MANAGEMENT OF HYPERKALEMIA

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POTASSIUM BALANCE

- K+ is the major **intracellular cation**
- Intracellular K+ is maintained at a high concentration by the 3Na, 2K-ATPase pump
- 95% to 98% of total body K+ is stored intracellularly
- 80% of K+ excretion occurs **via the kidney**, with the remainder in the stool and sweat
- Renal K+ excretion increased by aldosterone
- Increased Na+ and water delivery to the distal nephron increases K+ excretion
- A **healthy blood potassium level** is 3.6 to 5.2 millimoles per liter (mmol/L)

DEFINITIONS:

- **Hyperkalemia**: Serum potassium level > 5 mEq/L
- **Acute hyperkalemia**: Abnormal ↑ K+ not known to be chronic
- **Chronic hyperkalemia**: Recurrent episodic ↑ K+ that require ongoing treatment

ETIOLOGY

☒ Potassium Excess:

due to altered K+ metabolism or intake

- **Reduced excretion:** acute and chronic kidney disease
- **Endocrine causes:** hypocortisolism, hypoaldosteronism
- **Drugs:** potassium-sparing diuretics, ACE inhibitors, angiotensin receptor blockers, NSAIDs, and trimethoprim-sulfamethoxazole
- **Type IV renal tubular acidosis**
- **Increased intake :**

  - High potassium diet, e.g., fresh fruits, dried fruits and legumes, vegetables, nuts, seeds, bran products, milk, and dairy products
  - K+ containing IV fluids

ETIOLOGY

☒ Extracellular Shift:

- **Acidosis** → ↑ extracellular H+ → inhibition of the Na+/H+ antiporter → ↓ intracellular Na+ → ↓ sodium gradient inhibits the Na+/K+-ATPase → ↑ extracellular K+ concentration

- Hyperkalemia → ↑ extracellular K+ concentration → ↑ potassium gradient stimulates the Na+/K+-ATPase → ↑ extracellular Na+ → ↑ sodium gradient stimulates the Na+/H+ antiporter → ↑ extracellular H+ → acidosis

- **Exceptions:** In renal tubular acidosis and acetazolamide toxicity, findings include hypokalemia and metabolic acidosis.

- **Hyperosmolality**
- **Insulin deficiency** (manifests with hyperglycemia)

- **Drugs:**

- Beta blockers
- Succinylcholine: (esp. when given with preexisting burns and/or muscle trauma) ,
- Digoxin: inhibits the Na+/K+-ATPase → ↑ extracellular K+ concentration

ETIOLOGY

☒ Extracellular Release:

- **Pathological cell lysis**

- Rhabdomyolysis
- Tumor lysis syndrome
- Hemolysis

- **High blood cell turnover:** e.g., thrombocytosis, erythrocytosis, leukocytosis
- **Pseudohyperkalemia:** resulting from iatrogenic red blood cell lysis
  - Blood drawn from the side of IV infusion or a central line without previous flushing
  - Prolonged use of a tourniquet
  - Fist clenching during blood withdrawal
  - Delayed sample analysis

NOTES:

☒ **Errors in blood-drawing technique** may lead to red blood cell lysis and a falsely elevated serum potassium concentration **(pseudohyperkalemia)!**

☒ **When K+ shifts out of the cell,** it's a **BAD LOSS!** – **B**eta blockers, **A**cidosis, **D**igoxin, **L**ysis, hyper**O**smolality, high **S**ugar, **S**uccinylcholine

PATHOPHYSIOLOGY:

☒ Potassium is an important factor in maintaining the resting membrane potential

☒ ↑ Extracellular K+concentration → resting membrane potential becomes **less negative** than -90 mV → ↑ excitability

☒ Particularly **acute** extracellular changes in concentration influence excitability! Chronic changes lead to intracellular compensation!

CLINICAL FEATURES

☒ Symptoms usually occur if serum potassium levels are **> 7.0 mEq/L** or they change rapidly.

– Cardiac arrhythmias (e.g., atrioventricular block, ventricular fibrillation)

– Muscle weakness, paralysis, paresthesia

– ↓ Deep tendon reflexes

– Nausea, vomiting, diarrhea

☒ **Hyperkalemia (and hypokalemia)** can cause **cardiac arrhythmia** and lead to ventricular fibrillation!

LABORATORY STUDIES

- **BMP**
  - **Glucose:** If very high, consider spurious hyperkalemia secondary to hyperglycemic crisis.
  - **Serum electrolytes**
    - **Na+:** normal or can be ↓ in adrenal insufficiency
    - **K+:** Repeat to confirm the diagnosis and rule out pseudohyperkalemia
  - Renal function tests: often show renal impairment
- **CBC**: can show hemolytic anemia or thrombocytosis
- **Liver chemistries:** may be abnormal in hemolysis or tumor lysis syndrome
- **Blood gases** (venous or arterial): often show metabolic acidosis

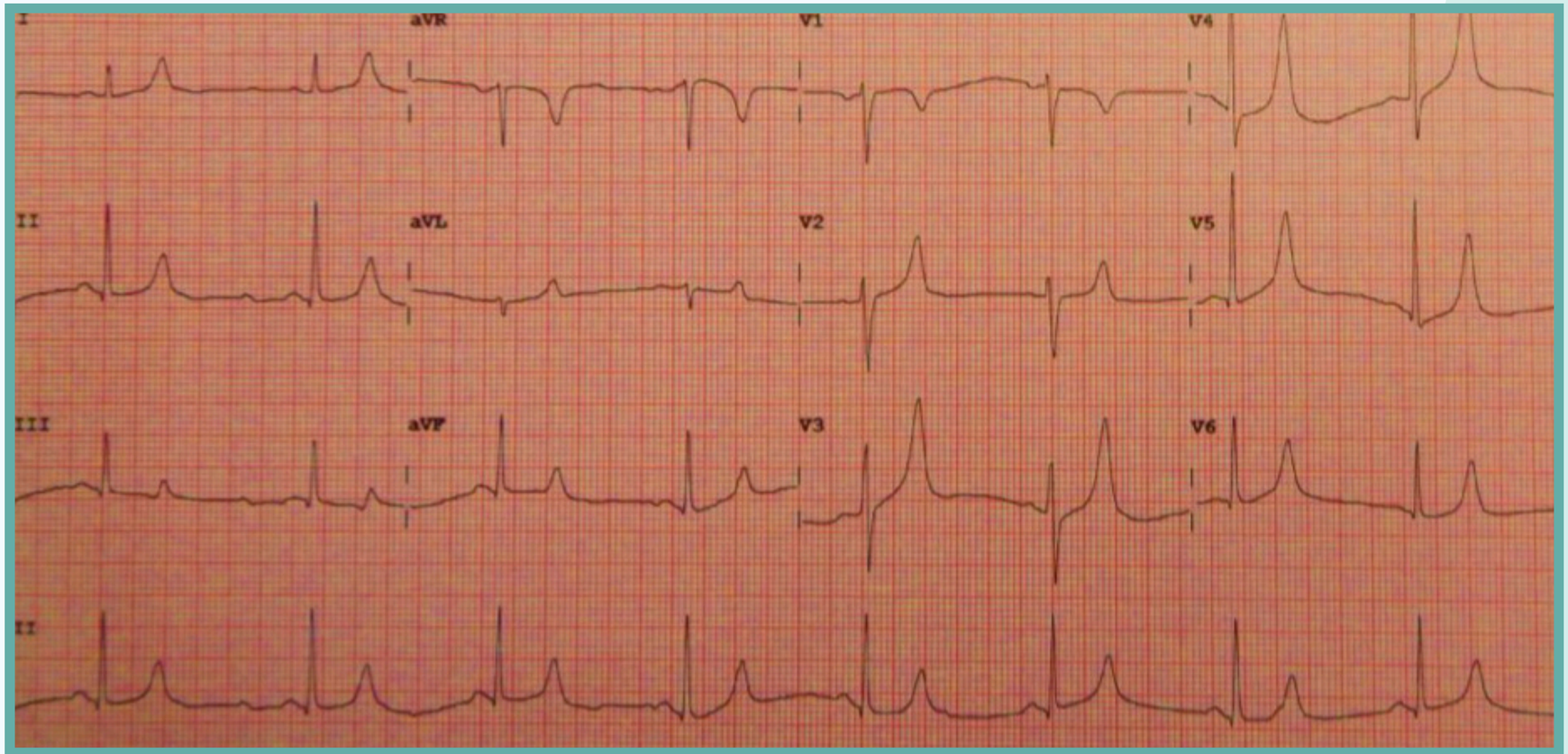
An inverse relationship between serum K+ and pH (e.g., **↓ pH → ↑ K+**) has previously been observed in specific types of metabolic acidosis. However, the underlying mechanisms
are complex and this association is inconsistent in clinical practice

DIAGNOSIS

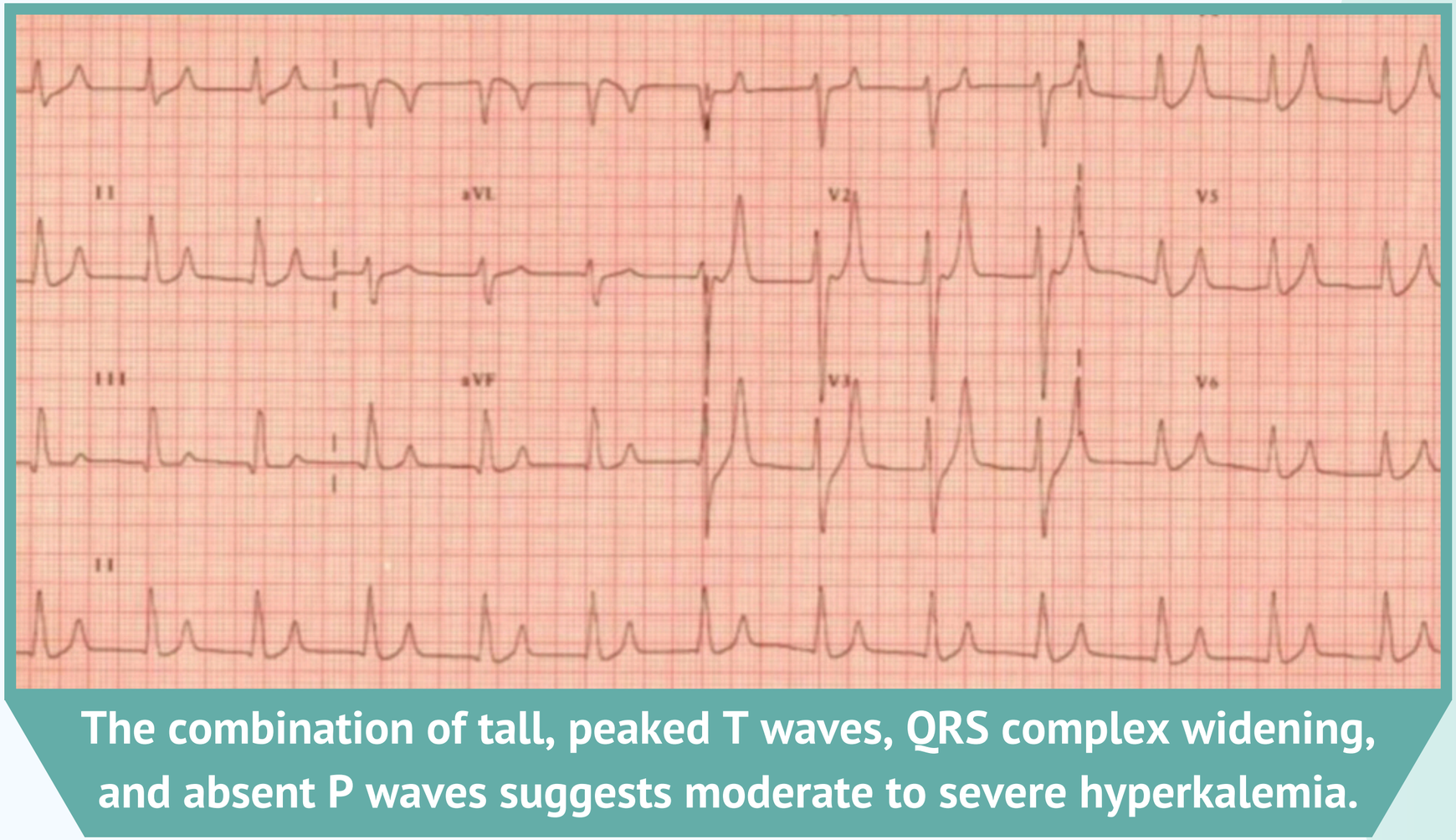
ECG Findings In Hyperkalemia

There is a weak correlation between serum K+ levels and the severity of ECG changes. Findings are more likely to occur with rapid-onset hyperkalemia.

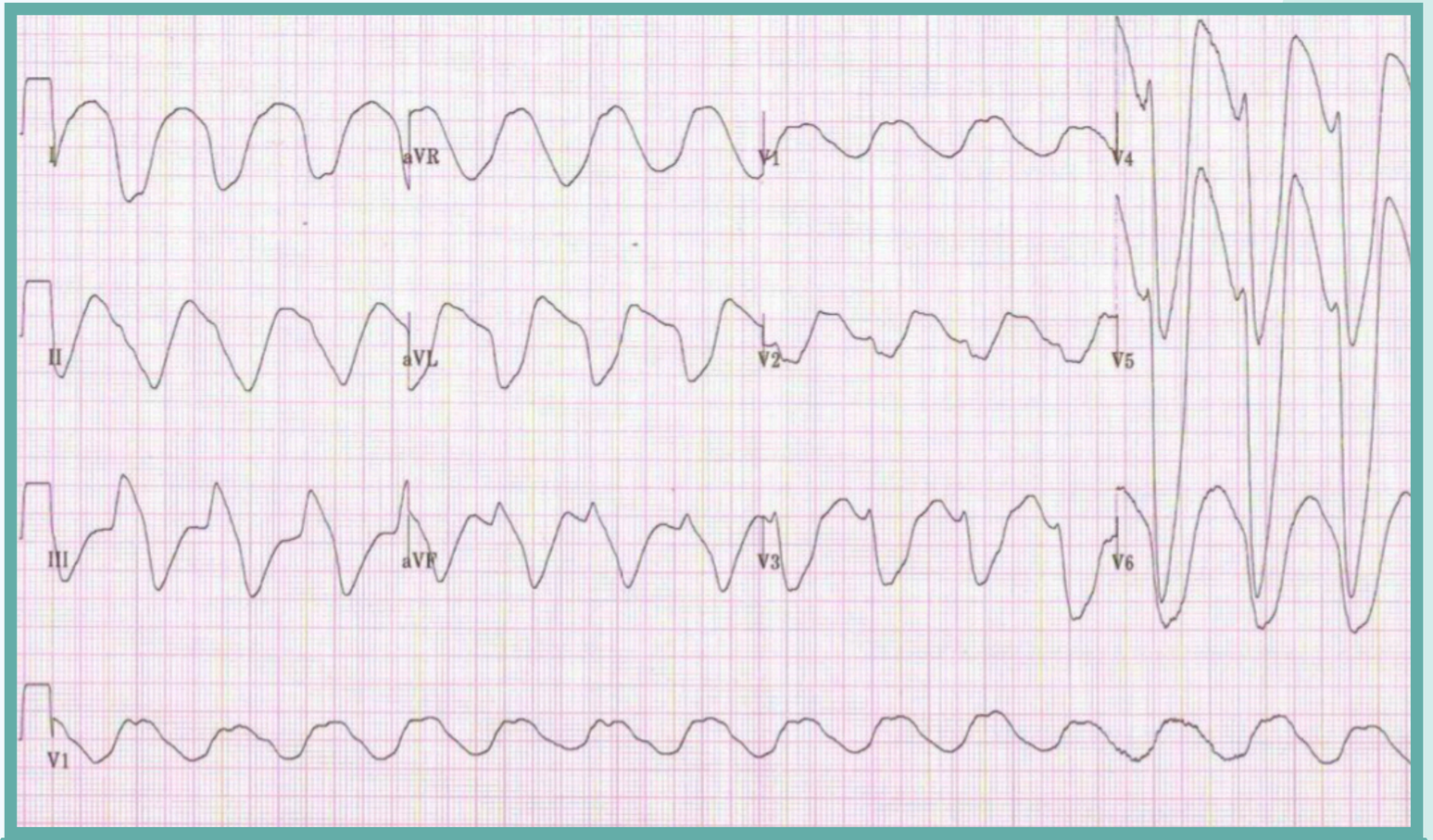
- **Mild hyperkalemia: 5.5–6.4 mEq/L**
  - Tall, peaked T waves
- **Moderate hyperkalemia: 6.5–8.0 mEq/L**
  - Lengthening of QRS interval (QRS complex widening)
  - Widening and flattening of P wave, which eventually disappears
- **Severe hyperkalemia: > 8.0 mEq/L**
  - Absent P wave
  - Intraventricular conduction block
  - Unusual QRS morphology
  - Sine wave pattern: a sinusoidal pattern with absent P waves and a wide QRS complex that merges with the T wave; a marker of impending V-Fib and asystole.
  - Cardiac arrhythmias (e.g., V-tach, V-fib), asystole



Tall, peaked T waves are suggestive of hyperkalemia



The combination of tall, peaked T waves, QRS complex widening, and absent P waves suggests moderate to severe hyperkalemia.



Fusion of the widened QRS complexes and the T waves creates a sinusoidal pattern

The sine wave pattern is suggestive of severe hyperkalemia and is a marker of impending ventricular fibrillation or asystole

DIAGNOSIS

Investigation of Underlying Causes

Depending on symptoms and risk factors, further testing may be appropriate, particularly if renal function is normal.

- ☒ **Creatine kinase:** ↑ in rhabdomyolysis
- ☒ **LDH:** ↑ in tumor lysis syndrome or hemolysis
- ☒ **Renin-angiotensin-aldosterone system :**
  - ↑ Aldosterone: suggestive of, e.g., pseudohypoaldosteronism or nephropathy due to sickle cell disease
  - ↓ Aldosterone: Assess plasma renin activity or plasma renin concentration
  - Normal or ↑ renin: suggests, e.g., hypoaldosteronism (e.g., due to Addison disease) or congenital adrenal hyperplasia
  - ↓ Renin: suggests, e.g., AIN, diabetic nephropathy
- ☒ **Cortisol:** can be ↓ in primary adrenal
- ☒ **Urine electrolytes:** rarely indicated

THERAPEUTIC APPROACH TO HYPERKALEMIA

☒ Determine severity: **By "Risk stratification."**

☒ **Hyperkalemic emergency**: Patients require immediate management.

- If there are ECG changes: Stabilize the cardiac membrane first, e.g., with IV calcium gluconate.
- Initiate treatment to shift potassium intracellularly, e.g.:
  - Short-acting insulin with glucose
  - Consider the addition of inhaled SABAs.

All Patients

☒ **Identify and treat underlying causes.**

☒ **Review medications and discontinue or modify dosing** of medications that may be contributing to hyperkalemia.

— Decrease dose (or consider discontinuation) of drugs required to treat underlying conditions, e.g., RAAS inhibitors.

— Avoid nonessential drugs associated with hyperkalemia (NSAIDs, over-the-counter supplements such as milkweed).

☒ **Start a low potassium diet and avoid salt substitutes .**

☒ **Consider treatment to remove potassium from the body**, e.g., cation-exchange resins, diuretics, hemodialysis.

☒ Repeat potassium regularly until it is within normal range

Risk Stratification

☒ **Hyperkalemic emergency** is an acute severe elevation that requires urgent lowering and occurs if any of the following are present:

- **Clinical manifestations:** ECG changes in hyperkalemia, muscle weakness, paralysis
- **Serum K+ > 6.0–6.5 mEq/L**
- **Comorbidities** that affect ongoing K+ influx and elimination: e.g., AKI, ESRD, GI bleeding, rhabdomyolysis, TLS

☒ **Less urgent hyperkalemia** (typically chronic elevations that can be lowered more slowly)

- **Patient is asymptomatic**
- **Serum K+ is 5.5–6.0 mEq/L**
- Patient has **no high-risk comorbidities**

☒ **Cardiac arrhythmias** due to hyperkalemia can cause sudden death.

Cardiac Membrane Stabilization

☒ Calcium salts reduce cardiac irritability.

☒ Indication: **ECG changes in hyperkalemia**

☒ Options :

- **10% calcium gluconate**
- **10% calcium chloride**

☒ Calcium salts have no influence on serum K+ levels and therefore should be paired with a K+ lowering agent.

INTRACELLULAR POTASSIUM SHIFTING

These drugs should be given in tandem with calcium salts (if calcium is indicated).

☒ **Insulin and Glucose**

- Preferred acute noninvasive K+ lowering treatment
- Sample agent: short-acting insulin combined with 50% dextrose

☒ **Inhaled SABAs**

- Consider as an adjunct to insulin (not effective as a monotherapy).
- Sample agent: nebulized albuterol

☒ **Sodium bicarbonate**

- Consider as an adjunct treatment only in patients with severe metabolic acidosis.
- Recommended concentration is IV 8.4% sodium bicarbonate

ENHANCED POTASSIUM ELIMINATION

Not required for all patients; treatment of the underlying cause may be sufficient.

The choice of treatment depends on underlying medical conditions and volume status.

☒ Cation-Exchange Medications :

- *Mechanism of action*: These drugs release Na+ or Ca2+ ions in the gut, which are exchanged for K+, thereby enhancing enteral K+ elimination.
- *Clinical applications*: nonurgent lowering of K+
- *Options* :
  - **Cation-exchange resins**

1. Sodium polystyrene sulfonate: falling out of favor due to adverse effects
2. Sodium zirconium cyclosilicate

  - **Cation-exchange polymers**, e.g., patiromer

- *Adverse effects :*
  - Gastrointestinal upset
  - Hypokalemia

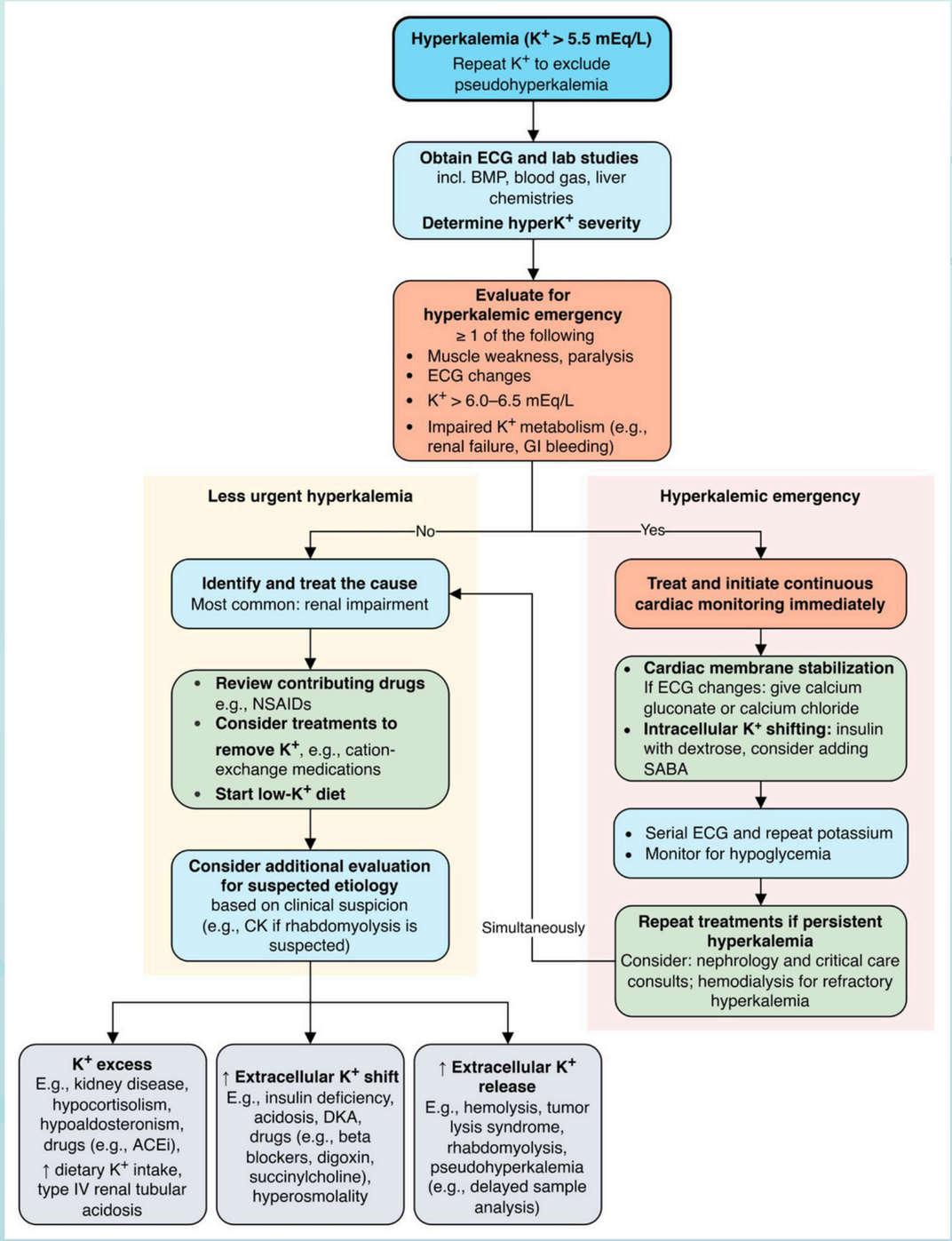
ENHANCED POTASSIUM ELIMINATION

☒ **Hemodialysis** :

- Most effective definitive therapy for refractory hyperkalemia
- Preferred option in patients with end-stage renal failure (particularly if already receiving renal replacement therapy), or oliguria.
- For all other patients, avoided as a first-line option because of its invasive nature and adverse effects

☒ **Loop Diuretics :**

- Consider loop diuretics, e.g., furosemide for patients with volume overload
- Closely monitor fluid balance and electrolytes due to unpredictable effects and risk of adverse events.


Hyperkalemia (K^+ > 5.5 mEq/L)
Repeat K^+ to exclude pseudohyperkalemia
Obtain ECG and lab studies
incl. BMP, blood gas, liver chemistries
Determine hyperK^+ severity
Evaluate for hyperkalemic emergency
≥ 1 of the following
Muscle weakness, paralysis
ECG changes
K^+ > 6.0–6.5 mEq/L
Impaired K^+ metabolism (e.g., renal failure, GI bleeding)
Less urgent hyperkalemia
Hyperkalemic emergency
No
Yes
Identify and treat the cause
Most common: renal impairment
Review contributing drugs
e.g., NSAIDs
Consider treatments to remove K^+, e.g., cation-exchange medications
Start low-K^+ diet
Consider additional evaluation for suspected etiology
based on clinical suspicion (e.g., CK if rhabdomyolysis is suspected)
Treat and initiate continuous cardiac monitoring immediately
Cardiac membrane stabilization
If ECG changes: give calcium gluconate or calcium chloride
Intracellular K^+ shifting: insulin with dextrose, consider adding SABA
Serial ECG and repeat potassium
Monitor for hypoglycemia
Repeat treatments if persistent hyperkalemia
Consider: nephrology and critical care consults; hemodialysis for refractory hyperkalemia
Simultaneously
K^+ excess
E.g., kidney disease, hypocortisolism, hypoaldosteronism, drugs (e.g., ACEi), ↑ dietary K^+ intake, type IV renal tubular acidosis
↑ Extracellular K^+ shift
E.g., insulin deficiency, acidosis, DKA, drugs (e.g., beta blockers, digoxin, succinylcholine), hyperosmolality
↑ Extracellular K^+ release
E.g., hemolysis, tumor lysis syndrome, rhabdomyolysis, pseudohyperkalemia (e.g., delayed sample analysis)

Hyperkalemia

Definition

Serum potassium (K^+) level > 5 mEq/L

Etiology

Reduced excretion (e.g., due to renal disease or drugs)
Extracellular shift (e.g., due to insulin deficiency or cell lysis)
Increased intake (e.g., due to high-potassium diet, K^+ containing IV fluids)
Pseudohyperkalemia (e.g., due to fist clenching during venipuncture, delayed analysis)

Clinical features

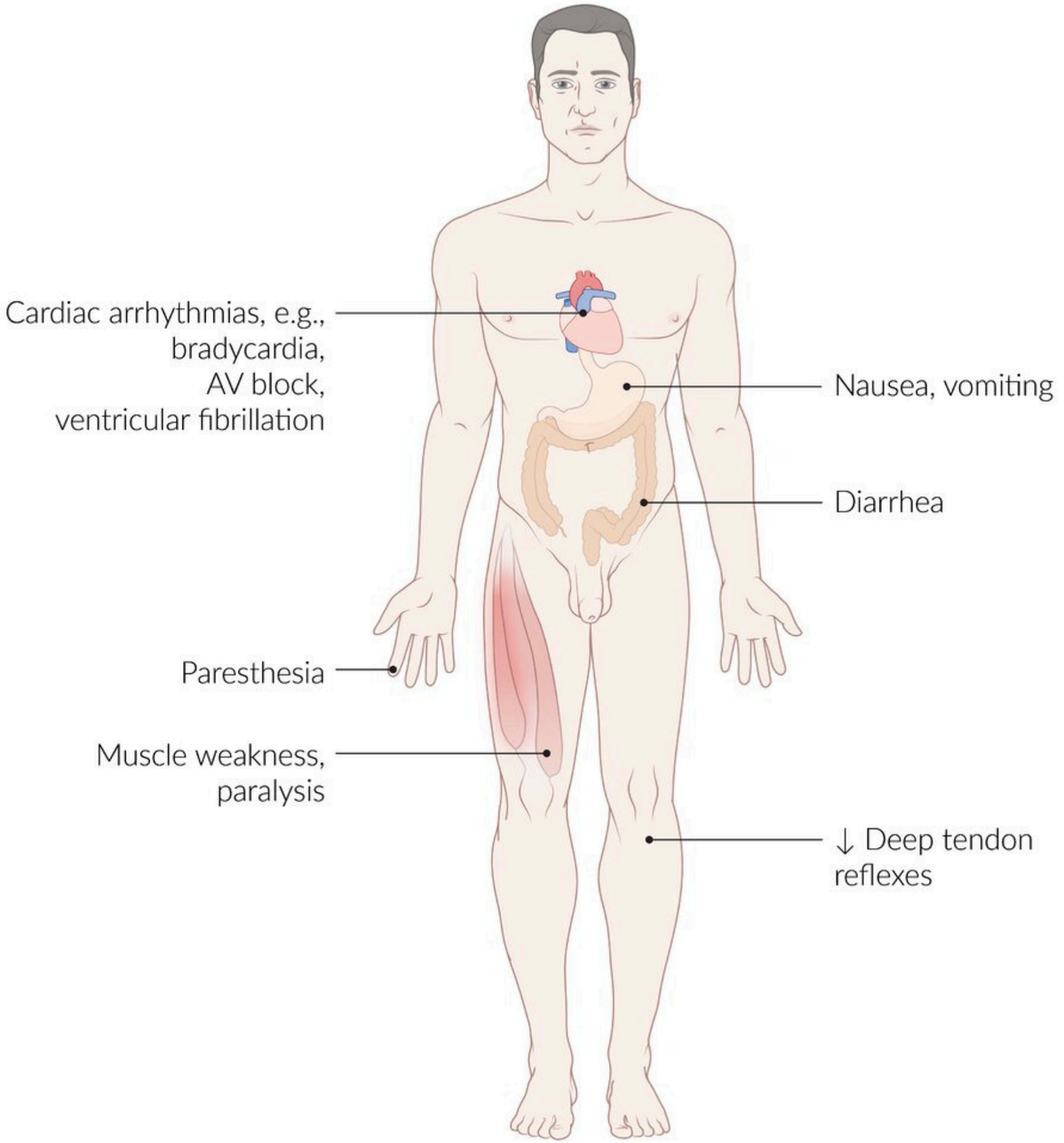
Symptoms usually occur if serum K^+ > 7.0 mEq/L or rapidly increasing

Diagnosis

Electrolytes, glucose, kidney and liver function, complete blood count, blood gas
ECG (e.g., peaked T-waves, QRS complex widening)

Treatment

Cardiac membrane stabilization with calcium salts
Intracellular K^+ shifting (e.g., with insulin and glucose)
Enhanced K^+ elimination (e.g., with hemodialysis)
Treatment of underlying cause



THANK YOU