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The Pennine Acute Hospitals NHS Trust

Reference Number: EDT019 **Version Number:** 2

Guideline for the Management of Electrolyte Disturbances in Adult Patients

What is this document for?	To assist in the management of adult patients with electrolyte disturbances.
Who needs to know?	The guideline can be used as a reference source for all healthcare professionals working within Pennine Acute Trust who are managing adult patients with an electrolyte disturbance, excluding those on critical care wards.
Related PAHNT Documents:	▪ Medicines Policy (EDC018) ▪ Intravenous Potassium Policy (EDT003) ▪ Policy for Training in Safe Administration of Medicines (EDN031) ▪ Procedure for Administration of Prescribed Medicines to Inpatients (EDT004) ▪ Accident & Incident Reporting Policy (EDQ008) ▪ Nutrition Support in Adults: hospital setting – Part 5 Guidelines for the Management Of Refeeding Syndrome In Adult Patients (CPDI148)
Related Legislation/ Obligations:	

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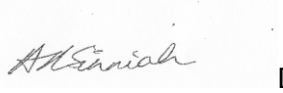
Ratified by:	 Dr Anton Sinniah, Acting Medical Director & Chair of Pennine Acute Drug and Therapeutics Committee
Date Ratified:	24/2/16
Replaces:	EDT019 Management of Electrolyte Disturbances; Guidelines for Treatment of Adult Patients version 1
How is this different from the previous document?	Hypokalaemia chapter: • Example of IV potassium infusion regime added for the treatment of severe hypokalaemia Hyperkalaemia chapter:

	• change in K^+ value at which to initiate treatment from 7.0 to 6.5mmol/L as per national guidelines • addition of severe symptoms which would prompt urgent treatment • Addition of alternative strengths of glucose solution Hypercalcaemia chapter: • change to max concentration of disodium pamidronate infusion as per SPC • clarification of when to administer IV bisphosphonates • Wording changed in rehydration paragraph from 'sodium chloride' to 'intravenous fluids' Hypocalcaemia chapter: • re-wording of the recommendation for ECG monitoring with IV calcium – bolus dose only • additional caution re: renal failure • addition of paragraph on underlying causes Hypomagnesaemia chapter: • removal of magnesium glycerophosphate as recommended oral treatment due to availability of a licensed product Magnaspartate® • addition of critical care regime for administration of IV magnesium Hypermagnesaemia – entirely new chapter Hyponatraemia chapter: • complete rewrite to reflect European Guidelines • separated into 'acute' and 'chronic hyponatraemia' • inclusion of an algorithm to guide diagnosis and management Hypophosphataemia chapter: • Directions for dissolving tablets added References updated
What dissemination & training arrangements have been made?	This Guideline will be available via the Document Management System.
Review arrangements:	The document will be reviewed every 3 years by the author. This will be done earlier If new information comes to light, or as considered appropriate by PADAT.
Safety Arrangements:	Compliance & effectiveness of this guideline will be via accident, incident & complaints monitoring. Staff experiencing difficulties with implementing this guideline should contact their line manager.

Priority Level: Priority 1
Impact Level: Trustwide
Keywords: Hypokalaemia, hyperkalaemia, hypocalcaemia, hypercalcaemia, hypomagnesaemia, hyponatraemia, hypermatraemia, hypophosphataemia, electrolyte, potassium, calcium, magnesium, sodium, phosphate

Contents

Page

1. What is this Guideline for?

1.1 The aim of this guideline is to assist doctors, nurses and pharmacists within Pennine Acute Trust in the management of adult patients with electrolyte disturbances.

2. Why do I need to know?

2.1 Electrolytes regulate the function of the neuromuscular, endocrine, and excretory systems. An electrolyte disturbance results from an imbalance in one or more of these ionised molecules, such as sodium, potassium, calcium or magnesium.

3. What is the guideline?

3.1 The guideline is presented as individual 'factsheets' that each describe the causes, symptoms, treatment and monitoring requirements of the following electrolyte disturbances: hypokalaemia, hyperkalaemia, hypocalcaemia, hypercalcaemia, hypomagnesaemia, hyponatraemia, hypernatraemia and hypophosphataemia.

3.2 In all cases, errors in serum electrolyte measurement should be excluded. An isolated deranged result may be spurious, and could be as a result of:

- blood samples been taken from a limb where there is already an infusion running
- haemolysed samples
- prolonged tourniquet time
- mislabelled samples

3.3 Where there is any question over compatibility of intravenous fluids with electrolytes, or for further advice regarding electrolyte disturbances, the Trust's Medicines Information Service can be contacted on ext 42152 or 78968.

Hypokalaemia

Potassium normal range: 3.5–5.4 mmol/L

Causes

- Medication – eg: thiazide diuretics, corticosteroids, salbutamol, theophylline
- Hypercorticolism – especially ectopic ACTH
- Renal tubular acidosis
- Intravenous fluids without potassium
- Laxative abuse
- Hyperaldosteronism
- Gastrointestinal losses
- Alkalosis

Symptoms

Usually asymptomatic, but severe hypokalaemia may cause muscle weakness, cramps, arrhythmias and polyuria.

Treatment

The underlying cause(s) should be identified and treated.
In most cases, withdrawal of diuretics or purgatives, accompanied by oral administration of potassium supplements is all that is required.

- **Mild hypokalaemia** (2.5–3.5 mmol/L, no symptoms)
  Give oral potassium chloride. Up to 80mmol per day may be required and should be guided by regular monitoring of serum potassium concentration.
  - eg: Sando-K® tablets (12mmol per tablet) 1–2 tablets two to three times a day
  - eg: Kay-Cee-L® syrup (1mmol/mL) 15–20mL three times a day

- **Severe hypokalaemia** (< 2.5 mmol/L or symptomatic)
  Urgent treatment is required. Give IV infusion of potassium.
  - eg: 40mmol potassium chloride in 1 litre sodium chloride 0.9% over 6–8 hours
  - Use readily-prepared IV fluids wherever possible - also see IV Potassium Policy.
  - Infuse at a maximum rate of 10mmol/hour in normal circumstances. A rate of 20mmol/hour may be used, but ECG monitoring is essential.
  - IV potassium must **never** be given as a fast bolus, as this may cause fatal arrhythmias.
  - Pain or phlebitis may occur during peripheral administration, particularly at higher concentrations, therefore it is generally recommended that infusion solutions containing > 40mmol of potassium per litre are given centrally.

Note: Inability to correct hypokalaemia may be due to concurrent hypomagnesaemia. Serum magnesium should be measured and any deficiency corrected.

Monitoring

Regular serum potassium concentrations – daily in severe hypokalaemia.
Renal function and electrolytes.

Caution

Hypokalaemia seriously increases the risk of digoxin toxicity.

Hyperkalaemia

Potassium normal range: 3.5–5.4 mmol/L

Causes

- Renal failure
- Addison's disease
- Medication – eg: ACE-inhibitors, angiotensin-II receptor antagonists, potassium-sparing diuretics, trimethoprim, NSAIDs, heparin, digoxin toxicity
- Burns
- Metabolic acidosis

Symptoms

May be asymptomatic, but can cause ECG changes, muscle weakness, hypotension or bradycardia.

Treatment

- **Severe hyperkalaemia** (≥ 6.5 mmol/L or ECG changes or severe symptoms [eg: muscle weakness / flaccid paralysis, palpitations, paraesthesia])
  Urgent treatment is required. Monitor ECG.
  - If there are ECG changes, give **10mls of 10% calcium gluconate** by IV bolus over 3 minutes*, preferably via a large vein, repeated as necessary after 5–10 minutes until ECG improves. This protects the cardiac membrane but has no effect on serum potassium concentration. Up to 50mL may be required.
  - Give **10 units of soluble insulin (eg: Actrapid®) with 25g of glucose** intravenously over 15 minutes.
    ie: 50ml of 50% glucose OR 125ml of 20% glucose OR 250ml 10% glucose
    This drives potassium into cells. Monitor hourly for hypoglycaemia after insulin administration, starting 15 minutes after the end of the infusion. Delayed hypoglycaemia may occur up to 6 hours after insulin administration.
  - **Nebulised salbutamol 10mg** (unlicensed use) can be added to help drive potassium into cells. This should have effect within 30 minutes and last for 2 hours.
  - If despite the above measures, potassium remains ≥ 6.5mmol/L or if ECG changes/symptoms persist, the renal or critical care team should be contacted for advice and to arrange urgent **dialysis** if appropriate.

- **Maintenance treatment / mild-moderate hyperkalaemia**
  Identify any underlying causes of hyperkalaemia and manage appropriately (eg: stop any offending drugs [see above] or foods such as bananas, chocolate).
  - Calcium polystyrene sulphonate resin (Calcium Resonium®) binds potassium in the gut and promotes potassium clearance.
  - Give 15g orally three to four times daily in water (not fruit squash which is high in potassium) until potassium levels are ≤ 5.5 mmol/L.
  - Can be administered concurrently with lactulose to increase potassium losses.

Monitoring

Serum potassium concentrations, renal function and electrolytes.
Blood glucose levels if insulin used.
Continuous cardiac monitoring in severe hyperkalaemia with ECG changes.

Caution

*Rapid calcium administration may precipitate myocardial digoxin toxicity, therefore if the patient is taking digoxin and the decision is made that calcium gluconate is required, it should be given slowly over 20 minutes mixed in 100ml of glucose 5%.

Hypocalcaemia

Calcium normal range: 2.10–2.60 mmol/L (**Note**: calcium is highly bound to plasma proteins, therefore if albumin is low, the corrected calcium should be used)

Causes

- Medication – eg: bisphosphonates, corticosteroids, oral contraceptives, phenytoin
- Hypoparathyroidism (eg: post-total thyroidectomy or post-parathyroidectomy)
- Chronic kidney disease
- Hyperphosphataemia
- Acute pancreatitis
- Vitamin D deficiency
- Hypomagnesaemia
- Over-hydration

Symptoms

May be asymptomatic, but can cause tetany, parasthesiae, cramps, anxiety, Chvostek and Trousseau signs, seizures, cardiac arrhythmias or vomiting.

Treatment

- **Severe / symptomatic hypocalcaemia** (≤1.9 mmol/L *or* with tetany)
  Medical emergency. Intravenous calcium required.
  - Add 10–20ml calcium gluconate 10% to 100mL sodium chloride 0.9% or glucose 5% and administer intravenously over 10 minutes with ECG monitoring, as there is a risk of arrhythmias if calcium given too rapidly.
  - Follow with an IV infusion of 100mL calcium gluconate 10% in 1L of sodium chloride 0.9% or glucose 5% at an initial rate of 50mL/hour, adjusted according to response.
  - The infusion may need to be repeated according to calcium levels and symptoms.

- **Mild / asymptomatic hypocalcaemia** (1.9–2.1mmol/L)
  Give oral calcium carbonate.
  - eg: Adcal® (15mmol per tablet) or Calcichew® (12.5mmol per tablet) 1 tablet two to three times a day, initially. Up to 100mmol per day may be required.

- **Identify and treat the underlying cause.**
  - If vitamin D deficiency (<25nmol/L) is the cause, consider commencing supplementation with oral colecalciferol 40,000 units per day for 7 days.
  - If hypomagnesaemic, correct as per page 10.

- **Patients with hypoparathyroidism or persistent hypocalcaemia post-parathyroidectomy / total thyroidectomy**
  - Larger doses of oral calcium supplements may be required (up to 150mmol/day)
  - If patient remains mildly hypocalcaemic (1.9–2.1mmol/L) beyond 72 hours post-operatively despite calcium supplementation, vitamin D supplementation (1α-derivatives) may be required eg: **alfacalcidol capsules 0.25–0.5microgram daily** initially, adjusted as necessary to achieve normocalcaemia and avoid hypercalcaemia.
  - Consider referring to Endocrinology team.

Monitoring

Serum calcium concentrations, renal function and electrolytes.
Parathyroid hormone and vitamin D levels should ideally be checked before initiating treatment for hypocalcaemia, but do not delay treatment by waiting for these results.

Caution

Rapid calcium administration may precipitate myocardial digoxin toxicity.
Large volume calcium infusions should not be used in patients with end stage renal failure or who are on dialysis – seek advice from renal team.

Hypercalcaemia

Calcium normal range: 2.10–2.60 mmol/L (*__Note__: calcium is highly bound to plasma proteins, therefore if albumin is low, the corrected calcium should be used)

Causes

- Hyperparathyroidism
- Addison's disease
- Medication – eg: thiazide diuretics, vitamin D, lithium, calcium-containing antacids
- Malignant disease
- Calcium-alkali syndrome

Symptoms

May be asymptomatic, but severe hypercalcaemia can cause bone pain, renal colic, nausea and vomiting, polyuria and altered consciousness.

Treatment

The underlying cause should be sought and managed appropriately. If possible, stop any offending drugs. Correct any hypokalaemia / hypomagnesaemia.

- **Rehydration**

Maintaining adequate hydration is the cornerstone of therapy, and may be all that is required.

- Give 4–6 litres of intravenous fluid over 24 hours, followed by 3–4 litres/day thereafter (caution in the elderly / patients with heart failure)
- Loop diuretics can be considered, only once rehydrated, if there is a danger of salt and water retention. eg: furosemide IV 40mg once or twice daily

- **Intravenous Bisphosphonates**

If the patient remains severely hypercalcaemic (>3.5mmol/L) or is symptomatic despite adequate rehydration, measures to reduce bone resorption will also need to be initiated.

- eg: Disodium pamidronate IV. Dose dependent on calcium levels:

Serum Calcium* (mmol/L)	Stat Dose of Disodium Pamidronate
< 3	15–30mg
3–3.5	30–60mg
3.5–4	60–90mg
> 4	90mg

- Dilute with sodium chloride 0.9% or glucose 5% to a concentration not exceeding 90mg/250mL.
- Infuse at a maximum rate of 60mg/hour (in renal impairment, reduce rate to 20mg/hour).
- Disodium pamidronate will lower calcium over 2–4 days, and achieve maximum effect within 1 week. If normocalcaemia is not achieved within this time, a further dose may be given.
- Zoledronic acid can be considered as an alternative in fluid-restricted patients.

Monitoring

Serum calcium concentration.
Renal function and electrolytes.

Hypomagnesaemia

Magnesium normal range: 0.7–1.0 mmol/L

Causes

- Deficient intake
- Gastrointestinal losses
- Diabetes
- Defective gut absorption
- Acute pancreatitis
- Alcoholism
- Medication – eg: diuretics, gentamicin, PPIs, ciclosporin, amphotericin B, cancer chemotherapy particularly cisplatin.

Symptoms

Irritability, tremor, cramps, hypokalaemia, parasthesiae, confusion, hallucinations, seizures, ECG changes.

Treatment

The underlying cause should be sought and treated if possible. Correct deficiency over 3–5 days, as magnesium equilibrates slowly within intracellular compartments.

- **Mild / asymptomatic hypomagnesaemia** (0.4–0.7 mmol/L)

Consider oral magnesium. Up to 50mmol / day may be required, but side effects, particularly diarrhoea, limit the dose.

  - Magnaspartate® (magnesium aspartate dihydrate) 10mmol sachets, 1 sachet once or twice daily.
  - Recheck serum magnesium levels after five days.

- **Severe** (<0.4mmol/L) **OR symptomatic hypomagnesaemia**

Give IV magnesium sulphate infusion. Up to 160mmol over 5 days may be required. Maximum rate of administration must not exceed 0.6mmol/minute.

  - Dilute to a convenient volume with sodium chloride 0.9% or glucose 5% (maximum concentration of 0.8mmol/mL)
    - Suggested regime for peripheral administration: Magnesium sulphate IV 20mmol in at least 250mL over 6–8 hours.
    - Administration on critical care areas and/or via a central line: Magnesium sulphate IV 20mmol in 100mL over 3 hours
  - May cause tissue damage if extravasation occurs.
  - After initial IV administration, it may be appropriate to give oral magnesium, as above, to correct the deficiency and replenish magnesium stores.

- **Life-threatening hypomagnesaemia** (eg: seizures / arrhythmias)

Urgent treatment is required. Give IV magnesium sulphate.

  - 8mmol in at least 10mL sodium chloride 0.9% IV over 10–15 minutes.
  - Follow with an infusion of IV magnesium sulphate, as above.

Monitoring

Serum magnesium concentration, daily in patients receiving IV magnesium.
Renal function and electrolytes.
Tendon reflexes.

Caution

Magnesium is renally excreted and may accumulate in patients with renal impairment. Reduce doses by 25–50% in patients with severe renal impairment and monitor carefully.

Hypermagnesaemia

Magnesium normal range: 0.7–1.0 mmol/L

Causes

- Excessive magnesium intake (including laxatives and antacids containing magnesium)
- Magnesium-containing enemas or bowel cleansing preparations in renal impairment
- Renal failure
- Lithium toxicity
- Rhabdomyolysis
- Diabetic ketoacidosis

Symptoms

Flushing, ECG changes, nausea, vomiting, drowsiness, absent deep tendon reflexes, hypotension, muscle paralysis, respiratory depression, coma.

Treatment

- **Mild / asymptomatic hypermagnesaemia** (2–4 mmol/L)

In patients with normal renal function, the following is all that is likely to be required:

  - Stop any sources of magnesium.
  - Maintain good urine output, with IV fluids +/- loop diuretic if necessary.
  - Re-check magnesium concentration after 24 hours.

In patients with impaired renal function, more intensive monitoring may be required.

- **Severe** (>4mmol/L) **OR symptomatic hypermagnesaemia**

Treat as above, plus give IV calcium to antagonise the neuromuscular and cardiovascular effects of magnesium.

  - Add 10ml calcium gluconate 10% to 100mL sodium chloride 0.9% or glucose 5% and administer intravenously over 10 minutes.
  - Should have an immediate effect on ECG changes, but the effect will be transient unless the serum magnesium concentration falls.
  - Repeat intravenous calcium gluconate if required.
  - Re-check magnesium concentration after 4 hours.

If the patient is still hypermagnesaemic, discuss with a renal or biochemistry consultant. Dialysis may be needed.

Monitoring

Serum magnesium and electrolyte concentrations – disturbances of magnesium rarely occur alone.
Renal function.
Arrhythmias will require continuous ECG monitoring

Caution

Rapid calcium administration may precipitate myocardial digoxin toxicity.

Acute Hyponatraemia (<48 hours*)

Sodium normal range: 136–145 mmol/L

**** If duration of hyponatraemia not known, and symptoms are consistent with severe, symptomatic hyponatraemia, then treat as below.***

Common Causes

Excessive fluids post-operatively, polydipsia, exercise, recently started drugs – eg: thiazide diuretics, bowel cleansing preparations, oxytocin, desmopressin, terlipressin, ecstasy/MDMA, PPIs

Symptoms

Severe: Vomiting, cardiorespiratory distress, severe somnolence, seizures, coma (GCS ≤8)
Moderate: Nausea, confusion, headache

Treatment

- **SEVERE, SYMPTOMATIC HYPONATRAEMIA (ie: Na^+<125mmol/L with severe symptoms)**

Call for senior help; initiation of treatment under the direction of a consultant only.
Patients should be treated where close clinical and biochemical monitoring can be achieved, such as ICU/HDU, A&E resus or MAU. Inform critical care team.

1. Immediate management

Give hypertonic saline to actively correct hyponatraemia:

- Infuse 250mL[#] sodium chloride 1.8% over 20 minutes[##] via a central line or large peripheral vein
- Recheck serum Na^+ concentration after 20 minutes, preferably using arterial blood gas (ABG) machine, whilst commencing a second infusion of 250mL sodium chloride 1.8% over 20 minutes.
- Repeat above steps twice, or until a rise in serum Na^+ concentration of 5mmol/L is achieved, or until the symptoms improve, whichever comes first.

[#] In patients with a deviant body composition (eg: low BMI), consider using 3mL/kg rather than fixed 250ml.
[##] Sodium chloride 1.8% is available as a 500mL polyfusor – it should be infused at a rate of 750mL/hr and stopped after 20 minutes. Alaris® pumps should be set to just deliver a fixed volume of 250mL.

2. Subsequent management

2.1. Symptoms improved:

- Stop hypertonic saline
- Establish cause of hyponatraemia and treat accordingly
- Recheck serum Na^+ concentration at 6, 12 and then every 24 hours
- Aim to increase serum Na^+ concentration by **no more than** 10mmol/L in the first 24 hours and by **no more than** 18mmol/L in the first 48 hours.

2.2. No symptom improvement:

- Continue sodium chloride 1.8% infusion (contains 308mmol/L Na^+), aiming for a 1mmol/L/hr rise, using the following equation to approximately calculate the rate:

$$\text{Initial infusion rate (mL/hr)} = \frac{[\text{body weight (kg)} \times 0.6\ (♂)\ \textbf{\textit{or}}\ 0.5\ (♀)] \times 1000}{\text{Infusate concentration (ie: 308)}}$$

- Recheck serum Na^+ concentration after 2 hours, and then every 4 hours.
- Aim to increase serum Na^+ concentration by **no more than** 10mmol/L in the first 24 hours and by **no more than** 18mmol/L in the first 48 hours.
- Stop hypertonic saline when a rise in serum Na^+ concentration of 10mmol/L (in total) is achieved, or when the symptoms improve, whichever comes first.
- If symptoms do not improve after a 10mmol/L rise in serum Na^+ concentration, it is likely they are caused by something other than hyponatraemia.

▪ MODERATE, SYMPTOMATIC HYPONATRAEMIA
(ie: Na^+<129mmol/L with moderate symptoms)

The aim here is to prevent a further decrease in serum Na^+ concentration rather than inducing a rapid increase.

- Start prompt diagnostic assessment and stop potentially causative medications, such as diuretics or ACE-inhibitors, where possible.
- On assessment, if there is a strong suspicion that new onset of symptoms and secondary to hyponatraemia, then consider administering a single infusion of 250mL sodium chloride 1.8% over 20 minutes *(NB: Sodium chloride 1.8% is available as a 500mL polyfusor. To administer 250mL, the polyfusor should be infused at a rate of 750mL/hr and stopped after 20 minutes. Alaris® pumps should be set to just deliver a fixed volume of 250mL.)*
- Patients should be treated where close clinical and biochemical monitoring can be achieved.
- The decision to use hypertonic saline in this group of patients should be made in agreement with the consultant responsible for the patient's care. Inform critical care team of potential need for further monitoring.
- Aim to increase serum Na^+ concentration by 5mmol/L in the first 24 hours, but by **no more than** 10mmol/L in the first 24 hours and by **no more than** 8mmol/L per 24 hours thereafter, until a serum Na^+ concentration of 130mmol/L is reached.
- Recheck serum Na^+ concentration at 1, 6 and 12 hours.

▪ NON-SYMPTOMATIC HYPONATRAEMIA
(ie: Na^+<135mmol/L without severe or moderate symptoms)

The absence of symptoms indicates that the brain has not yet developed clinically important oedema, but any further decline in serum Na^+ concentration may rapidly worsen the clinical situation.

- Treatment to be diagnosis-specific, as there is time for diagnostic testing
- Start prompt diagnostic assessment and stop potentially causative medications, such as diuretics, ACE-inhibitors or hypotonic fluids.

Monitoring

CNS observations.
Biochemical monitoring as specified above.

Caution

If hypokalaemia is also present, correction of the serum K^+ will contribute to an increase in serum Na^+ concentration.

Chronic Hyponatraemia (≥48 hours*)

Sodium normal range: 136–145 mmol/L

Common Causes

See table below and algorithm on page 15

Symptoms

Can include: gait disturbances, falls, mental slowing, concentration deficits, osteoporosis / bone fractures.

***NB**: symptoms of acute and chronic hyponatraemia may overlap, and chronicity might not always be known. If duration of hyponatraemia not known, and symptoms are consistent with severe, symptomatic hyponatraemia, then treat as per page 11.*

Treatment

Correct the underlying cause; never base treatment on sodium concentration alone. The treatment depends on the volume status of the patient, the presence of symptoms and the chronicity of the hyponatraemia.

Investigations: if possible, stop diuretics/angiotensin-II antagonists/ACE-inhibitors for 48 hours and then measure serum and urine osmolality, and urinary sodium. Assess clinically for volume status.

Volume status	Causes		Treatment
	Urine Na^+ ≤30mmol/L	Urine Na^+ >30mmol/L	
Hypovolaemic	▪ Vomiting ▪ Diarrhoea ▪ Fluid shifts (eg: pancreatitis ▪ Diuretics (urinary sodium measured 48hr after these stopped)	▪ Diuretics (if still taking) ▪ Salt-wasting renal disease ▪ Nephropathy (analgesics, pyelonephritis) ▪ Adrenal insufficiency	▪ Stop relevant drugs ▪ Restore volume with sodium chloride 0.9% ▪ If there is any suspicion of adrenal insufficiency, give hydrocortisone 100mg IV 6 hourly (without waiting for results of short Synacthen test)
Euvolaemic	▪ Acute water overload ▪ Excessive administration of hypotonic IV fluids	▪ Chronic water overload ▪ Chronic kidney disease ▪ Hypothyroidism ▪ Adrenal insufficiency ▪ Severe stress (eg: pain, surgery) ▪ SIADH (serum osmolarity <270mOsm/kg; urine osmolarity >100mOsm/kg) ▪ Ecstasy ingestion	▪ Stop relevant drugs ▪ Water restriction (1L/day) ▪ Oral sodium chloride (Slow Sodium®, starting at 600mg qds) +/- low-dose furosemide ▪ Consider demeclocycline (300mg tds–qds) if no improvement ▪ If demeclocycline ineffective, consider contacting Endocrinology team for advice on further management
Hypervolaemic	▪ Excess water intake ▪ Liver cirrhosis ▪ Heart failure ▪ Nephrotic syndrome	▪ Chronic kidney disease	▪ Treat underlying cause ▪ Sodium and water restriction (800mL–1L/day)

Monitoring

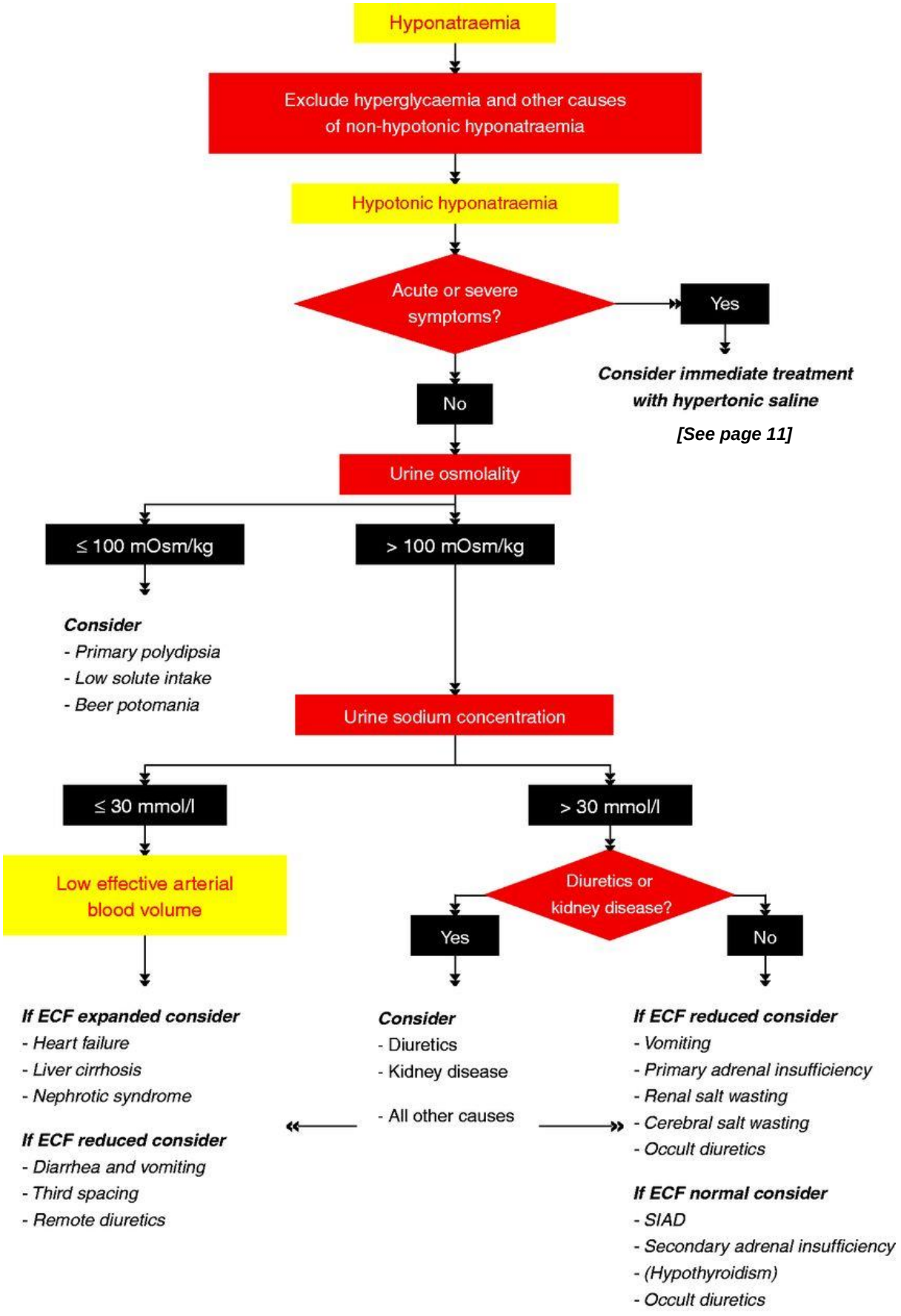
CNS observations.
Serum sodium concentration: re-assess every 6 hours when giving active treatment, until stabilised.
Fluid balance: take care to avoid fluid overload – cautious addition of a loop diuretic may need to be considered.

Caution

The rate of correction of hyponatraemia is important, since overly rapid correction of severe, chronic hyponatraemia can lead to the **osmotic demyelination syndrome** (central pontine myelinolysis):

- Typically begins 2–4 days after correction of severe, chronic (ie: >48hours) hyponatraemia where serum sodium is ≤120mmol/L.
- Symptoms include dysarthria, dysphasia, seizures, altered mental status, quadriparesis, 'locked-in syndrome' and hypotension.
- Can occur if chronic, severe hyponatraemia is corrected too quickly.
- May be complicated by hypoxia, so adequate oxygenation should be ensured.
- Serum sodium concentrations should not be increased by >10mmol in the first 24 hours.
- Therapeutic relowering of the serum sodium concentration with hypotonic fluids and desmopressin may be required if chronic hyponatraemia is inadvertently corrected too quickly. This should be under the direction of a specialist only.

Algorithm for the Diagnosis of Hyponatraemia, adapted from:
Clinical Practice Guideline on Diagnosis and Treatment of Hyponatraemia. European Journal of Endocrinology 2014; 170: G1-G47



Hypernatraemia

Sodium normal range: 136–145 mmol/L

Causes

- Diarrhoea and vomiting
- Burns
- Nephrogenic/central diabetes insipidus (which may follow head injury or pituitary surgery)
- Glucosuria in uncontrolled diabetes mellitus (eg: hyperosmolar hyperglycaemia status)
- Medication – eg: lithium, phenytoin, demeclocycline, corticosteroids
- Excessive administration of saline infusion fluids or drugs with a high sodium content (eg: IV benzylpenicillin, soluble / effervescent preparations)

Symptoms

Lethargy, thirst, confusion, fever, irritability, signs of dehydration.

Treatment

The treatment depends on the volume status of the patient and the underlying cause.
Aim to correct hypernatraemia over 48 hours.

- **Mild hypernatraemia** (<155mmol/L)

Drinking water may be enough in some patients.

- **Severe hypernatraemia / hypovolaemic** (ie: both salt and water loss; >155mmol/L)

In haemodynamically compromised patients, slow infusion of sodium chloride 0.9% should be used initially until systemic haemodynamics are stabilised.
Thereafter, glucose 5% or sodium chloride 0.45% can be used to correct the water deficit.

- **Severe hypernatraemia / dehydration** (ie: water loss; >155mmol/L)

Slow infusion of glucose 5%.

The following equation can be used to determine the initial fluid regimen:

a) **Water deficit (mL)** = [3mL x body weight (kg) x (serum sodium – 145)]

b) **Length of time to replace deficit (hr)** = $\frac{\text{(serum sodium – 145)}}{10}$ x 24

c) **Rate of replacement using glucose 5% (mL/hr)*** = $\frac{a}{b}$

+ water losses from stool and skin (40mL/hr)
+ ongoing water losses from GIT / urine, if can be estimated

*NB: double this rate if using sodium chloride 0.45%

Monitoring

Serial measurements of serum sodium concentration are required as these equations only provide a guide to initial therapy. Adjust rate of fluid replacement as appropriate.
Renal function, urea and electrolytes.

Caution

Too rapid correction of hypernatraemia can induce cerebral oedema – maximum rate of change is 10mmol/L per day.

Hypophosphataemia

Phosphate normal range: 0.8–1.45 mmol/L

Causes

- Malnutrition
- Hyperparathyroidism
- Vitamin D deficiency
- Diabetic ketoacidosis
- Alcohol abuse
- Refeeding syndrome
- Medication – eg: antacids, acetazolamide, phosphate binders

Symptoms

Muscle weakness, paraesthesia, tremor, confusion, arrhythmias

Treatment

- **Mild–moderate hypophosphataemia** (≤0.72 mmol/L)

Give oral phosphate.

  - Phosphate-Sandoz® tablets (16mmol phosphate per tablet) 1–2 tablets three times a day.
  - Dissolve tablets in approx 100mL of water.

  Note: each tablet also contains 20mmol sodium and 3mmol potassium

- **Severe / symptomatic hypophosphataemia** (≤0.32 mmol/L)

Give IV phosphate.

  - Phosphates polyfusor® (50mmol phosphate, 9.5mmol potassium, 81mmol sodium in 500mL)
  - Give 9mmol over 12 hours (ie: administer at a rate of 7.5mL/hr for 12 hours and then stop the infusion).
  - Infusion may need to be repeated on subsequent days, based on serum phosphate levels and symptoms.
  - Doses of 2–5mL/kg given over 12 hours may be required in more severe hypophosphataemia (max 30mmol/day).
  - In critical care areas, up to 50mmol may be given over 12 hours.

Monitoring

Serum phosphate, calcium and potassium concentration.
Renal function.
If IV phosphate is given peripherally, veins should be monitored for irritation.

Caution

Excessive doses of phosphate may cause hyperkalaemia, hypocalcaemia and metastatic calcification.

4. What do I need to do?

4.1 This document provides guidance to all staff dealing with patients with electrolyte disturbances. Treatment decisions should still be made on an individual basis and will ultimately be the responsibility of the doctor under which that patient is being cared.

4.2 Ward/Unit Managers and Clinical leads are responsible for investigating accidents, incidents and near misses in relation to failure to follow these guidelines and for reporting up to their Divisional Governance Committees where appropriate.

4.3 The relevant Divisional Governance Committees are responsible for reviewing incident reports within their remit and for the development, implementation and monitoring of action plans to address concerns where appropriate to ensure that lessons are learnt.

5. Abbreviations & Definitions of terms used

<	Less than
>	Greater than
≤	Less than or equal to
≥	Greater than or equal to
♀	Female
♂	Male
ACE	Angiotensin-converting enzyme
ACTH	Adrenocorticotropic hormone
BNF	British National Formulary
ECF	Extra-cellular fluid
ECG	Electrocardiogram
g	Gram
GIT	Gastrointestinal tract
hr	Hour
IV	Intravenous
kg	Kilogram
L	Litre
mg	Milligram
mL	Millilitre
mmol	Millimole
mOsm	Milliosmole
NSAID	Non-steroidal anti-inflammatory drug
PADAT	Pennine Acute Drug and Therapeutics Committee
PPI	Proton pump inhibitor

qds	Four times daily
SIAD	Syndrome of inappropriate antidiuresis
SIADH	Syndrome of inappropriate antidiuretic hormone secretion
tds	Three times daily

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7. Appendices

Appendix 1 – Equality Impact Assessment

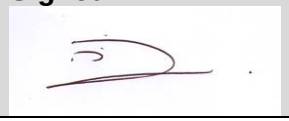
The Pennine Acute Hospitals NHS Trust

Equality Impact Assessment for Guideline for the Management of Electrolyte Disturbances in Adult Patients

For each of the Protected Characteristics & equality & diversity streams listed answer the questions below using Y to indicate yes and N to indicate no:	Age	Disability	Ethnicity / Race	Gender	Gender Reassignment	Marriage & Civil Partnership	Pregnancy & Maternity	Religion/belief	Sexual orientation	Human Rights	Carers	Please explain your justification
1. Does the practice covered have the potential to affect individuals or communities differently or disproportionately, either positively or negatively (including discrimination)?	N	N	N	N	N	N	N	N	N	N	N	These guidelines are intended for all and therefore do not discriminate against any of the protected characteristics.
2. Is there potential for, or evidence that, the proposed practice will promote equality of opportunity for all and promote good relations with different groups?	N	N	N	N	N	N	N	N	N	N	N	These guidelines are intended for all and therefore do not discriminate against any of the protected characteristics.
3. Is there public concern (including media, academic, voluntary or sector specific interest) in the document about actual, perceived or potential discrimination about a particular community?	N	N	N	N	N	N	N	N	N	N	N	These guidelines are intended for all and therefore do not discriminate against any of the protected characteristics.

Your Name: Sarah Boulger	**Your Designation:** Medicines Information Pharmacist	**Signed*:** 	**Date:** 27.11.15

To be completed by the relevant Equality Champion following satisfactory completion & discussion of answers above with author

Equality Champion: Michelle S Davis	**Directorate: Support Services**	**Signed*:** 	**Date:** 27.11.15