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

**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

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

**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

**Hyperkalemia**

<table>
<thead>
<tr>
<th>Classification:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild: 5.5 – 6.5 mmol/L</td>
</tr>
<tr>
<td>Moderate: 6.5 – 6.9 mmol/L</td>
</tr>
<tr>
<td>Severe: &gt; 7 mmol/L</td>
</tr>
</tbody>
</table>

**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
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**

<table>
<thead>
<tr>
<th>Salt</th>
<th>Route</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>K acetate</td>
<td>IV</td>
<td>4 mmol/mL</td>
</tr>
<tr>
<td>K chloride</td>
<td>IV</td>
<td>2 mmol/mL</td>
</tr>
<tr>
<td></td>
<td>SR capsule</td>
<td>8 mmol/cap</td>
</tr>
<tr>
<td></td>
<td>Liquid</td>
<td>20 mmol/15 mL</td>
</tr>
<tr>
<td></td>
<td>SR tablet</td>
<td>20 mmol/tab</td>
</tr>
<tr>
<td></td>
<td>Slow-release tablet</td>
<td>8 mmol/tab</td>
</tr>
<tr>
<td>K citrate</td>
<td>SR tablet</td>
<td>5 mmol/tab</td>
</tr>
<tr>
<td></td>
<td>Effervescent tab</td>
<td>10 mmol/tab</td>
</tr>
<tr>
<td></td>
<td>Liquid</td>
<td>10 mmol/5 mL</td>
</tr>
<tr>
<td>K gluconate</td>
<td>Caplet</td>
<td>2.5 mmol/cap</td>
</tr>
<tr>
<td>K phosphate</td>
<td>IV</td>
<td>4.4 mmol/mL</td>
</tr>
</tbody>
</table>

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 non-dextrose containing solution

4. **Selection of appropriate rate of administration**

<table>
<thead>
<tr>
<th>Mild-mod</th>
<th>40-100 mEq/day in divided doses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mod-sev</td>
<td>IV intermittent General (central)</td>
</tr>
<tr>
<td></td>
<td>General (peripheral)</td>
</tr>
<tr>
<td></td>
<td>Critical/special care (central)</td>
</tr>
<tr>
<td>IV infusion</td>
<td>Peripheral line</td>
</tr>
<tr>
<td></td>
<td>Central line</td>
</tr>
</tbody>
</table>

   | 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

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

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