Hypokalaemia

EXCLUSIONS

- IV replacement only: no ECG monitoring or arterial line (regular monitoring of ABGs required)
- Creatinine >150mmol/l except when continuous veno-venous haemodiafiltration (CVVHDF) used
- Haemodialysis
- Chloride > 110mmol/l
- pH <7.25
- Diabetic ketoacidosis (DKA) or hyperosmotic hyperglycaemic state (HHS)
- Burns patients
- Patients receiving potassium containing fluids (except Hartmanns)
- Fluid restriction where patient only has peripheral access

If the patient is receiving Addiphos® or Potassium Acid Phosphate take a potassium level after these have been given to determine if potassium supplementation is still required.

Aim to maintain potassium between 3.8 – 5.0 mmol/l

Dosing and Administration

Route	Preparation	Notes
Enteral/oral	Sando-K: 2 tablets every 8 hours	Each tablet contains 12 mmol of potassium and 8 mmol of chloride
Peripheral	40mmol potassium chloride in 1000ml sodium chloride 0.9% (0.3%) via a volumetric pump over 4 hours	
Central venous	40mmol potassium chloride in 100ml sodium chloride 0.9% (3%) via a volumetric pump over 2 hours	**40mmol potassium chloride in 100ml sodium chloride 0.9% is CVC only. Only for critical care areas**

Potassium level	Action	Monitor
>3.9mmol/L	Nil	Every 24 hours unless otherwise directed
2.9 – 3.8 mmol/L	Administer 40mmol **OR** Sando-K: 2 tablets 8 hourly if K^+ >3.0mmol/L	Check Potassium after 24 hours
≤2.8mmol/L	Administer 40mmol Another dose may need to be given depending on results of ABG / Formal labs taken after administration	Repeat ABG and send blood away for formal labs after administration Multiple administration of K^+ may be required to correct deficit

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Additional information

- Hypomagnesaemia should be corrected in hypokalaemic patients.
- Urinary losses of potassium increase with diuretics (furosemide, thiazides) amphotericin, hypomagnesaemia, polyuria and dialysis.
- Vomiting, diarrhoea or laxative use increase gastrointestinal potassium losses.
- Increased potassium entry into cells is caused by catecholamines, salbutamol, insulin, metabolic or respiratory acidosis.
- An acute increase in haematopetic cell production causes hypokalaemia, this most commonly occurs when treatment with hydroxocobalamin, folic acid or GCSF is started.