Appendix A Drugs Used in the Treatment of Cardiac Arrest

Drug	Shockable (VF/Pulseless VT)	Non-Shockable (PEA/Asystole)
Adrenaline	• Dose: 1 mg (10 ml 1:10,000 or 1 ml 1:1,000) IV • Given after the 3rd shock once compressions have been resumed • Repeated every 3 - 5 min (alternate loops) • Give without interrupting chest compressions	• Dose: 1 mg (10 ml 1:10,000 or 1 ml 1:1,000) IV • Given as soon as circulatory access is obtained • Repeated every 3 - 5 min (alternate loops) • Give without interrupting chest compressions
	Adrenaline has been the primary sympathomimetic drug for the management of cardiac arrest for 40 years. Its alpha-adrenergic effects cause systemic vasoconstriction, which increases coronary and cerebral perfusion pressures. The beta-adrenergic actions of adrenaline (inotropic, chronotropic) may increase coronary and cerebral blood flow, but concomitant increases in myocardial oxygen consumption and ectopic ventricular arrhythmias (particularly in the presence of acidaemia), transient hypoxaemia because of pulmonary arteriovenous shunting, impaired microcirculation, and increased post cardiac arrest myocardial dysfunction may offset these benefits. Although there is no evidence of long-term benefit from the use of adrenaline, the improved short-term survival documented in some studies warrants its continued use.	
Amiodarone	• Dose: 300 mg bolus IV • Given after the 3rd shock once compressions have been resumed • Further dose of 150 mg if VF/VT persists	• Not indicated for PEA or asystole
	Amiodarone is a membrane-stabilising anti-arrhythmic drug that increases the duration of the action potential and refractory period in atrial and ventricular myocardium. Atrioventricular conduction is slowed, and a similar effect is seen with accessory pathways. Amiodarone has a mild negative inotropic action and causes peripheral vasodilation through non-competitive alpha-blocking effects. The hypotension that occurs with intravenous amiodarone is related to the rate of delivery and is caused by the solvent, rather than the drug itself. Amiodarone should be flushed with 0.9% sodium chloride or 5% dextrose. When amiodarone is unavailable, consider an initial dose of 100 mg (1 - 1.5 mg kg^{-1}) of lidocaine for VF/VT refractory to three shocks. Give an additional bolus of 50 mg if necessary. The total dose should not exceed 3 mg kg^{-1} during the first hour.	
Magnesium	• Dose: 2 g given peripherally IV • May be repeated after 10 - 15 min • Indicated for VT, torsade de pointes, or digoxin toxicity associated with hypomagnesaemia	• Dose: 2 g given peripherally IV • May be repeated after 10 - 15 min • Indicated for supraventricular tachycardia or digoxin toxicity associated with hypomagnesaemia
	Magnesium facilitates neurochemical transmission: it decreases acetylcholine release and reduces the sensitivity of the motor endplate. Magnesium also improves the contractile response of the stunned myocardium, and may limit infarct size.	

Drug	Shockable (VF/Pulseless VT) Non-Shockable (PEA/Asystole)
Calcium	• Dose: 10 ml 10% calcium chloride (6.8 mmol Ca^{2+}) IV • Indicated for PEA caused specifically by hyperkalaemia, hypocalcaemia or overdose of calcium channel blocking drugs
	Calcium plays a vital role in the cellular mechanisms underlying myocardial contraction. High plasma concentrations achieved after injection may be harmful to the ischaemic myocardium and may impair cerebral recovery. Do not give calcium solutions and sodium bicarbonate simultaneously by the same route.
Sodium Bicarbonate	• Dose: 50 mmol (50 ml of an 8.4% solution) IV • Routine use not recommended • Consider sodium bicarbonate in shockable and non-shockable rhythms for ○ cardiac arrest associated with hyperkalaemia ○ tricyclic overdose. Repeat the dose as necessary, but use acid-base analysis to guide therapy.
	Cardiac arrest results in combined respiratory and metabolic acidosis as pulmonary gas exchange ceases and cellular metabolism becomes anaerobic. The best treatment of acidaemia in cardiac arrest is chest compression; some additional benefit is gained by ventilation. Bicarbonate causes generation of carbon dioxide, which diffuses rapidly into cells. This has the following effects: • it exacerbates intracellular acidosis; • it produces a negative inotropic effect on ischaemic myocardium; • it presents a large, osmotically-active sodium load to an already compromised circulation and brain; • it produces a shift to the left in the oxygen dissociation curve, further inhibiting release of oxygen to the tissues. Do not give calcium solutions and sodium bicarbonate simultaneously by the same route.
Fluids	Infuse fluids rapidly if hypovolaemia is suspected. During resuscitation, there are no clear advantages in using colloid, so use 0.9% sodium chloride or Hartmann's solution. Avoid dextrose, which is redistributed away from the intravascular space rapidly and causes hyperglycaemia, which may worsen neurological outcome after cardiac arrest.
Thrombolytics	• Tenecteplase 500 - 600 mcg kg^{-1} IV bolus • Alteplase (r-tPA) 50 mg IV bolus (British Thoracic Society guidelines for the management of suspected acute pulmonary embolism. Thorax 2003;58:470–484)
	Fibrinolytic therapy should not be used routinely in cardiac arrest. Consider fibrinolytic therapy when cardiac arrest is caused by proven or suspected acute pulmonary embolus. If a fibrinolytic drug is given in these circumstances, consider performing CPR for at least 60 - 90 min before termination of resuscitation attempts. Ongoing CPR is not a contraindication to fibrinolysis.