



## POTASSIUM

Fluids 360

Electrolyte priorities and Imbalances

K pp. 42-44

B pp 290-292;

Potassium and Potassium Imbalances

K pp. 45-61

C (5<sup>th</sup> ed) pp. 39-43

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## Objectives

Upon completion of this lecture the learner should be able to:

- Discuss the functions, physiology, and pathophysiology of potassium .
- Discuss the incidence, morbidity, and mortality of potassium emergencies .
- Identify etiologies and risk factors predisposing to potassium emergencies.

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## Objectives (cont.)

- Identify abnormal signs, symptoms, and clinical features of potassium emergencies .
- Correlate abnormal findings with clinical situations of patients at risk for sudden death
- Identify the need for rapid intervention in a patient with a hyperkalemic emergency
- Discuss the prehospital management, in detail, of a probable hyperkalemic emergency

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## Priorities - review

When fluids and electrolytes are altered, they should be corrected in the following order:

- Volume
- pH
- **potassium**, calcium, and magnesium
- sodium and chloride

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## Potassium – distribution

- Intracellular
  - 97-98% ICF (muscle, liver, and blood cells)
  - $K^+$  is the most abundant intracellular cation
- Extracellular
  - 2% ECF (3rd space + serum)
  - very small percentage of the total body  $K^+$
  - small change in serum levels may represent :
    - a huge deficiency in the total body  $K^+$
- Normal extracellular (serum)  $K^+$  :
  - 3.5 – 5.3 mEq/L

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## Potassium $K^+$

Significant and rapid  $K^+$  shifting from intracellular to extracellular occurs in response to:

- severe injury ( surgical stress, trauma, or burns)
- acid-base imbalance
- catabolic states ( homeless ; cancer ; cocaine addiction )
- insulin deficiency

These shifts are important :

- propagation of electrical impulses

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## Potassium - function

- Muscle
  - neuromuscular conduction
  - smooth and skeletal
- Cardiac
  - conduction
  - contraction
- Cellular
  - acid-base buffering

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## Potassium pathophysiology

- Skeletal and Smooth Muscle Sn. + Sx.
- Cardiac
  - conduction abnormalities
- Acid-base abnormalities
  - Acidosis → ↑ K<sup>+</sup>

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## Potassium Abnormalities

- Hypokalemia
  - (less than 3.5mEq/L)
  - not symptomatic if more than 2.5 mEq/L
- Hyperkalemia
  - (greater than 5.5mEq/L)
  - not symptomatic if less than 7.5 mEq/L
  - especially if chronic / gradual
- Result from:
  - conditions of excessive losses or retention

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## Hypokalemia

### Causes

- inadequate intake
- losses
  - renal
  - GI
  - shift
    - from ECF into the cell

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## Hypokalemia

### Renal losses

- diuretics
- aldosterone
- osmotic (sugar-diuresis)
- post-obstructive diuresis

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## Hypokalemia causes

- Losses due to intracellular shift
  - alkalosis
  - drugs
    - insulin
    - beta agents (albuterol)
- GI losses
  - vomiting
  - diarrhea

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## Hypokalemia causes

- Insufficient intake
  - starvation
  - *alcoholism*
  - iatrogenic
- Miscellaneous
  - licorice OD
  - Mg <sup>++</sup> deficiency
  - drugs

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## Hypokalemia Clinical Presentation

- Neuromuscular :
  - weakness
  - paralysis
- GI :
  - N, V
  - constipation
  - SBO (ileus)

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## Hypokalemia Clinical Presentation

Cardiovascular :

- AV block
- accentuates the Dig effect
- EKG findings: "everything low"
  - low-voltage QRS
  - *low, flat T wave*
  - depressed ST segment
  - prolonged QT, PR ; U wave
  - tachycardias
- PVCs, *V tach* ; *V fib*

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## Hypokalemia – Treatment

- CO<sub>2</sub>M<sub>3</sub>E BI<sub>NS</sub>G
- Correct volume status
- Correct acid-base status \
- KCl po
  - i.e., non-urgent
- KCl IV [ **NEVER** as a bolus ]
  - 40-50 mEq / 1000mL NS / 1 hr.

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## Hyperkalemia

- Biggest emergency of electrolyte abnormalities !!
- Causes
  - pseudohyperkalemia (factitious)
  - transcellular shift (i.e., acid-base)
  - excessive intake
  - potassium loading
  - decreased excretion
  - drugs

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## Hyperkalemia

### Etiologies

- excessive intake
  - salt substitute
- potassium loading
  - succinylcholine
  - massive transfusion of stored blood
  - tissue injury
    - burns, crush, rhabdomyolysis, ischemia
    - hemolysis, tumor lysis
    - GI bleed

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## Hyperkalemia

### Etiologies (cont.)

- decreased excretion
  - renal failure
  - decreased mineralocorticoid
- drugs
  - digoxin (esp overdose) most common drug
  - induced
  - spironolactone
  - b-blockers
  - NSAIDs
  - heparin

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## Hyperkalemia Clinical Presentation

- Neuromuscular :
  - cramps, tetany, quadriplegia
  - paresthesias, lethargy
- Cardiac :
  - Peaked T
  - Bradycardia, sinus arrest
  - Late ( at levels of 7-10)
    - ↓ P wave, QRS widening, HB
    - sine wave, V fib

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## Hyperkalemia - Treatment

- $\text{CO}_2\text{M}_3\text{E BI}_{\text{NS}}\text{G}$
- Correct volume and acid-base status then
- Calcium IV
- Saline flush IV
- Bicarb. IV
- D 50 IV
- Lasix IV
- Albuterol Nebulizer

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## Hyperkalemia – Treatment

- $\text{CO}_2$ ,  $\text{M}_3\text{E}$ ,  $\text{B}_{\text{NS}}\text{G}$  ;  
Correct volume and acid-base status , then
- $\text{CaCl}_2$  10% 5 mL IV
- NS IV [ as a rapid 250 mL bolus ]
- $\text{NaHCO}_3$  (50-100 mEq ) IV
- $\text{D}_{50}\text{W}$  50 mL IV
- Furosemide 40 mg IV
- Albuterol 0.5 mL (2.5mg) with NS by nebulizer

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## Summary

We have discussed :

- Functions of and homeostasis of  $\text{K}^+$
- Regulators of  $\text{K}^+$  balance
- Pathophysiology and causes of  $\text{K}^+$  abnormalities
- Clinical presentations
- Treatment [ *know, in detail !!* ]

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