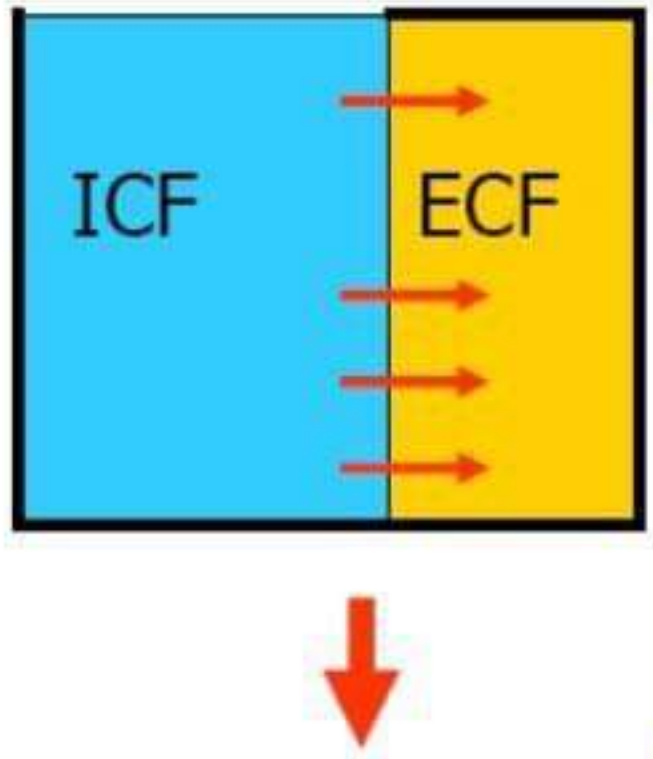
Hyperkalemia

Hyperkalemia- Etiology



- Intake (never alone)
- Shift (Acute)
  - Acidosis
  - Insulin lack
  - Tissue Lysis
  - Beta blockade
  - Digitalis o.d.
  - Succinylcholine
- Excretion (Chronic)
  - Advanced renal failure
  - Hypoaldosteronism
  - Volume depletion

Pseudohyperkalemia

- Hemolysis via small needle (traumatic Venipuncture)

The most common cause is lysis of red cells in a phlebotomy specimen.

- Delayed analysis.
- Excessive tourniquet .
- Severe Thrombocytosis. Potassium released from platelets
- Severe Leukocytosis.

Pseudohyperkalemia can be excluded by repeating the sample as atraumatically as possible .

Redistribution (ICF to ECF)

- Acidosis
- Hyperkalemic periodic paralysis (AD disease)
- Insulin deficiency (DKA).
- Tissue damage (Tumor lysis ,Burn injury ,Crush injury, Surgery , Massive Hemolysis)
- **Medications:**
  - Beta Blockers
  - Digitalis intoxication (Digoxin Toxicity)

Decreased renal excretion

Renal Insufficiency or Renal Failure.
Renal Tubular Acidosis (Type 4) =(hyporeninimic hypoaldostronism)
Adrenal Insufficiency (Addison's).

Medication.

- Heparin .
- Cyclosporine
- ACE Inhibitor
- Potassium sparing Diuretics
- NSAIDS

- The initial diagnostic approach begins with the clinical history, review of medications,

and physical examination.

- Symptoms and signs include muscular weakness or flaccid paralysis, paralytic ileus, and characteristic ECG changes.

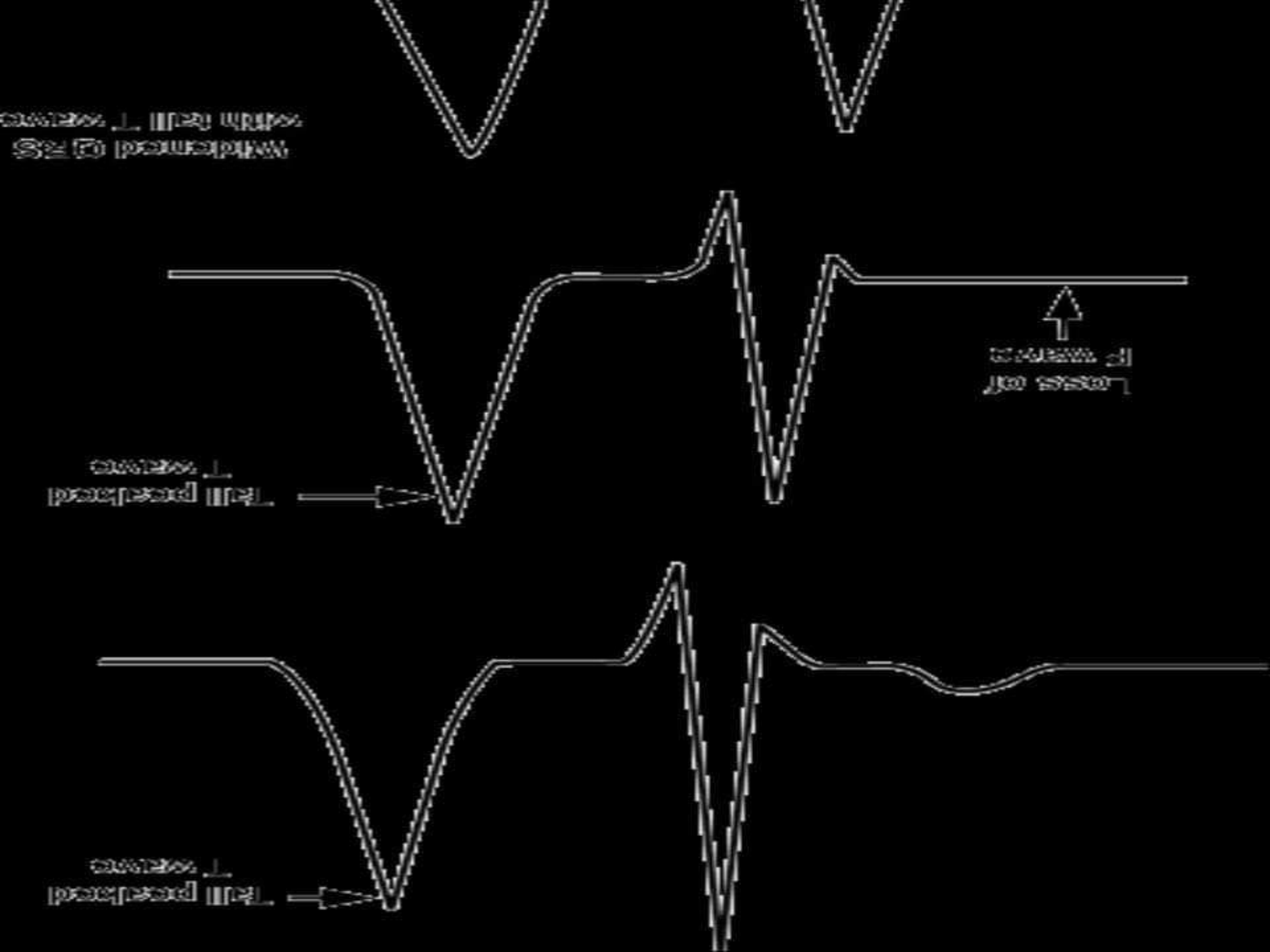
Symptoms and Signs

3 types of muscle fibres (skletal, smooth ,cardiac).

- May be asymptomatic
- **Irregular heartbeat**
- Fatigue
- **Tingling, numbness, or paraethesia**
- **Weakness**
- **Paralysis (in some population with AD** hyperkalemic periodic paralysis)
- **Difficult breathing**

ECG

- Mild hyperkalemia 5.5-6 mmol/L **peaked T**
- Moderate 6-8 mmol/L, peaked T + **absent P wave**
- Severe > 8 mmol/L , peaked T **absent P wave** , wide QRS , Vent **arrhythmia, VF, asystole**



TARGET OF TREATMENT

- Minimize membrane depolarization(Ca gluconate)
- Shifting k into cells (glucose- insulin , bicarb, B2 agonist)
- Promote k loss (diuretics) but be sure of functioning kidney
- Correction of acidosis (Na bicarb.)
- D/C exogenous k sources , antikaliuretic drugs (stop aldacton ,NSAID & ACE I)

Serious hyperkalemia =

Plasma K> 7.5 mmol/L

Profound weakness

Absent P wave , wide QRS , vent arrhythmia.

Intravenous calcium is administered to **stabilize the myocardium;** it lowers the threshold potential, thus counteracting the toxic effect of high potassium.

Calcium does not have any effect on the serum potassium level. **Improvement in the ECG** changes should be visible **within two to three minutes of administration of calcium**

Medications Used in Acute Treatment of Hyperkalemia	Dosage	Onset	Length of effect	Mechanism of action	Cautions
Calcium gluconate	10 to 20 mL of 10 percent solution IV over two to three minutes	Immediate	30 minutes	Protects myocardium from toxic effects of K; no effect on serum potassium level	Can worsen digoxin toxicity
Insulin	**Regular insulin 10 units IV with 50 mL of 50 percent glucose**	**15 to 30 minutes**	Two to six hours	Shifts potassium out of the vascular space and into the cells; no effect on total body potassium	Consider 5 percent dextrose solution infusion at 100 mL per hour to prevent hypoglycemia with repeated doses. Glucose unnecessary if blood sugar elevated above 250 mg per dL

Albuterol (Ventolin)	10 to 20 mg by nebulizer over 10 minutes (use concentrated form, 5 mg per mL)	15 to 30 minutes	Two to three hours	Shifts potassium into the cells, additive to the effect of insulin; no effect on total body potassium	May cause a brief initial rise in serum potassium
Furosemide (Lasix)	20 to 40 mg IV, give with saline if volume depletion is a concern	15 minutes to one hour	Four hours	Increases renal excretion of potassium	Only effective if adequate renal response to loop diuretic
Sodium polystyrene Cation exchange resin (sorpstret powder) **(Q) in resistant hyperkalemia**	Oral: 50 g in 30 mL of sorbitol solution Rectal: 50 g in a retention enema	One to two hours (rectal route is faster) 1gm binds 1mmol k	Four to six hours	**Removes potassium from the gut in exchange for sodium**	**Sorbitol may be associated with bowel necrosis. May lead to sodium retention**
hemodiatysis	The most rapid and effective	PD is 15% as effective as HD			

Hyporeninemic **hypoaldosteronism**(RTA type 4) should be considered in patients with diabetes and hyperkalemia, a trial of **oral fludrocortisone (asetonin H)**^{R} (aldosteron like miniralocorticoides) to establish this diagnosis;

potassium levels will return to **normal** in a **day or two** after initiation of fludrocortisone.