Lec. 16

Electrolytes Disturbance

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

- *Decreased intake*.
- *Increased K+ loss*—vomiting or nasogastric suctioning, diarrhea, pyloric stenosis, diuretics, renal tubular acidosis, hyperaldosteronism, Mg2+ depletion, leukemia.
- *Intercompartmental shift*—insulin, alkalosis (0.1 increase in pH decreases K+ by 0.6mmol/L), β2 -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, growth hormone 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+ >5.5mmol/L.

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

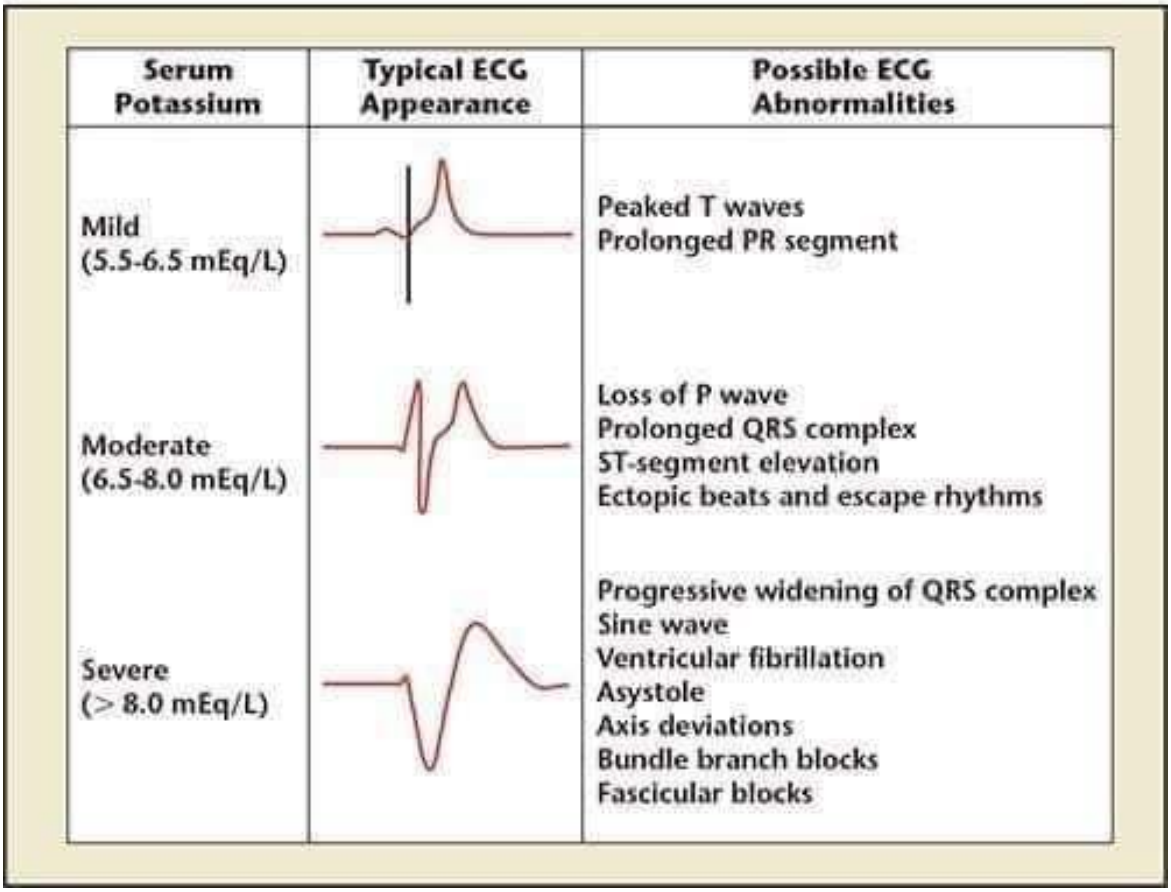
Causes

- *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

Promote Transcellular Shift	Impair Renal K^+ Execretion
β-Blockers	**ACEI (**Angiotensin-converting enzyme inhibitor)
Digitalis	**ARB (**Angiotensin receptor blockers)
Succenylcholine	**K^+ Sparing Diuretics**
	NSAIDs
	Heparin
	TMP/SMX (Trimethoprim/sulfamethoxazole)

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 Ca2+, low Na+, and acidosis.



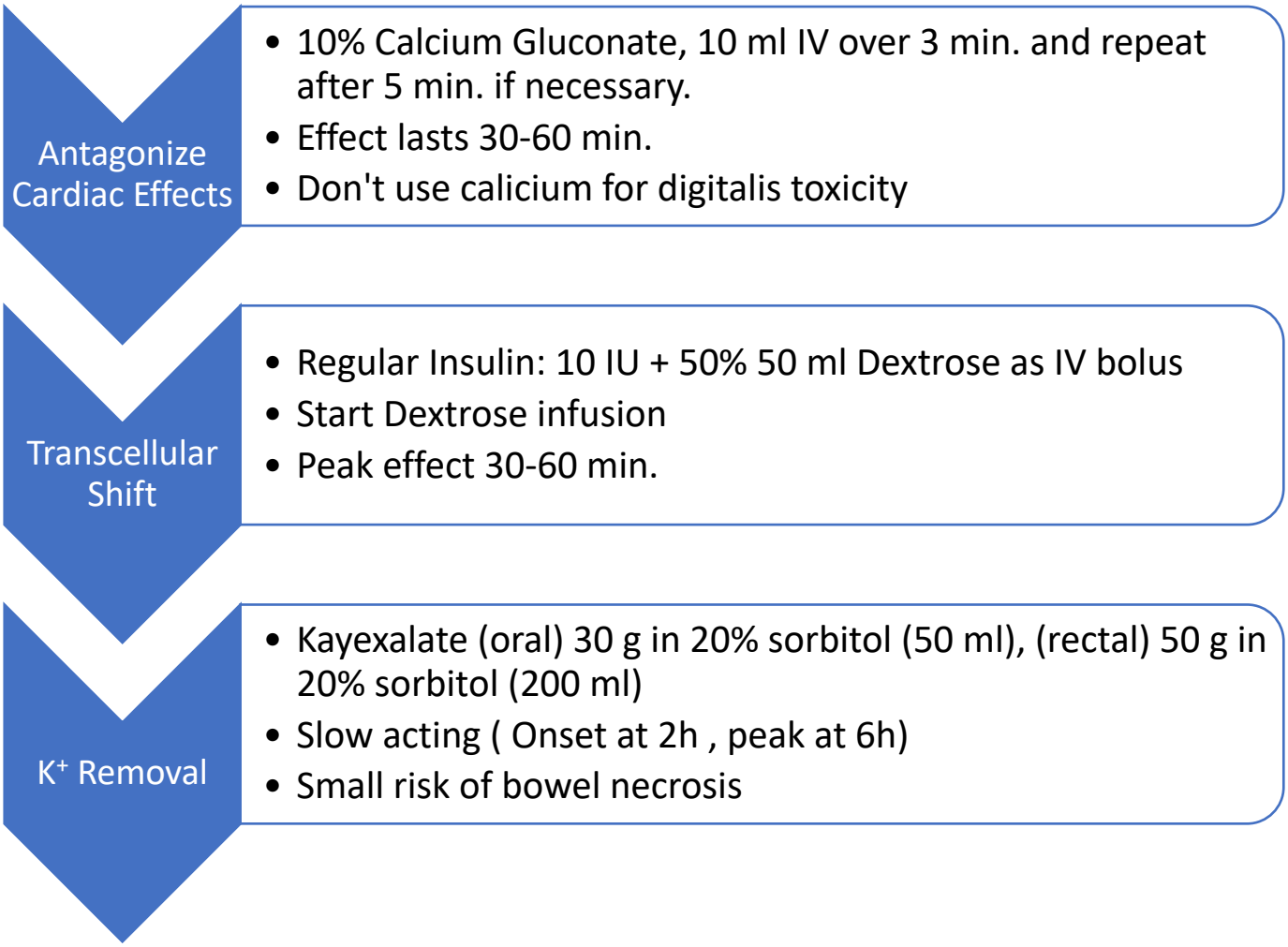
Serum Potassium	Typical ECG Appearance	Possible ECG Abnormalities
Mild (5.5-6.5 mEq/L)		Peaked T waves Prolonged PR segment
Moderate (6.5-8.0 mEq/L)		Loss of P wave Prolonged QRS complex ST-segment elevation Ectopic beats and escape rhythms
Severe (> 8.0 mEq/L)		Progressive widening of QRS complex Sine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

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

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.
- β2 -agonist—salbutamol (5–10mg nebulized—beware tachycardia). Should see a response at 30min and has a longer duration of action than insulin.5

- 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, short-lived.
- If acidotic, give HCO3 − (50mmol IV).
- Ion exchange resin—calcium resonium (15g PO 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/L6
- 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

Hypovolaemic hyponatraemia:

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

Euvolaemic hyponatraemia

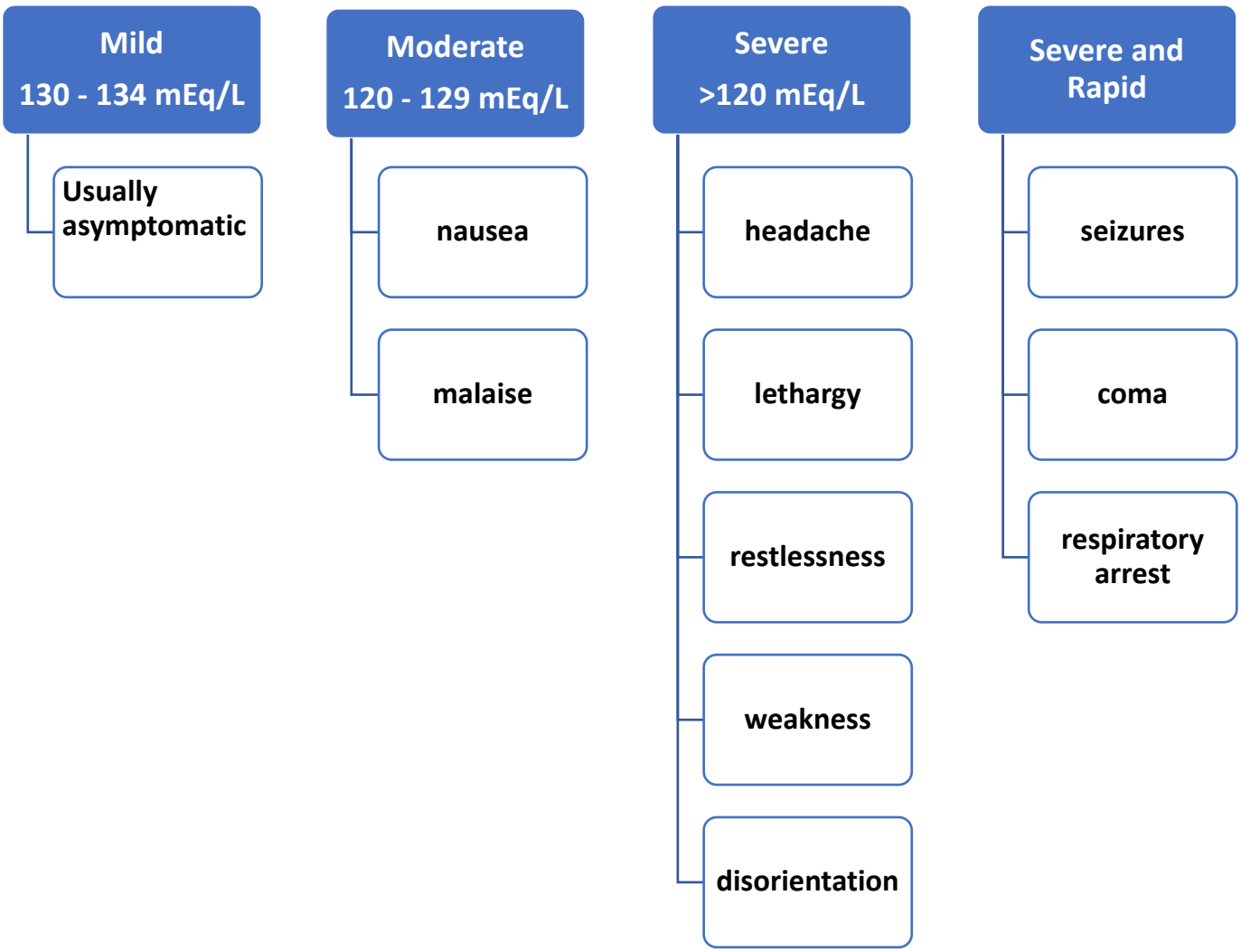
Hypotonic fluid replacement post-surgery, hypothyroidism, glucocorticoid deficiency, syndrome of inappropriate antidiuretic hormone secretion (SIADH), psychogenic polydipsia.

Hypervolaemic hyponatraemia

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+ >125mmol/L.7
- Na+ 125–130mmol/L causes mostly GI symptoms, i.e. nausea/vomiting.
- Na+ <125mmol/L—neuropsychiatric symptoms, nausea/vomiting, muscular weakness, headache, lethargy, psychosis, raised intracranial pressure, seizures, coma, and respiratory depression. Mortality high, if untreated.



Treatment of symptomatic hyponatraemia

- **Acute symptomatic hyponatraemia** (develops in <48hr), e.g. TURP syndrome, hysteroscopy-induced 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.

Hypernatraemia

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

Hypovolaemic

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

Euvolaemic

- Diabetes insipidus, insensible losses.

Hypervolaemic

- 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 hypo- tension, 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.
- Hypervolaemic (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