Emergency ALS Drugs

Adrenaline

Dose – IV/IO 10 mcg/kg (max. single dose of 1 mg); ETT 100mcg/kg
Effect lasts 3 – 5 minutes; therefore, dose can be repeated every 2nd cycle of 2-minutes of CPR

Use	Action	Side Effects
Pulseless VT, VF – after 2nd shock once compressions have resumed Pulseless Electrical Activity (PEA) & Asystole – give immediately Repeat every 4 minutes (every 2nd loop of 2 minutes of CPR) Instead of repeated bolus doses, a continuous infusion of 0.1 – 0.2 mcg/kg/min may be given (preferably into a large vein)	Stimulates alpha & beta receptor sites Peripheral vasoconstriction, increasing cerebral and coronary blood flow Increases contractility (positive inotrope) Increases heart rate (positive chronotrope) Increases cardiac output, blood pressure	Tachydysrhythmias Hypertension Ischaemia Cold peripheries Extravasation produces tissue necrosis

Amiodarone

Dose – IV/IO bolus 5mg/kg in 5% Dextrose (dose may be repeated)

Use	Action	Side Effects
Broad-spectrum antiarrhythmic Used to treat refractory pulseless VT & VF arrests (administered after 3rd failed defibrillation attempt once compression have resumed) Prophylaxis of recurrent VT/VF	Effects sodium, potassium & calcium channels Delays repolarization, increasing refractory period of atrial, nodal and ventricular tissues Slows the speed of conduction Slows the automaticity of the Sinoatrial Node Slows conduction through accessory pathways Alpha- & Beta-adrenergic blocking properties	Vasodilatory effects & negative inotropic properties Bradycardia Hypotension Heart block

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Lignocaine

Dose – IV/IO 1mg/kg; ETT 2 – 3 mg/kg
Inferior to amiodarone as an antiarrhythmic
Not recommended in paediatric cardiac arrest unless amiodarone is not available

Use	Action	Side Effects
Shock resistant Ventricular Tachycardia (VT) Given if defibrillation & subsequent administration of adrenaline with further defibrillation has failed to revert VT if amiodarone is unavailable	Membrane stabilising agent Decreases the automaticity of conducting tissues Elevates the fibrillation threshold Inhibits the fast sodium channels Suppresses ventricular irritability Increases defibrillation threshold	Hypotension Bradycardia Heart blocks Asystole Central nervous system effects

Atropine

Dose – IV/IO 20mcg/kg, ETT 30mcg/kg

Use	Action	Side Effects
May be indicated only if bradycardia caused by vagal stimulation or cholinergic drug toxicity	Parasympathetic blockade Anti-cholinergic Increases sinoatrial node rate and speed of conduction through the atrioventricular node	Tachycardia Decreased urine output

Calcium Chloride

Dose – IV/IO 0.2 mL/kg 10% Calcium Chloride (or 0.7 mL/kg 10% Calcium Gluconate)
Not routinely given at cardiac arrest, has been associated with worse outcomes

Use	Action	Side Effects
Pulseless Electrical Activity (PEA) arrests due to: Hyperkalaemia Hypocalcaemia Hypermagnesaemia Calcium channel blocker overdose	Essential for normal muscle and nerve activity Increases myocardial contractile force Aids in peripheral vasoconstriction	Extravasation produces tissue necrosis May increase myocardial & cerebral injury by mediating cell death

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Magnesium

Dose – IV/IO bolus 0.1 – 0.2 mmol/kg Magnesium Sulphate.
Followed by an infusion 0.3 mmol/kg over 4 hours.

Use	Action	Side Effects
Ventricular tachyarrhythmias unresponsive to other treatment Documented hypomagnesaemia Polymorphic VT *(Torsade de pointes)*	Essential electrolyte Stabilizes the myocardium	Hypotension Muscle weakness Paralysis Respiratory failure

Potassium

Dose – IV/IO bolus 0.03 – 0.07 mmol/kg by slow injection
NB: a small bolus injection may cause a dangerous rise in serum potassium,
e.g.: a 1 mmol bolus in a 5 kg infant theoretically will raise the serum level about 4 mmol/L

Use	Action	Side Effects
Hypokalaemia	Essential for normal membrane stability	Hyperkalaemia Bradycardia, hypotension Asystole Extravasation produces tissue necrosis

Sodium Bicarbonate

Dose – 0.5 - 1 mmol/kg IV/IO
Ensure CPR with adequate ventilation is continued to expel CO_2

Use	Action	Side Effects
Protracted arrests Severe metabolic acidosis, pH < 7.1	Alkalinising solution To correct acidosis by "mopping up" hydrogen ions (H^+) $H^+ + HCO^- \leftrightarrow H_2CO_3 \leftrightarrow H_2O + CO_2$ Hydrogen + bicarbonate → weak → carbonic acid → water + carbon dioxide Sodium bicarbonate combines with hydrogen ions to produce carbon dioxide (CO_2) and water (H_2O) Carbon dioxide is a by-product of the equation, which quickly diffuses into cells. This can exacerbate the intracellular acidosis and have a negative inotropic effect on the myocardium	Worsening intracellular acidosis as excess CO_2 diffuses into myocardial and cerebral cells Extracellular alkalosis shifts the O_2 dissociation curve to the left, inhibiting the release of oxygen to the tissues Hypernatraemia Hyperosmolality

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