Post-resuscitation Care

CHAPTER 13

Learning outcomes

To understand:

- **The need for continued resuscitation after return of spontaneous circulation**
- **How to treat the post-cardiac arrest syndrome**
- **How to facilitate transfer of the patient safely**
- **The role and limitations of assessing prognosis after cardiac arrest**

Introduction

Return of a spontaneous circulation (ROSC) is an important step in the continuum of resuscitation. However, the next goal is to return the patient to a state of normal cerebral function, and to establish and maintain a stable cardiac rhythm and normal haemodynamic function. This requires further treatment, tailored to each patient's individual needs. The quality of treatment provided in this post-resuscitation phase - the final ring in the Chain of Survival - significantly influences the patient's ultimate outcome. The post-resuscitation phase starts at the location where ROSC is achieved but, once stabilised, the patient needs transfer to the most appropriate high-care area (e.g. intensive care unit (ICU), coronary care unit (CCU)) for continued monitoring and treatment.

The post-cardiac arrest syndrome

The post-cardiac arrest syndrome, which comprises post-cardiac arrest brain injury, post-cardiac arrest myocardial dysfunction, the systemic ischaemia/reperfusion response, and persistence of the precipitating pathology, often complicates the post-resuscitation phase. The severity of this syndrome will vary with the duration and cause of cardiac arrest. It may not occur at all if the cardiac arrest is brief. Post-cardiac arrest brain injury manifests as coma, seizures, myoclonus, varying degrees of neurological dysfunction and brain death. Post-cardiac arrest brain injury may be exacerbated by microcirculatory failure, impaired autoregulation, hypercarbia, hypoxaemia and hyperoxaemia, pyrexia, hyperglycaemia and seizures. Significant myocardial dysfunction is common after cardiac arrest but typically recovers by 2 - 3 days. The whole body ischaemia/reperfusion that occurs with resuscitation from cardiac arrest activates immunological and coagulation pathways contributing to multiple organ failure and increasing the risk of infection. Thus, the post-cardiac arrest syndrome has many features in common with sepsis, including intravascular volume depletion and vasodilation.

Continued resuscitation

In the immediate post-resuscitation phase, pending transfer to an appropriate high-care area, treat the patient by following the ABCDE approach (Figure 13.1).

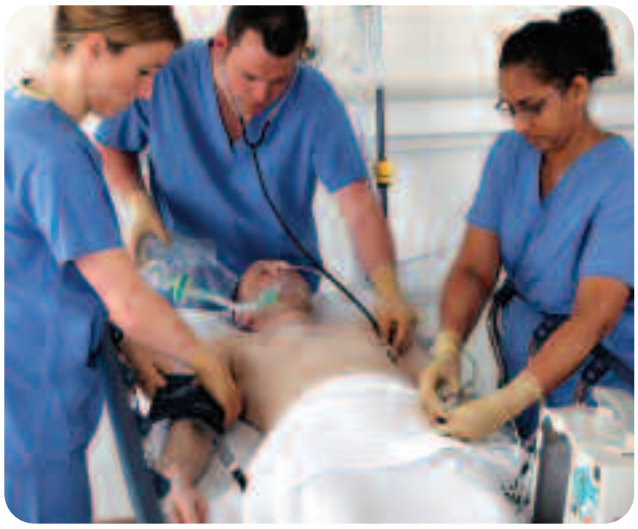

Figure 13.1 Immediate post-resuscitation care using the ABCDE approach

Airway and breathing

Patients who have had a brief period of cardiac arrest and have responded immediately to appropriate treatment (e.g. witnessed ventricular fibrillation (VF) reverting to sinus rhythm after early defibrillation) may achieve a rapid return of normal cerebral function. These patients do not require tracheal intubation and ventilation, but should be given oxygen by face mask to maintain a normal arterial oxygen saturation.

Hypoxaemia and hypercarbia both increase the likelihood of a further cardiac arrest and may contribute to secondary brain injury. Several animal studies indicate that hyperoxaemia causes oxidative stress and harms post-ischaemic neurones. One clinical study has shown that post-resuscitation hyperoxaemia is associated with worse outcome, compared with both normoxaemia and hypoxaemia. As soon as arterial blood oxygen saturation can be monitored reliably (by blood gas analysis and/or pulse oximetry [SpO_2]), titrate the inspired oxygen concentration to maintain the arterial blood oxygen saturation in the range of 94 - 98%. Consider tracheal intubation, sedation and controlled ventilation in any patient with obtunded cerebral function. Adjust ventilation to achieve normocarbia and monitor this using the end-tidal carbon dioxide ($ETCO_2$) with waveform capnography and arterial blood gas values.

Examine the patient's chest and look for symmetrical chest movement. Listen to ensure that the breath sounds are equal on both sides. A tracheal tube that has been inserted too far will tend to go down the right main bronchus and fail to ventilate the left lung. If ribs have been fractured during chest compression there may be a pneumothorax (reduced or absent breath sounds) or a flail segment. Listen for evidence of pulmonary oedema or pulmonary aspiration of gastric contents. Insert a gastric tube - this will decompress the stomach following mouth-to-mouth or bag-mask ventilation, prevent splinting of the diaphragm, and enable drainage of gastric contents.

If the intubated patient regains consciousness soon after ROSC, and is cooperative and breathing normally, consider immediate extubation: coughing on the tracheal tube will increase the patient's plasma catecholamine concentrations significantly, which may provoke arrhythmias and/or hypertension. Ensure that rigid suction is available. If immediate or early extubation is not possible sedate the patient to ensure the tracheal tube is tolerated, and provide ventilatory support.

Circulation

Cardiac rhythm and haemodynamic function are likely to be unstable following a cardiac arrest. Continuous monitoring of the ECG is essential. Seek evidence of poor cardiac function. Record the pulse and blood pressure and assess peripheral perfusion: warm, pink digits with a rapid capillary refill usually imply adequate perfusion. Grossly distended neck veins when the patient is semi-upright may indicate right ventricular failure, but in rare cases could indicate pericardial tamponade. Left ventricular failure may be indicated by fine inspiratory crackles heard on auscultation of the lung fields, and the production of pink frothy sputum. Try to optimise right and left heart filling pressures: measurement of central venous pressure will guide this. If the facility for direct continuous arterial blood pressure monitoring is available (e.g. in the emergency department) insert an arterial cannula to enable reliable monitoring during transfer. Once in a high-care area, the use of non-invasive cardiac output monitoring devices may be valuable. Infusion of fluids may be required to increase right heart filling pressures or conversely, diuretics and vasodilators may be needed to treat left ventricular failure.

Record a 12-lead ECG as soon as possible. Acute ST-segment elevation or new left bundle branch block in a patient with a typical history of acute myocardial infarction (AMI) is an indication for treatment to try to re-open an occluded coronary artery (reperfusion therapy), either with fibrinolytic therapy or by emergency percutaneous coronary intervention (PCI) (Chapter 4). Primary PCI is the preferred treatment for STEMI if it can be performed by an experienced team in a timely manner. If primary PCI is not feasible in an appropriate time frame (within 90 min of first medical contact), give fibrinolytic therapy (Chapter 4). Cardiopulmonary resuscitation, even if prolonged, is not a contraindication to fibrinolytic therapy.

In post-cardiac arrest patients, chest pain and/or ST elevation are relatively poor predictors of acute coronary occlusion; for this reason primary PCI should be considered in all post-cardiac arrest patients who are suspected of having coronary artery disease as the cause of their arrest, even if they are sedated and mechanically ventilated. Several studies indicate that the combination of therapeutic hypothermia (see below) and PCI is feasible and safe after cardiac arrest caused by AMI.

Disability and exposure

Although cardiac arrest is frequently caused by primary cardiac disease, other precipitating conditions must be excluded, particularly in hospital patients (e.g. massive blood loss, respiratory failure, pulmonary embolism). Assess the other body systems rapidly so that further resuscitation can be targeted at the patient's needs. To examine the patient properly full exposure of the body may be necessary.

Although it may not be of immediate significance to the patient's management, assess neurological function rapidly and record the Glasgow Coma Scale score (Table 13.1). The maximum score possible is 15; the minimum score possible is 3.

Glasgow Coma Scale score		
Eye Opening	Spontaneously	4
	To speech	3
	To pain	2
	Nil	1
Verbal	Orientated	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
	Nil	1
Best Motor Response	Obeys commands	6
	Localises	5
	Normal flexion	4
	Abnormal flexion	3
	Extension	2
	Nil	1

Table 13.1 The Glasgow Coma Scale score

Consider the need for inducing mild hypothermia in any patient that remains comatose after initial resuscitation from cardiac arrest (see below). When therapeutic hypothermia is considered an appropriate treatment, it should be started as soon as possible - do not wait until the patient is in the ICU before starting to cool.

Further Assessment

History

Obtain a comprehensive history as quickly as possible. Those involved in caring for the patient immediately before the cardiac arrest may be able to help (e.g. emergency medical personnel, general practitioner, and relatives). Specifically, symptoms of cardiac disease should be

sought. Consider other causes of cardiac arrest if there is little to suggest primary cardiac disease (e.g. drug overdose, subarachnoid haemorrhage). Make a note of any delay before the start of resuscitation, and the duration of the resuscitation; this may have prognostic significance, although is generally unreliable and certainly should not be used alone to predict outcome. The patient's baseline physiological reserve (before the cardiac arrest) is one of the most important factors taken into consideration by the ICU team when determining whether prolonged multiple organ support is appropriate.

Monitoring

Continuous monitoring of ECG, arterial and possibly central venous blood pressures, respiratory rate, pulse oximetry, capnography, core temperature and urinary output is essential to detect changes during the period of instability that follows resuscitation from cardiac arrest. Monitor continuously the effects of medical interventions (e.g. assisted ventilation, diuretic therapy).

Investigations

Several physiological variables may be abnormal immediately after a cardiac arrest and urgent biochemical and cardiological investigations should be undertaken (Table 13.2).

Arterial blood gases

Guidance on the interpretation of arterial blood gas values is given in Chapter 15.

Hypoperfusion during the period of cardiac arrest will usually cause a metabolic acidosis. This will cause a low pH (acidaemia), low standard bicarbonate and a base deficit. The rate at which the acidaemia resolves in the post-resuscitation period is an important guide to the adequacy of tissue perfusion. The most effective way of correcting any acidaemia is by addressing the underlying cause. For example, poor peripheral perfusion is treated best by giving fluid and inotropic drugs and not by giving sodium bicarbonate.

The normal physiological response to a metabolic acidosis is to reduce the $PaCO_2$ by an increase in ventilation (respiratory compensation). The patient who is breathing spontaneously may fail to achieve this if ventilation is depressed by sedatives, a reduced conscious level, or significant pulmonary disease. In these cases, the $PaCO_2$ may increase, causing a combined respiratory and metabolic acidosis and profound acidaemia.

Giving bicarbonate may, paradoxically, increase intracellular acidosis, as it is converted to CO_2 with the release of hydrogen ions within the cell. Indications for bicarbonate include cardiac arrest associated with hyperkalaemia or tricyclic overdose. Do not give bicarbonate routinely to correct acidaemia after cardiac arrest.

Full blood count
• To exclude anaemia as contributor to myocardial ischaemia and provide baseline values.
Biochemistry
• To assess renal function. • To assess electrolyte concentrations (K^+, Mg^{2+}, and Ca^{2+}).* • To ensure normoglycaemia. • To commence serial cardiac troponin measurements. • To provide baseline values.
12-lead ECG
• To record cardiac rhythm.** • To look for evidence of acute coronary syndrome. • To look for evidence of old myocardial infarction. • To provide a baseline record.
Chest radiograph
• To establish the position of a tracheal tube, a gastric tube, and/or a central venous catheter. • To check for evidence of pulmonary oedema. • To check for evidence of pulmonary aspiration. • To exclude pneumothorax. • To assess cardiac contour (accurate assessment of heart size requires standard PA erect radiograph - not always practicable in the post-resuscitation situation).
Arterial blood gases
• To ensure adequacy of ventilation and oxygenation. • To ensure correction of acid/base imbalance.
Echocardiography
• To identify contributing causes to cardiac arrest. • To assess LV and RV structure and function.

*Immediately after a cardiac arrest there is typically a period of hyperkalaemia. However endogenous catecholamine release promotes influx of potassium into cells and may cause hypokalaemia. Hypokalaemia may cause ventricular arrhythmias. Give potassium to maintain the serum potassium between 4.0 - 4.5 mmol l^{-1}.

**Normal sinus rhythm is required for optimal cardiac function. Atrial contraction contributes significantly to ventricular filling, especially in the presence of myocardial disease and valve disease. Loss of the sequential atrial and ventricular contraction of sinus rhythm may reduce cardiac output substantially in some patients.

Table 13.2 Investigations after restoration of circulation

Patient transfer

Following the period of initial post-resuscitation care and stabilisation, the patient will need to be transferred to an appropriate critical care environment (e.g. ICU or CCU). The decision to transfer a patient from the place where stabilisation has been achieved should be made only after discussion with senior members of the admitting team. Continue all established monitoring during the transfer and secure all cannulae, catheters, tubes and drains. Make a full re-assessment immediately before the patient is transferred. Ensure that portable suction apparatus, an oxygen supply and a defibrillator/monitor accompany the patient and transfer team.

The transfer team should comprise individuals capable of monitoring the patient and responding appropriately to any change in patient condition, including a further cardiac arrest. The Intensive Care Society (UK) has published guidelines for the transport of the critically ill adult (www.ics.ac.uk). These outline the requirements for equipment and personnel when transferring critically ill patients.

Optimising organ function

The extent of secondary organ injury after ROSC depends on the ability to minimise the harmful consequences of post-cardiac arrest syndrome (Figure 13.2). There are opportunities to limit the insult to organs following cardiac arrest.

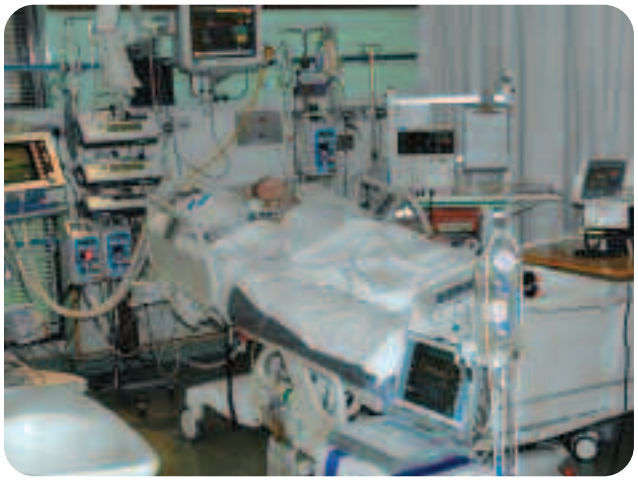

Figure 13.2 Multiple organ support for a patient with the post-cardiac arrest syndrome

Heart and cardiovascular system

Post-cardiac arrest myocardial dysfunction causes haemodynamic instability, which manifests as hypotension, a low cardiac output and arrhythmias. Early echocardiography will enable the degree of myocardial dysfunction to be quantified. In the ICU an arterial line for continuous blood pressure monitoring is essential. Treatment with fluid, inotropes and vasopressors may be guided by blood pressure, heart rate, urine output, and rate of plasma lactate clearance and central venous oxygen saturations. Non-invasive cardiac output monitors may help to guide treatment but there is no evidence that their use affects outcome. If treatment with fluid resuscitation and vasoactive drugs is insufficient to support the circulation, consider insertion of an intra-aortic balloon pump. Infusion of relatively large volumes of fluids is tolerated remarkably well by patients with post-cardiac arrest syndrome.

In the absence of definitive data supporting a specific goal for blood pressure, target the mean arterial blood pressure to achieve an adequate urine output (1 ml kg^{-1} h^{-1}) and normal or decreasing plasma lactate values, taking into consideration the patient's normal blood pressure, the cause of the arrest and the severity of any myocardial dysfunction. Importantly, hypothermia (see below) may increase urine output and impair lactate clearance.

Referral for implantable cardioverter defibrillator

Consider the possible requirement for an implantable cardioverter defibrillator (ICD) in any patient who has been resuscitated from cardiac arrest in a shockable rhythm outside the context of proven acute ST segment elevation myocardial infarction. All such patients should be referred before discharge from hospital for assessment by a cardiologist with expertise in heart rhythm disorders (Chapter 10).

Brain: optimising neurological recovery

Cerebral perfusion

Immediately after ROSC there is a period (about 15 min) of cerebral hyperaemia. After asphyxial cardiac arrest, brain oedema may occur transiently after ROSC but it is associated only rarely with clinically relevant increases in intracranial pressure. Autoregulation of cerebral blood flow is impaired for some time after cardiac arrest, which means that cerebral perfusion varies with cerebral perfusion pressure instead of being linked to neuronal activity. Following ROSC, maintain mean arterial pressure near the patient's normal level.

Sedation

Although it has been common practice to sedate and ventilate patients for at least 24 h after ROSC, there are no data to support a defined period of ventilation, sedation and neuromuscular blockade after cardiac arrest. Patients need to be well-sedated during treatment with therapeutic hypothermia, and the duration of sedation and ventilation is therefore influenced by this treatment. There are no data to indicate whether or not the choice of sedation influences outcome, but a combination of opioids and hypnotics is usually used. Short-acting drugs (e.g. propofol, alfentanil, remifentanil) will enable earlier neurological assessment. Adequate sedation will reduce

oxygen consumption. During hypothermia, optimal sedation can reduce or prevent shivering, which enables the target temperature to be achieved more rapidly.

Control of seizures

Seizures or myoclonus or both occur in 5% - 15% of adult patients who achieve ROSC and 10% - 40% of those who remain comatose. Seizures increase cerebral metabolism by up to three-fold and may cause cerebral injury: treat promptly and effectively with benzodiazepines, phenytoin, sodium valproate, propofol, or a barbiturate. Myoclonus can be particularly difficult to treat; phenytoin is often ineffective. Clonazepam is the most effective antimyoclonic drug, but sodium valproate, levetiracetam and propofol may also be effective. Start maintenance therapy after the first event once potential precipitating causes (e.g. intracranial haemorrhage, electrolyte imbalance) are excluded. No studies directly address the use of prophylactic anticonvulsant drugs after cardiac arrest in adults.

Glucose control

There is a strong association between high blood glucose after resuscitation from cardiac arrest and poor neurological outcome. However, severe hypoglycaemia is associated with increased mortality in critically ill patients, and comatose patients are at particular risk from unrecognised hypoglycaemia. Based on the available data and expert consensus, following ROSC, blood glucose should be maintained at ≤10 mmol l^{-1}. Hypoglycaemia (< 4.0 mmol l^{-1}) must be avoided. Strict glucose control (4.5 - 6.0 mmol l^{-1}) should not be implemented in adult patients with ROSC after cardiac arrest because of the increased risk of hypoglycaemia.

Temperature control

Treatment of hyperpyrexia

A period of hyperthermia (hyperpyrexia) is common in the first 48 h after cardiac arrest. Several studies document an association between post-cardiac arrest pyrexia and poor outcome. Although the effect of elevated temperature on outcome is not proved, treat any hyperthermia occurring after cardiac arrest with antipyretics or active cooling.

Therapeutic hypothermia

Mild hypothermia is neuroprotective and improves outcome after a period of global cerebral hypoxia-ischaemia. Cooling suppresses many of the pathways leading to delayed cell death, including apoptosis (programmed cell death). Hypothermia decreases the cerebral metabolic rate for oxygen by about 6% for each 1°C reduction in temperature and this may reduce the release of excitatory amino acids and free radicals.

Which post-cardiac arrest patients should be cooled?

All studies of post-cardiac arrest therapeutic hypothermia have included only patients in coma. There is good evidence supporting the use of induced hypothermia in comatose survivors of out-of-hospital cardiac arrest caused by VF. Two randomised trials demonstrated improved neurological outcome at hospital discharge or at 6 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 h. Extrapolation of these data to other cardiac arrests (e.g. other initial rhythms, in-hospital arrests, children) seems reasonable but is supported only by data derived from non-randomised trials.
Based on the evidence available and expert consensus, consider therapeutic hypothermia for any mechanically ventilated patient admitted to the ICU for post resuscitation organ support.

How to cool

The practical application of therapeutic hypothermia is divided into three phases: induction, maintenance, and rewarming. Animal data indicate that earlier cooling after ROSC produces better outcome. External and/or internal cooling techniques can be used to initiate cooling. An infusion of 30 ml kg^{-1} of 4°C 0.9% sodium chloride or Hartmann's solution decreases core temperature by approximately 1.5°C and this technique can be used to initiate cooling prehospital. Other methods of inducing and/or maintaining hypothermia include:

- Simple ice packs and/or wet towels are inexpensive; however, these methods may be more time consuming for nursing staff, may result in greater temperature fluctuations, and do not enable controlled rewarming.
- Ice-cold fluids alone cannot be used to maintain hypothermia, but even the addition of simple ice packs may control the temperature adequately.
- Cooling blankets or pads.
- Water or air circulating blankets.
- Transnasal evaporative cooling.
- Water circulating gel-coated pads.
- Intravascular heat exchanger, placed usually in the femoral or subclavian veins.
- Cardiopulmonary bypass.

In most cases, it is easy to cool patients initially after ROSC because the temperature usually decreases spontaneously within this first hour. Initial cooling is facilitated by neuromuscular blockade and sedation, which will prevent shivering. Magnesium sulphate (e.g. 5 g infused over 5 h), can also be given to reduce the shivering threshold.

In the maintenance phase, a cooling method with effective temperature monitoring that avoids temperature fluctuations is preferred. This is achieved best with external or internal cooling devices that include continuous temperature feedback to achieve a set target temperature. The temperature is typically monitored from a thermistor placed in the bladder and/or oesophagus. There are no data indicating that any specific cooling technique increases survival when compared with any other cooling technique; however, internal devices enable more precise temperature control compared with external techniques. Once the temperature is in the target range (32 - 34°C), maintain this temperature for 24 h. Rewarming must be achieved slowly: the optimal rate is not known, but expert consensus supports about 0.25 - 0.5 °C of warming per hour and strict avoidance of hyperthermia. Plasma electrolyte concentrations, effective intravascular volume and metabolic rate can change rapidly during rewarming, as they do during cooling.

Physiological effects and complications of hypothermia

The well-recognised physiological effects of hypothermia need to be managed carefully.

- Shivering will increase metabolic and heat production, thus reducing cooling rates - strategies to reduce shivering are discussed above.
- Mild hypothermia increases systemic vascular resistance, causes arrhythmias (usually bradycardia).
- Hypothermia causes a diuresis and electrolyte abnormalities such as hypophosphataemia, hypokalaemia, hypomagnesaemia and hypocalcaemia.
- Hypothermia decreases insulin sensitivity and insulin secretion, hyperglycaemia, which will need treatment with insulin (see glucose control).
- Mild hypothermia impairs coagulation and increases bleeding although this has not been confirmed in many clinical studies.
- Hypothermia can impair the immune system and increase infection rates.
- The serum amylase concentration is commonly increased during hypothermia but the significance of this unclear.
- The clearance of sedative drugs and neuromuscular blockers is reduced by up to 30% at a core temperature of 34°C.

Contraindications to hypothermia

Generally recognised contraindications to therapeutic hypothermia, but which are not applied universally, include: severe systemic infection, established multiple organ failure, and pre-existing medical coagulopathy (fibrinolytic therapy is not a contraindication to therapeutic hypothermia).

Prognostication

Two thirds of those dying after admission to ICU following out-of-hospital cardiac arrest die from neurological injury. A quarter of those dying after admission to ICU following in-hospital cardiac arrest die from neurological injury. A means of predicting neurological outcome that can be applied to individual patients immediately after ROSC is required. Many studies have focused on prediction of poor long term outcome (severe cerebral disability or death), based on clinical or test findings that indicate irreversible brain injury, to enable clinicians to limit care or withdraw organ support. The implications of these prognostic tests are such that they should have 100% specificity or zero false positive rate, i.e. no individuals should have a 'good' long-term outcome if predicted to have a poor outcome.

Clinical examination

There are no clinical neurological signs that predict reliably poor outcome (severe cerebral disability or death) less than 24 h after cardiac arrest. In adult patients who are comatose after cardiac arrest, and who have not been treated with hypothermia and who do not have confounding factors (such as hypotension, sedatives or muscle relaxants), the absence of both pupillary light and corneal reflex at ≥ 72 h predicts poor outcome reliably. Absence of vestibulo-ocular reflexes at ≥ 24 h and a GCS motor score of 2 or less (extension or no response to pain) at ≥ 72 h are less reliable. Other clinical signs, including myoclonus, are not recommended for predicting poor outcome. The presence of myoclonic status in adults is strongly associated with poor outcome but rare cases of good neurological recovery from this situation have been described and accurate diagnosis of myoclonic status is problematic.

Biochemical markers

Serum (e.g. neuronal specific enolase, S100 protein) or cerebrospinal fluid (CSF) biomarkers alone are insufficient as predictors of poor outcomes in comatose patients after cardiac arrest with or without treatment with therapeutic hypothermia.

Neurophysiological studies

No neurophysiological study predicts outcome for a comatose patient reliably within the first 24 h after cardiac arrest. If somatosensory evoked potentials (SSEPs) are measured after 24 h in comatose cardiac arrest survivors not treated with therapeutic hypothermia, bilateral absence of the N20 cortical response to median nerve stimulation predicts poor outcome. Very few hospitals in the UK have the resources to enable SSEPs to be measured.

Imaging studies

Many imaging modalities (magnetic resonance imaging [MRI], computed tomography [CT], single photon emission computed tomography [SPECT], cerebral angiography, transcranial Doppler, nuclear medicine, near infra-red spectroscopy [NIRS]) have been studied to determine their utility for prediction of outcome in adult survivors of cardiac arrest. Based on the available evidence, none of these imaging modalities will predict reliably outcome of comatose cardiac arrest survivors.

Impact of therapeutic hypothermia on prognostication

Most prognostication studies have been undertaken before implementation of therapeutic hypothermia and there is evidence that this therapy makes these tests less reliable even when undertaken after normothermia has been restored. Potentially reliable predictors of poor outcome in patients treated with therapeutic hypothermia after cardiac arrest include bilateral absence of N20 peak on SSEP ≥ 24 h after cardiac arrest and the absence of both corneal and pupillary reflexes 3 or more days after cardiac arrest. Given the limited available evidence, decisions to limit care should not be made based on the results of a single prognostication tool.

Organ donation

Post-cardiac arrest patients who do not survive should be considered as potential organ donors, either after brain death or as non-heart-beating donors.

Care of the resuscitation team

Audit all resuscitation attempts and, ideally, send these data to the National Cardiac Arrest Audit (Chapter 2). Feedback for the resuscitation team should be constructive and not based on a fault/blame culture. Whether the resuscitation attempt was successful or not, the patient's relatives will require considerable support. Consider the pastoral needs of all those associated with the arrest.

Key learning points

- After cardiac arrest, return of spontaneous circulation is just the first stage in a continuum of resuscitation.
- The quality of post-resuscitation care will influence significantly the patient's final outcome.
- These patients require appropriate monitoring, safe transfer to a critical care environment, and continued organ support.
- The post-cardiac arrest syndrome comprises post-cardiac arrest brain injury, post-cardiac arrest myocardial dysfunction, the systemic ischaemia/reperfusion response, and persistence of precipitating pathology.
- Our ability to predict the final neurological outcome for those patients remaining comatose after cardiopulmonary resuscitation remains very poor.

Further reading

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