17.2 Cardiac arrest 3839

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ESSENTIALS Cardiovascular disease is the most common cause of sudden cardiac arrest, which causes over 60% of adult coronary heart disease deaths. Most cardiac arrests are preventable. Survival depends on early recognition and

prompt initiation of chest compressions and ventilations (cardiopulmonary resuscita- tion), and early defibrillation if appropriate. High-quality cardiopulmonary resuscitation is defined by com- pressions to a depth of 5–6 cm, at a rate of 100–120 per minute, full release of pressure between compressions and minimal interrup- tions to chest compression. The compression to ventilation ratio is 30:2 if the airway is not protected. Give continuous chest compressions with no pause for

ventilations once the trachea is intubated or an appropriate supraglottic airway has been inserted. Treat shockable cardiac arrest rhythms (ventricular fibrillation/ pulseless ventricular tachycardia) with attempted defibrillation. In ventricular fibrillation refractory to defibrillation attempts, iden- tify and treat reversible causes. Treat nonshockable rhythms (asys- tole and pulseless electrical activity) by identifying and treating the underlying cause. Drugs have a limited role in treating cardiac

arrest. If initial resuscitation is successful the quality of post-resuscitation care determines the patient’s final outcome. In patients who are co- matose after cardiac arrest, outcome may be improved by inter- ventions such as primary percutaneous coronary intervention in patients with ST elevation in the post-arrest 12-lead electrocar- diogram, targeted temperature management, and multimodal prognostication. Use emergency care treatment plans to record in advance, re-commendations for emergency

treatments, including CPR. Introduction Survival from cardiac arrest depends on a sequence of interventions— the Chain of Survival (Fig. 17.2.1)—all four links in the chain must be strong: • early recognition and call for help • early cardiopulmonary resuscitation (CPR) • early defibrillation • post-resuscitation care Historical perspective Current cardiopulmonary resuscitation techniques were first de- scribed relatively recently: the first report of external defibrillation was in

1956, mouth-to-mouth ventilation in 1958, and chest com- pressions in 1960. Epidemiology Sudden cardiac arrest causes over 60% of adult coronary heart dis- ease deaths. In the United Kingdom the annual incidence of ambu- lance service treated out-of-hospital cardiac arrests for all rhythms is 52 per 100 000. The presenting cardiac arrest rhythm is shockable (ventricular fibrillation or pulseless ventricular tachycardia [VF/ pVT]) in about a quarter of patients, 25–30% of whom survive to hospital discharge. The remainder

of cases are nonshockable—asystole in about 50% and pulseless electrical activity (PEA) in about 25% of cases—and have much poorer survival (less than 5%). The incidence of in-hospital cardiac arrest is difficult to assess be- cause it is influenced by factors such as the criteria for hospital ad- mission and implementation of do-not-attempt-cardiopulmonary resuscitation (DNACPR) decisions. UK National Cardiac Arrest 17.2 Cardiac arrest Gavin D. Perkins, Jasmeet Soar, Jerry P. Nolan, and David A.

Gabbott E a r l y r e c o g n i t i o n a n d c a l l f o r h e l p

t o p r e v e n t c a r d i a c a r r e s t E a r l y C P R

t o b u y t i m e E a r l y d e f i b r i l l a t i o n

t o r e s t a r t t h e h e a r t P o s t-r e s u s c i t a t i o n c a r e

t o r e s t o r e q u a li t y o f l i f e Fig. 17.2.1 Chain of survival.

Section 17 Critical care medicine 3840 Audit data from 2011 to 2013 for arrests attended by the hospital resuscitation team showed an overall incidence of adult in-hospital cardiac arrest of 160 per 100 000 hospital admissions. The presenting rhythm was VF/pVT in 16.9% and nonshockable in 72.3% of cases. Survival to hospital discharge associated with these rhythms was 49.0% and 10.5%, respectively, but varied substantially between hospitals. Prevention Out-of-hospital, most sudden cardiac death (SCD) victims have cor- onary artery disease, and have a history of heart disease and warning symptoms, most commonly chest pain, in the hour before cardiac arrest. Calling an ambulance early after the onset of symptoms can improve survival. Other causes of sudden cardiac death are com- moner in adults younger than 35 years and children, and include cardiomyopathies, valve disease, inherited ion channel disorders, and congenital heart disease. Symptoms and signs include syncope (in the supine position, or without prodrome, or during exercise), chest pain, palpitation, and heart murmur. Those at risk of sudden cardiac death, or are family members of victims of sudden cardiac death, should be assessed in a specialist clinic. In-hospital cardiac arrests are usually not sudden or unpredict- able: in up to 80% there is deterioration in clinical signs during the preceding few hours. Hypoxaemia and hypotension are often unnoticed, or are detected but not treated appropriately. The car- diac arrest rhythm is

usually pulseless electrical activity or asys- tole and prognosis is poor. Prevention of in-hospital cardiac arrest requires staff education, monitoring of patients, recognition of patient deterioration, a system to call for help, and an effective response. Use of early warning scores, such as the National Early Warning Score (NEWS) in the United Kingdom, based on vital signs can help identify those at risk of deterioration, cardiac ar- rest, or unplanned intensive care unit admission. Earlier recogni- tion also enables proactive decision-making about emergency care treatment plans, including whether CPR should be attempted in the event of cardiac arrest. Cardiopulmonary resuscitation The division between basic life support and advanced life support is arbitrary: the resuscitation process is a continuum. The keys steps are: • cardiorespiratory arrest is recognized immediately; • help is summoned; • CPR is started immediately and, if indicated, defibrillation at- tempted as soon as possible (within 3 minutes of collapse). The sequence of actions and outcome depends on: • Location—out-of-hospital/in-hospital? Witnessed/unwitnessed? Monitored/unmonitored? • Skills of the responders—in public places (e.g. airports, railway stations) access to automated external defibrillators (AEDs) en- ables a bystander to deliver the first shock before an ambulance arrives. Defibrillation within 3–5 minutes of collapse can produce survival rates of 50–70% for shockable rhythms. • Number of responders—single responders must ensure that help is coming. If others are nearby, several actions can be undertaken simultaneously. • Equipment available—AEDs are available in some public places. Hospital staff should have immediate access to resuscitation equipment and drugs. General practitioners and dental practi- tioners should have an automated external defibrillator on their premises. • Response system to cardiac arrest and medical emergencies— outside hospital, call for an ambulance. In hospital, the resus- citation team can be a traditional cardiac arrest team (called when cardiac arrest is recognized). Alternatively, hospitals can have strategies to recognize patients at risk of cardiac arrest and summon a team (e.g. medical emergency team, rapid response team, or critical care outreach team) before cardiac arrest occurs. Risks to the rescuer There are very few reports of harm to rescuers from doing CPR. The personal safety of rescuers is the first priority during any re- suscitation attempt. Check that the patient’s surroundings are safe. Put on gloves as soon as possible, and use other personal protective equipment (PPE) (e.g. eye protection, face masks, aprons, gowns) when the patient has a serious infection such as tuberculosis. Follow local infection control measures to minimize risks. Barrier devices decrease transmission of bacteria during mouth-to-mouth rescue breathing in controlled laboratory set- tings, but their effectiveness in actual CPR is unknown. Be careful with sharps, and use safe techniques for moving victims during resuscitation. Starting CPR CPR should be started as shown in Box 17.2.1. Cardiopulmonary resuscitation—mechanism of action Chest compressions create blood flow by increasing intrathoracic pressure and compressing the heart directly. However, perfusion of the brain and myocardium is at best 25% of normal. High-quality CPR leads to better outcomes and is defined by compressing the chest 5–6 cm at a rate of 100–120 compressions a minute and en- suring full release of pressure between compressions. Minimize interruptions in chest compressions to avoid harmful drops in coronary perfusion and systemic blood flow (Fig. 17.2.2). Avoid hyperventilation, which increases intrathoracic pressure and thus reduces coronary perfusion pressure. Ventilate the lungs at 10 breaths a minute. Mechanical chest compression devices can deliver consistent, high-quality CPR, but require a highly trained team to avoid harmful interruptions to CPR during device deployment. Restrict their use to situations where high-quality manual CPR is impos- sible and a trained team is available (e.g. transfer in a moving am- bulance, during cardiac catheterization, as a bridge to starting extracorporeal CPR). Advanced life support The advanced life support (ALS) algorithm enables a standardized approach to cardiac arrest management (Fig. 17.2.3). Once CPR

17.2 Cardiac arrest 3841 has started, assess the patient’s rhythm as soon as possible. Heart rhythms associated with cardiac arrest comprise: • Shockable rhythms—VF/pVT. Ventricular fibrillation is identi- fied by a characteristic pattern of random, disorganized electrical activity on the electrocardiogram (ECG). Pulseless ventricular tachycardia is a broad complex tachycardia with no palpable pulse (or other signs of life). • Nonshockable rhythms—asystole and pulseless electrical activity. Pulseless electrical activity is cardiac electrical activity in the absence of any palpable pulses (or other signs of life). Asystole is the absence of electrical activity (other than electrical activity of <0.2 mV which could represent atrial complexes) for at least 6 seconds. Treatment of shockable rhythms (VF/pVT) Shockable rhythms should be treated as shown in Box 17.2.2. Defibrillation The definitive treatment for VF/pVT is defibrillation. Maintain high- quality CPR while the defibrillator is retrieved, pads applied, and the defibrillator is charged. Defibrillation success can be optimized by delivering high-quality chest compressions with minimal interrup- tions, particularly immediately before and after shock delivery. When used for initial monitoring of a rhythm and shock delivery, both pads and paddles enable quicker delivery of the first shock compared with separate initial attachment of standard ECG elec- trodes. The use of self-adhesive defibrillation pads makes it easier to minimize interruption to chest compressions and are preferred over paddles. Place defibrillation pads or paddles in the standard sternal (to the right of the sternum below the clavicle) and apical (mid- axillary line level with the ECG V6 lead) positions. Alternative posi- tions if the standard position is not possible (or does not terminate VF/pVT) are bi-axillary, or apical-posterior (one electrode over the left precordium and the other posteriorly, and inferior to the left scapula). Transthoracic impedance is minimized by ensuring good contact between pads and skin (shaving excessive chest hair if necessary), orientating the apical pad in a cranio-caudal direction, avoiding placement of the pad on breast tissue, and in a ventilated patient delivering a shock at the end of expiration and avoiding PEEP (posi- tive end-expiratory pressure). The energy levels and configurations of waveforms vary among defibrillators, hence follow the manufacturers’ guidance for energy levels for first and subsequent shocks. If the appropriate energy levels are unknown, for adults use the highest available shock energy for all shocks. Minimize interruptions to CPR during attempted defibrillation by adopting the shock sequence described in Box 17.2.2. Resume chest compressions immediately after a shock. Even if the defib- rillation attempt is successful in restoring a perfusing rhythm, it is rare for a pulse to be palpable immediately after defibrillation. Box 17.2.1 Starting CPR (for healthcare professionals in hospital) Check the patient for a response • If you see a patient collapse or apparently unconscious: — assess responsiveness (shake their shoulders) and seek a verbal response. If the patient does not respond • Alert other members of staff. • With the patient supine, open airway using head tilt and chin lift. • Assess for signs of life such as normal breathing, coughing, movement, and if trained and experienced, palpate for a central pulse. Take no more than 10 seconds to decide if signs of life are present. • Agonal breathing (occasional gasps, slow, laboured, or noisy breathing) is common immediately after cardiac arrest—do not mistake this for a sign of life. In addition, immediately after cardiac arrest the sudden ces- sation of cerebral blood flow can cause an initial short seizure-like epi- sode that can be confused with epilepsy. If the patient has no signs of life, no pulse, or if there is any doubt, start CPR immediately • Get a colleague to call the resuscitation team and collect the resuscita- tion equipment and a defibrillator. • If alone, leave the patient to get help and equipment. • Give 30 chest compressions: • The hand position for chest compression is the middle of the lower half of the sternum • Depth 5–6 cm • Rate 100–120 compressions min–1 • Allow the chest to recoil completely after each compression • After 30 compressions give two ventilations (compression-ventilation ratio = 30:2). • Once chest compressions have started, all

interruptions in compression must be kept to a minimum, of short duration, and tasks for the interrup- tion planned before stopping compressions. • Take the same amount of time for compression and relaxation. • Use whatever equipment is available immediately for airway and ven- tilation. Use a pocket mask (which can be supplemented with an oral airway), a supraglottic airway (e.g. laryngeal mask airway or i-gel) and self-inflating bag, or bag-mask. Attempt tracheal intubation only if trained and competent to do so with minimal interruption (less than 5 seconds) to chest compressions. Use waveform capnography routinely for confirming that a tracheal tube is in the patient's airway and subse- quent monitoring during CPR. Waveform capnography can also be used to monitor the quality of CPR, as an indicator of a return of spontaneous circulation and to help with determining prognosis during CPR. • Use an inspiratory time of one second and enough volume to produce a normal chest rise. Add supplemental oxygen as soon as possible. • Avoid rapid or forceful breaths to prevent gastric distension and prevent raised intrathoracic pressure. • Once the patient's trachea has been intubated, continue chest compres- sions uninterrupted at a rate of 100–120 min–1, and ventilate the lungs at approximately 10 breaths min–1. • If airway and ventilation equipment are unavailable, give mouth-to- mouth ventilation. If there are clinical reasons to avoid mouth-to-mouth contact, do chest compressions until help or airway equipment arrives. • When the defibrillator arrives, apply the electrodes (self-adhesive defib- rillator pads) to the patient and analyse the rhythm. Do not pause chest compressions to apply defibrillator pads. See advanced life support for further steps. • Providing CPR is tiring—change the person undertaking compressions every 2 min. 5 If the patient is not breathing and has a pulse (respiratory arrest) • Ventilate the patient's lungs (as just described) and check for a circulation every 10 breaths (about every minute). • If there are any doubts about the presence of a pulse, start chest compressions.

Section 17 Critical care medicine 3842 The duration of asystole before return of spontaneous circulation (ROSC) can be longer than two minutes in as many as 25% of suc- cessful shocks, and delay in trying to palpate a pulse will further compromise the myocardium if a perfusing rhythm has not been restored. If a perfusing rhythm has been restored, giving chest com- pressions does not increase the chance of ventricular fibrillation recurring. Defibrillator safety The operator must ensure that everyone is clear of the patient be- fore delivering a shock. Ensure there is no oxygen flowing across the chest. Remove oxygen masks to greater than 1 metre away, but leave any tracheal tube or supraglottic airway device connected to its breathing circuit, bag device, or mechanical ventilator during at- tempted defibrillation. Percutaneous coronary intervention during CPR Percutaneous coronary intervention (PCI) may be indicated if the patient remains in persistent VF/pVT following a suspected acute coronary syndrome. A mechanical chest compression device can be used during transfer to the catheter lab and through the procedure. Nonshockable rhythms (pulseless electrical activity
and asystole) Identify and treat reversible causes during CPR (see Box 17.2.3). Airway and ventilation Airway management and ventilation options during CPR can vary according to patient factors, the phase of the resuscitation (during CPR, after return of spontaneous circulation), and the rescuers' skill. Basic airway options include no airway and no ventilation (compression-only CPR by untrained individuals), compression- only CPR with the airway held open (with or without supplementary oxygen), mouth-to-mouth breaths, mouth-to-mask, and bag-mask ventilation with simple airway adjuncts. Advanced options include supraglottic airways and tracheal intubation (inserted with the aid of direct laryngoscopy or videolaryngoscopy, or via a supraglottic airway). Most patients are treated with more than one airway man- agement option during CPR, and a

stepwise approach is recom- mended. Tracheal intubation provides the most reliable airway during CPR, but should be attempted only by trained rescuers. If intubation is attempted, confirm tube position using clinical as- sessment and waveform capnography. In the absence of rescuers skilled in tracheal intubation, insertion of a supraglottic airway (e.g. laryngeal mask airway (LMA), i-gel, laryngeal tube) is safer than unskilled rescuers attempting tracheal intubation. Compared with bag-mask ventilation, early ventilation with a supraglottic airway reduces the incidence of gastric distension and subsequent regurgitation, and enables more effective ventilation of the lungs of an unconscious patient. If an alternative airway has been in- serted, attempt continuous chest compressions without stopping for ventilations. If excessive gas leakage results in inadequate ventilation of the patient's lungs, interrupt the chest compressions to enable ventilation. During CPR Ensure high-quality CPR is maintained throughout a resuscitation attempt. Rotate the CPR provider at least every two minutes when Fig. 17.2.2 Effect of chest compressions on coronary perfusion pressure. Coronary perfusion pressure (CPP) is determined by the difference between aortic diastolic pressure and right atrial pressure. The lower border of the dark band (marked by the orange ellipse) depicts the aortic diastolic pressure and thus CPP. This increases progressively as chest compressions are continued but decreases to base levels each time compressions are stopped. Note also that CPP continues to increase and does not plateau after 15 compressions. Uninterrupted chest compressions will generate a higher CPP. From Kern KB, et al. (1998). Efficacy of chest compression-only BLS CPR in the presence of an occluded airway. Resuscitation, 39, 179–88, with permission from Elsevier.

17.2 Cardiac arrest 3843 possible. The quality of CPR can be monitored by CPR feedback and prompt devices, supplemented by regular review of downloads and post-event debriefing. Waveform capnography may be helpful as a monitor of CPR quality (compression depth and ventilation rate). A rapid rise in the end-tidal CO2 value may indicate return of spontaneous circulation. A low end-tidal CO2 (<1.3 kPa) after at least 20 minutes of advanced life support can indicate a poor prognosis, although this should not be the sole factor taken into account when deciding to terminate a CPR attempt. Check the rhythm every two minutes and check the pulse if the rhythm is compatible with a perfusing rhythm. Reversible causes Identify and treat reversible causes during CPR for all cardiac arrests. These are divided into two groups of four based upon their initial letter—either H or T. Key treatments are summarized in brackets. • Hypoxia (secure airway, administer oxygen) • Hypovolaemia (intravenous fluids/blood, treat cause) • Hyperkalaemia, hypokalaemia, hypocalcaemia, acidaemia and other metabolic disorders (treat specific abnormality) • Hypothermia (rewarming) • Tension pneumothorax (thoracostomy or needle thoracocentesis) Fig. 17.2.3 The advanced life support algorithm. Reproduced with permission of the Resuscitation Council (UK).

Section 17 Critical care medicine 3844 • Tamponade (resuscitative thoracotomy) • Toxic substances (consult poison advice centre, consider antidote) • Thromboembolism—pulmonary embolism/coronary throm- bosis (thrombolysis or embolectomy/percutaneous coronary intervention) Peri-arrest ultrasound by a trained rescuer with minimal interrup- tion in CPR can be used to identify and treat some reversible causes of cardiac arrest. Drugs The available evidence suggests drugs have a limited role in the management of cardiac arrest. Adrenaline 1 mg every 3–5 minutes is retained in current guidelines on the basis that it improves the short-term outcomes of return of spontaneous circulation and admission to hospital, but it remains uncertain whether there is benefit or harm in terms of survival to discharge or neurologic outcome. There is no

evidence that giving any antiarrhythmic drug rou- tinely during cardiac arrest increases survival to hospital discharge. In comparison with placebo, the use of amiodarone (300 mg IV) in shock-refractory ventricular fibrillation (three failed defibrillation attempts) in the prehospital setting improves the short-term out- come of survival to hospital admission. The routine use of thrombolytic drugs in cardiac arrest is not recommended. Where the index of suspicion is high that car- diac arrest has been caused by a pulmonary embolus, thrombo- lytic therapy and prolonged CPR (up to 60–90 minutes) may be considered. Drug delivery Peak drug concentrations are higher and circulation times are shorter when drugs are injected into a central vein compared with a peripheral vein. Insertion of a central venous catheter requires interruption of CPR and is associated with several po- tential complications. Peripheral venous cannulation is quicker, easier, and safer. Flush drugs injected peripherally with at least 20 ml of fluid and elevate the extremity for 10 to 20 seconds to facilitate drug delivery to the central circulation. If intravenous access is difficult or impossible, the intraosseous route is a rea- sonable alternative. Extracorporeal CPR (e-CPR) Smaller portable pump devices and refinements to circuits, anticoagulation and vascular access have made the emergency use of extracorporeal circulatory support feasible for both in-hospital and out-of-hospital cardiac arrest. Extracorporeal CPR is a rescue therapy that can facilitate specific interventions (e.g. Box 17.2.2 Treatment of shockable rhythms (VF/pVT) 1 Start CPR, and once pads applied, stop chest compressions to confirm VF/pVT from the ECG. This pause in chest compressions should be brief and no longer than five seconds. 2 Resume chest compressions immediately; warn all rescuers other than the individual performing the chest compressions to ‘stand clear’ and remove any oxygen delivery device as appropriate. 3 The designated person selects the appropriate energy on the defib- rillator and presses the charge button. Choose an energy setting of at least 150 J for the first shock, the same or a higher energy for subse- quent shocks, or follow the manufacturer’s guidance for the particular defibrillator. If unsure of the correct energy level for a defibrillator, in adults choose the highest available energy. 4 Ensure that the rescuer giving the compressions is the only person touching the patient. 5 Once the defibrillator is charged and the safety check is complete, tell the rescuer doing the chest compressions to ‘stand clear’; when clear, give the shock. 6 After shock delivery immediately restart chest compressions. Do not pause to reassess the rhythm or feel for a pulse. The total pause in chest compressions should be brief and no longer than 5 seconds. 7 Continue CPR for 2 min; the team leader prepares the team for the next pause in CPR. 8 Pause briefly to check the monitor. • If VF/pVT, repeat steps 2–8 and deliver a second shock. • If VF/pVT persists after a second shock, repeat steps 2–6 and deliver a third shock, then 9 Resume chest compressions immediately. Give adrenaline 1 mg IV and amiodarone 300 mg IV while performing a further 2 min CPR. Withhold adrenaline if there are signs of return of spontaneous circulation (ROSC) during CPR. 10 Repeat this 2 min CPR—rhythm/pulse check—defibrillation sequence if VF/pVT persists. 11 Give further adrenaline 1 mg IV after alternate 2 min periods of CPR (i.e. approximately every 3–5 min). 12 If organized electrical activity compatible with a cardiac output is seen during a rhythm check, seek evidence of ROSC (check for signs of life, a central pulse, and end-tidal CO2 if available). a If there is ROSC, start post-resuscitation care. b If there are no signs of ROSC, continue CPR, and switch to the non- shockable algorithm. 13 If asystole is seen, continue CPR, and switch to the nonshockable algorithm. Box 17.2.3 Treatment for pulseless electrical activity (PEA) and asystole 1 Start CPR 30:2. 2 Give adrenaline 1 mg IV as soon as intravascular access is achieved. 3 Continue CPR 30:2 until the airway is secured—then continue chest compressions without pausing during ventilation. 4 Recheck the rhythm after 2 min: a If organized electrical activity is seen, check for a pulse and/or signs of life: i If pulse and/or signs of life are present, start post-resuscitation care. ii If

no pulse and/or signs of life are present (PEA): 1 Continue CPR 2 Recheck the rhythm after 2 min and proceed accordingly 3 Give further adrenaline 1 mg IV every 3–5 min (alternate 2 min loops of CPR). b If VF/pVT at rhythm check, change to the shockable side of algorithm. c If asystole or an agonal rhythm seen at rhythm check: 1 Continue CPR 2 Recheck the rhythm after 2 min and proceed accordingly 3 Give further adrenaline 1 mg IV every 3–5 min (alternate 2 min loops of CPR).

17.2 Cardiac arrest 3845 percutaneous coronary intervention, surgical treatment of mas- sive pulmonary embolus, rewarming after severe hypothermia) where standard advanced life support interventions have failed to achieve a sustained return of spontaneous circulation. Routine use outside of these settings requires further research to opti- mize case selection criteria and determine clinical and cost effectiveness. Post-resuscitation care The quality of post-resuscitation care significantly influences the patient's ultimate outcome. ABCDE approach The ABCDE (airway, breathing, circulation, disability, exposure) system approach should be applied after resuscitation: Airway and breathing Consider tracheal intubation, sedation, and lung protective venti- lation in patients with obtunded cerebral function after return of spontaneous circulation. Maintain the oxygen saturation of arterial blood between 94–98%, avoiding hypoxaemia and hyperoxaemia, both of which may exacerbate brain injury and impair cardiac function. After cardiac arrest, hypocapnia induced by hyperventi- lation causes cerebral ischaemia. There are few data to support the targeting of a specific $PaCO_2$ after resuscitation from cardiac arrest, but it is reasonable to adjust ventilation to achieve normocapnia. Decompress the stomach by placing a nasogastric tube. Obtain a chest x-ray to check tracheal tube, NG tube and central line pos- ition, and to identify any complications of CPR (fractured ribs, pneumothorax). Circulation Haemodynamic instability is common after cardiac arrest and manifests as hypotension, low cardiac output, vasodilation, and arrhythmias. This is partly caused by reperfusion injury and is usu- ally transient, often reversing within 24–48 hours. Fluids and vaso- active drugs (noradrenaline and/or dobutamine), with or without an intra-aortic balloon pump, may be required to maintain perfu- sion of the vital organs. Echocardiography and monitoring cardiac output, urine output, and lactate clearance can be used to guide therapy. Bradycardia may occur as a consequence of targeted tem- perature management; this should be treated only if it adversely affects haemodynamics. Coronary reperfusion Acute coronary syndrome is a common cause of cardiac arrest (up to 70% of out-of-hospital cardiac arrests). Record a 12-lead electrocardiogram as soon as possible. Observational studies in- dicate that patients with acute ST-segment elevation or new left bundle branch block benefit from urgent coronary angiography and (when indicated) percutaneous coronary intervention, ir- respective of conscious level. The evidence is less certain for pa- tients without ST elevation but with a history consistent with a cardiac event. Such patients should be discussed with an inter- ventional cardiologist and considered for emergency cardiac catheterization. Other clinical issues Sedation and brain imaging Sedate the patient with short acting drugs. Consider a neuromus- cular blocking drug in the event of patient-ventilator asynchrony or uncontrolled shivering. Continuous electroencephalography (EEG) is recommended if an infusion of a neuromuscular blocker is used because this may mask seizure activity. Consider a computed tomography (CT) scan of the brain or chest if history of presentation suggests a possible cerebral or respiratory cause of the cardiac arrest. Cerebral perfusion Immediately after return of spontaneous circulation there is a period of cerebral hyperaemia, but after 15–30 minutes of reperfusion global cerebral blood flow decreases and there is generalized hypoperfusion. In about one-third of post cardiac arrest patients normal cerebral autoregulation is lost, leaving cerebral

perfusion dependent on mean arterial pressure. Under these circumstances, hypotension will compromise cerebral blood flow severely and can worsen any neurological injury. After return of spontaneous cir- culation, aim to maintain mean arterial pressure at the patient's usual level. Control of seizures Seizures and/or myoclonus occur in 5–15% of patients who achieve return of spontaneous circulation, and in approximately 40% of those who remain comatose. Seizures increase cerebral metabolism by up to fourfold and may worsen brain injury. The available data do not support prophylactic treatment with anticonvulsants. If seizures occur, treat with benzodiazepines, sodium valproate, levetiracetam, phenytoin, or propofol. Treat myoclonus with sodium valproate, levetiracetam, clonazepam, or propofol; phenytoin is rarely effective. Clinical or EEG evidence of seizures, myoclonus, and status epilep- ticus are associated with a poor outcome, particularly if these occur in the first 24–48 hours. Nevertheless, survival with good outcome despite early seizures does occur, hence the presence of seizures should not alone determine a decision to withdraw life-sustaining treatment. Glucose control Hyperglycaemia during post-resuscitation care phase is associ- ated with poor neurological outcome. Maintain blood glucose at ≤10 mmol L–1, but avoid hypoglycaemia. Temperature control and management Hyperthermia is common in the first 48 hours after cardiac arrest. The risk of a poor neurological outcome increases for each degree of body temperature over 37°C. Although there are no random- ized trials comparing treatment of hyperthermia with no treat- ment, there is consensus that hyperthermia occurring in the first 72 hours after cardiac arrest should be treated with antipyretics or active cooling. Mild hypothermia suppresses many of the chemical reactions associated with reperfusion injury. These reactions include free radical production, excitatory amino acid release, and calcium shifts, which can in turn lead to mitochondrial damage and apop- tosis (programmed cell death).

Section 17 Critical care medicine 3846 Randomized trials from 2002 reported improved neurological outcomes in patients treated with therapeutic hypothermia (32– 34°C) for 12–24 hours after out-of-hospital cardiac arrest. The more recent Targeted Temperature Management (TTM) trial re- ported similar outcomes with temperature targets of 33°C versus 36°C in comatose out-of-hospital cardiac arrest survivors. Current guidelines recommend maintaining a constant target temperature value between 32–36°C for 24 hours in all cardiac arrest survivors who remain unresponsive after return of spontaneous circulation, irrespective of cardiac arrest location and initial cardiac arrest rhythm. Therapeutic hypothermia reduces overall metabolic rate; glucose should be closely monitored during rewarming as it may precipitate hypoglycaemia in patients being treated with insulin. Use targeted temperature management cautiously in patients with severe systemic infection and pre-existing coagulopathy; in such patients consider using a target of 36oC instead of a lower temperature. Prognosis Survival For out-of-hospital cardiac arrest, among those in whom resus- citation is attempted, 25–30% initially achieve return of spon- taneous circulation but only about 8% survive to go home from hospital. For in-hospital cardiac arrest, the rate of return of spontaneous circulation is 45%, with 18.5% surviving to hospital discharge. Some patients with out-of-hospital cardiac arrest have recovered consciousness by admission to hospital, while many have obtunded conscious levels requiring admission to intensive care. Of 8664 patients admitted to intensive care units in the United Kingdom after in- or out-of hospital cardiac arrest in 2014, 47% survived to leave intensive care and 37% survived to hospital discharge. Most (>90%) of patients who survive to discharge have a cerebral per- formance category of 1 or 2, indicating a moderate to good neuro- logical outcome. Prediction of outcome Unless return of spontaneous circulation is achieved rapidly after the onset of cardiac arrest, most patients who achieve return of spontaneous circulation will have an obtunded conscious level. There are no tests to predict

reliably which of those patients may benefit from intensive care. The decision about whether to admit to intensive care is informed by a careful assessment of the patient's prearrest functional status, comorbidities, and the cause of the car- diac arrest. Of those admitted to intensive care after cardiac arrest in the United Kingdom, 35–40% survive to hospital discharge. Two-thirds of those dying after admission to intensive care following out-of- hospital cardiac arrest die from neurological injury, compared to a quarter of those dying after admission following in-hospital cardiac arrest. International guidelines recommend a multimodal approach to prognostication in the comatose post-cardiac arrest patient, which includes clinical, radiological and electrophysio- logical tests and measure of biomarkers (Fig. 17.2.4). Assessment is made only after sufficient time has passed to enable clearance of sedatives and neurological recovery (usually at least 72 hours from cardiac arrest). Clinical tests There are no neurological signs that can predict outcome in the first few hours after return of spontaneous circulation. By three days after the onset of coma relating to cardiac arrest, 50% of patients with no chance of ultimate recovery have died. Brain stem reflexes may recover up to 72 hours after cardiac arrest. The bilateral absence of the pupillary reflex to light and/or corneal reflex 72 hours after return of spontaneous circulation predicts poor outcome with a low false positive rate, yet sensitivity is low (about 19%), that is, these signs are only present in one in five patients with a poor outcome. An absent or extensor motor response to pain at 72 hour is more sensitive (75%) but has a high false positive rate (27%). An extended period of assessment is required in cases of residual sedation or muscle relaxants. Status myoclonus suggests significant cerebral insult but is not universally associated with adverse outcome. Some variants of myo- clonus (e.g. Lance Adams syndrome, a chronic action myoclonus beginning within days or weeks after CPR) can be associated with a good outcome and it is important to involve experienced clinicians in making the diagnosis. Clinical tests are relatively easy to perform, yet lack sufficient precision to be relied upon in isolation, hence the presence of ad- verse clinical signs should prompt multimodal imaging and electro- physiological assessments. Imaging The evidence supporting the use of diagnostic imaging is less certain than with other test modalities. Sulcal effacement and loss of grey/ white matter differentiation on CT scan of the brain suggests cere- bral hypoxic-ischaemic injury. Magnetic resonance imaging (MRI) is more sensitive than CT for detecting cerebral hypoxic-ischaemic injury. Cerebral/CT or MRI angiography may also play a role as an- cillary tests for brain stem death. Electrophysiological tests The bilateral absence of the N20 component median nerve somato- sensory evoked potentials in normothermic patients, comatose for at least 72 h after cardiac arrest, predict poor outcome with high reliability (false positive rate 0–2%). Electro-encephalographic (EEG) patterns associated with poor outcomes are absence of EEG reactivity, status epilepticus, and burst suppression. Biomarkers Neuron-specific enolase and S-100B proteins are markers of neuro- logical injury. High and sustained (or increasing) values are associ- ated with poor outcome.

17.2 Cardiac arrest 3847 Management of cardiac arrest survivors Rehabilitation Cognitive impairment, emotional and memory problems, and fa- tigue are common after cardiac arrest and adversely effect recovery and quality of life. Early evidence suggests post-discharge assess- ment and rehabilitation improves outcomes. Cardiac electrophysiological assessment 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 Other considerations Organ donation Depending on national laws and codes of practice, organ dona- tion

can be considered after brain stem death is confirmed (dona- tion after neurological determination of death) or after withdrawal of life-sustaining treatment (controlled donation after circulatory Fig. 17.2.4 Prognostication for comatose survivors of cardiac arrest. Reproduced with permission of the Resuscitation Council (UK).

Section 17 Critical care medicine 3848 death) in those who achieved initial return of spontaneous circula- tion after cardiac arrest but remained comatose. In some countries, when advanced life support fails to achieve return of spontaneous circulation, organ donation may be considered (uncontrolled dona- tion after circulatory death). Audit The Utstein template provides a standardized approach to auditing demographic, process, and outcomes from cardiac arrest. Structured quality improvement initiatives linked to local performance data such as the Resuscitation Academy concept (http://www. resuscitationacademy.eu) have great potential to improve quality and outcomes. Data from regional, national, and international registries allow evaluation of the safety and effectiveness of interventions diffi- cult to test in a randomized controlled trial. Decisions relating to cardiopulmonary resuscitation It is essential to identify patients for whom cardiopulmonary arrest represents an anticipated terminal event and in whom CPR is in- appropriate. All healthcare settings (hospital and community) should ensure that there are clear and explicit resuscitation plans for those at risk of cardiac arrest. Generally accepted reasons for withholding CPR are (i) when it is deemed that it will be ineffective (i.e. will not restart the heart and breathing for a sustained period; (ii) CPR will not provide overall benefit (informed by discussion with the pa- tient or their family or other nominated surrogate decision makers); (iii) the patient has refused CPR. Emergency care treatment plans allow resuscitation decisions to be considered as part of an overall treatment plan. Observational data suggest that this approach is associated with more patient- centred decision-making, better communication, and less risk that a decision to withhold resuscitation will mean an overall reduction in the quality and quantity of care. Likely developments over the next 5–10 years The International Liaison Committee on Resuscitation (ILCOR) coordinates evidence-based reviews of resuscitation science. Technology advances will enable a more continuous review of emerging science to feed into clinical guidelines in future years. A greater emphasis on training the community to recognize cardiac arrest and how to initiate CPR and use an automated ex- ternal defibrillator will be key to saving more lives. National and international initiatives to introduce CPR and automated external defibrillator training in schools, optimizing dispatcher recognition of cardiac arrest, and provision of telephone CPR instruction are likely to have significant impact. The registration of automated ex- ternal defibrillators in the community and intelligent systems (using app-based technology) to support their deployment may increase the proportion of patients receiving early defibrillation before the emergency services arrive. Few advanced life support interventions are based on robust evi- dence. Large multicentre trials are in progress and will provide de- finitive information on the optimal advanced airway technique, and whether drugs are safe and effective as treatments for cardiac ar- rest. Research will also define if percutaneous coronary intervention during cardiac arrest improves survival. Global (e.g. blood pressure/flow, lactate clearance) and regional measurements (e.g. end-tidal CO_2, cerebral oximetry, coronary perfusion pressure, ventricular fibrillation waveform analysis) are likely to play a growing role in tailoring specific interventions to the patient’s physiological status. The increased portability of technology that can provide extracor- poreal resuscitation means that it is now feasible to initiate e-CPR at the scene of a cardiac arrest. Further research is required to deter- mine for which patients it is clinically and cost effective to deploy this technology. FURTHER READING Fritz Z, Slowther AM, Perkins GD (2017). Resuscitation policy should focus on the patient, not the decision. BMJ, 356, j813. Monsieurs KG, et

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Revision #1
Created 2026-01-22 16:39:50 UTC by Omar Ayman
Updated 2026-01-22 16:39:50 UTC by Omar Ayman