Electrolyte Disturbances

Part 1: Potassium

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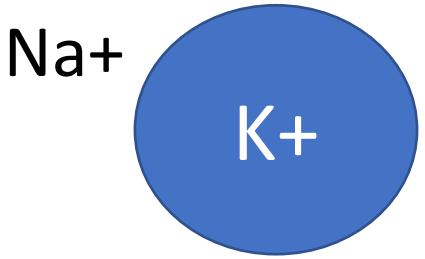
Objectives:

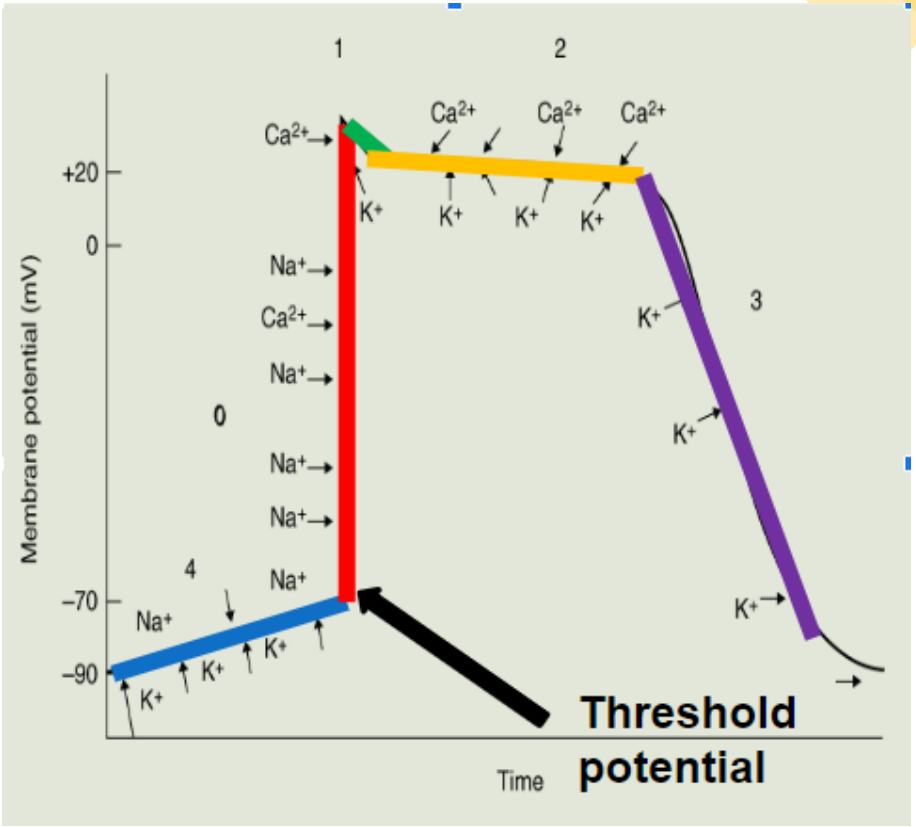
- Recognize the signs and symptoms of hypokalemia and hyperkalemia
- Determine the etiology of serum potassium derangements
- Develop a pharmacotherapeutic plan for the management of serum potassium disturbances

POTASSIUM
K^+

POTASSIUM: Background

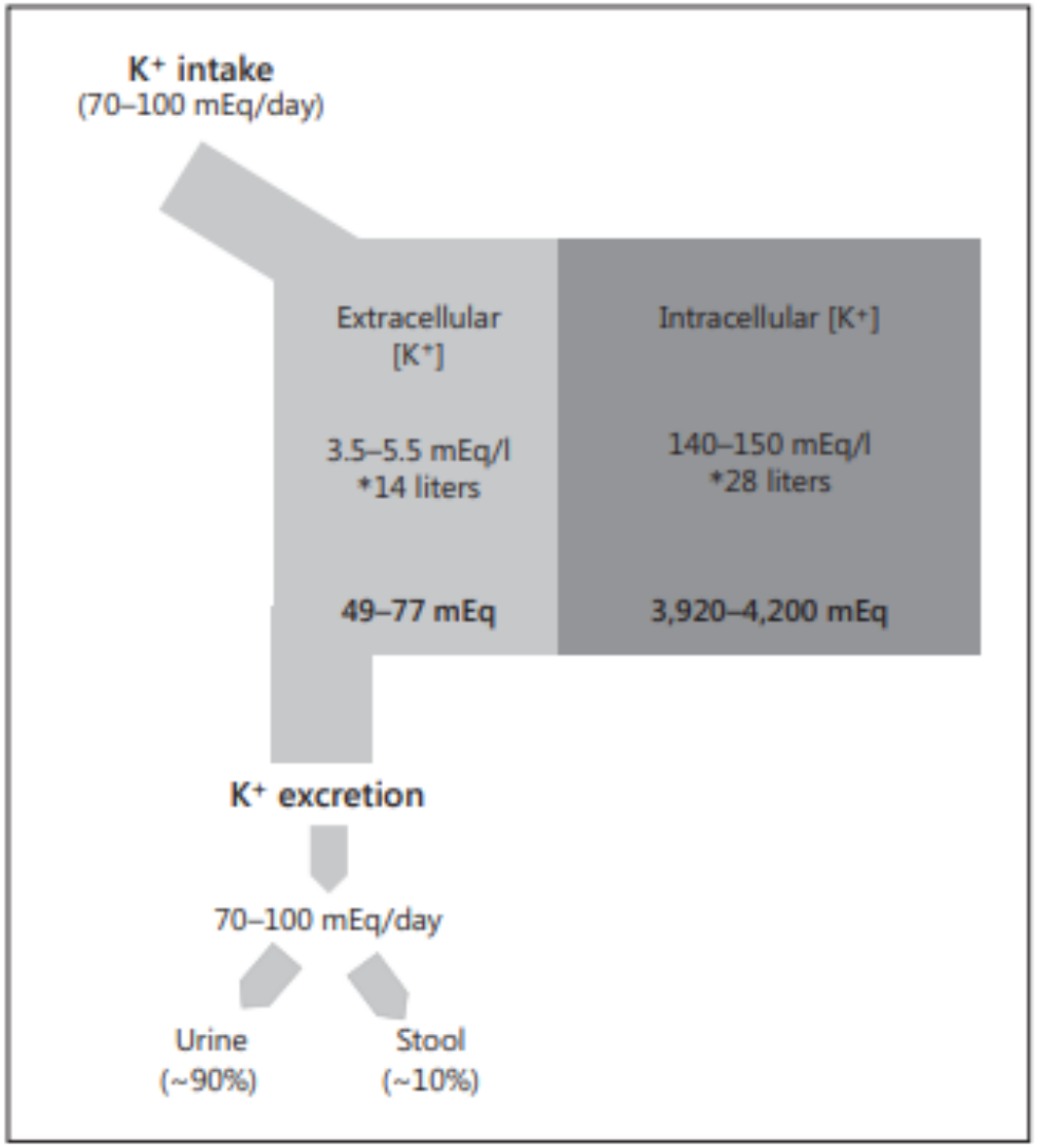


- Potassium is the most abundant intracellular cation (only 2% extracellular)
- **Normal serum potassium: 3.6 – 5.1 mMol/L**
- 98% of total body potassium is <u>intracellular</u>
- Extracellular potassium concentration is under tight control to maintain the resting membrane potential of excitable cells (at -90mV)
- Potassium homeostasis is largely determined by the kidneys, but regulation also involves many distribution buffers



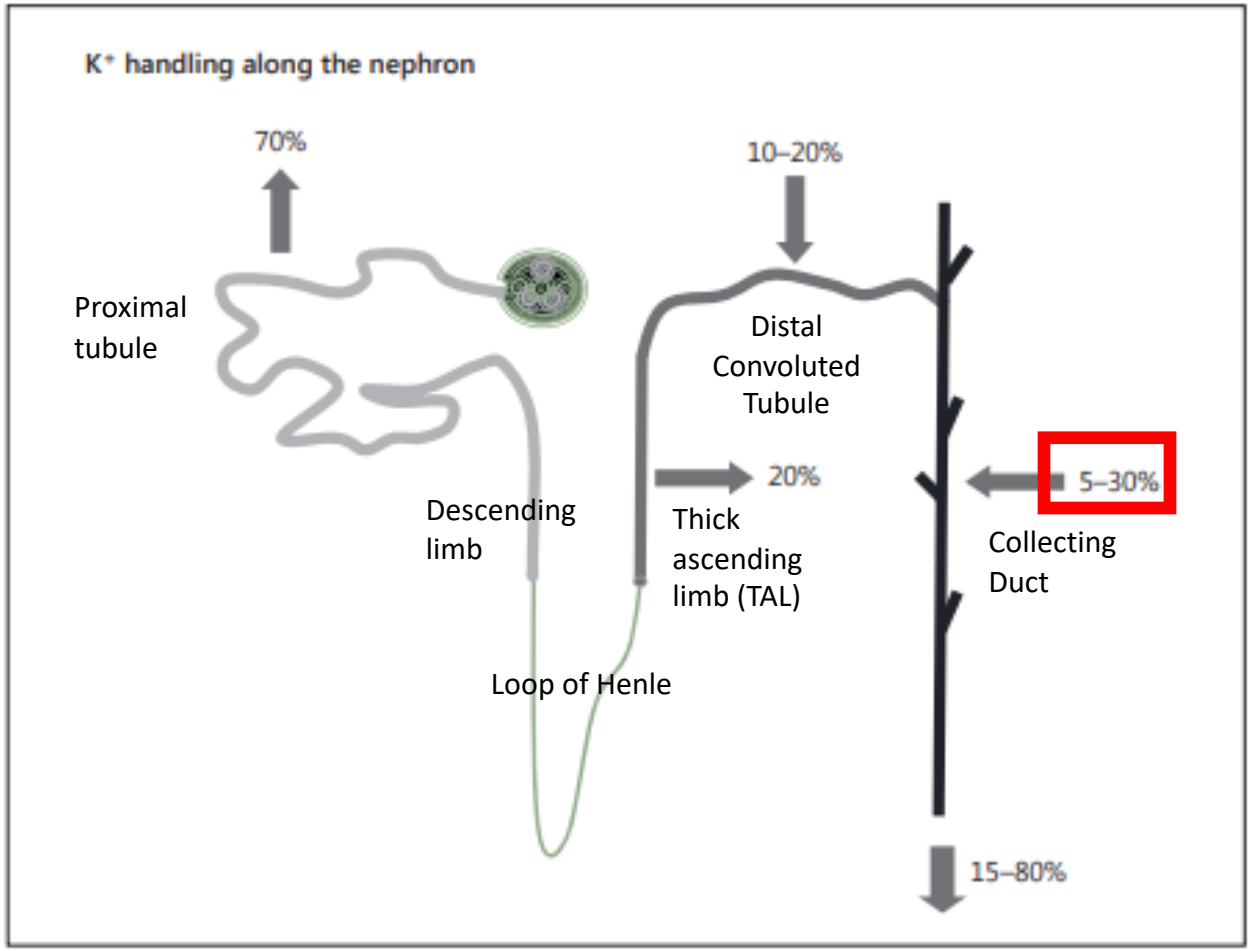
Potassium Homeostasis

- The K+ gradient between the ICF and the ECF is dependent upon the Na-K ATPase pump located on all plasma membranes
  - Several factors affect the activity of this pump:
    - Acid-base status
    - Plasma osmolality
    - Insulin and glucagon
    - Catecholamines
    - Aldosterone



Potassium Excretion

- The kidney is the main determinant of potassium excretion
- Potassium is freely filtered across the glomerulus and then reabsorbed in the proximal tubule (PT) and thick ascending loop (TAL) of Henle with some distal secretion
- Final potassium excretion is 10-20% of filtered load, since reabsorption of K+ at the PT and TAL is largely fixed, the main final excretion is determined by distal secretion (mainly under aldosterone control)



HypERkalemia

Potassium: HypERkalemia

Etiology

Symptoms

Treatment

HypERkalemia: Etiology

Renal failure/dysfunction
Excessive potassium intake
Metabolic acidosis
Hypertonicity (hyperglycemia, mannitol, sucrose)
Muscle tissue injury (Rhabdomyolysis, hemolysis, tumor lysis)
Adrenal insufficiency
Medications

Medication-Induced Hyperkalemia

- Potassium supplementation
- ACE inhibitors
- Digoxin toxicity
- Succinylcholine
- Potassium-sparing diuretics (aldosterone antagonist such as spironolactone, eplerenone)
- Trimethoprim (a component of Bactrim®)
- Angiotensin receptor blockers (ARB)
- Heparin
- NSAIDs
- Beta blockers

Table 1. Medications That Commonly Cause Hyperkalemia*

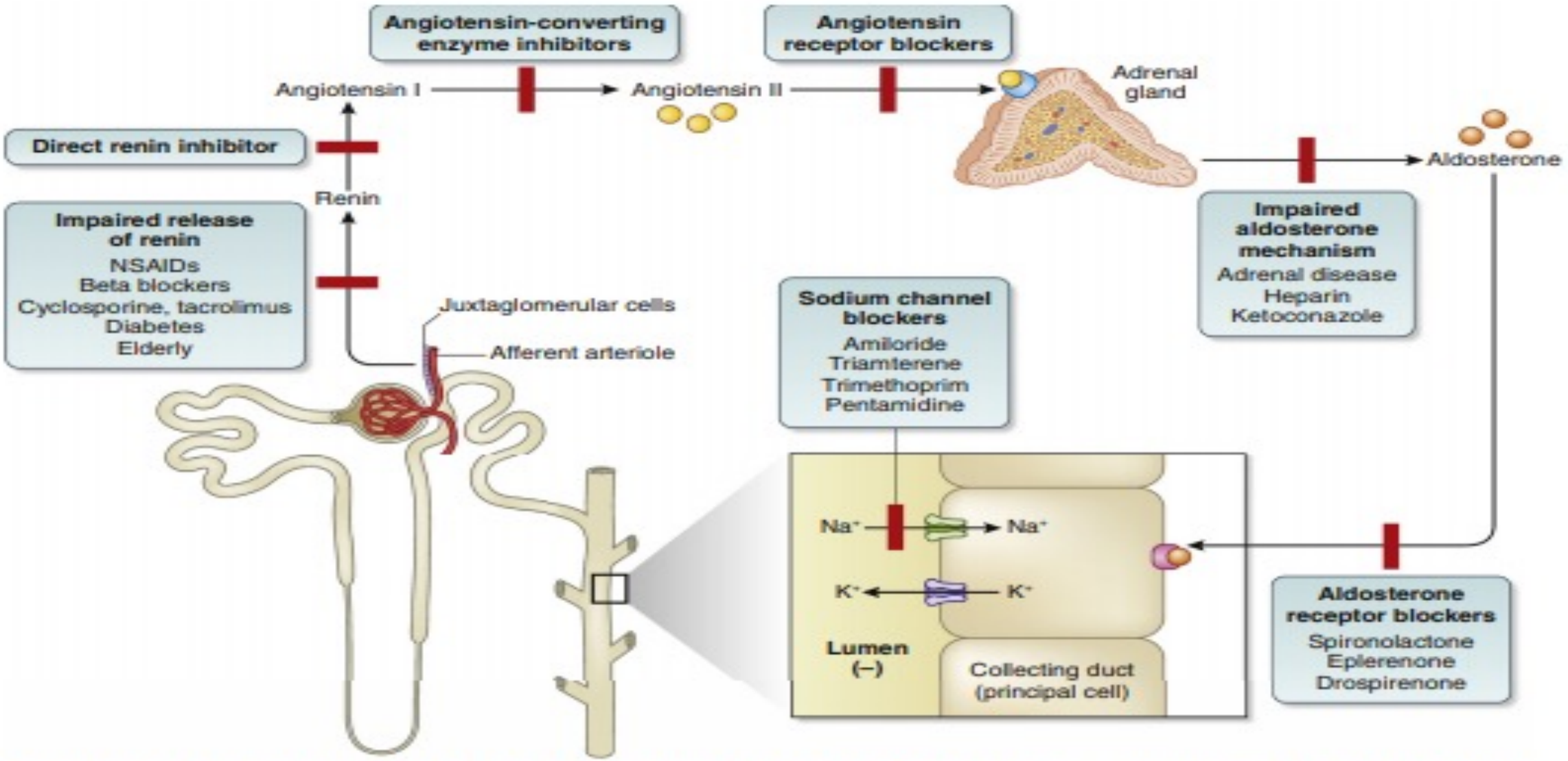
Medication	Reported Percentage of Patients Who Develop Hyperkalemia from the Drug	Reported Percentage of Hyperkalemia Resulting from the Medication†
Potassium supplements	3–24	11–58
Beta blockers	1–5	4–17
Digoxin	2–15	12–19
Potassium-sparing drugs	2–19	9–21
NSAIDs	10–46	9–18
ACE inhibitors	10–38	8–37
Angiotensin-II blockers	2–7	—
Trimethoprim	6–21	14–29
Pentamidine	5–24	0–5
Cyclosporine	11–44	5–28
Tacrolimus	15–53	8–28
Heparin	8–17	1–20

* Data from references 5, 6, 8, 9–11, 14, 48, 53, 59–61, 63, 66–71, 77–80 and from personal experience. Hyperkalemia defined as serum potassium level greater than 5.3 mEq/L.

† Usually reported among hospitalized patients.

ACE = angiotensin-converting enzyme; NSAID = nonsteroidal anti-inflammatory drug.

POTASSIUM: Background



Medication-Induced Hyperkalemia

Medication	Mechanism of Hyperkalemia
ACE inhibitors	Decrease aldosterone synthesis and decease renal blood flow and glomerular filtration rate
Angiotensin-II Receptor Antagonist (ARBs)	Decrease adrenal synthesis of aldosterone but may not cause clinically significant hyperkalemia as compared to ACEI
Spironolactone, amiloride	Potassium-sparing diuretic blocks aldosterone
NSAIDs	Inhibit renal prostaglandin synthesis which decreases renin release with subsequent decrease aldosterone synthesis
Trimethoprim	Structurally similar to amiloride, block sodium transport channels in the distal collecting duct which hyperpolarizes the luminal membrane and secondarily blocks potassium secretion via the Na-K ATPase pump
Beta blockers	Block catecholamine mediated renin release which decreases aldosterone synthesis. Non-selective beta blockers decrease beta-2 driven potassium update
Heparin	Decreases the number and affinity of angiotensin II receptors in the adrenal zona glomerulosa thus reducing aldosterone synthesis which reduces renal potassium excretion.
Digoxin	Block Na-K ATPase pump
Succinylcholine	Depolarizes cell membrane which allows for potassium leakage from cells

HypERkalemia: Symptoms

Muscle twitching

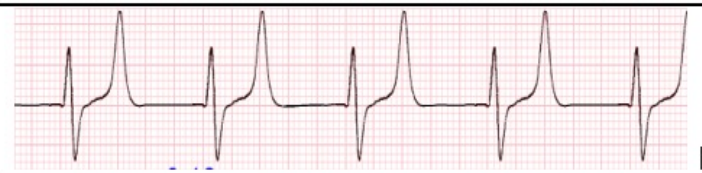
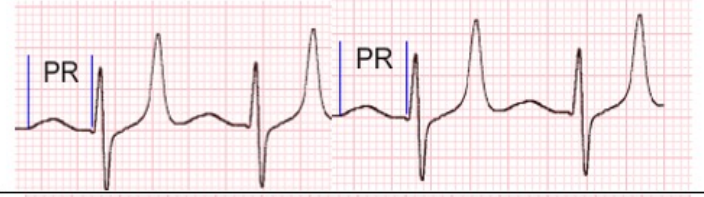
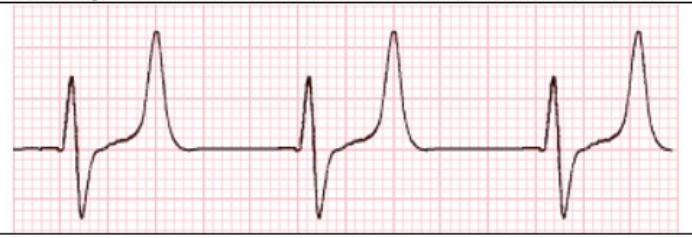
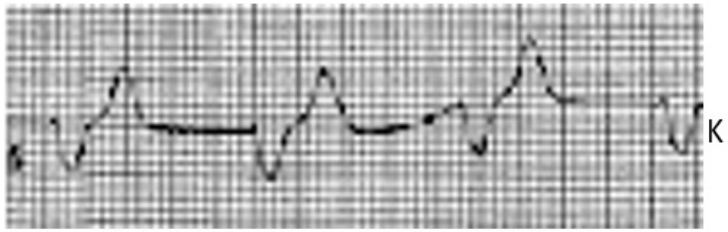
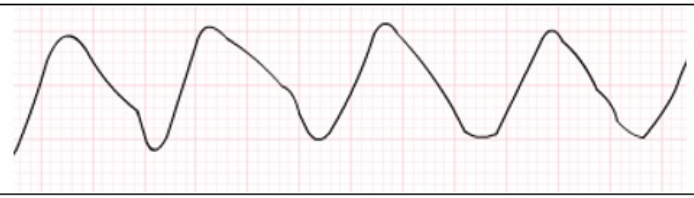
Cramping

Muscle Weakness

Ascending paralysis

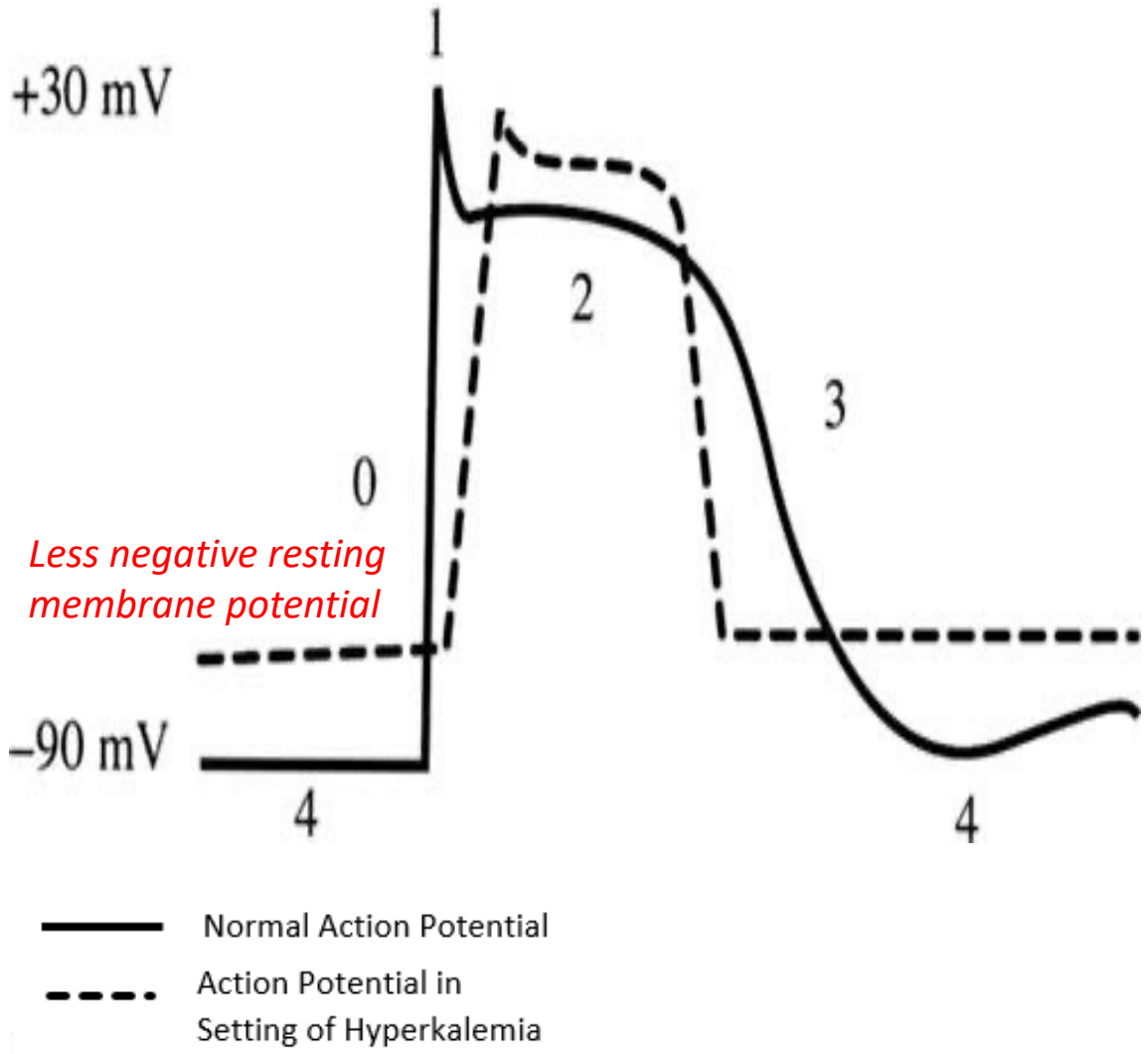
ECG changes

HyperERkalemia: ECG Changes

1) Peaked T waves (defined as ≥ ½ height of QRS with a height of ≥ 5mm or 1 large block	 K =5.5-6.5
2) PR Prolongation (Defined as PR Interval > 200 msec)	
3) Loss of P Wave	 K = 6.5 - 8
4) Wide QRS (> 120 msec).	 K = 6.5 - 8
5) Sine Wave	 K > 8

Etiology of ECG Changes in Hyperkalemia

- Phase 0 of the action potential represents sodium influx through voltage gated sodium channels
- Hyperkalemia raises resting membrane potential making depolarization easier (membrane destabilization)
- Increasing resting membrane potential results in fewer voltage gated sodium channels opening during phase 0 of the action potential
- Fewer open voltage gated sodium channels causing depolarization to occur more slowly, resulting in a QRS complex with a longer duration (wide QRS)



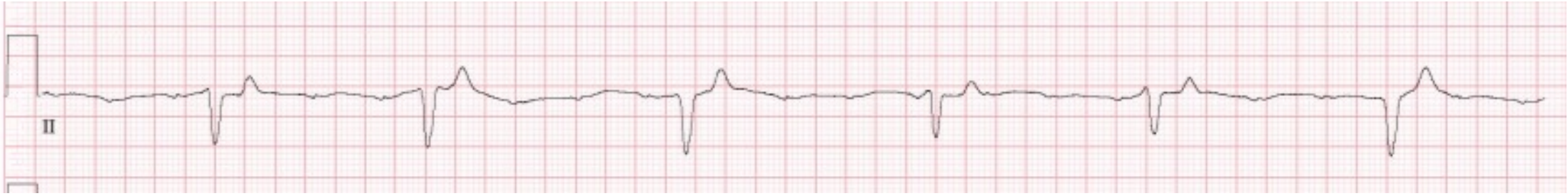
HypERkalemia: TREATMENT

1. “Stabilize” the cardiac membrane to prevent ventricular arrhythmia
2. Drive potassium into the cell
3. Eliminate potassium from the body

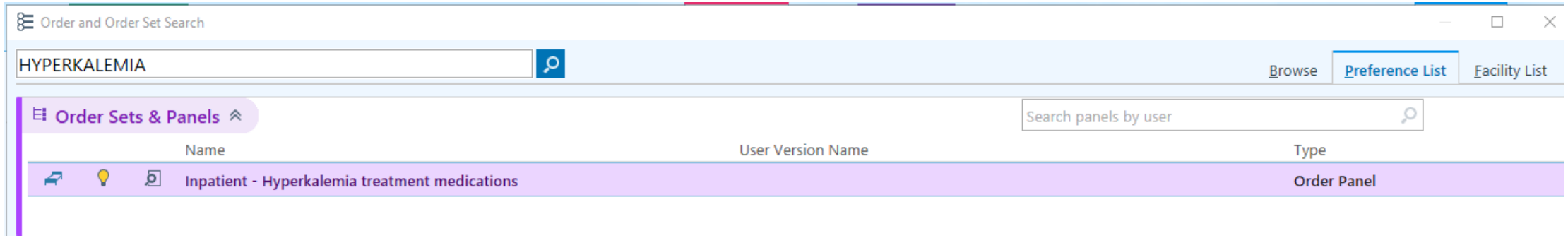
Example Case:

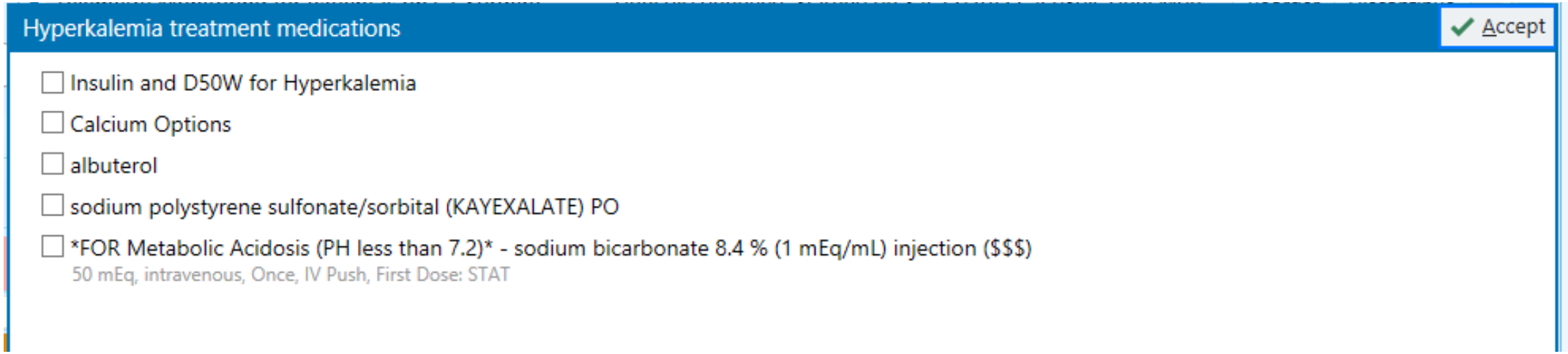
LAB		
Chemistry-General	Sodium Level	Potassium Level
06/08/2021 05:27 EDT	L 133 mMol/L	* C 7.8 mMol/L

Chemistry-General	BUN	Creatinine
06/07/2021 21:43 EDT	H 68 mg/dL	H 4.10 mg/dL



Example ORDER PANEL: HYPERKALEMIA

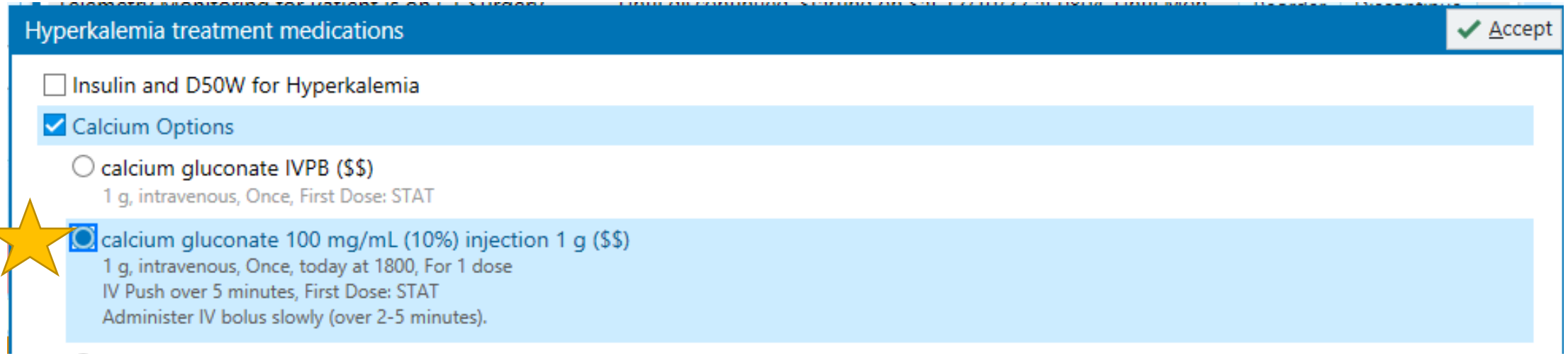




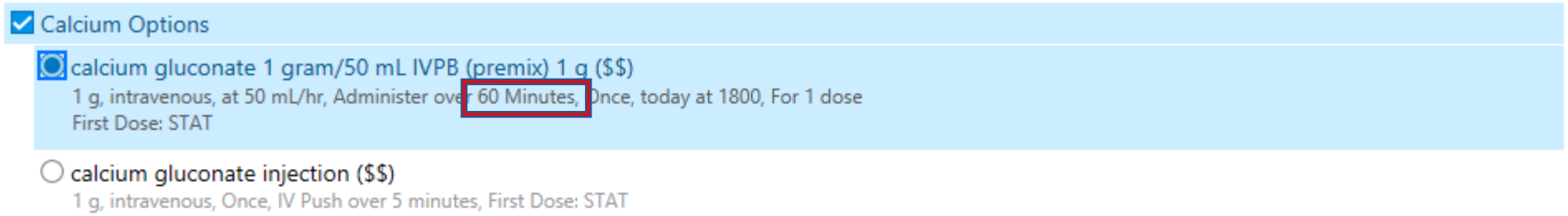
HypERkalemia: TREATMENT Options

Medication	Dose	Pharmacokinetics	Mechanism of Action
Calcium Gluconate	1 – 2 g IV once administered slow IVP 5-10 minutes or IVPB *(Given first priority for administration if ECG changes)*	Onset: 1 – 2 minutes Duration: 10 – 30 minutes	Increases threshold potential (less negative than – 70mV), which restores the normal gradient between threshold potential and resting membrane potential (RMP is elevated (less negative) in hyperkalemia)
Insulin regular IV	5 – 10 units IV	Onset: 15 minutes Duration: 4 – 6 hours	Potentiates the placement of Na/K ATPase Pumps on cellular membranes, causing K influx
Dextrose 50%	25 grams IV once	Onset: immediate (prevent hypoglycemia), 30 minutes (insulin release to treat hyperkalemia)	Prevention of hypoglycemia and stimulation of endogenous insulin release
Sodium Bicarbonate 8.4%	50 – 100 mEq IVP over 2 – 5 minutes	Onset: 30 minutes Duration: 2-6 hours	HCO_3 initially causes Na influx via the Na/ HCO_3 cotransporter. Additional influx of Na subsequently increases activity of the Na/K ATPase, leading the increased K influx
Albuterol	10 – 20 mg Nebulized over 10 minutes	Onest: 30 minutes Duration: 1-2 hours	Increases activity of the Na/K ATPase, causing intracellular influx of K+
Sodium polystyrene sulfonate (Kayexalate)	15 – 30 g PO or Rectally may repeat (max daily dose 60 g)	Onset: 1 hour	Sodium cation exchange resin that binds potassium in the intestinal lumen, allowing for fecal excretion of potassium.
Sodium Zirconium Cyclosilicate (Lokelma)	10 g PO TID	Onset: 1 hour	Potassium binder that preferentially exchanges potassium for hydrogen and sodium to reduce free potassium in GI lumen to allow further shift for elimination in stool

ECG Changes → Order Calcium Gluconate



NOTE: IVPB defaults to 60 minutes infusion time. Max administration rate is 200 mg/min → so 1000 mg IV can be given over as fast as 5 minutes → ok to change infusion time if concern for arrhythmia



Calcium if ECG Changes Present

- Calcium gluconate 1 gram may be ordered as slow IV push over 5 minutes or ordered as an IVBP
  - In the setting of hyperkalemia with ECG changes, slow IV push administration preferred to expedite treatment
  - Onset: 1 – 2 minutes Duration: 30 minutes
  - DO NOT exceed administration rate of 200 mg/min → 1 gram calcium gluconate (10 mL) administered over 5 minutes
    - Overly rapid administration of calcium IV can result in vasodilation, hypotension, bradycardia, arrhythmias, syncope, and cardiac arrest
  - Calcium gluconate is a vesicant (but safer than calcium chloride)

Extravasation management:

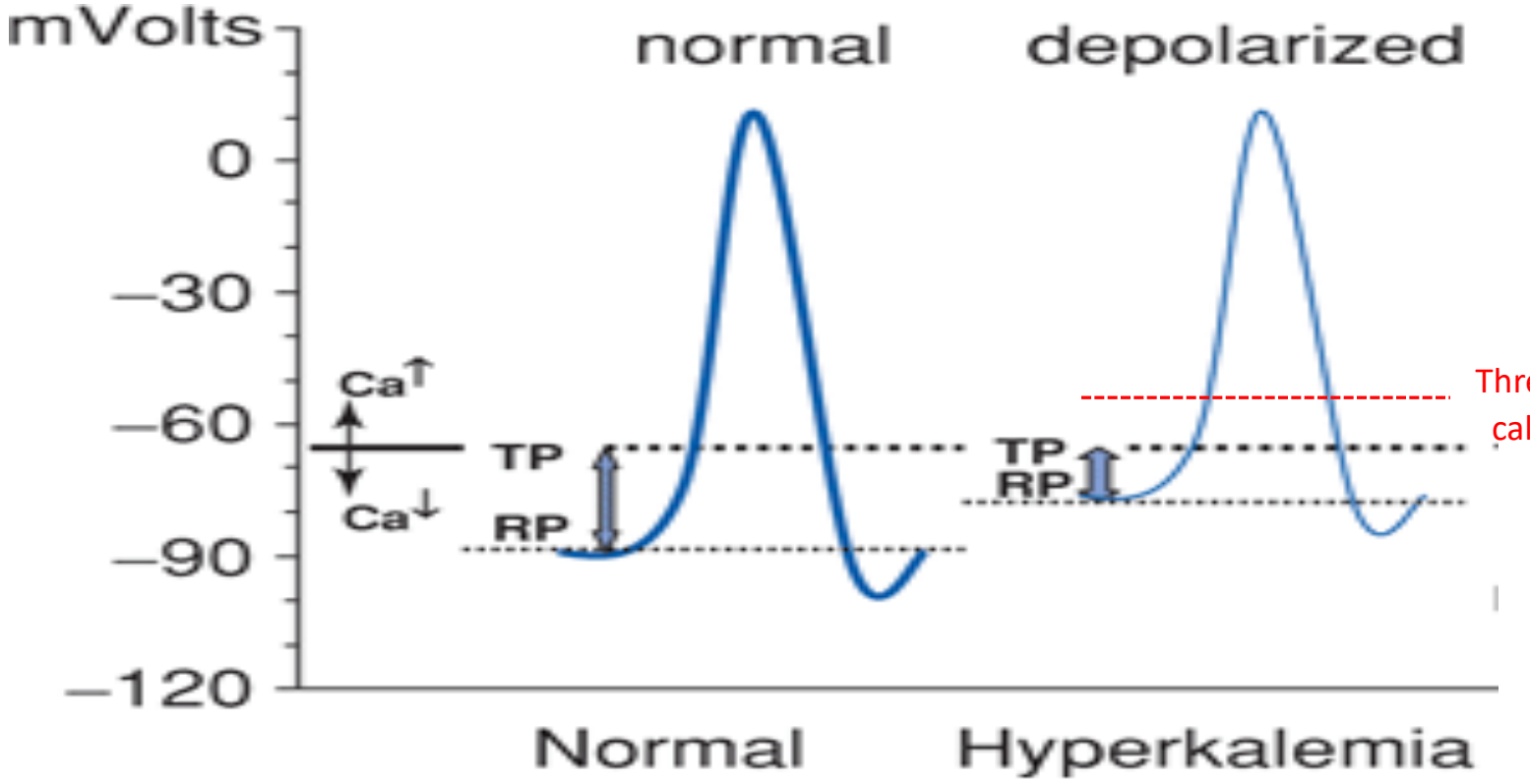
Calcium solutions/vesicant	Electrolytes	Hyaluronidase	Apply ice pack for 15 minutes every 6 hours for 48 hours (if ordered)

Calcium Chloride vs. Calcium Gluconate

- Calcium Chloride contains three times more calcium per gram than calcium gluconate (13.6 mEq vs 4.56 mEq per gram of each calcium salt respectively)
- Calcium chloride stored in crash carts
- Administration:
  - ACLS → rapid IV push
  - Non-arrest → pharmacy will compound IVPB if emergency situation administer no faster than 100 mg/min
  - In non- arrest: Central line preferred if since chloride salt more likely to cause tissue necrosis if extravasation occurs
- INCOMPATIBLE with sodium bicarbonate (flush between each dose if separate administration sites not possible)

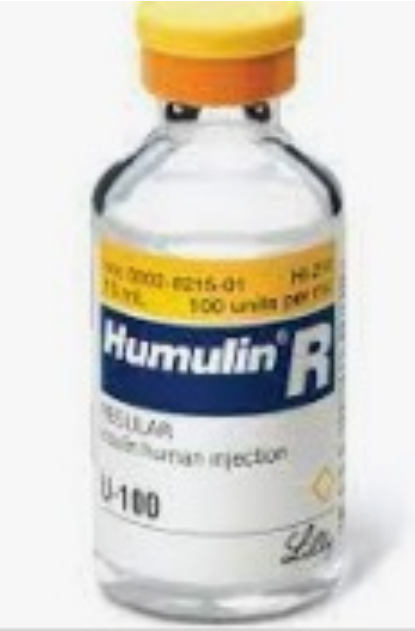
Mechanism of Calcium in Hyperkalemia

- Calcium changes the threshold potential to restore normal gradient with the distorted resting potential induced by hyperkalemia



RP = resting potential
TP = threshold potential

Use IV luer compatible insulin syringe to avoid dosing errors



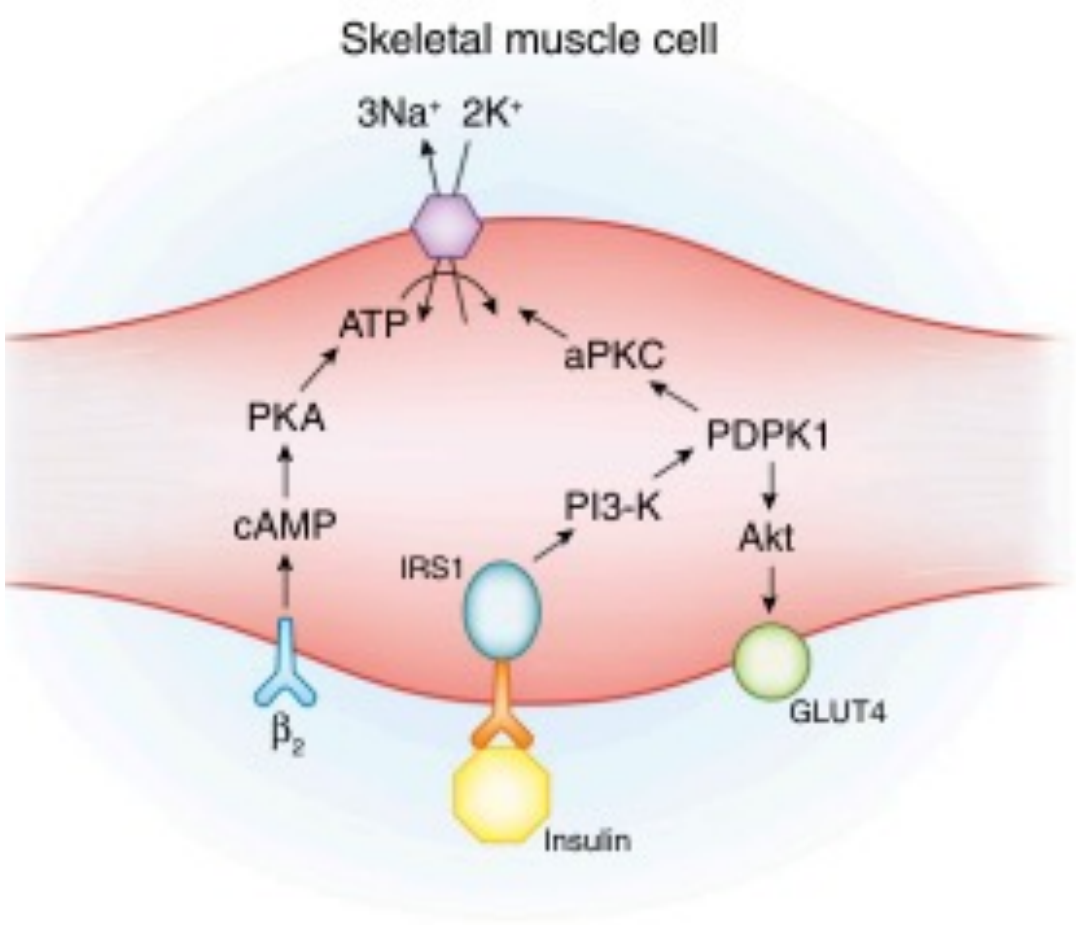
Insulin Regular 100 units/mL
→ 10 units = 0.1 mL

IV Regular Insulin + Dextrose 50% IV

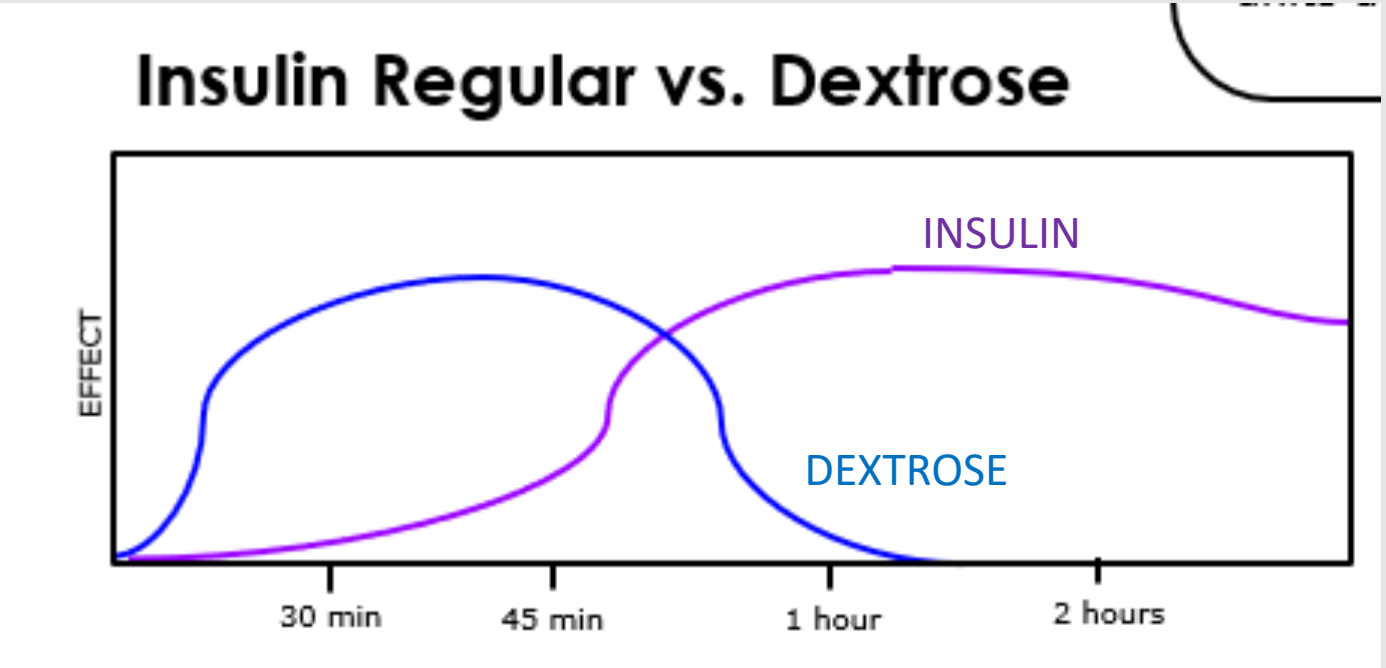
Insulin regular IV 5 – 10 units ONCE + Dextrose 50% 25 grams (50 mL)

Insulin Regular IV for Hyperkalemia: Mechanism

- After a meal, postprandial release of insulin regulates serum glucose and also allows for dietary potassium to shift into the cells until kidney excretion
- Insulin binding to skeletal muscle causes phosphorylation of insulin receptor substrate protein (IRS1) which binds to phosphatidylinositide 3-kinase (PI3-K) which activates 3-phosphoinosidtide-dependent protein kinase (PDK1) which activates atypical protein kinase C (aPCK) which leads to membrane insertion of the Na-K ATPase pump to drive potassium intracellularly



Insulin IV: Risk of Hypoglycemia



Bottonline: Even when D50% is administered with IV insulin, there is still a significant risk of hypoglycemia due to differences in duration

- IV insulin onset is fast (within 15 minutes) and long lasting, up to 6-8 hours (prolonged in renal insufficiency)
- IV Dextrose effect is to prevent hypoglycemia (but also increase insulin release)
  - Onset is slower, stimulating insulin release (onset hyperkalemia: 30 – 45 min)
  - The “protecting” effect to prevent hypoglycemia are short-lived, approximately an hour

Hypoglycemia after IV Insulin for Hyperkalemia

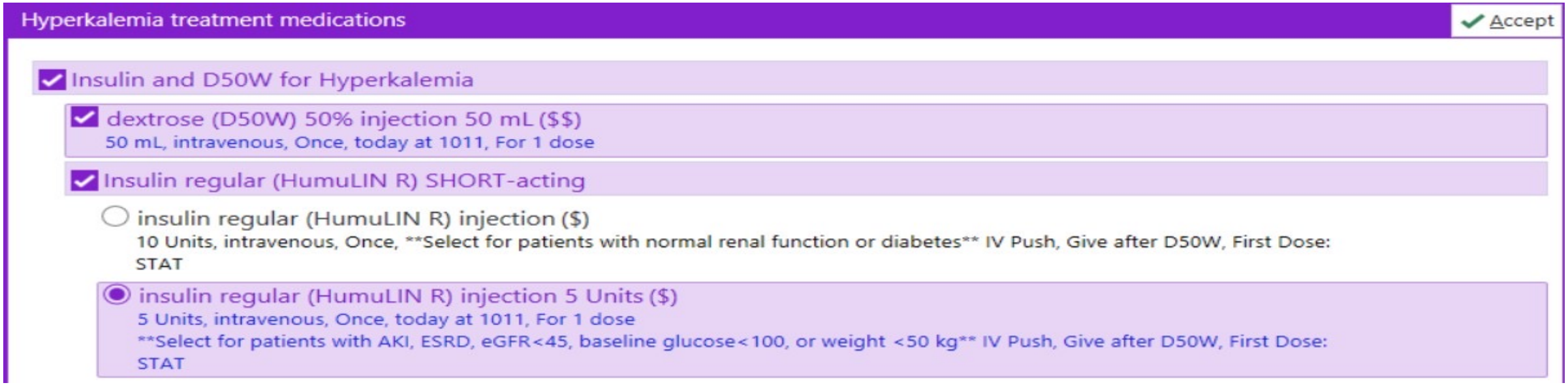
- Insulin regular 10 units IV for hyperkalemia → incidence of hypoglycemia:
  - 6 – 20% for all patients
  - 17 – 28% for subgroup with renal dysfunction
- In a trial of assessing only patients with renal dysfunction treated with IV regular insulin for hyperkalemia → incidence of hypoglycemia:
  - 5 units IV regular insulin = 19.5% hypoglycemia
  - 10 units IV regular insulin = 28.6% hypoglycemia

Crnobrnja L,et al. Scientific Reports. 2020 Dec 16;10(1):1-9.
Humphrey, et al. European Journal of Internal Medicine. 2022 Jan;95:87-92.
Chothia M, et al. PLoS ONE. 2022 May 12;17(5):1-17.

Regular Insulin IV Dose for Hyperkalemia

Hyperkalemia order panel update will include decision support

Use insulin lower dose of 5 units IV if "AKI, ESRD, eGFR < 45, baseline BG < 100 mg/dL, weight < 50 kg"



Note: current guidelines recommend patients presenting with a glucose ≥ 300 mg/dL DO NOT need to have dextrose administered with the insulin dose for treatment of hyperkalemia

Monitoring after IV Insulin Dose

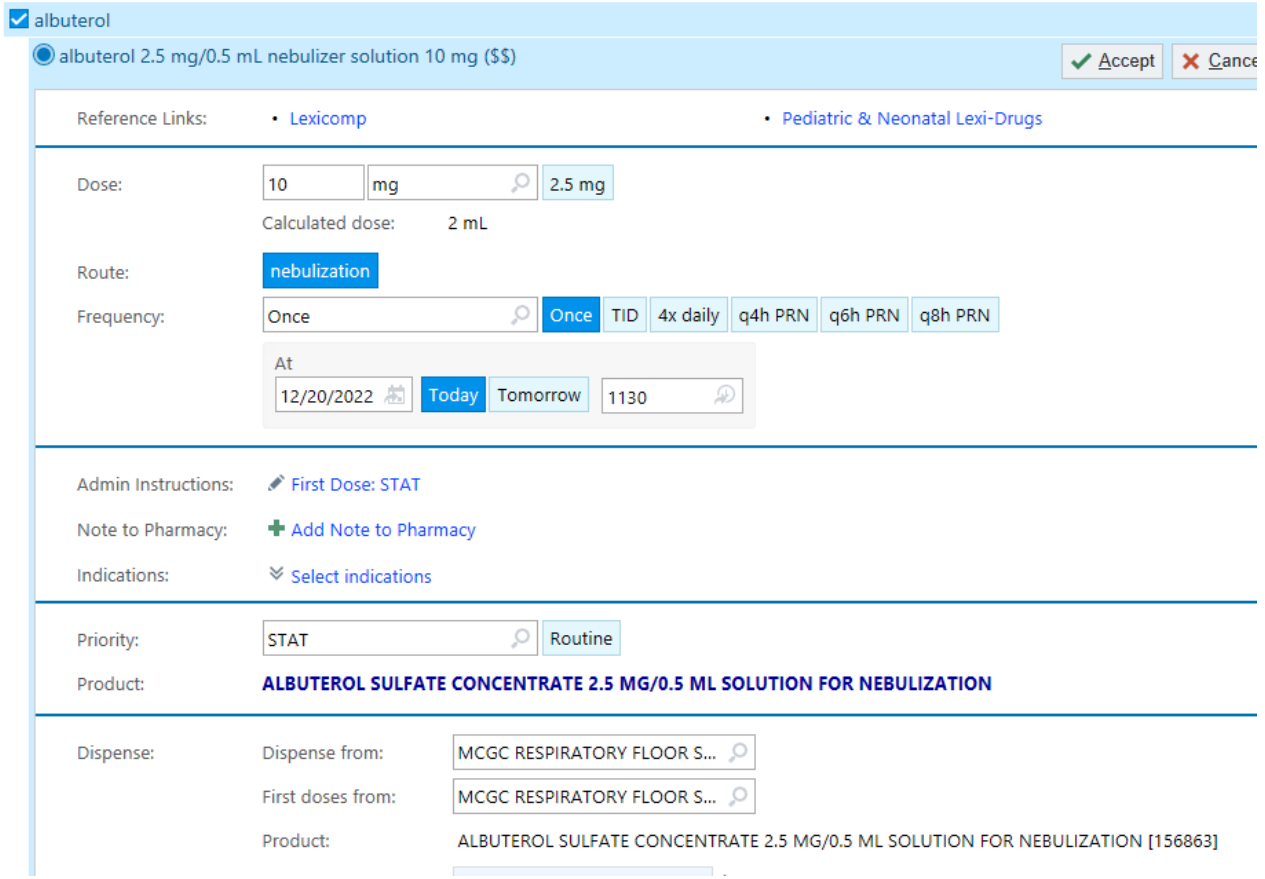
If patient not making urine or dialyzed or given exchange resin with subsequent bowel movement → remember, insulin only transiently moves potassium in the cell

- If the underlying etiology of hyperkalemia was not corrected, recheck potassium in 4 – 6 hours to ensure hyperkalemia has not returned

Blood glucose should be monitored for most patients who receive IV insulin, even when they are given dextrose to prevent hypoglycemia since the effects of regular insulin far outlast the effects of IV dextrose

- Hyperkalemia order panel should include hourly BG checks for several hours after IV insulin

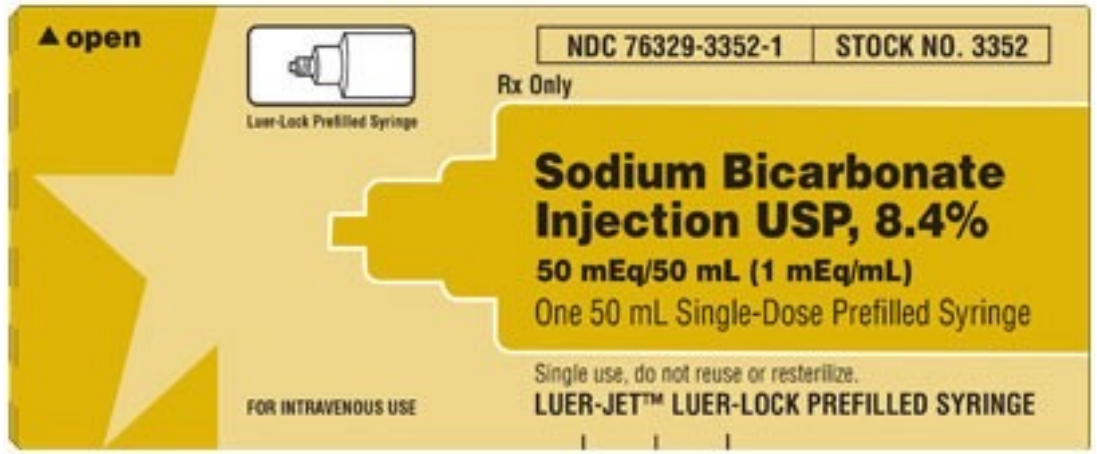
Albuterol for Hyperkalemia



- Note: albuterol dose is 10 mg = 4 pouches of albuterol 2.5 mg/0.5 mL nebulized

Sodium Bicarbonate for Hyperkalemia

- Should be reserved for hyperkalemia in the setting of concomitant metabolic acidosis (pH < 7.2)
- Randomized controlled trials have failed to show a benefit of hypertonic bicarbonate (50 meq IVP of 8.4% NaHCO3) → likely this is due to the hypertonicity pulling potassium out of cells ("solvent drag") counteracting the effect of increasing pH to move potassium intracellularly with an overall neutral effect on potassium
- Mechanism: HCO_3 initially causes Na influx via the Na/ HCO_3 cotransporter. Additional influx of Na subsequently increases activity of the Na/K ATPase, leading the increased K influx



Non-absorbable Cation Exchange Resins: Kayexalate vs. Lokelma

- Sodium polysyrene sulfonate (SPS) (Kayexalate) functions via a sodium ion-exchange mechanism in which sodium ions are released and replaced with potassium ions
- Sodium zirconium cyclosilicate (SZC) (Lokelma) works similarly, but also exchanges hydrogen ions, in addition to sodium ions, for potassium
- Similar efficacy, delayed onset for full effect (beginning within 1 hour and increasing over 48 hours)
- Kayexalate with warning for intestinal necrosis (especially when administered with sorbitol)
  - Use only inpatients with normal bowel function, avoid in postoperative patients at high risk for constipation, history of constipation (due to opioids), underlying bowel disease (ulcerative colitis, C.diff colitis)
- Lokelma now preferred agent even though off-label for acute hyperkalemia: dose 10 grams PO TID for up to 48 hours

Kayexalate

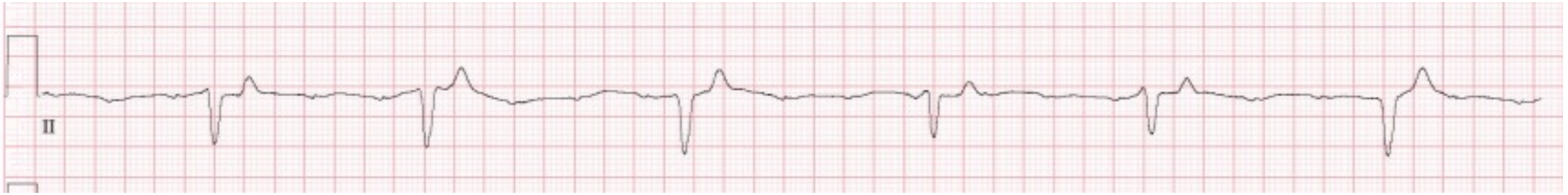
Potassium Elimination: Kaliuresis or Dialysis

- Elimination of excess potassium from the body via the kidneys (kaliuresis) or via dialysis
- Patients with ESRD on chronic dialysis will require emergent dialysis session
  - Serum potassium expected to fall by 1 mmol/L within first hour and 2 mmol/L by 190 minutes, plateau, then rebound expected over the next 1 – 6 hours so patients should be monitored on telemetry
- For patients not requiring dialysis, eliminate potassium by administering potassium wasting diuretic such as furosemide 40 mg IV

Example Case: MCGC

LAB		
Chemistry-General	Sodium Level	Potassium Level
06/08/2021 05:27 EDT	L 133 mMol/L	* C 7.8 mMol/L

Chemistry-General	BUN	Creatinine
06/07/2021 21:43 EDT	H 68 mg/dL	H 4.10 mg/dL



What will you order for this patient to treat hyperkalemia?

Example Case:

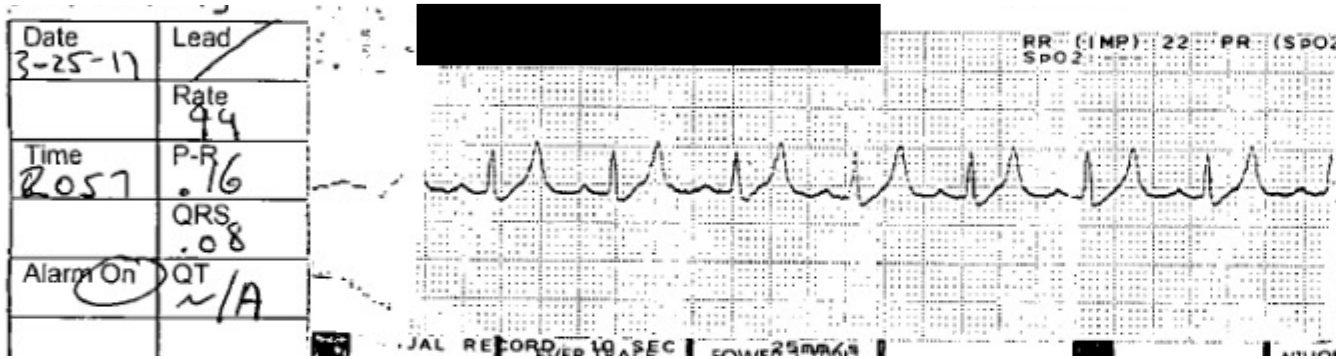
What order will these medications be administered in if the nurse asks for clarification?

What dose of regular insulin IV will you order?

What monitoring will you order for this patient?

Case #2: CG, 44/M Unresponsive at home

- CG is a 44/M patient who was found down after unknown amount of time and minimally responsive in his bathroom after reportedly snorting heroin, no known past medical history, unable to interview patient. Narcan administered on the scene, minimal response
- Patient remains obtunded on arrival to ED, decision made to intubate, patient is being ventilated with bag mask
- Ht: 5’5” Wt: 80.4 kg
- Vital signs: BP: 68/41 HR: 87 RR: 29 O2 sat 93% Temp 96.8ºF
- Stat labs are obtained
- Crash cart brought to room for suspected impending cardiac arrest
- Rhythm on the monitor at that time:



[redacted]

ID:01131875

25-MAR-2017 20:46:56

[redacted]

Vent. rate	192	BPM
PR interval	166	ms
QRS duration	170	ms
QT/QTc	166/296	ms
P-R-T axes	73 82	0

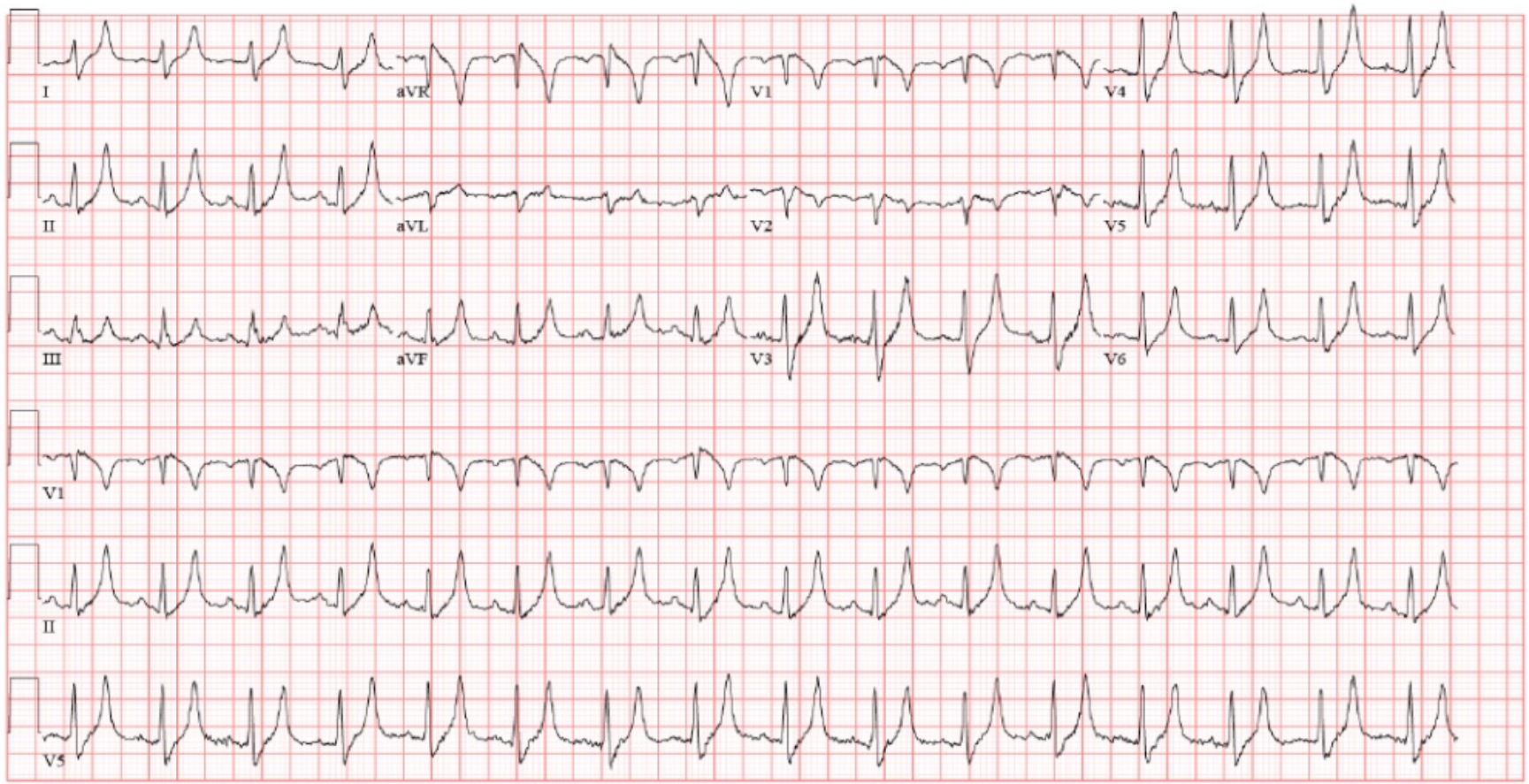
Sinus tachycardia with frequent premature ventricular complexes in a pattern of bigeminy
Nonspecific intraventricular block
Abnormal ECG
No previous ECGs available

[redacted]

Room:4402
Loc:6

Technician: all
Test ind:Emergency

Referred by: [redacted]

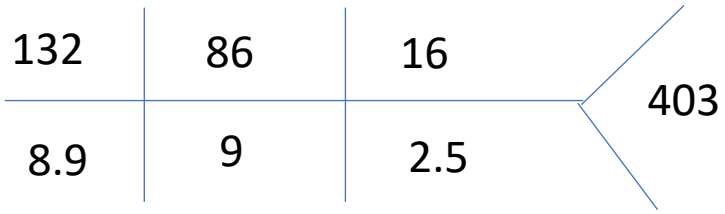


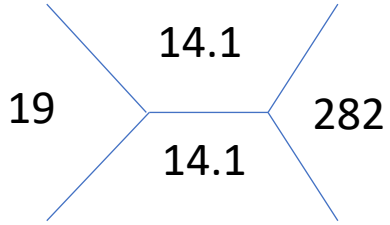
25mm/s 10mm/mV 150Hz 8.0 SP2 12SL 241 HD CID: 108

[redacted]

Case #2: CG, 44/M Unresponsive at home

ABG results: pH 6.793 CO2 41.1 O2 154 HCO3 6.2 (bagging)





Ca: 7.8
Mg: 3.8
Phos: 8.4
Lactate: 22
Ketones: neg

Alb: 2.1
Alk phos: 45
AST: > 7000
ALT: 2469
Bili: 0.7

Toxicology screen: positive only for opiates

Cortisol: 41
TSH: 5.6

PT: 13.6 INR: 1.3
aPTT: 30.3 seconds

Troponin: 0.18
CKMB: 247.3
CK: > 20,000

Rhabdomyolysis-induced AKI and severe hyperkalemia (exacerbated by severe metabolic acidosis)
How to treat severe hyperkalemia in this case?
What medication should be avoided for intubation?

HypOkalemia

Hypokalemia: General Overview

- Hypokalemia (K < 3.5 mEq/L) is a common problem in hospitalized patients
- Causes of hypokalemia:
  - Magnesium depletion
  - Potassium depletion: potassium-wasting diuretics (loop and thiazide), decreased intake of potassium, malnutrition, GI losses (diarrhea/laxatives), renal loss (hyperaldosteronism, amphotericin B)
  - Intracellular shifting of potassium: severe alkalosis, insulin, beta agonists, sodium bicarbonate, theophylline, caffeine
- Pathophysiology: hypokalemia results in membrane hyperpolarization and impaired muscle contraction
- Signs/symptoms: nausea, vomiting, weakness, constipation, paralysis, respiratory compromise

Kjeldsen K. 2010 Winter;15(4):e96-9.

Kraft MD, et al. Am J Health Syst Pharm. 2005 Aug 15;62(16):1663-82.

Potassium: HypOkalemia

Etiology

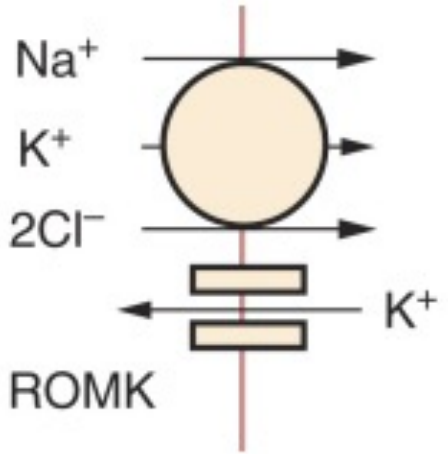
Symptoms

Treatment

HypOkalemia: Etiology

Urinary losses (diuretics, mineralocorticoid excess, polyuria, amphotericin B, salt wasting (Barter's or Gitelman's syndrome, renal tubular acidosis)
Gastrointestinal losses (vomiting, diarrhea, gastric tube drainage (NG/OG, ileostomy), laxative abuse)
Metabolic alkalosis
Hypomagnesemia
Poor oral intake
Hypothermia
Medications: diuretics, insulin, sodium bicarbonate, beta agonist (albuterol)

Hypokalemia in the Presence of Hypomagnesemia



- Hypomagnesemia is often associated with hypokalemia because it causes urinary potassium wasting
- Remember: refractory hypokalemia may be the result of low magnesium levels
  - Mechanism: magnesium regulates the activity of ROMK (renal outer medullary potassium channel) where low magnesium causes high efflux activity and potassium losses

Huang CL, et al. Mechanism of hypokalemia in magnesium deficiency. J Am Soc Nephrol. 2007 Oct;18(10):2649-52.

HypOkalemia: Symptoms

Decreased extracellular potassium hyperpolarizes cell membranes (more electronegative) impairing the ability of the muscle to depolarize or contract

Nausea/Vomiting

Muscle weakness

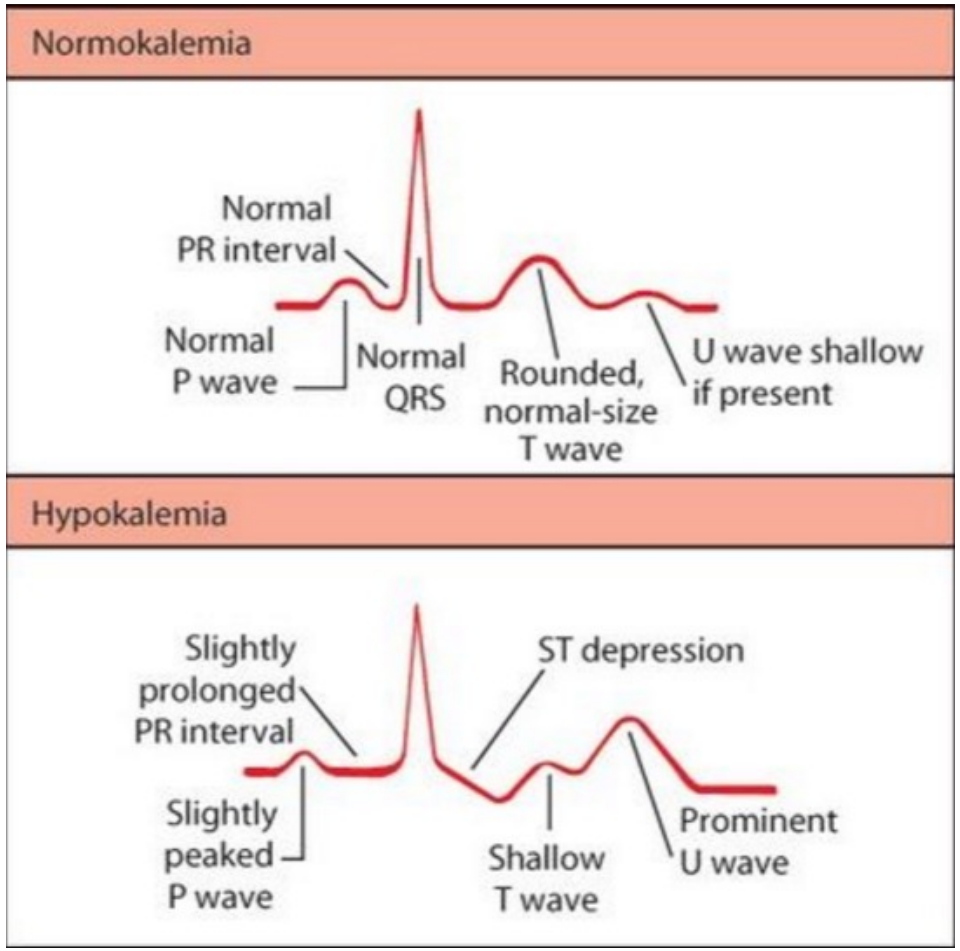
Respiratory depression

Constipation

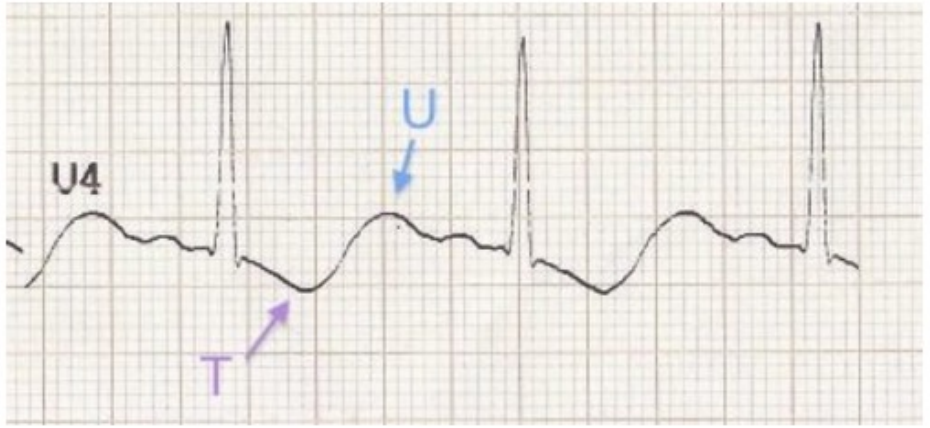
Rhabdomyolysis

ECG changes

Hypokalemia: ECG Changes



- Increased amplitude and width of the P wave
- Prolonged PR interval
- T wave flattening and inversion
- ST depression
- Prominent U waves (may see fusion of the T and U waves)
- May progress to life threatening ventricular arrhythmias



T wave inversion and prominent U waves in hypokalaemia

Levis JT. Perm J. 2012 Spring; 16(2): 57.

[redacted]

[redacted]

13-OCT-2016 13:41:23

[redacted]

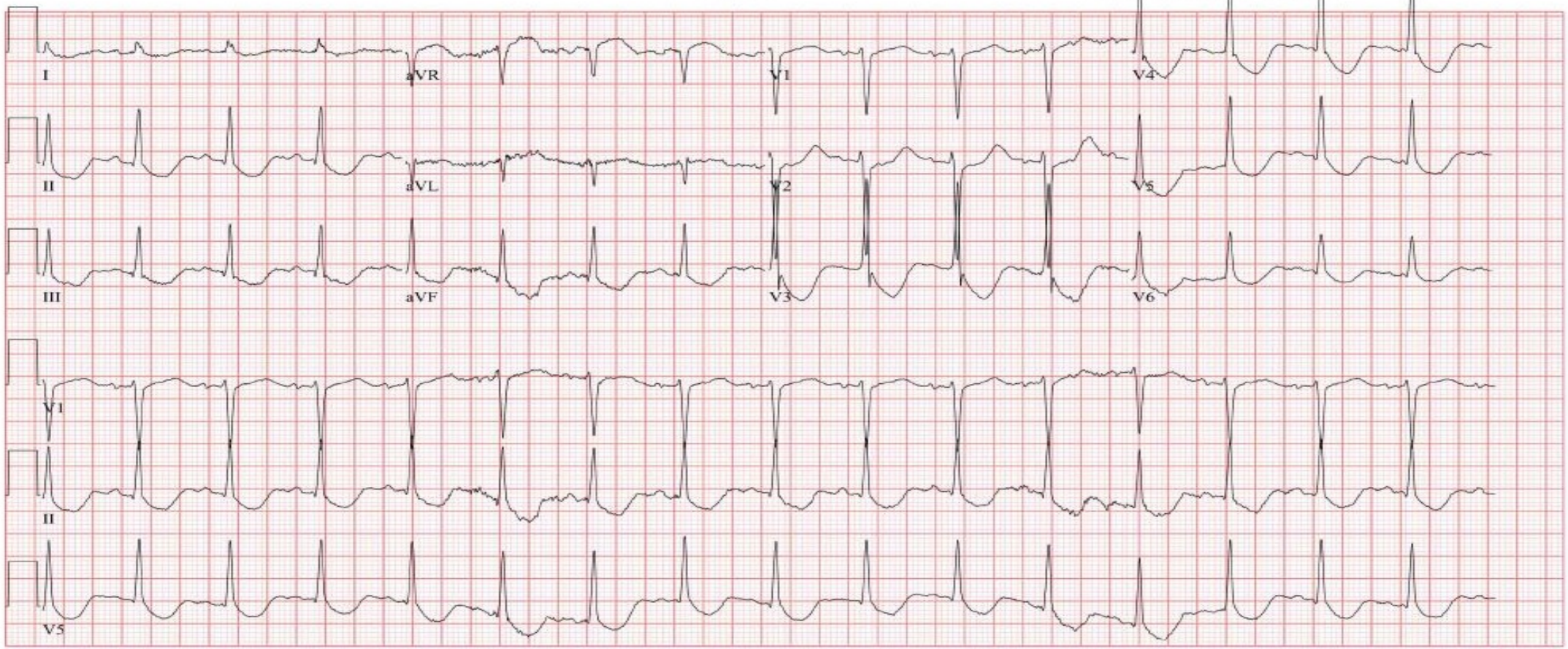
Vent. rate	96	BPM
PR interval	156	ms
QRS duration	94	ms
QT/QTc	418/528	ms
P-R-T axes	60 79	266

Normal sinus rhythm
Diffuse ST changes
Prolonged QT
Abnormal ECG
When compared with ECG of 13-OCT-2016 09:12,
No significant change was found

Technician: allegretto
Test ind:Other~

[redacted]

Referred by: [redacted]



25mm/s 10mm/mV 150Hz 8.0 SP2 12SL 241 HD CID: 104

[redacted]

Hyperglycemic Crises in Adult Patients With Diabetes

- IV insulin can cause significant hypokalemia, therefore, it is important to check serum potassium level before initiating insulin therapy during the treatment of DKA
- Insulin treatment should be delayed until potassium concentration is restored to > 3.3 mEq/L to avoid life threatening arrhythmias and respiratory muscle weakness

Potassium

Despite total-body potassium depletion, mild-to-moderate hyperkalemia is common in patients with hyperglycemic crises. Insulin therapy, correction of acidosis, and volume expansion decrease serum potassium concentration. To prevent hypokalemia, potassium replacement is initiated after serum levels fall below the upper level of normal for the particular laboratory (5.0–5.2 mEq/l). The treatment goal is to maintain serum potassium levels within the normal range of 4–5 mEq/l. Generally, 20–30 mEq potassium in each liter of infusion fluid is sufficient to maintain a serum potassium concentration within the normal range. Rarely, DKA patients may present with significant hypokalemia. In such cases, potassium replacement should begin with fluid therapy, and insulin treatment should be delayed until potassium concentration is restored to >3.3 mEq/l to avoid life-threatening arrhythmias and respiratory muscle weakness (4,13).

Kitabchi AE, et a. Diabetes Care. 2009 Jul; 32(7): 1335-1343.

Hypokalemia Treatment

- It takes significant loss in potassium stores to see a drop in serum sodium (since most potassium is in the intracellular space compensating for losses
- It is estimated that for every 0.3 mEq/L decrease in serum potassium concentration → the total body potassium deficit is approximately 100 mEq
  - This may be an overestimate in patients who have alkalosis or have received medications that caused an intracellular shift of potassium → in that case the total body potassium is unchanged and correction of alkalosis or removal of offending medication will result in redistribution of potassium into the extracellular space

Aboujamoush H, et al. Evaluation fo the change in serum potassium levels after potassium administration. J Clin Neph Ren Care. 2016. 2(2):JCNRC-3-013,

Potassium Replacement

- For every 10 mEq potassium administered → expected increase in serum potassium = 0.1 mEq/L
- If potassium is refractory to replacement, assess for ongoing potassium losses (urinary wasting due to diuretics or hypomagnesemia), or intracellular shifting (alkalosis or medication-induced)

K+ (mEq/L)	Replacement
≥4	None
3.7-3.9	20 mEq KCl
3.4-3.6	40 mEq KCl
3-3.3	60 mEq KCl
< 3	80 mEq KCl

Potassium is NOT given all at once for doses > 20 mEq

Aboujamoush H, et al. Evaluation fo the change in serum potassium levels after potassium administration. J Clin Neph Ren Care. 2016. 2(2):JCNRC-3-013,

Hypokalemia Treatment

Hospital Policy Potassium Administration Guidelines:

	Maximum Bolus Concentration	Maximum Administration Rate NOT on Cardiac Monitor	Maximum Administration Rate ON Cardiac Monitor	Maximum Maintenance IV Fluid concentration
Central Line Administration	20 mEq per 50 mL 40 mEq per 100 mL	10 mEq per hour	20 mEq per hour	60 mEq per Liter
Peripheral Line Administration	10 mEq per 100 mL	10 mEq per hour	10 mEq per hour	40 mEq per Liter

- Maximum rate of potassium supplementation in central line with telemetry monitoring is 20 mEq/hr → in life-threatening (peri-cardiac arrest) situations 40 mEq/hr is recommended max (document that rapid replacement is intentional due to emergent situation)

Vanden Hoek TL, et al. Circulation. 2010 Nov 2;122(18 Suppl 3):S829-61.

The Joint Commission

Sentinel Event Alert

Medication Error Prevention -- Potassium Chloride

In the two years since the Joint Commission enacted its Sentinel Event Policy, the Accreditation Committee of the Board of Commissioners has reviewed more than 200 sentinel events. The most common category of sentinel events was medication errors, and of those, the most frequently implicated drug was potassium chloride (KCl). The Joint Commission has reviewed 10 incidents of patient death resulting from misadministration of KCl, eight of which were the result of direct infusion of concentrated KCl. In all cases, a contributing factor identified was the availability of concentrated KCl on the nursing unit. In six of the eight cases, the KCl was mistaken for some other medication, primarily due to similarities in packaging and labeling. Most often, KCl was mistaken for sodium chloride, heparin or furosemide (Lasix).

Issue For Consideration

In light of this experience, the Joint Commission suggests that health care organizations NOT make concentrated KCl available outside of the pharmacy unless appropriate specific safeguards are in place.

"The way to prevent tragic deaths from accidental intravenous injection of concentrated KCl is excruciatingly simple -- organizations must take it off the floor stock of all units. It is one of the best examples I know of a 'forcing function' -- a procedure that makes a certain type of error impossible."

Lucian L. Leape, M.D. Harvard School of Public Health

"Unfortunately, there are too many in health care who feel that if it hasn't happened to them, the adverse experiences of others do not apply. That is why potassium chloride concentrate vials can still be found in patient care areas."

Michael Cohen, MS, FASHP, President, Institute for Safe Medication Practices

ISMP
Institute for Safe Medication Practices
An ECRI Affiliate

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FEATURED ARTICLES

Administration of Concentrated Potassium Chloride for Injection During a Code: Still Deadly!

June 3, 2021

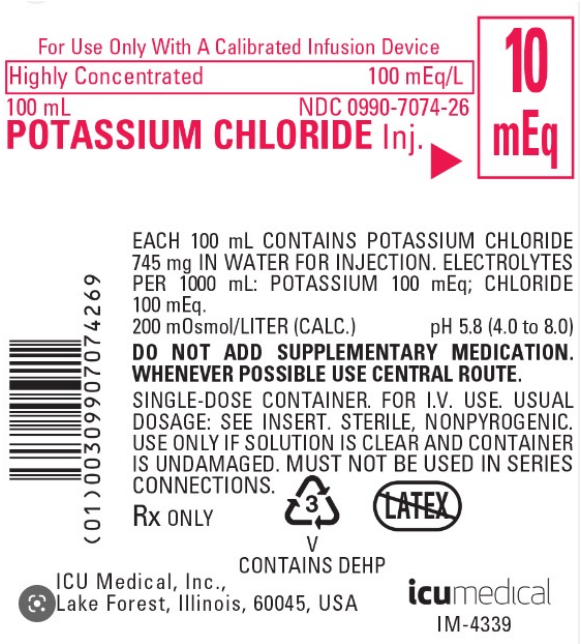
- 2021 case where a provider gave verbal order for potassium chloride 20 mEq IV during the course of a code blue for a patient who had experienced cardiac arrest. The pharmacist incorrectly assumed the provider wanted the potassium to be given as IV push and requested it from the pharmacy, drew it up from the vial, gave it to the nurse for administration, and the patient immediately developed asystole and was unable to be resuscitated

Administration of Concentrated Potassium Chloride for Injection During a Code: Still Deadly! | Institute For Safe Medication Practices (ismp.org)

HypOkalemia: TREATMENT IV

Potassium Level	Recommended Replacement
3.7 – 3.9	KCl 20 mEq
3.4 – 3.6	KCL 40 mEq
3 – 3.3	KCl 60 mEq
< 3	KCl 80 mEq

PERIPHERAL LINE: KCL 10 mEq/100 mL INFUSED OVER 1 HOUR



CENTRAL LINE AND TELEMETRY: KCL 20 mEq/100 mL INFUSED OVER 1 HOUR

EXAMPLE: A patient with K = 3.5 mmol/L needs 40 mEq KCL → If this patient only has peripheral IV access this is ordered as KCL 10 mEq/100 mL q1hr x 4 doses for total of 40 mEq

REMINDER: DKA Potassium Replacement Utilizes a More Aggressive Proactive Scale

Insulin causes intracellular shifts of potassium. Hypokalemia is an expected consequence of continuous infusion IV insulin. For this reason, potassium chloride is proactively administered to any patient on DKA insulin infusion unless they already have hyperkalemia

DO NOT use the non-DKA potassium Replacement" order panel → it is reactive replacement once K < 3.9 mEq/L

K+ (mEq/L)	Replacement
≥4	None
3.7-3.9	20 mEq KCl
3.4-3.6	40 mEq KCl
3-3.3	60 mEq KCl
< 3	80 mEq KCl

Use the DKA Potassium Replacement Scale → it is a proactive replacement for K ≤ 5.2 mEq/L

K+ (mEq/L)	DKA Potassium Replacement Scale
>5.2	None
4-5.2	20 mEq KCl
3-3.9	40 mEq KCl
≤2.9	60 mEq KCl

HypOkalemia: Oral Products

- Patient can swallow whole tablets:
  - K-Dur = Potassium Chloride SR (sustained release) 10 mEq and 20 mEq tablets
  - Many patients find K-Dur tablets difficult to swallow because they are large
  - DO NOT crush or break tablets (ruins the release mechanism)
- Potassium oral replacement with feeding tube or unable to swallow whole tablets:
  - KLOR-KON packets: Potassium chloride 20 mEq oral packet
    - Dissolve each packet in 4 oz of water (120 mL) = 5 mEq per 1 oz fluid (30 mL)
  - Potassium chloride oral solution cups are very expensive → many hospitals use packets and dissolve

Oral Potassium Pearls

- Various potassium salts available (check hospital formulary) such chloride, bicarbonate, citrate, phosphate
  - Potassium chloride most common
  - Potassium bicarbonate PO can be used if correction of acidosis is also necessary
  - Potassium phosphate can be used when concurrent hypophosphatemia
- Maximum single oral dose of oral potassium products = 40 mEq
- Take oral potassium with meals and a glass of water or other liquid to minimize GI irritation

Hypokalemia and Hypophosphatemia

Available products to replace both potassium and phosphate

Oral phosphorus replacement options and electrolyte content:

Each dosage form contains combination potassium phosphate and sodium phosphate	**Total PHOSPHORUS per tablet/packet**	**Total POTASSIUM per tablet/ packet**	**Total SODIUM per tablet/ packet**
K-Phos Neutral 250 mg tablet	250 mg **(8 mmol)**	45 mg **(1.1 mEq)**	298 mg **(13 mEq)**
Phos-NaK 250 mg powder for solution	250 mg **(8 mmol)**	280 mg **(7.1 mEq)**	160 mg **(6.9 mEq)**

Potassium phosphate IV:

	Phosphate	**Potassium**	**Volume**
Potassium Phosphate	**15 mmol**	22 mEq K	250 mL NS
	30 mmol	44 mEq K	500 mL NS

- **Phosphorus level 2.3—3 mg/dL**:
  - **IV**: recommend 15 mmol phosphorus
  - **PO**: recommend K-Phos tablet or phos-NaK packet (500 mg (contains 16 mmol phosphorus)
- **Phosphorus level 1.6—3.3 mg/dL**:
  - **IV**: recommended 15—30 mmol phosphorus
  - **PO**: recommend K-Phos tablet or Phos-NaK packet (500 mg (contains 16 mmol phosphorus)
- **Phosphrours level LESS than 1.6 mg/dL**:
  - **IV**: recommended 60 mmol phosphorus

Hypokalemia: Example

- Patient presented with agitation, confusion, and tremor → history of alcohol abuse and symptoms are consistent with alcohol withdrawal
- Laboratory values pictured
- What would you order for IV potassium replacement through peripheral line?
  - Potassium of 3.1 mmol/L → start with 60 mEq KCl

K+ (mEq/L)	Replacement
≥4	None
3.7-3.9	20 mEq KCl
3.4-3.6	40 mEq KCl
3-3.3	60 mEq KCl
< 3	80 mEq KCl

Order as KCl 10 mEq IV over 1 hour per dose x 6 doses → then recheck potassium

Need to also replace Magnesium 2 g IVPB

CHEM PROFILE	
Sodium	130
Potassium	3.1
Chloride	94
CO2	26
Anion Gap	10
Glucose	118
BUN	9
Creatinine	0.77
Calculated GFR	101
BUN/Creatinine Ratio	11.7
Calcium	8.0

Magnesium	1.2

Questions