke, thromboembolic disease, overdose or poisoning, sepsis or hypovolaemia among others, may be an ongoing source of hypoperfusion if not resolved at the point of return of spontaneous circulation. The significance of this secondary injury may in part explain the large discrepancy between the numbers of patients surviving to return of spontaneous circulation and those surviving to hospital discharge.

Figure 1. The post-cardiac arrest syndrome: pathophysiological mechanisms. From Nolan et al (2008).



Management of the post-cardiac arrest patient focuses on treating the cause of the cardiac arrest and minimizing early secondary injury, so this article covers the first 72 hours of care post-return of spontaneous circulation. While management of the post-cardiac arrest patient is not infrequent for the hospital doctor, resuscitation training tends to focus on the use of the advanced life support algorithm to achieve return of spontaneous circulation in cardiac arrest (Soar et al, 2015). This article systematically addresses the early priorities and challenges which a junior doctor should focus on when managing patients post-cardiac arrest (*Table 1*).

Airway

Anaesthetic or intensive care registrars form part of hospital cardiac arrest teams. They attend in-hospital cardiac arrests and will receive out of hospital cardiac arrests in either the emergency department or percutaneous coronary intervention centre. They are responsible for securing a definitive airway, in the form of tracheal intubation, when required. After return of spontaneous circulation, tracheal intubation is indicated in the event of ongoing coma (a Glasgow coma scale value of <8 is commonly used), physiological instability, if further imaging or procedures are indicated, and for airway protection. In addition to other precipitants of cardiac arrest, usual indications for tracheal intubation and mechanical ventilation remain applicable in this setting. The position of an endotracheal tube should be confirmed, with a chest X-ray, promptly post-intubation (Nolan et al, 2015).

Breathing

Pulmonary complications of cardiopulmonary resuscitation include lung contusions, pneumothoraces and rib fractures following chest compressions. These should be identified by performing a chest X-ray early in the post-return of spontaneous circulation phase. A degree of

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Table 1. Target physiological parameters and key investigations in post-cardiac arrest management

Ventilation and oxygenation	Without ventilator	SaO_2 94–98% (88–92% if hypercapnic respiratory failure)	
	With ventilator	Protective mechanical ventilation: tidal volume 6–8 ml/kg/ideal body weight + high positive end expiratory pressure	Normoxia: PaO_2 >8 kPa and PaO_2 <30 kPa or SaO_2 94–98%
			Normocapnia: $PaCO_2$ 4.5–6.0 kPa
Circulation	Heart rate 60–100 bpm		
	Blood pressure: systolic blood pressure >90 mmHg or mean arterial pressure >65 mmHg		
	Electrolytes: K^+ 4.5–5.0 mmol/litre, ionized Ca^{2+} >1.2 mmol/litre, Mg^{2+} >1.2 mmol/litre		
	Targeted temperature management 33–36°C		
	Blood glucose 4–10 mmol/litre		
Investigations	Basic bloods (full blood count, urea and electrolytes, liver function tests, C-reactive protein), electrocardiogram, chest X-ray, echocardiography, ± coronary angiography, ± computed tomography pulmonary angiography ± computed tomography head		

$PaCO_2$ = partial pressure of carbon dioxide; PaO_2 = partial pressure of oxygen; SaO_2 = oxygen saturation

aspiration is to some extent inevitable in view of the impaired Glasgow coma scale during the arrest period but prophylactic antibiotic treatment is not recommended (Nolan et al, 2015). Hospital-acquired pneumonia, atelectasis and acute respiratory distress syndrome are also prevalent in this population, particularly among those who are mechanically ventilated (Sutherasan et al, 2015b).

Breathing: without a ventilator

Self-ventilating patients should receive supplemental oxygen if oxygen saturation is below 94%. Supplemental oxygen should be provided via non-invasive devices and titrated to maintain arterial oxygen saturations between 94 and 98%. In patients with hypercapnic respiratory failure target saturations may need to be adjusted to 88–92% (O'Driscoll et al, 2017). Peripheral vasoconstriction is common post-return of spontaneous circulation and as such caution must be applied to titrating oxygen based purely on pulse oximetry recordings. Arterial blood gas analysis, via an arterial line, is more reliable and the preferred means of titrating supplemental oxygen therapy in the initial post-return of spontaneous circulation period (Nolan et al, 2015).

Patients should initially be managed in high dependency environments and closely monitored for signs of respiratory deterioration. Non-invasive or high-flow oxygen ventilatory support may be a bridge, but should not delay invasive ventilation where it is indicated.

Breathing: with a ventilator

Injudicious invasive positive pressure ventilation may result in a host of pulmonary and systemic complications in any setting. A protective mechanical ventilation strategy, which involves using lower tidal volume (6–8 ml/kg based on ideal body weight), peak pressure limitation (≤30 cmH_2O) and positive end-expiratory pressure titration, is the preferred means of ventilating patients post-cardiac arrest and has been associated with a decrease in pulmonary complications (Sutherasan et al, 2015b). Adjustments to ventilation in view of existing lung pathology, notably restrictive and obstructive lung disease, may be required.

Targets: normoxia and normocapnia

Adequate oxygenation is the primary concern in the ventilation of the post-return of spontaneous circulation patient. Arterial hypoxia (partial pressure of oxygen (PaO_2) <8 kPa) is associated with an increased risk of re-arrest and neuronal injury, because of impaired delivery of oxygen to the myocardium and brain respectively. Arterial hyperoxia (PaO_2 >30 kPa) can lead to vasoconstriction and is therefore associated with worse neurological outcomes (Llitjos et al, 2016). Normoxia is therefore the recommendation and avoids the harmful effects of both. This explains the strategy of oxygen therapy recommended in patients breathing both with or without a ventilator. Where possible, fraction of inspired oxygen is maintained below 0.6 (Aggarwal and Brower, 2014).

Carbon dioxide control is generally a secondary priority, but normocapnia (partial pressure of carbon dioxide ($PaCO_2$) 4.5–6 kPa) is the recommendation for the partial pressure of carbon dioxide. Hypercapnia has not been associated with a particular outcome and may be accepted to avoid barotrauma. Hypocapnia is associated with a risk of cerebral vasoconstriction but may be useful in the short-term control of cerebral oedema (Callaway et al, 2015).

Overall the recommended targets of both normoxia and normocapnia may need to be sacrificed, in certain circumstances, to prevent greater harm, in the form of further secondary injury to the brain or lungs. Evidence characterizing the optimal settings for mechanical ventilation, target PaO_2 and $PaCO_2$ in patients post-cardiac arrest is still lacking (Sutherasan et al, 2015a).

Circulation

Identification and reversal of the cause of the cardiac arrest should be an early priority in care post-return of spontaneous circulation. These can be classified into cardiac and non-cardiac causes.

Cardiac causes

Over 60% of all cardiac arrests (out of hospital and in-hospital) are attributable to coronary artery disease. Acute coronary syndrome has been identified as the cause in up to 50% of patients undergoing coronary angiography and percutaneous coronary intervention immediately post-return of spontaneous circulation (Wallmuller et al, 2012; Patterson et al, 2017).

> ...immediate coronary angiography and percutaneous coronary intervention have been associated with reduced risk of re-arrest and myocardial dysfunction

Early reperfusion can treat the underlying precipitant, promote early haemodynamic stability and reduce the propensity for arrhythmogenic conditions. As such it can protect against an additional primary hypoxic injury or further secondary hypoxaemic injury. The evidence seems to support these assertions: immediate coronary angiography and percutaneous coronary intervention have been associated with reduced risk of re-arrest and myocardial dysfunction (Patterson et al, 2017). Early coronary angiography is also an independent predictor for survival to hospital discharge and neurological outcome irrespective of initial electrocardiogram findings.

Ischaemic causes

Working in a percutaneous coronary intervention centre: Guidelines currently recommend that out of hospital cardiac arrest patients, with ST elevation on electrocardiogram, should be immediately transferred for coronary angiography and percutaneous coronary intervention (Nolan et al, 2015). There is an increasing focus within the NHS to place emergency specialist care into regional centres. Ambulance services increasingly take out of hospital cardiac arrest patients with ST elevation directly to a cardiac arrest centre, so junior doctors may not begin managing these patients until they are transferred to a high dependency unit or intensive care unit setting post-percutaneous coronary intervention. If in-hospital cardiac arrests with a suspected ischaemic cause occur within a percutaneous coronary intervention centre, early coronary angiography and percutaneous coronary intervention should be arranged without significant delay following urgent cardiology review.

Working in a centre which does not perform percutaneous coronary intervention: Managing patients with a suspected ischaemic cause of cardiac arrest in a hospital without primary percutaneous coronary intervention facilities is more of a challenge. It is not routine for out of hospital cardiac arrest patients without ST elevation to be transferred to primary percutaneous coronary intervention centres and most are transferred to emergency departments. Furthermore, a ST segment elevation myocardial infarction is identified in over 50% of patients who have in-hospital cardiac arrests, with an underlying cardiac aetiology (Wallmuller et al, 2012). Urgent review by cardiology, echocardiography and early discussion with primary percutaneous coronary intervention centres are all priorities. Thrombolysis is an alternative option if a patient can not be transferred for coronary angiography and percutaneous coronary intervention (Nolan et al, 2015).

Referral for cardiac surgery: A single centre observational study has demonstrated that coronary artery bypass surgery performed within 48 hours of return of spontaneous circulation was associated with improved survival to discharge and neurological outcome (Grothusen et al, 2017). Early discussion with cardiac surgery centres should therefore be considered for a small subset of patients with coronary artery disease, in whom primary percutaneous coronary intervention is either not appropriate or fails.

Non-ischaemic causes

Other cardiac causes of arrest which are likely to have required control before return of spontaneous circulation include cardiac tamponade and electrolyte disturbances. Structural heart disease, e.g. cardiomyopathies, valvular heart disease or aortic dissection, may precipitate unstable rhythms leading to a cardiac arrest. Early echocardiography may reveal structural, muscle or valve disease, and timely referral to a tertiary cardiac centre will allow for definitive management (Nolan et al, 2015).

Non-cardiac causes

Computed tomography scans of the head and chest, including computed tomography pulmonary angiography, can readily identify neurological and respiratory causes of cardiac arrest, notably stroke, traumatic brain injury, pulmonary embolus and pneumonia. In the absence of clear evidence to suggest a neurological or respiratory cause, coronary angiography should be the first-line management (Nolan et al, 2015).

Haemodynamic targets

Post-cardiac arrest myocardial instability may occur as a result of 'myocardial stunning', defined as temporary cardiac muscle dysfunction which occurs secondary to hypoxic injury. There may be an associated fall in cardiac output. Clinical manifestations include hypotension, dysrhythmias and cardiogenic shock (Nolan et al, 2008).

Blood pressure

It is recommended that hypotension (minimum systolic blood pressure of <90 mmHg or mean arterial pressure of <65 mmHg) should be avoided in all patients post-return of spontaneous circulation (Callaway et al, 2015). However, appropriate targets must account for the patient's premorbid autoregulatory range, the cause of the arrest and the post-cardiac arrest cardiac function. Continuous blood pressure monitoring and cardiac output monitoring allow direction of haemodynamic stability in the post-cardiac arrest patient. Urine output, lactate and mixed venous oxygen saturations are surrogate markers of end-organ oxygen delivery used routinely in clinical practice.

Therapies including fluid resuscitation and vasopressor and inotropic drugs should be initiated and titrated in response to these parameters. The use of an intra-aortic balloon pump as an adjunct to medical interventions, in patients with cardiogenic shock following myocardial infarction, did not show any survival benefit in a randomized controlled trial. The Impella device is another adjunct in cardiogenic shock. A randomized controlled trial is awaited to determine if it confers a survival benefit (Nolan et al, 2015). Extracorporeal membrane oxygenation is currently only commissioned in five UK hospitals but may be beneficial in carefully selected cases of cardiogenic shock following a cardiac arrest (Bougouin et al, 2017).

Heart rate

Dysrhythmias may be the underlying cause of the arrest but are also a manifestation of myocardial instability post-return of spontaneous circulation.

Tachycardia should be avoided post-return of spontaneous circulation as it increases the risk of coronary ischaemia and re-arrest, because of the decreased time spent in diastole and therefore decreased

time for coronary perfusion. Dysrhythmias, including atrial fibrillation, atrial flutter and sustained ventricular tachycardia, which are common following an arrest, can compromise cardiac output and increase the propensity for re-arrest. They should therefore be promptly identified and aggressively managed, cardioverting when safe to do so. Abnormalities in electrolytes are a common source of electrical instability and should be kept within target ranges (*Table 1*), supplementing where necessary (Nolan et al, 2008).

Neuroprotection

Hypoxic ischaemic brain injury is the main cause of death following cardiac arrest. Unfortunately, perfusion is often impaired post-return of spontaneous circulation and altered cerebral autoregulation can cause insufficient cerebral blood flow to keep up with oxygen demand. Neuroprotection underpins most decisions in patient care post-return of spontaneous circulation. Patients with a Glasgow coma scale <8 are intubated and ventilated, with targets to avoid hypoxaemia and deliver controlled systemic hypertension, to ensure adequate cerebral perfusion and therefore tissue oxygenation. Advanced techniques including insertion of an intracranial bolt and use of transcranial Doppler allow continuous monitoring of intracranial pressure, global or regional metabolic state and/or blood flow. Where elevated intracranial pressure is unresponsive to medical management, decompressive craniectomy may be considered.

Oxygen demand is supraphysiological in the presence of seizures and pyrexia. Prevention of seizures, avoidance of hyperthermia and glucose control are therefore essential to prevent ongoing ischaemia and further neuronal injury (Nolan et al, 2008).

Sedation

Of the intravenous hypnotic agents commonly used on intensive care units, barbiturates, benzodiazepines and propofol reduce cerebral oxygen demand. Infusion of these agents is therefore neuroprotective, but systemic blood pressure may require augmentation to counteract vasodilatory and negative inotropic effects. There is no evidence to support a predetermined duration of sedation post-arrest. Shorter acting agents are usually preferred because earlier awakening allows earlier assessment of neurological function and reduces the likelihood of ventilator-associated complications (Paul et al, 2018).

> “Neuroprotection underpins most decisions in patient care post-return of spontaneous circulation.”

Seizure control

Seizures are a frequent clinical presentation in the post-cardiac arrest population (Nolan et al, 2015). Seizures increase cerebral metabolic demands so can be detrimental to neurological recovery and potentiate further neurological damage. Electroencephalography can help identify and characterize epileptic activity, although corresponding epileptiform activity may be absent on electroencephalography in the majority of patients who exhibit seizure-like activity, in particular myoclonus. Post-anoxic myoclonus is often resistant to standard antiepileptic medications.

Failure to wake off-sedation may indicate non-convulsive status in the absence of clinical manifestations and early electroencephalography is advised. Therapeutic options include antiepileptic drugs (most commonly sodium valproate, phenytoin, barbiturates and/or levetiracetam) or hypnotics (benzodiazepines and propofol). It remains unclear whether timely identification and treatment of seizures improves outcomes (Nolan et al, 2015).

Temperature control

Pyrexia is associated with worse outcomes in patients post-cardiac arrest. The preference has been to keep patients cool, by inducing hypothermia (32–34°C) in the immediate post-arrest period. The targeted temperature management randomized controlled trial heralded a new approach to temperature management in the post-cardiac arrest patient. The study compared temperature controlling patients to either 33°C or 36°C for 48 hours, followed by a slow rewarming phase and then subsequent avoidance of pyrexia (<37.5°C) with fever controlling strategies until 72 hours post-return of spontaneous circulation (Nielsen et al, 2013). There was no significant difference in mortality or neurological outcome between the groups.

The ideal duration of targeted temperature management is undefined, but current guidelines recommend targeted temperature management of 33–36°C for at least 24 hours post-cardiac arrest (Nolan et al, 2015). Hyperthermia should be aggressively treated.

In practice, there has been a growing trend for targeted temperature management at 36°C. This avoids the logistics and complications of inducing hypothermia, the risks of rapid rebound hyperthermia and also a reduced need for inotropic, sedatives and neuromuscular-blocking agents associated with hypothermia. A retrospective analysis of changes in temperature management, with targeted temperature management at 36°C being preferred, has shown an increased incidence of fever. A modest increase in annual mortality over the same time period was also observed. The relationship of this to the higher incidence of fever is unclear but should perhaps warrant a degree of caution (Salter et al, 2018). Targeting normothermia should not result in a laxity in temperature management, allowing for fever, in patients post-cardiac arrest.

Glucose control

Normoglycaemia (4–10 mmol/litre) is currently the recommended target in post-cardiac arrest patients (Nolan et al, 2015). Hyperglycaemia is associated with poorer neurological outcomes post-return of spontaneous circulation and strict glycaemic control regimens are fraught with a higher incidence of hypoglycaemia, which is independently associated with increased mortality. Blood glucose variability (the degree to which a person's blood glucose concentration fluctuates between high and low) may be a more important predictor of outcome: high blood glucose variability has been linked to poorer neurological outcomes. Similar to pyrexia, hyperglycaemia and high blood glucose variability may also represent an epiphenomenon, rather than an independent contributing factor to neurological injury (Taccone et al, 2014).

Prognostication

Withdrawal of life-sustaining therapy, as a result of a poor neurological outcome following hypoxic ischaemic brain injury,

is the main cause of death following out of hospital cardiac arrest (Sandroni et al, 2018). Neurological outcome is generally divided into good or poor, and more formally defined with a cerebral performance category score. The ideal timing of an assessment of neurological outcome is unclear and neurological status can improve up to 6 months after the cardiac arrest (Sandroni et al, 2018). Predictors of neurological outcome include findings on clinical examination, imaging, electrophysiological studies and biomarkers (*Table 2*). In view of accessibility, clinical examination, computed tomography scan and an electroencephalogram are likely to be the most widely used.

It is recommended that neurological prognostication with these markers should begin at a minimum of 72 hours after return of spontaneous circulation and may need to be further delayed if confounding variables (sedation, neuromuscular blockade, hypothermia, severe haemodynamic, metabolic or respiratory derangements) remain present (Sandroni et al, 2018). Clear improvement or deterioration of the patient can also be considered before this time point.

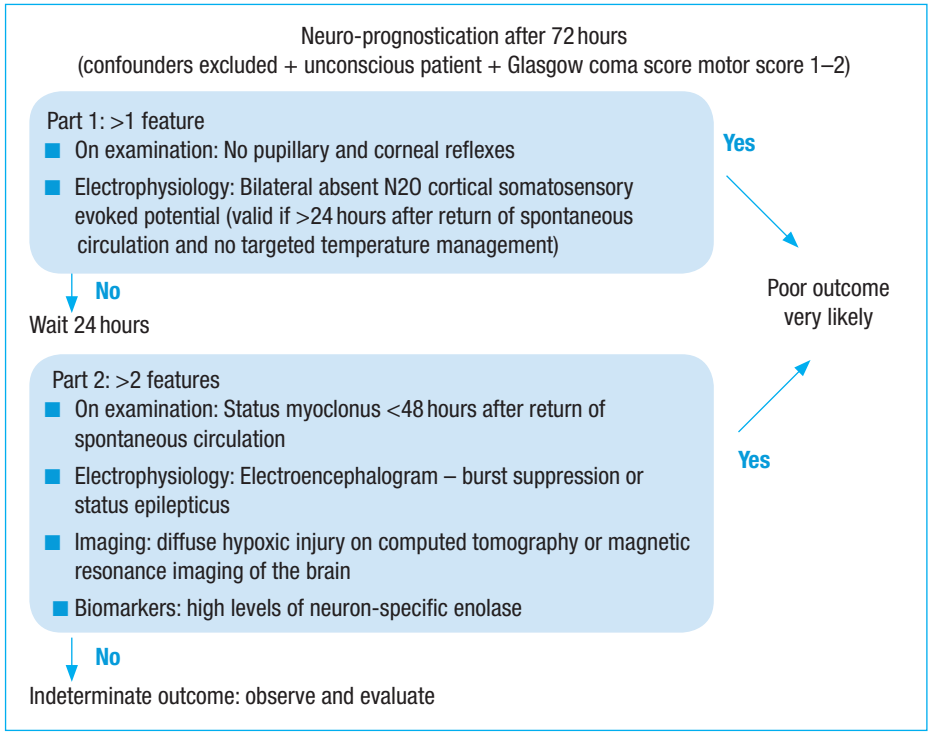

Figure 2. Prognostication strategy. From Sandroni et al (2018).

As there is no ideal single prognostic marker, a multi-modal approach to prognostication is favoured, as summarized in *Figure 2*.

Outcomes

Organ donation

Organ donation is an important consideration early in the care of patients in whom

Table 2. Predictors of neurological outcome: markers of hypoxic-ischaemic brain injury

Marker			Limitations
Clinical examination	■ Bilateral absence of pupillary light reflex and/or corneal reflex ■ Extensor motor response to pain ■ Myoclonus		■ Pupillary light reflex is a subjective assessment ■ Pharmacological interference: residual sedation, neuromuscular blockade
Neuroimaging	■ Computed tomography of the brain: attenuation of grey-white matter differentiation (ratio between grey and white matter densities) ■ Diffusion-weighted magnetic resonance imaging of the brain: hyperintense lesions, calculated as apparent diffusion coefficient		■ Variability in methods used to calculate grey-white matter differentiation and apparent diffusion coefficient ■ Magnetic resonance imaging scans can be logistically difficult in unstable patients
Electrophysiology	Electroencephalogram	■ Status epilepticus ■ Absence of background electroencephalogram activity ■ Burst suppression >50% of electroencephalogram record voltage <10 mV with alternating bursts	■ Pharmacological interference with electroencephalogram ■ Residual sedation ■ Neuromuscular blockade ■ Electrical interference of somatosensory evoked potentials
	Somatosensory evoked potential	■ Bilateral absence of N20 cortical somatosensory evoked potentials, response of primary somatosensory cortex	
Biomarkers	■ Neuron-specific enolase released following injury to neurons ■ S100 calcium-binding protein (S-100B) released following injury to glial cells		■ Threshold values determined by timing of sample ■ Variability in laboratory analysis ■ Biomarkers non-specific; extra-cerebral release source of false positive

From Sandroni et al (2018)

withdrawal of life-sustaining treatment is being considered. Cardiopulmonary resuscitation has not been shown to adversely affect graft function when compared at immediate, 1-year and 5-year follow-up time points (West et al, 2016). As such meticulous care of physiological parameters post-return of spontaneous circulation, even when withdrawal of life-sustaining treatment seems inevitable, is essential to protect organ quality and ensure that precious resources are not wasted.

Early contact with specialist nurses in organ donation and transplantation should be made when considering withdrawal. They should initiate the discussion regarding organ donation with families and will proceed to coordinate the process on behalf of NHS Blood and Transplant (National Institute for Health and Care Excellence, 2011).

Conclusions

Post-return of spontaneous circulation care is focused on identifying and treating the cause of the arrest and limiting further hypoxic ischaemic injury. Limiting hypoxic–ischaemic brain injury with neuroprotective measures underpins many treatment decisions post-return of spontaneous circulation. This article provides junior doctors with an understanding of early priorities and an approach with which to address challenges while managing this group of patients.

An improvement in delivery of care post-return of spontaneous circulation may be a small step towards improving the discrepancy in outcomes from return of spontaneous circulation to survival. The impetus for this surely lies in reports that post-cardiac arrest patients who survive to discharge generally have a good quality of life, albeit if not the same quality of life as before the arrest (Elliott et al, 2011). **BJHM**

Conflict of interest: none.

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KEY POINTS

- Identify and treat the cause of the cardiac arrest early with targeted investigations and interventions.
- Minimize the post-cardiac arrest syndrome which comprises secondary brain injury, myocardial dysfunction and systemic ischaemic reperfusion injury.
- Target normal physiological parameters for oxygen, carbon dioxide (not at the expense of causing further harm), blood pressure, blood glucose and temperature.
- Neuro-protection underpins many decisions in post-cardiac arrest care.
- Adopt a multimodal approach to prognostication and delay withdrawal until at least 72 hours.