4H 4T

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H-K, Ca , Mg T-toxiny

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European Resuscitation Council Guidelines for Resuscitation 2015 Section 4. Cardiac arrest in special circumstances

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be identified or excluded during any resuscitation. For improving recall during ALS, these are divided into two groups of four, based upon their initial letter – either H or T – and are called the '4Hs and 4Ts': Hypoxia; Hypo-/hyperkalaemia and other electrolyte disorders; Hypo-/hyperthermia; Hypovolaemia; Tension pneumothorax; Tamponade (cardiac); Thrombosis (coronary and pulmonary); Toxins (poisoning). The second part covers cardiac arrest in special environments, where universal guidelines have to be modified due to specific locations or location-specific causes of cardiac arrest. The third part is focused on patients with spe-

72year men with DM II type- insulin depend., CKD on chronic haemodialysis emergency bcs.- dyspnoe, 2 min CPR

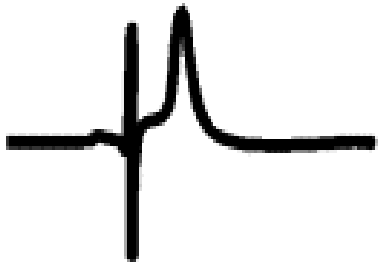
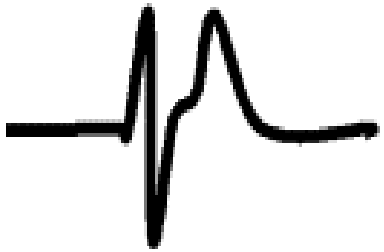
- A+B :oti+VCV
- C :PEA
- D : coma

Hyperkalaemia. This is the most common electrolyte disorder associated with cardiac arrest. It is usually caused by impaired excretion by the kidneys, drugs or increased potassium release from cells and metabolic acidosis. Hyperkalaemia occurs in up to 10% of hospitalised patients.[13–15] Chronic kidney disease (CKD) is common in the general population and the incidence of hyperkalaemia increases from 2 to 42% as glomerular filtration rate (GFR) drops from 60 to 20 mL min^{-1}.[16] Patients with end-stage renal disease are particularly susceptible, particularly following an OHCA.[17] Prolonged hyperkalaemia is an independent risk factor for in-hospital mortality.[18] Acute hyperkalaemia is more likely than chronic hyperkalaemia to cause life-threatening cardiac arrhythmias or cardiac arrest.

Definition. There is no universal definition. We have defined hyperkalaemia as a serum potassium concentration higher than 5.5 mmol L^{-1}; in practice, hyperkalaemia is a continuum. As the potassium concentration increases above this value the risk of adverse events increases and the need for urgent treatment increases. Severe hyperkalaemia has been defined as a serum potassium concentration higher than 6.5 mmol L^{-1}.

Causes. The main causes of hyperkalaemia are:

- renal failure (i.e. acute kidney injury or chronic kidney disease);
- drugs (e.g. angiotensin converting enzyme inhibitors (ACE-I), angiotensin II receptor antagonists (ARB), potassium-sparing diuretics, non-steroidal anti-inflammatory drugs, beta-blockers, trimethoprim);
- tissue breakdown (e.g. rhabdomyolysis, tumour lysis, haemolysis);
- metabolic acidosis (e.g. renal failure, diabetic ketoacidosis);
- endocrine disorders (e.g. Addison's disease);
- diet (may be sole cause in patients with advanced chronic kidney disease) and
- spurious – pseudo-hyperkalaemia (suspect in cases with normal renal function, normal ECG and/or history of haematological disorder). Pseudo-hyperkalaemia describes the finding of a raised serum (clotted blood) K^+ value concurrently with a normal plasma (non-clotted blood) potassium value. The clotting process releases K^+ from cells and platelets, which increases the serum K^+ concentration by an average of 0.4 mmol/L. The most common cause of pseudo-hyperkalaemia is a prolonged transit time to the laboratory or poor storage conditions.[19,20]

Serum potassium	Typical ECG appearance	Possible ECG abnormalities
Mild (5.5-6.5 mEq/L)		Peaked T waves Prolonged PR segment
Moderate (6.5-8.0 mEq/L)		Loss of P wave Prolonged QRS complex ST-segment elevation Ectopic beats and escape rhythms
Severe (>8.0 mEq/L)		Progressive widening of QRS complex Sine wave Ventricular fibrillation Asystole Axis deviations Bundle branch blocks Fascicular blocks

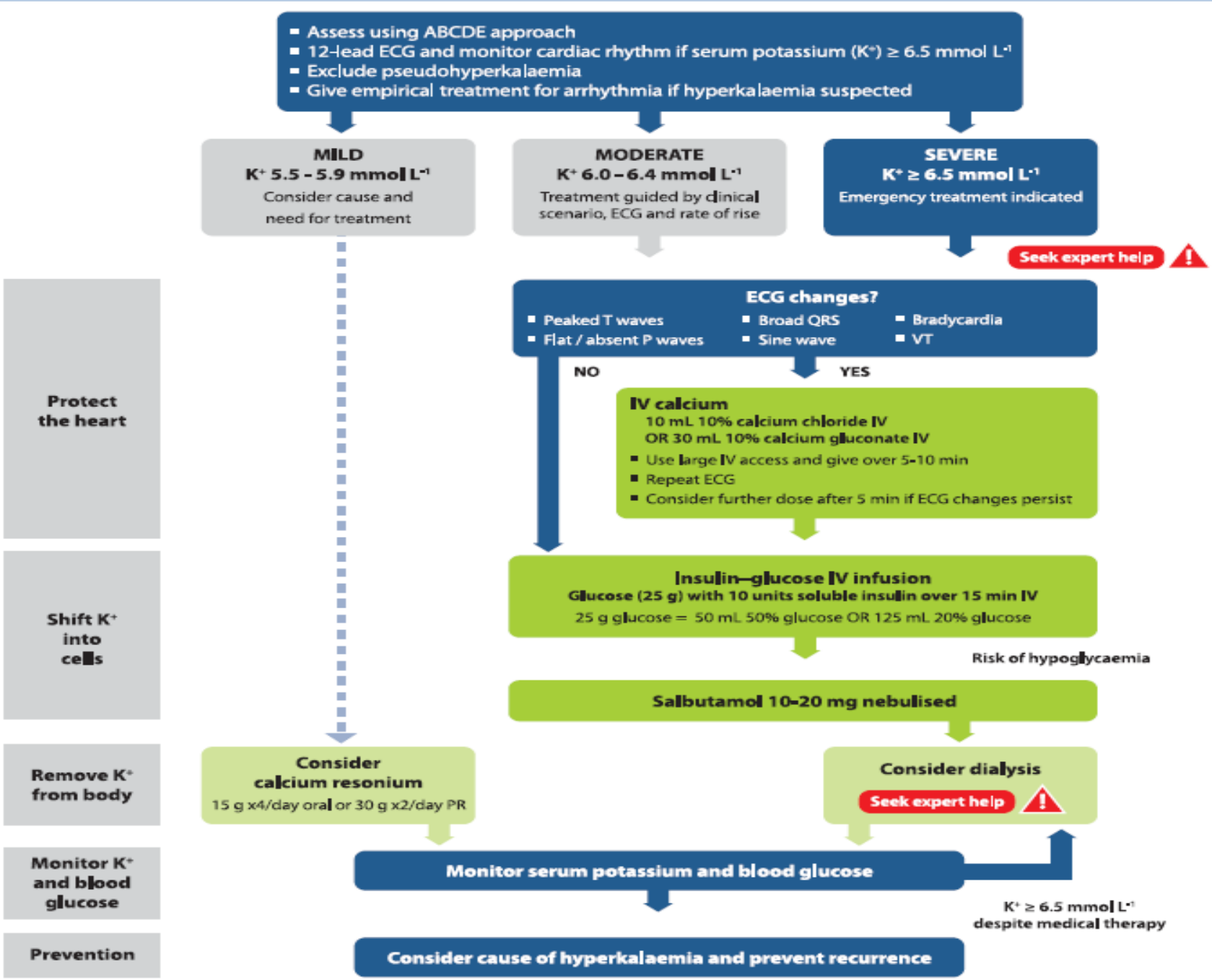


Fig. 4.1. Emergency treatment of hyperkalaemia. PR per rectum; ECG electrocardiogram; VT ventricular tachycardia. Reproduced with permission from Renal Association and Resuscitation Council (UK).

27 year men , psoriasis emergency bcs.- coma, hypotension, bradycardia

- A+B : spont ventilation-RF 10, spo293%,
- C : BP 75/45 HR sinus.bradycardia 42/min
- D : GCS E1 V2 M5

Glasgow Coma Scale						
	1	2	3	4	5	6
Eye	Does not open eyes	Opens eyes in response to painful stimuli	Opens eyes in response to voice	Opens eyes spontaneously	N/A	N/A
Verbal	Makes no sounds	Incomprehensible sounds	Utters incoherent words	Confused, disoriented	Oriented, converses normally	N/A
Motor	Makes no movements	Extension to painful stimuli (decerebrate response)	Abnormal flexion to painful stimuli (decorticate response)	Flexion / Withdrawal to painful stimuli	Localizes painful stimuli	Obeys commands

cardiac arrest and intoxication

- Rare cause
- Young people
- Cause of CPR- cardiotoxicity +secondary insult- airway obstruction , aspiration, hypotension
- Prolonged CPR –how long?
- Toxicology center in CR

Table 1. Agents associated with cardiac toxicity

Sodium channel blockers	Tricyclic antidepressants
	Type 1 anti-arrhythmics
	Local anaesthetic agents
	Anti-malarials (e.g. quinine, chloroquine, hydroxychloroquine)
	Propranolol
	Carbamazepine
	Bupropion
Calcium channel blockers	Diltiazem
	Verapamil
β-blockers	Propranolol
	Sotalol
	Metoprolol
Potassium channel blockers	Amiodarone
	Sotalol
	Amisulpride
	Citalopram
	Phenothiazine antipsychotics
	Tricyclic antidepressants
	Venlafaxine
Other pharmaceuticals	Digoxin
	Theophylline
	Potassium chloride
Chemicals	Organophosphate and carbamate pesticides
Drugs of abuse	Amphetamines (including MDMA)
	Cocaine
Natural toxins	Snake venom
	Funnel-web spider venom
	Box jellyfish (*C. fleckeri*) toxin
	Oleander plant

Table 2. Potential antidotes in toxic cardiac arrest

Antidote/inotrope	Agent/toxin	Indications
Atropine	Acetylcholinesterase inhibitors (e.g. organophosphates) β-blockers Calcium channel blocking agents	Bradycardia Hypotension Excess secretions
Sodium bicarbonate	Sodium channel blocking agents (e.g. tricyclic antidepressants)	Arrhythmias (usually wide complex tachyarrhythmias) Intractable hypotension Seizures
Calcium	Calcium channel blocking agents	Hypotension Bradycardia
High-dose insulin/dextrose	β-blockers Calcium channel blocking agents	Intractable hypotension Heart block
Digoxin antibody fragments	Digoxin Other cardiac glycosides (e.g. oleander)	Life-threatening arrhythmias
Lipid emulsion rescue	Local anaesthetic agents	Cardiac arrest
Antivenom	Snake, funnel-web spider, box jellyfish venom	Clinical signs of envenoming

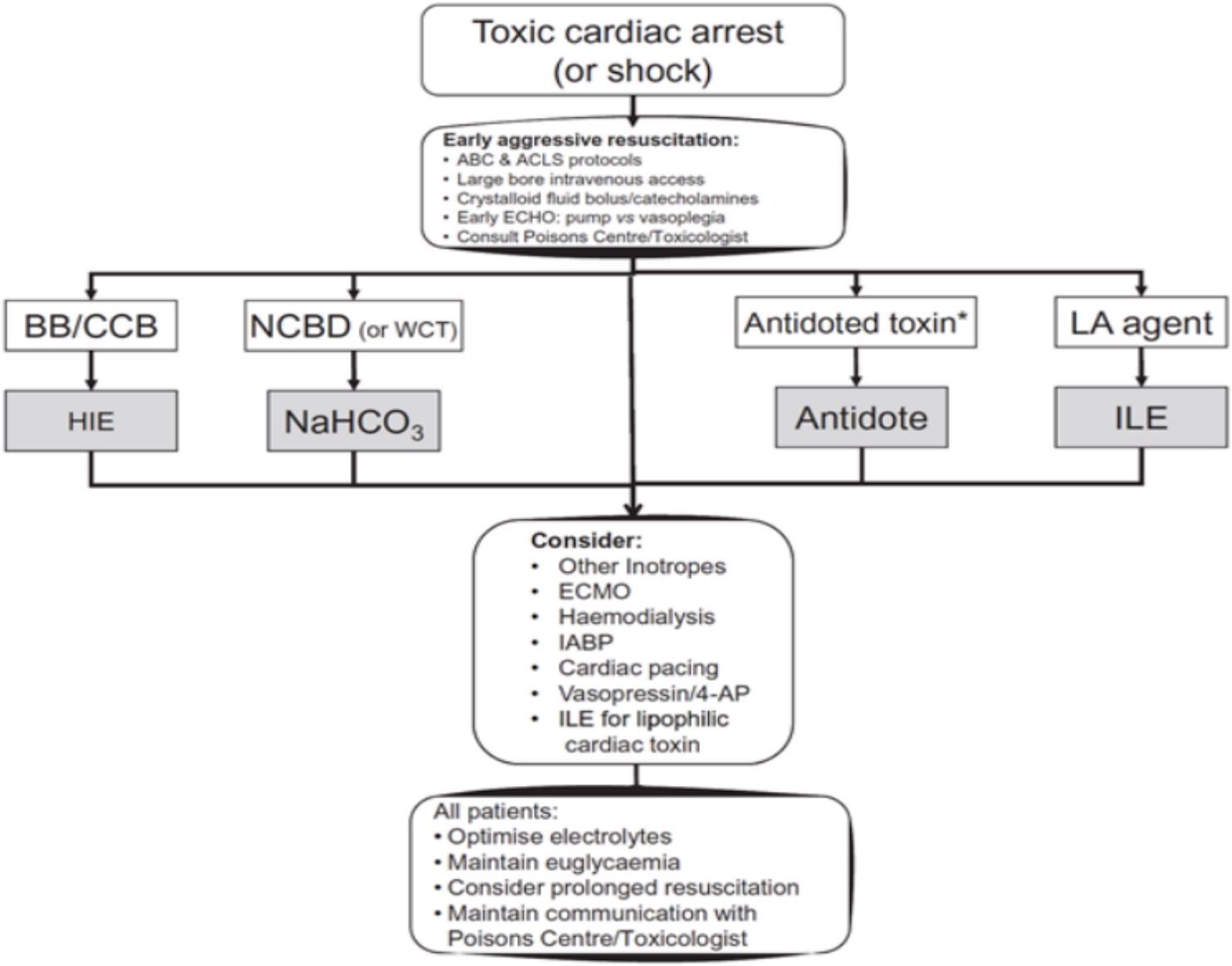


Figure 1. Management flowchart. *Toxin with available antidote, e.g. natural toxin, digoxin, organophosphates. 4-AP, 4-aminopyridine; BB, β-blocker; CCB, calcium channel blocker; ECMO, extra-corporeal membrane oxygenation; HIE, high-dose insulin euglycaemia; IABP, intra-aortic balloon pump; ILE, intravenous lipid emulsion; LA, local anaesthetic; NCBD, sodium channel blocker; WCT, wide complex tachycardia.