

Department of Anesthesia Techniques

Electrolytes disturbance



Dr. Mohammed Sami Mohammed.sami.hasan@uomus.edu.iq

Hypokalaemia

Defined as plasma K⁺ <3.5mmol/L.

- Mild 3.0–3.5mmol/L
- Moderate 2.5–3.0mmol/L
- Severe <2.5mmol/L

Causes

1- Decreased intake.

2- Increased K+ loss—vomiting or nasogastric suctioning, diarrhea, pyloric stenosis, diuretics, renal tubular acidosis, hyperaldosteronism, Mg²⁺ depletion, leukemia.

3- Intercompartmental shift—insulin, alkalosis (0.1 increase in pH decreases K⁺ by 0.6mmol/L), β_2 -agonists, and steroids.

Clinical manifestations

ECG changes:

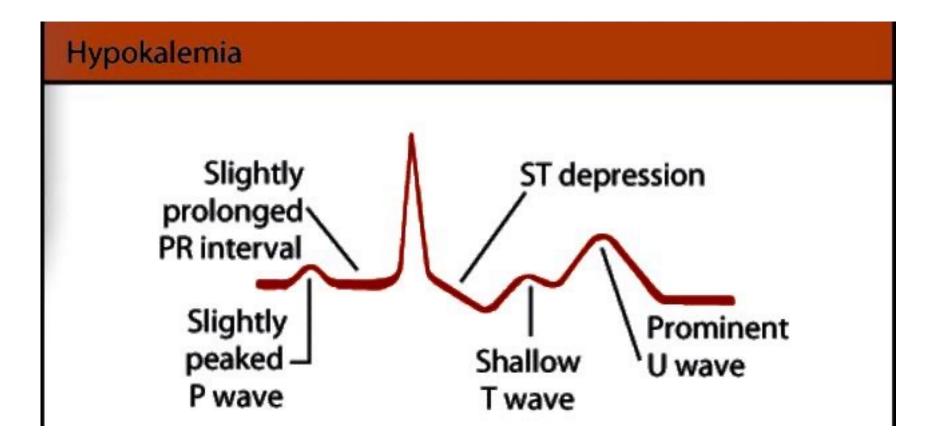
T wave flattening and inversion prominent U wave ST-segment depression prolonged P–R interval.

Dysrhythmias

decreased cardiac contractility.

Skeletal muscle weakness: tetany, ileus, polyuria, impaired renal concentrating ability, decreased insulin secretion, GH secretion, aldosterone secretion, negative nitrogen balance.

Encephalopathy in patients with liver disease.



Management

Check U&Es, creatinine, Ca²⁺, phosphate, Mg2+, HCO₃, and glucose if other electrolyte disturbances suspected.
Hypokalemia resistant to treatment may be due to concurrent hypomagnesaemia.

- Exclude Cushing's and Conn's syndromes.
- **Oral replacement** is safest, up to 200mmol/d, e.g. KCl two tablets qds = 96mmol K⁺.
- **IV replacement**—essential for patients with cardiac manifestations, skeletal muscle weakness, or where oral replacement not appropriate.

Aim to increase K^+ to 4.0mmol/L if treating cardiac manifestations.

Maximum concentration for peripheral administration is 40mmol/L (greater concentrations than this can lead to venous necrosis)

40mmol KCl can be given in 100mL of 0.9% NaCl over 1hr, but only via an infusion device, with ECG monitoring, in HDU/ICU/theatre environment, and via a central vein.

Plasma K⁺ should be measured at least hourly during rapid replacement.

 K^+ depletion sufficient to cause 0.3mmol/L drop in serum K^+ requires a loss of ~100mmol of K^+ from total body store.

Hyperkalaemia:

Defined as plasma $K^+ > 5.5 \text{mmol/L}$.

- Mild 5.5–6.0mmol/L
- Moderate 6.1–7.0mmol/L
- Severe >7.0mmol/L

Causes

- *Increased intake*—IV administration, rapid blood transfusion.
- *Decreased urinary excretion*—renal failure (acute or chronic), adrenocortical insufficiency

Drugs (K⁺-sparing diuretics, ACE inhibitors, ciclosporin, etc.).

• Intercompartmental shift of K^+

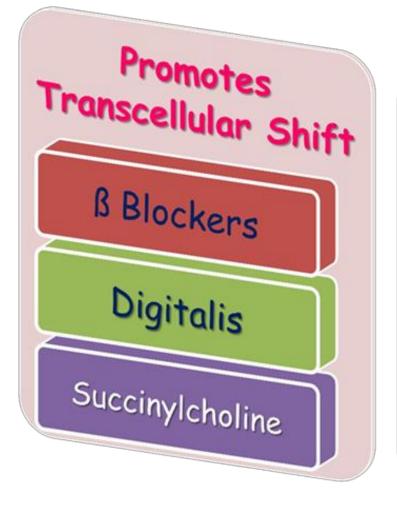
acidosis (H⁺ is taken into the cell, in exchange for K+), rhabdomyolysis

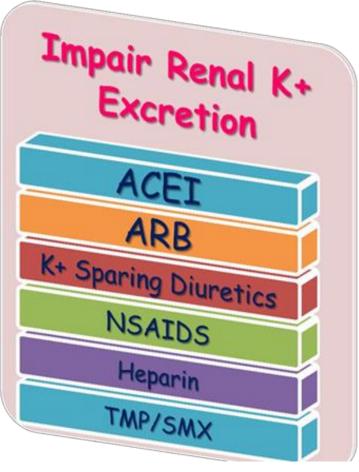
trauma

malignant hyperthermia (MH)

suxamethonium (especially with burns or denervation injuries) familial periodic paralysis.

• *Pseudohyperkalaemia*—due to in vitro haemolysis





Clinical manifestations

• ECG changes, progressing through peaked T waves widened QRS prolonged P–R interval loss of P wave loss of R wave amplitude ST depression? VF, asystole. ECG changes potentiated by low Ca^{2+,} low

Na⁺, and acidosis.

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

- Muscle weakness at K⁺ >8.0mmol/L.
- Nausea, vomiting, diarrhea.

Management

• Treatment should be initiated if K+>6.5mmol/L or ECG changes present.

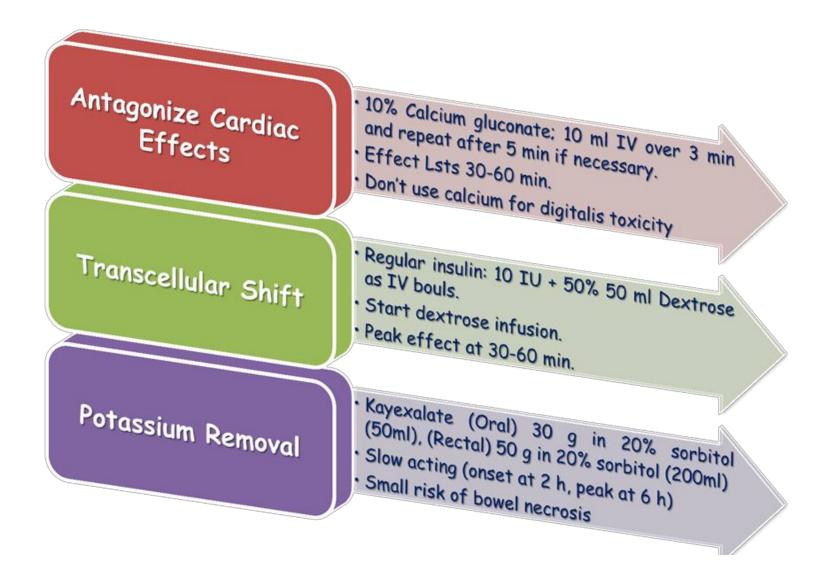
• Unlike hypokalaemia, the incidence of serious cardiac compromise is high, and therefore intervention is important. Treat the cause, if possible.

Ensure IV access and cardiac monitor.

• Insulin (10U in 50mL of 50% glucose IV over 30– 60min). This has the fastest onset of action and is very effective in reducing serum K⁺ by shifting the K⁺ into the cells. Beware rebound occurs within 2hr.

• β 2 -agonist—salbutamol (5–10mg nebulized—beware tachycardia). Should see a response at 30min and has a longer duration of action than insulin.

- •Ca²⁺ (5–10mL of 10% calcium gluconate or 3– 5mL of 10% calcium chloride).
- •Ca²⁺ stabilizes the myocardium by increasing the threshold potential. Rapid onset, shortlived.
- If acidotic, give HCO3⁻ (50mmol IV).
- Ion exchange resin—calcium resonium (15g P.O. or 30g per rectum (PR) 8-hourly). This binds K+ in the gut.
- If initial management fails, consider dialysis or haemofiltration.



Hyponatraemia

Defined as serum Na⁺ <135mmol/L.

- Mild 125–134mmol/L
- Moderate 120–124mmol/L
- Severe <120mmol/L

- •ECF volume is directly proportional to total body Na⁺ content.
- •Renal Na⁺ excretion ultimately controls the ECF volume and total body Na⁺ content.
- •To identify the causes of abnormalities of Na⁺ homeostasis, it is important to assess plasma and urinary Na⁺ levels, along with the patient's state of hydration (hypo-/eu-/hypervolaemic).

Causes

•Hypovolemic hyponatremia:

- •Urinary Na⁺ <30mmol/L suggests an extrarenal cause, i.e. diarrhea, vomiting, burns, pancreatitis, trauma.
- •Urinary Na⁺ >30mmol/L suggests a 1° renal problem, i.e. diuretic excess, osmotic diuresis, mineralocorticoid deficiency, saltwasting nephropathy, proximal renal tubular acidosis.

•Euvolemic hyponatremia

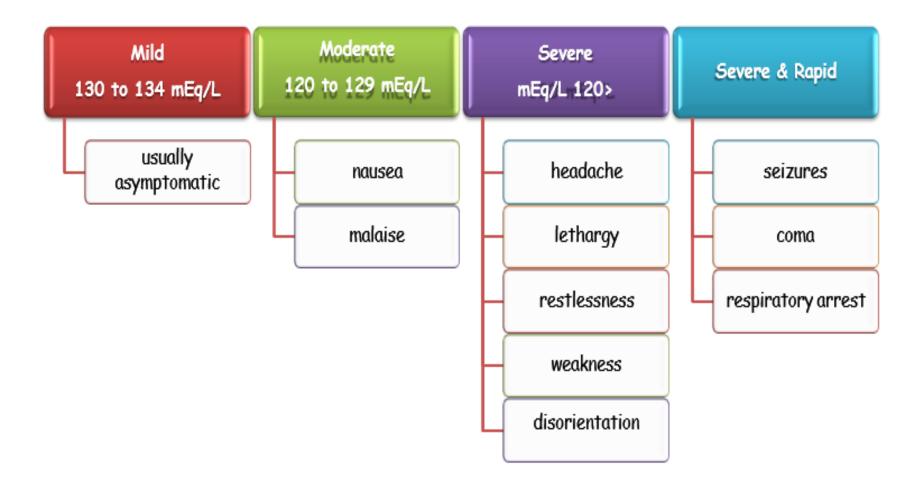
- Hypotonic fluid replacement post-surgery,
- hypothyroidism
- glucocorticoid deficiency
- syndrome of inappropriate antidiuretic hormone secretion (SIADH)
- psychogenic polydipsia.

•Hypervolemic hyponatremia

- ARF or CRF
- CCF
- Cirrhosis
- nephrotic syndrome
- transurethral resection of the prostate (TURP) syndrome.

Presentation

- Important to differentiate between **acute** and **chronic** hyponatraemia.
- Speed of onset is much more important for the manifestation of symptoms than the absolute Na⁺ level.
- Rare to get clinical signs if $Na^+ > 125 \text{mmol/L}$.
- Na⁺ 125–130mmol/L causes mostly GI symptoms, i.e. N&V.
- Na⁺ <125mmol/L—neuropsychiatric symptoms, N&V, muscular weakness, headache, lethargy, psychosis, raised ICP, seizures, coma, and respiratory depression.
- Mortality high, if untreated.



Treatment of symptomatic hyponatremia

• Acute symptomatic hyponatraemia

(develops in <48hr), e.g. TURP syndrome, hysteroscopyinduced hyponatraemia, SIADH.

Aim to raise serum Na⁺ by 2mmol/L/hr until symptoms resolve.

- Complete correction is unnecessary, although not unsafe.
- Infuse hypertonic saline (3% NaCl) at a rate of
- 1.2–2.4mL/kg/hr through a large vein.
- Measure Na⁺ levels hourly.
- In cases of fluid excess, give furosemide (20mg IV) to promote diuresis.
- If there are severe neurological symptoms (seizures, coma) 3% NaCl may be infused at 4–6mL/kg/hr.
- Electrolytes should be carefully monitored.

Chronic symptomatic hyponatraemia

- (present for >48hr or duration unknown).
- Aim to correct serum Na⁺ by 5–10mmol/d.
- Rapid correction (serum Na⁺ rise of >0.5mmol/L/hr) can lead to central pontine myelinolysis, subdural haemorrhage, and cardiac failure.
- If hypovolaemia is present, correct with 0.9% NaCl. This removes the antidiuretic hormone (ADH) response that accentuates the Na⁺/ water imbalance.

If hypervolaemic, treat with fluid restriction and furosemide.

Monitor electrolytes and urine output every 12hr.

For SIADH—fluid-restrict, and give demeclocycline (300–600mg daily).

- Consult with an endocrinologist.
- Watch for resolution of symptoms.
- Treat the cause.

Asymptomatic hyponatraemia (often chronic)

- Fluid-restrict to 1L/d.
- Treat the cause.

Hypernatremia

- Defined as serum Na⁺ >145mmol/L
 - Mild 145–150mmol/L
 - Moderate 151–160mmol/L
 - Severe >160mmol/L
- Caused by excessive salt intake or, more frequently, inadequate water intake.
- Important to assess the volume status.

Causes

Hypovolemic

- Renal—loop/osmotic diuretics, intrinsic renal disease, post- obstruction
- Extrarenal—diarrhoea/vomiting, burns, excessive sweating, fistulae.

Euvolemic

• Diabetes insipidus, insensible losses.

Hypervolemic

• Na⁺ ingestion/administration of hypertonic saline, Conn's syndrome, Cushing's syndrome.

Presentation

CNS symptoms likely if serum Na⁺ >155mmol/L due to hyperosmolar state and cellular dehydration, e.g. thirst, confusion, seizures, and coma.

Features depend on the cause, e.g. water deficiency will present with hypotension, tachycardia, and decreased skin turgor.

Management

Correct over at least 48hr to prevent occurrence of cerebral oedema and convulsions.

Treat the underlying cause. Give oral fluids (water), if possible.

- Hypovolaemic (Na⁺ deficiency): 0.9% NaCl until hypovolaemia corrected, then consider 0.45% saline.
- Euvolaemia (water depletion): estimate the total body water (TBW) deficit; treat with 5% glucose.
- Hypervolemic (Na+ excess): diuretics, e.g. furosemide (20mg IV) and 5% glucose;

dialysis if required.

• Diabetes insipidus—replace urinary losses, and give desmopressin (1–4 micrograms daily SC/IM/IV).

MCQ TEST

1- Causes of hypokalemia (all true except one)

- a) Vomiting
- b) Diarrhea
- c) diuretics,
- d) insulin
- e) acidosis
- 2- Signs and symptoms of severe hyponatremia (all true except one)a) neuropsychiatric symptoms
 - b) nausea/vomiting
 - c) muscular weakness
 - d) headache, lethargy
 - e) decreased intracranial pressure.
- 3- ECG changes in hyperkalemia (which one is true)
 - a) Absent T waves
 - b) Narrow QRS
 - c) short P–R interval
 - d) normal ST segment,
 - e) ECG changes potentiated by acidosis.

4- Management of hyperkalemia (all true except one)

- a) Salbutamol
- b) Calcium
- c) Insulin
- d) Intravenous potassium
- e) Dextrose
- 5- All the following drugs causes hyperkalemia except one (all true except one)
 - a) rapid blood transfusion.
 - b) renal failure (acute or chronic),
 - c) potassium sparing diuretics
 - d) thiopental
 - e) Succinylcholine