Potassium: primary intracellular cation
→ 98% of total conc. is contained in ICF

Potassium physiological function
- Protein & glycogen synthesis
- Cell metabolism & growth
- Determination of resting membrane potential across membranes

Potassium homeostasis
- Na/K ATPase pump: responsible for maintaining basal intracellular to extracellular ratio
- Insulin and catecholamines: promote shift of K into cells
- Kidney: primary route of K elimination (long-term balance)
→chronic K disorder usually have renal/adrenal abnormality

Hyperkalemia

Classification:

Mild: 5.5 – 6.5 mmol/L
Moderate: 6.5 – 6.9 mmol/L
Severe: > 7 mmol/L

Common causes:
- ↑ K intake
- ↓ K excretion
- Tubular unresponsive to aldosterone
- Redistribution of K into extracellular space
- Drugs:
  - K supplements
  - B-blockers
  - Digoxin
  - NSAIDs
  - ACE inhibitors
  - Trimethoprim
  - Pentamidine
  - Cyclosporine
  - Tacrolimus

Clinical presentation
- CNS: paresthesia
- RESP: resp. difficulties
- CVS: palpitations, **ECG changes,** arrhythmia
- MSK: fatigue, weakness, leg cramps, muscular paralysis, depressed tendon reflexes

Goals of therapy
1. Antagonize adverse cardiac effects
2. Reverse any symptoms that may be present
3. Return serum & total body stores of K to normal

Therapeutic approach:
1. Verify hyperkalemia is true (not due to pseudohyperkalemia).
2. Determine acuity of hyperkalemia. Is it emergency situation?
3. Determine rate at which hyperkalemia developed.

Acute therapy treatment options:
1. Antagonist membrane toxic effects of potassium
   a. Calcium chloride: 10 mL of 10% IV
   b. Calcium gluconate: 10 mL of 10% IV
2. Promote cellular uptake of potassium
   a. Insulin/50% glucose: 10 units in 50g IV
   b. Salbutamol: 0.5 mg IV or 20 mg via neb
3. Remove potassium directly from the body
   a. Calcium resonium: 15-30 g PO/PR
   b. Sodium polystyrene sulfonate: 15g od-qid or 30-50g PR q6h
   c. Hemodialysis

Pseudohyperkalemia: produced due to release of potassium in process of drawing blood or from lysis of cell in blood prior to assay
→ associated with normal total body stores
→ can exclude by simultaneous measurement of K in plasma & serum [if serum K > plasma K by > 0.3 mmol/L]

Causes:
- Collection & storage of specimen
  - Pt clenched fist
  - Sample shaken or squirted through needle
  - Contamination w/ anticoagulant (potassium EDTA)
  - Cooling
  - Deterioration of specimen due to length of storage
- Pre-existing conditions
  - Thrombocytosis
  - Severe leukocytosis
  - Hereditary and acquired red cell disorders

Hypokalemia: most common electrolyte abnormality in hospitalized patients (20%)

Classification:

Mild: 3 – 3.5 mmol/L
Moderate: 2.5 – 3 mmol/L
Severe: < 2.5 mmol/L

Clinical presentation:

- CNS: ascending paralysis
- RESP: resp. difficulties
- CVS: ECG changes, arrhythmias
- GI: constipation
- GU: acute renal failure
- MSK: fatigue, weakness, leg cramps, rhabdomyolysis

Common causes

Decreased net intake (usual: 4700mg/d)
- Starvation
- Clay ingestion

Redistribution into cells
- Metabolic alkalosis from H+ loss (vomiting, NG suction)
- Hormonal (insulin, β2 agonists)
- Anabolic state

Increased net loss
- Non-renal
  - Direct loss of K+ from GI fluids
  - Plasma volume contraction → increase in aldosterone
  - Integumentary loss (sweat)
- Renal
  - Increase distal flow
  - Increase secretion of potassium

Drugs
- Transcellular K shift: pseudoephedrine, salbutamol, theophylline, insulin overdose
- Increased renal K loss: thiazides, furosemide, high-dose glucocorticoids (prednisone)
- Excess K in stool: sodium polystyrene sulfonate

Goals of therapy:

- To treat or prevent severe life-threatening signs & symptoms
- To restore serum potassium concentrations to normal
- To correct underlying cause of hypokalemia
- To prevent hyperkalemia

Therapeutic approach:

1. Estimation of the potassium deficit: serum K decreases by 0.3 mmol/L on average for every 100 mmol reduction in total body K stores

   Ex: serum K of 3.2 with a target-K of 4 = 0.8 deficit
   → ~ 300 mmoL reduction

2. Selection of appropriate preparation

Salt	Route	Dosage
K acetate	IV	4 mmol/mL
K chloride	IV	2 mmol/mL
	SR capsule	8 mmol/cap
	Liquid	20 mmol/15 mL
	SR tablet	20 mmol/tab
	Slow-release tablet	8 mmol/tab
K citrate	SR tablet	5 mmol/tab
		10 mmol/tab
	Effervescent tab	25 mmol/tab
	Liquid	10 mmol/5 mL
K gluconate	Caplet	2.5 mmol/cap
K phosphate	IV	4.4 mmol/mL

3. Selection of appropriate route of administration
   a. Oral route preferred
      → liquid poorly tolerated due to unpleasant taste, aftertaste, N, D, heartburn
   b. Life-threatening, use IV in <u>non-dextrose</u> containing solution

4. Selection of appropriate rate of administration

Mild-mod	40-100 mEq/day in divided doses		
Mod-sev	IV intermittent	General (central)	20 mEq/50 mL over 1h
		General (peripheral)	20 mEq/250 mL over 1h
		Critical/special care (central)	40 mEq/100 mL over 1h
	IV infusion	Peripheral line	20-40 mEq/L at max rate 10 mEq/hr
		Central line	20-60 mEq/L at max rate 20 mEq/h
Preventative	20-40Eq/day		

5. Monitoring plan
   a. Outpatient: serum creatinine q1-2 months
   b. Inpatient: serum K on daily basis
   c. Low K & IV replacement: K 30 min after infusion

Hypomagnesemia: < 0.75 mmol/L

Status	Route	Dosage
Mild-mod (0.5-0.69)	PO	25-35 mEq/day
		Mg glucoheptonate: 60-90 mL/day in 3-4 div doses
		Mg complex: 300-400 mg/day in 2-3 div doses
	IV	5g Mg sulphate (20 mmol) in 100 mL D5W/ NS over 3-4 h (daily x 1-3 doses)
Severe (<0.5)	IV	5g Mg sulphate (20 mmol) in 100 mL D5W/ NS over 3-4 h (q12-24h x 1-3 doses)
Renal insufficiency	IV	2 g Mg sulphate (8 mmol) in 50 mL D5W/ NS over 30-60 min
		5 g Mg sulphate (20 mmol) in 100 mL D5W/ NS over 3-4 h x 1 dose

NOTE: in patients with hypokalemia and hypomagnesia, if you don't correct the Mg, you will never be able to rectify the hypokalemia

Hypermagnesemia: > 0.85 mmol/L

- Discontinuation of magnesium
- If neuromuscular and cardiovascular effects: IV administration of calcium
- Hemodialysis
- ICU support