Derangements of Potassium

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KEYWORDS

• Potassium • Hypokalemia • Hyperkalemia • Peaked T waves

KEY POINTS

- Potassium balance regulates the excitability of cardiac cells, and both hypokalemia and hyperkalemia can cause cardiac arrest when severe.
- Treatment of hyperkalemia includes cardiac membrane stabilization, transcellular shifting, and total body potassium elimination. Sodium bicarbonate and Kayexalate are not recommended for management.
- Treatment of symptomatic hypokalemia consists of repletion with potassium chloride, which is available in liquid, pill, and intravenously (IV) administrable forms. Magnesium should be repleted simultaneously to potentiate potassium absorption and avoid further potassium losses.
- Determine and treat the underlying cause of potassium derangement to prevent recurrence.
  - Avoid potentiating medications.
  - Consider the dietary potassium contribution.
  - Consider problems with potassium excretion from the gastrointestinal (GI) tract or kidneys.
  - Consider transcellular potassium shifts across cell membranes.

INTRODUCTION AND PATHOPHYSIOLOGY

About 98% of total body potassium (K+) is intracellular,[1,2] and 75% of the intracellular potassium is contained in skeletal muscle cells.[3,4] The body maintains the remaining 2% extracellular component within a tight range of 3.5 to 5.0 mEq/L (1 mmol equals 1 mEq K+).[3] The main mechanism for maintaining this transcellular ratio is the sodium-potassium (Na-K) adenosine triphosphatase (ATPase) pump, which uses energy in the form of adenosine triphosphate to drive K+ into cells in exchange for sodium (Na). The resulting K+ gradient creates a resting membrane potential that determines cardiac and neuromuscular cell excitability and signal conduction.

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Because the extracellular K+ level is proportionally so much less than the intracellular level, even a small change in the extracellular level significantly alters the resting membrane potential. Hyperkalemia is less tolerated by the body and causes more significant extracellular shifts than hypokalemia. A 100 mEq excess of total body K+ increases extracellular levels by 0.5 mEq, whereas a 100 mEq deficit decreases extracellular levels by just 0.3 mEq.[3] Three different mechanisms alter the extracellular K+ concentration: K+ intake, K+ excretion, and transcellular shift of K+ into or out of cells.[3] Many common medications affect one of these 3 mechanisms and can provoke a potassium imbalance (**Boxes 1** and **2**).

Usually the body's regulatory mechanisms can resist large fluctuations in daily potassium intake. However, over time or in those persons predisposed to K+ disorders, diet can affect the extracellular K+ level. Patients with altered total-body potassium stores, who chronically take medications that alter K+ balance, or who have a disease predisposing them to K+ imbalance may need to either increase or avoid intake of potassium-rich foods (**Box 3**).

Excretion of K+ from the body is primarily managed by the kidneys, which are responsible for 90% of excretion in normal physiology.[2,4] The other 10% is excreted mostly by the intestine into stool, with a small contribution from sweat. In cases of severe burns or extreme exercise, sweat and skin losses increase. Similarly, in end-stage renal disease when the kidneys no longer function, the gut upregulates to perform 25% of excretion.[2]

Box 1
Medications causing hyperkalemia

- Inhibit excretion
  - Decrease aldosterone
    - Angiotensin converting enzyme inhibitors
    - Angiotensin receptor blockers
    - Potassium-sparing diuretics (spironolactone)
    - Nonsteroidal antiinflammatory drugs (NSAIDs)
    - Heparin
    - Nonselective β-blockers
  - Block sodium channels
    - Potassium-sparing diuretics (amiloride, triamterene)
    - Antibiotics (trimethoprim, pentamidine)
- Transcellular shift
  - Inhibit Na-K ATPase pump
    - Digoxin (dose dependent)
    - NSAIDs
    - Nonselective β-blockers
    - Anesthetics (succinylcholine, suxamethonium)

Data from Alfonzo AV, Isles C, Geddes C, et al. Potassium disorders—clinical spectrum and emergency management. Resuscitation 2006;70:10–25; and Pepin J, Shields S. Advances in diagnosis and management of hypokalemic and hyperkalemic emergencies. Emerg Med Pract 2012;14(2):1–17.