Hyperkalemia.

- Common electrolyte disorder, serum potassium >5 mmol/L (3.5 to 5 mmol/L).

Causes Potassium inside cell - 98%, outside - 2%.

$2K^+ \rightleftarrows 3Na^+$.

1) External Balance shift.
   1) Artefactual → During Venab puncture,
   2) Increased intake
      → Diet
      → ↑ Intravenous fluids.
   3) Decreased urinary excretion
      → Addisons disease
      → Kidney injury
      → Drugs inhibiting Renin, Angiotens
      → Tubulointerstitial disease.

2) Internal Balance shift.
   1) Diabete Mellitus Type I
   2) Acidosis
   3) Hyperosmolarity
   4) β_2 adrenergic receptor blocker
   5) Cell lysis
   6) Exercise.

Clinical features ↑ Resting Membrane Potential → ↑ Depolarization (Contraction)
↓ Repolarization

1) Smooth Muscle.
   ↓
   Severe Intestinal Cramps

2) Skeletal muscle
   ↓
   i) Weakness
   ii) Flaccid paralysis (Starts from lower and upward)

3) Cardiac muscle
   ↓
   - Arrhythmia
   - Cardiac arrest.

Investigations

ECG → ↑ T wave, ST depression. Short QT interval,
↓ Sodium → Addisons disease, Renal function.

Severe
↓
Prolonged PR Interval
Absent P wave
Widened QRS complex

Treatment

→ Insulin, Calcium, β adrenergic agonists, ^ Sodium Bicarbonate → Shift K^+ into cells.

1) External Balance shift

1) Artefactual → During venus puncture or thrombocytosis,
   -> Injury to cells → ↑ Potassium outflow in blood.

2) ↑Intake → Potassium rich intravenous fluids.
   Diet → Rich in potassium.

3) ↓ Excretion →

1) Hypoaldosteronism.
   (Adrenal insufficiency)

2) Addisons disease
   (Primary adrenocortical insufficiency)

↓ Aldosterone from Zona glomerulosa
↓
↑ Hyperkalemia & ↓ Hypokalemia.

2) Drugs inhibiting Renin, angiotensin, aldosterone.
   → Renin inhibitors, ACE inhibitors, Angiotensin II inhibitors, Selective aldosterone inhibitors, Potassium sparing diuretics

3) Acute & Chronic Kidney Injury
   ↳ ↓ GFR → ↓ oliguria & Hyperkalemia.

4) Tubulointerstitial disease →
   ↳ Interstitial nephritis, Diabetic nephropathy.

2) Internal Balance shift

1) Insulin deficiency (Diabetes Type I) → ↓ Intake of Glucose & Potassium by cells.

2) Acidosis → Too acidic (↓ PH) → ↑ H^+ ions in blood
   ↳ Compensatory mechanism → ↑ H^+ ions inside cells
   to increase pH. ↓
   ↑ K^+ outside cells in Blood

3) β 2 adrenergic receptors usually stimulates Na^+/K^+ pump increased Na^+ outside cells by catecholamines

↓

β 2 antagonists (β Blockers).

4) Hyperosmolarity inside cell → Throws it out in Blood

5) Cell lysis (Burns, Rhabdomyolysis (Breakdown of skeletal muscle), Tumor lysis (Radiotherapy).

6) During Exercise → Potassium comes outside cell due to usage of ATP.

Treatment

1) Calcium → Stabilize myocardial cell membrane.

2) IV Insulin, IV Glucose, Inhale β-adrenergic agonists, IV Sodium Bicarbonate → Shifts K^+ into cells.

↓ Shifting into cells.

3) Potassium Wasting Diuretics,

4) Excretion → Dialysis

Resins binding to K^+ and excrete through GIT.