POTASSIUM ON THE WARDS

QUICK POTASSIUM BASICS

98% intracellular. Driven there by Na-K-ATPase pump in the cell membrane

2% Extracellular and tightly controlled at ~3.7-5.2 mEq/L. K levels outside of this range potentially life threatening

	Mild-to-Moderate	Severe
Hyperkalemia	6-7 mEq/L	>7 mEq/L and/or symptomatic
Hypokalemia	3-3.4 mEq/L	< 2.5-3 mEq/L and/or symptomatic

IS IT REAL? PSEUDOHYPERKALEMIA

Pseudohyperkalemia: lab findings of **falsely** \uparrow serum K due to K movement out of the cells during or after a blood draw. Suspect in an asymptomatic patient with no apparent cause for K elevation

- 1. Lysis of rbc
- 2. Specimen deterioration (cooling, prolonged storage)
- 3. ↑wbc, ↑plt
- 4. Drawing blood downstream from a vein into which K is infusing
- 5. Trauma: forcible expression of blood (milking a heel stick)
- 6. Exercise: fist clenching with blood draws

HYPERKALEMIA CAUSES

I. Shifting of K into extracellular space

- A. Tissue (lots of cells) damage: burns, crush injury, rhabdo, tumor lysis
- B. Acidosis
- C. Hyperosmolar states
- D. Insulin deficiency

II. Impaired Renal Excretion (↑ total body K)

- A. Renal insufficiency/failure
- B. Endocrine: \downarrow renin, \downarrow aldosterone, adrenal insufficiency, pseudohypoaldosteronism

III. Iatrogenic

- A. K in IVF/TPN
- B. Lots of meds (NSAIDS, ACE inhibitors, beta blockers, K sparing diuretics, trimethoprim)

HYPERKALEMIA SIGNS/SYMPTOMS

Resolve with hyperkalemia correction

I. Muscle: ascending weakness and paralysis

sphincter tone, cranial nerves, and respiratory muscles typically preserved

II. Cardiac

- A. Conduction abnormalities and arrhythmias
- B. EKG changes
 - 1. Peaked T waves→ Loss of P wave→ Widened QRS→ Sine wave pattern

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2. Rough (NOT perfect) correlation b/w EKG changes and ↑K Hyperkalemia can be life-threatening even if EKG nl
Any EKG changes should be treated as an emergency

HYPERKALEMIA TREATMENT

4 Basic Approaches

- 1. **Do no harm:** remove any exacerbating factors: K containing IVF, TPN, meds known to cause hyperkalemia
- 2. Stabilize cell membranes: Calcium
- 3. **Drive K into cells:** insulin/glucose, Beta2 agonist (albuterol)
- 4. **Remove excess K from the body:** furosemide, Kayexalate, dialysis

Monitoring: continuous cardiac monitors, serial EKG's, and q1hr K for pt's who require rapidly acting therapies

POTASSIUM ON THE WARDS

MEDICATION	DOSING	MECHANISM	Notes
Calcium IV	10-20mg/kg	Stabilizes myocardium	-Give if any EKG changes and/or K is rapidly increasing (if peaked T waves alone and rapidly acting methods
			being initiated could consider holding Ca)
			Onset: immediate
			Duration: up to 1hr
Insulin/Glucose	Insulin: 0.1		Onset: minutes
	units/kg +		Duration: Peak ~1hr. Lasts 4-6hrs
	D 25W 2m/kg or	Drives K into cells	
	D10W 5ml/kg	↑ activity of the Na-K-ATPase	
Beta2 agonist	10-20mg neb	pump	Onset: minutes
(Albuterol)			Peak: ~90 min
Loop diuretic	1mg/kg/dose	↓ total body K by ↑ renal K	Consider adding NS bolus to maximize distal sodium
(furosemide)		excretion	delivery and flow
			Onset: 15 min to 1hr
Cation exchange resin	1-2g/kg	↓ total body K by ↑ GI K	-PO/NG: Peak 4-6hrs
Sodium polystyrene	PO/NG/PR	excretion	Enema 1-2hrs
sulfonate (Kayexalate)		Binds K in the colon in exchange	
		for Na	
Dialysis		↓ total body K	Use if:
		-	1) anuric
			2) K↑rapidly
			3) Above measures ineffective

HYPOKALEMIA CAUSES

I. Shifting of K into intracellular space

- A. Alkalosis
- B. Insulin
- C. ↑ Beta-adrenergic activity

II. \uparrow K losses (\downarrow total body K)

- A. GI track
- B. Urine

HYPOKALEMIA SIGNS/SYMPTOMS

Resolve with hypokalemia correction

I. Muscle

- A. Ascending weakness and paralysis. Can include respiratory muscles→resp failure, and GI muscles→ileus
- B. Ischemia: cramping, rhabdomyolysis, myoglobinuria

II. Cardiac

- A. Conduction abnormalities and arrhythmias
- B. EKG Changes (not seen in all pts): ST segment depression and prominent U wave



TREATMENT OF HYPOKALEMIA

- I. Investigate and manage any underlying causes
- II. Investigate and manage any coexisting alkalosis and/or ↓ Mg
- III. Replace K
 - A. Enteral (preferred unless pt symptomatic or unable to tolerate)
 - 1-4 mEq of K/kg/day divided QID-BID
 - B. Parenteral: 0.3mEq/kg per dose. Worry about over-correcting and causing hyperkalemia. Pt needs to be on a CR monitor during K bolus infusion. Recheck K following infusion.