**Hypernatremia**

- (Plasma Na > 148 mmol/L)
- Reflects inadequate concentration of the urine in the face of restricted water intake.
- **Causes include:**
  - Failure of the ADH, either no ADH released from the pituitary (central or ‘cranial’ diabetes insipidus) or renal collecting duct cells are unable to respond to circulating ADH (nephrogenic diabetes insipidus, inherited or acquired).
  - Failure to generate an adequate medullary concentration gradient (low GFR states, loop diuretic therapy).

### Causes of hypernatremia

<table>
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<th>Volume status</th>
<th>Examples</th>
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| Hypovolemic (Na deficit with a relatively greater water deficit) | Renal Na losses
  Diuretic therapy (especially osmotic diuretic, or loop diuretic during water restriction)
  Glycosuria (HONK)
  Gastrointestinal Na losses
  Colonic diarrhea
  Skin Na losses
  Excessive sweating |
| Euvolemic (water deficit alone)   | Diabetes insipidus (central or nephrogenic)                               |
| Hypervolemic (Na retention with relatively less water retention) | Enteral or parenteral feeding
  I.V. or oral salt administration
  Chronic renal failure (during water restriction) |

### Hyponatremia and hypernatremia in old age

- **Decline in GFR:** older patients are predisposed to both hyponatremia and hypernatremia, mainly because, as GFR declines with age, the capacity of the kidney to dilute or concentrate the urine is impaired.
- **Hyponatremia:** occurs when free water intake continues in the presence of a low dietary salt intake and/or diuretic drugs (particularly thiazides).
- **ADH release:** water retention is aggravated by any condition which stimulates ADH release, especially heart failure. Moreover, the ADH response to non-osmotic stimuli may be brisker in older subjects. Appropriate water restriction may be a key part of the management.
- **Hypernatremia:** occurs when water intake is inadequate, due to physical restrictions preventing access to drinks and/or blunted thirst. Both are frequently present in patients with advanced dementia or following a severe stroke.
- **Dietary salt:** hypernatremia is aggravated if dietary supplements or medications with a high Na content (especially effervescent preparations) are administered. Appropriate prescription of fluids is a key part of the management.

### Clinical Features

- Reduced cerebral function, either as a primary problem or as a consequence of the hypernatremia itself, which results in dehydration of cerebral neurons & brain shrinkage.
- In the presence of an intact thirst & preserved capacity to obtain/ingest water, hypernatremia not progress very far.
- If adequate water is not obtained, dizziness, confusion, weakness & ultimately coma& death can result.

### Management

- Depends on both the rate of development & underlying cause.
- If the condition has developed rapidly, cerebral shrinkage may be acute, so relatively rapid correction with appropriate volumes of IVF (isotonic 5% dextrose or hypotonic 0.45% saline) can be attempted.
- In older, institutionalized patients it is more likely that the disorder has developed slowly & extreme caution should be used, to lower the plasma sodium slowly, to avoid the risk of cerebral edema in the osmotically adapted cerebral neurons.
- Where possible, the underlying cause should also be addressed.
- Elderly patients are predisposed, in different circumstances, to both hypernatremia & hyponatremia & a high index of suspicion is appropriate in aged patients with recent alterations in behavior.
Disorders of Potassium Balance: Hypokalemia

Investigations

- Occasionally the cause is obscure, especially with incomplete or unreliable history & when urine potassium is indeterminate.
- Many such cases are associated with metabolic alkalosis & urine chloride can be helpful.
- A low urine chloride (< 30 mmol/L) is characteristic of vomiting (spontaneous or self-induced, in which chloride is lost in HCl in the vomit), while a urine chloride > 40 mmol/L suggests diuretic therapy (acute phase) or a tubular disorder such as Bartter’s or Gitelman’s syndrome.
- Differentiation between these latter possibilities can be assisted by performing a screen of urine for diuretic drugs.

Features

- Patients with mild hypokalemia (plasma K 3.0–3.5 mmol/L) are generally asymptomatic.
- With more severe falls in the plasma potassium there is often muscular weakness, tiredness & typical ECG changes.
- Cardiac effects include ventricular ectopic beats or more serious arrhythmias & potentiation of the adverse effects of digoxin.
- Functional bowel obstruction may occur due to paralytic ileus.
- Long-standing hypokalemia damages renal tubular structures (hypokalemic nephropathy) & interferes with the tubular response to ADH.
Management

- Correction of the underlying cause.
- K replacement:
  - Slow-release KCl (if acidosis KHCO3) tablets in less severe, less acute cases.
  - MAGNESIUM SUPPLEMENTATION IF THERE IS Hypomagnesemia.
- Potassium-sparing diuretics as amiloride if it is due to loop diuretics
- In more acute circumstances IV potassium chloride infusion is necessary. Generally, not exceed 10 mmol /hour.
- If higher rates needed, K infusion may be increased to 40 mmol/L if a peripheral vein is used, but higher concentrations must be infused into a large ‘central’ vein with continuous cardiac monitoring.
- K infusions never given by direct IV & even never infused if there is no urine output or oliguria BZ it causes sudden death.

Hyperkalemia

- Significant hyperkalemia can be dangerous, because of the risk of cardiac arrest caused by the marked slowing of action potential conduction in the presence of potassium >7 mmol/L.
- Patients typically present with progressive muscular weakness, but sometimes there are no symptoms until cardiac arrest occurs.
- Typical ECG changes occur.
Investigations

Measurement of plasma electrolytes, bicarbonate, urine potassium & sometimes of plasma calcium & magnesium is usually sufficient to establish the diagnosis.

Plasma renin activity is low in patients with primary hyperaldosteronism & other forms of mineralocorticoid excess; in other causes of hypokalemia renin is elevated.

<table>
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<tr>
<th>Treatment of severe hyperkalemia</th>
<th>Mechanism</th>
<th>Therapy</th>
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<tbody>
<tr>
<td>Stabilize cell membrane potential 1</td>
<td>I.V. calcium gluconate (10 mL of 10% solution)</td>
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<tr>
<td>Shift K into cells</td>
<td>Inhaled B2 agonist, e.g. salbutamol</td>
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<td>I.V. glucose (50 mL of 50% solution) and insulin (5 U Actrapid)</td>
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<tr>
<td></td>
<td>Intravenous sodium bicarbonate 2</td>
<td></td>
</tr>
<tr>
<td>Remove K from body</td>
<td>I.V. furosemide and normal saline 3</td>
<td></td>
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<tr>
<td></td>
<td>Ion-exchange resin (e.g. Resonium) orally or rectally</td>
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<tr>
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<td>Dialysis</td>
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</table>

1 If ECG changes suggestive of hyperkalemia (K typically > 7 mmol/L).
2 If acidosis present.
3 If adequate residual renal function.

Questions (Answer with T or F):

- Both hypo & hypernatremia can be associated with all types of circulatory volume deficits. T
- There is risk of brain edema in hypernatremia & risk of brain shrinkage in hyponatremia. F
- Insulin excess, alkalosis & beta agonists cause hyperkalemia. F
- Pseudo-hyperkalemia occurs with hemolysis, leukocytosis & thrombocytosis. T
- Acidosis & hyperglycemia can cause hyperkalemia. T
- K should never be infused. F
- K should never be given directly intravenously. T
- The first treatment of hyperkalemia is to protect the heart by IV calcium.