

Electrolytes and Acid-Base Practice for the Boards II

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 - Clinical focus: Nephrology
 - Research focus: Kidney physiology, PKD

Disclosures

- I have no financial disclosures

Objectives

- Use ABIM-style MCQs to:
 - Review the diagnostic approach to common electrolyte disorders, including hyperkalemia and acid-base disturbances
 - Review the approach to management of common electrolyte disorders

A 41-year-old female with lupus nephritis and chronic renal insufficiency maintained on prednisone and candesartan presents for a routine clinic visit. On exam, BP is 146/95 and there is moderate dependent edema.

Laboratory studies:

Serum sodium	136 mEq/L
Serum potassium	6.3 mEq/L
Serum chloride	109 mEq/L
Serum bicarbonate	18 mEq/L
Blood urea nitrogen	25 mg/dL
Serum creatinine	1.6 mg/dL

Which of the following would NOT be appropriate in the management of this patient:

- (A) Hydrochlorothiazide
- (B) Discontinue candesartan
- (C) Spironolactone
- (D) Low potassium diet
- (E) Potassium-binding resin

Which of the following would NOT be appropriate in the management of this patient:

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- (D) Low potassium diet
- (E) Potassium-binding resin

Hyperkalemia

↑ Intake

Decreased urinary
K⁺ excretion

24 hr urine K⁺ < 40 mEq

TTKG < 6

Cell shift

Metabolic acidosis

Hyperglycemia

β-blocker

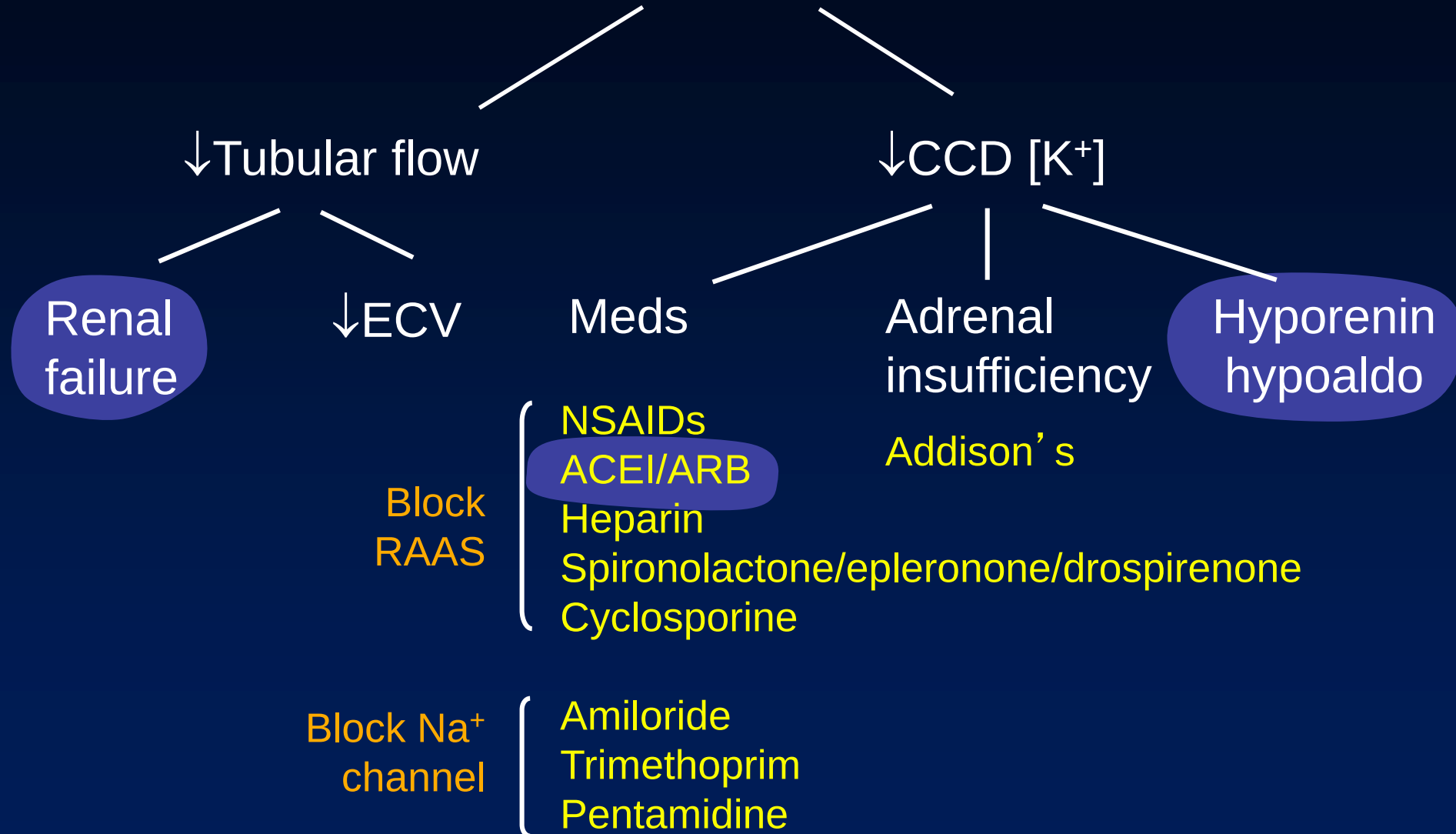
Digitalis

Hyperkalemic periodic

paralysis

Cell lysis

Decreased urinary K⁺ excretion



A 48 yr-old female was evaluated for several weeks of fatigue, nausea and mild generalized abdominal pain. Exam revealed a thin, tanned female with blood pressure of 90/45 mm Hg. Her palmar creases are hyperpigmented.

Serum sodium	131 mEq/L
Serum potassium	6.1 mEq/L
Serum chloride	105 mEq/L
Serum bicarbonate	20 mEq/L
Blood urea nitrogen	11 mg/dL
Serum creatinine	0.5 mg/dL
Serum glucose	40 mg/dL

Which of the following tests would be most likely to establish the diagnosis in this patient:

- (A) Plasma renin and aldosterone
- (B) Serum cortisol at baseline and after ACTH
- (C) Serum C-peptide
- (D) Iothalamate GFR determination
- (E) Genetic testing of the sodium channel gene, SCN4A

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- (E) Genetic testing of the sodium channel gene, SCN4A

A 26-year-old male with AIDS is admitted with *Pneumocystis jirovecii* pneumonia and treated with prednisone and intravenous trimethoprim-sulfamethoxazole. On examination, BP is 115/65, HR 90, RR 24. He appears tachypneic with diffuse rales on chest auscultation. EKG appears normal.

Serum sodium	134 mEq/L
Serum potassium	5.6 mEq/L
Serum chloride	105 mEq/L
Serum bicarbonate	22 mEq/L
Blood urea nitrogen	8 mg/dL
Serum creatinine	0.7 mg/dL

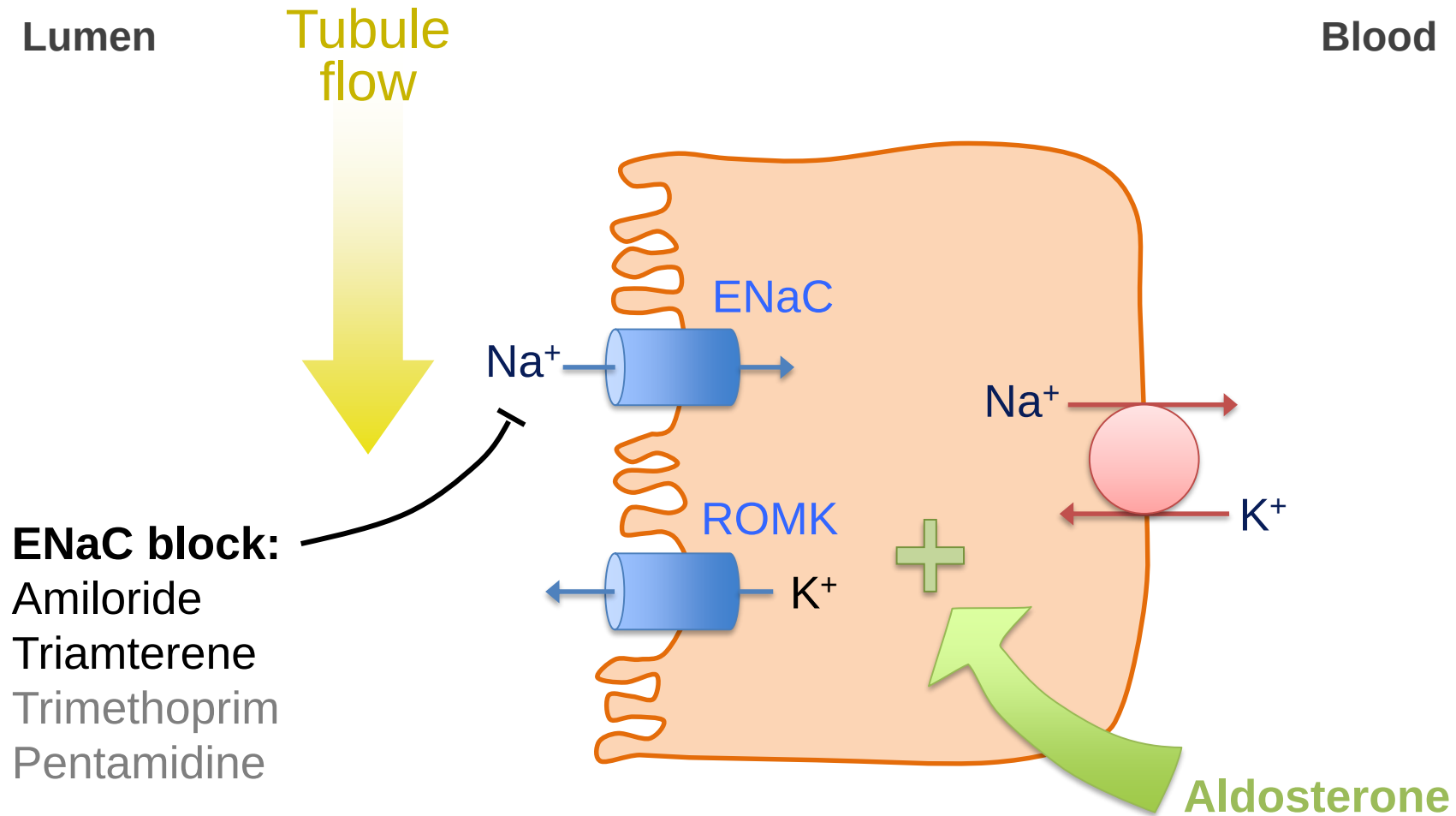
Which of the following would be the best treatment for the hyperkalemia in this patient:

- (A) Fludrocortisone
- (B) Sodium bicarbonate
- (C) Discontinue trimethoprim-sulfamethoxazole and start pentamidine
- (D) Discontinue trimethoprim-sulfamethoxazole and start clindamycin-primaquine
- (E) None of the above

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- ☒ (D) Discontinue trimethoprim-sulfamethoxazole and start clindamycin-primaquine
- (E) None of the above

Collecting duct K^+ secretion



A 57-year-old female with end-stage renal disease secondary to diabetic nephropathy maintained on chronic hemodialysis is seen on a non-dialysis day.

Serum sodium	139 mEq/L
Serum potassium	7.1 mEq/L
Serum chloride	108 mEq/L
Serum bicarbonate	20 mEq/L
Blood urea nitrogen	26 mg/dL
Serum creatinine	4.5 mg/dL

All of the following would lower the serum potassium substantially EXCEPT:

- (A) Insulin and glucose
- (B) Sodium bicarbonate
- (C) Albuterol
- (D) Sodium zirconium silicate
- (E) Hemodialysis

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- (C) Albuterol
- (D) Sodium zirconium silicate
- (E) Hemodialysis

Treatment of hyperkalemia

- Stabilize membrane excitability
 - Calcium chloride or gluconate, 1 g IV
- Increase K^+ entry into cells
 - Glucose 25 g and insulin 10 U
 - β_2 -adrenergic agonist (albuterol 10-20 mg inh)
 - $NaHCO_3$ (*poor efficacy in ESRD patients*)
- Removal of excess K^+
 - Cation exchange resin (Kayexalate)
 - Diuretics
 - Dialysis
- Dietary K^+ restriction

Efficacy of treatments for hyperkalemia in dialysis patients

Treatment	↓Serum K ⁺
Insulin + glucose	0.85
Albuterol/epinephrine	0.3
Sodium bicarbonate	0
Hemodialysis	1.3

A 23 year-old male with diabetes mellitus presents with 5 days of polyuria, polydipsia and abdominal pain.

Serum sodium	134 mEq/L
Serum potassium	6.5 mEq/L
Serum chloride	99 mEq/L
Serum bicarbonate	15 mEq/L
Serum glucose	296 mg/dL
Blood urea nitrogen	38 mg/dL
Serum creatinine	1.6 mg/dL

Urinalysis:

Specific gravity 1.028, pH 4.5, 1+ protein, 2+ ketones

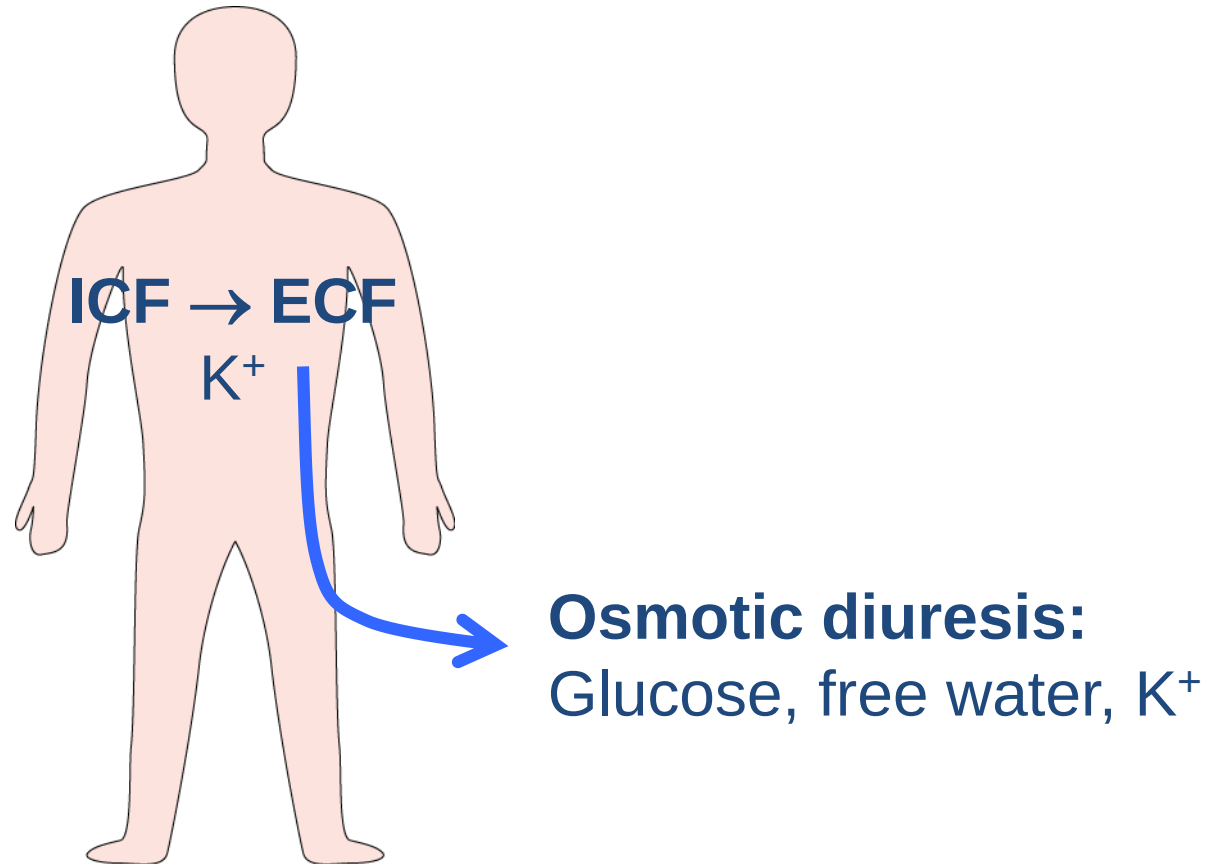
Which of the following statements about the total body stores in this patient is most likely to be correct:

- (A) Total body potassium depleted
- (B) Total body potassium overloaded
- (C) Normal total body potassium stores but redistributed into the extracellular space
- (D) Normal total body potassium stores but redistributed into the intracellular space
- (E) None of the above

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- (E) None of the above

Electrolyte imbalance in diabetic ketoacidosis



An 18-year-old female is brought in after a suspected suicidal attempt. Unidentified pills were found in her pocket. On exam she is obtunded. BP 101/64 mm Hg, HR 112/min, RR 30/min, T 101.8° C.

Serum sodium	140 mEq/L
Serum potassium	3.8 mEq/L
Serum chloride	99 mEq/L
Serum bicarbonate	15 mEq/L
Blood urea nitrogen	9 mg/dL
Serum creatinine	0.8 mg/dL
Serum glucose	81 mg/dL
Arterial pH	7.35
Arterial PCO ₂	25 mm Hg

Case A1

Which of the following is most likely to be helpful in this patient:

- (A) Forced alkaline diuresis
- (B) Intravenous ethanol
- (C) Insulin drip
- (D) Glucagon
- (E) Potassium citrate

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- (B) Intravenous ethanol
- (C) Insulin drip
- (D) Glucagon
- (E) Potassium citrate

pH 7.35, HCO_3^- 15

Actual $\text{PCO}_2 = 25$ (expected: $1.5(15)+8 \sim 30$)

Primary metabolic acidosis and primary respiratory alkalosis

$\text{AG} = 140 - 99 - 15 = 26$

$\Delta\text{AG}/\Delta\text{HCO}_3^- = (26-10)/(24-15) \sim 2$

Anion gap acidosis and metabolic alkalosis (i.e. triple acid-base disorder)

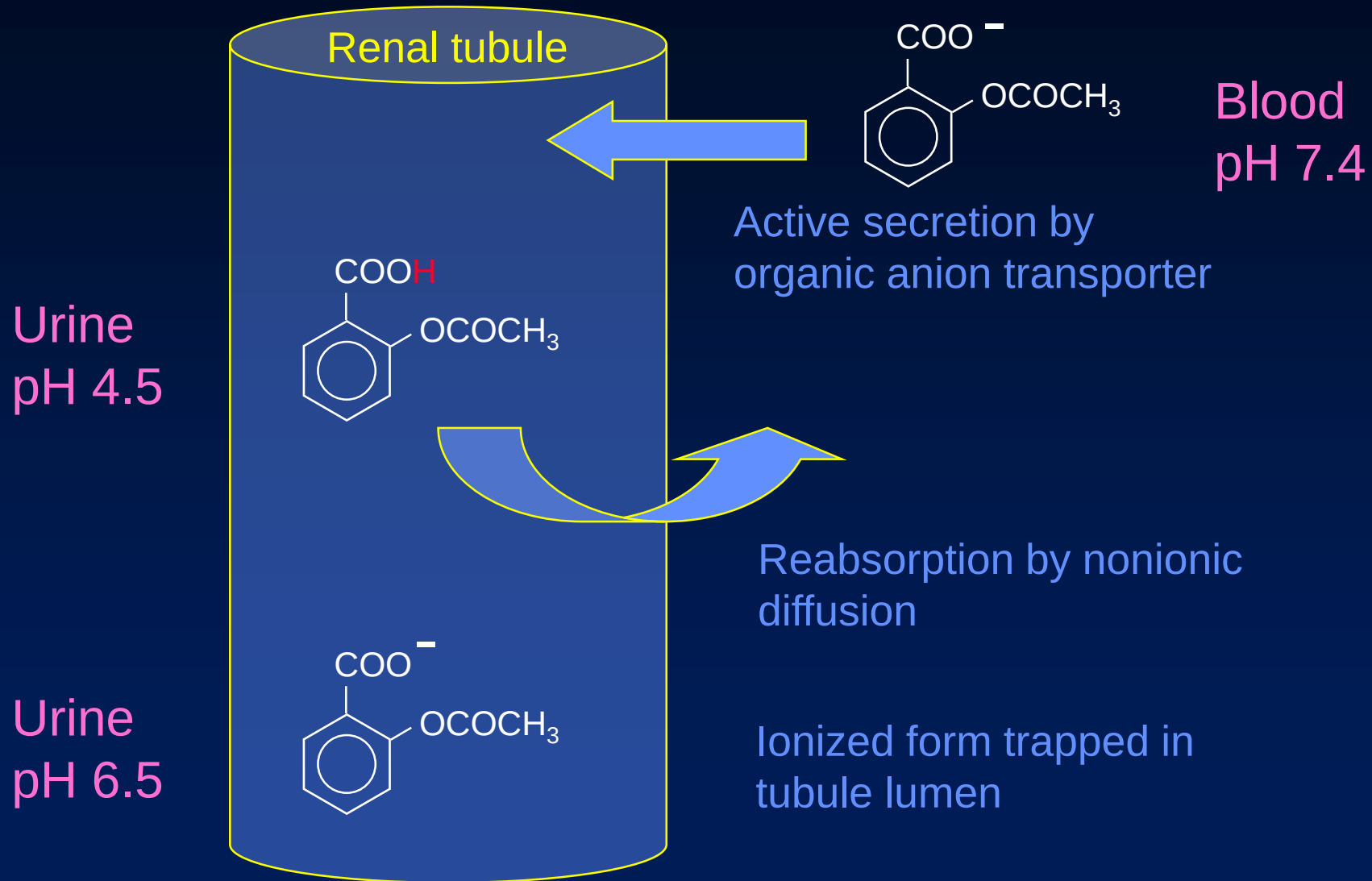
Pt is febrile, tachycardic, tachypneic and had probable drug overdose

Suspect salicylate poisoning

Rx is alkaline diuresis +/- hemodialysis

Case A1

Alkaline diuresis increases urinary salicylate excretion by ion trapping



A 29-year-old female is found unconscious and brought into the emergency room. BP 105/67 mm Hg. She is comatose and has an alcoholic fetor.

Serum sodium	129 mEq/L
Serum potassium	3.4 mEq/L
Serum chloride	94 mEq/L
Serum bicarbonate	11 mEq/L
Blood urea nitrogen	11 mg/dL
Serum creatinine	1.6 mg/dL
Serum glucose	72 mg/dL
Serum lactate	1 mmol/L
Serum creatine kinase	20 mU/mL
Serum osmolality	300 mOsm/kg
Arterial pH	7.22
Arterial PCO ₂	24 mm Hg

Serum levels of ethanol, ketones by the nitroprusside test, β -hydroxybutyrate, and salicylate were all negative.

Case A2

The most appropriate first step in the management of this patient is:

- (A) Forced alkaline diuresis
- (B) Dopamine
- (C) Fomepizole
- (D) Thiamine
- (E) Hemodialysis

The most appropriate first step in the management of this patient is:

(A) Forced alkaline diuresis

(B) Dopamine

(C) Fomepizole

(D) Thiamine

(E) Hemodialysis

pH 7.22, HCO₃ 11

Actual PCO₂ = 24 (expected: 1.5(11)+8 = 24)

Pure metabolic acidosis

$$AG = 129 - 94 - 11 = 24$$

$$\Delta AG / \Delta HCO_3 = (24-10)/(24-11) = 1.1$$

Pure anion gap acidosis

Case A2

Serum osmolal gap

$$\text{Osmolal gap} = \text{Measured } S_{\text{osm}} - \text{Calc } S_{\text{osm}}$$

$$\begin{aligned} &\text{Calculated } S_{\text{osm}} : \\ &2 [\text{Na}^+] + [\text{glucose}]/18 + [\text{BUN}]/2.8 \end{aligned}$$

$$\begin{aligned} \text{Calculated serum osmolality} &= 2(129) + 72/18 + 11/2.8 \\ &= 266 \end{aligned}$$

$$\text{Osmolal gap} = 300 - 266 = 34 \text{ (normal } < 10)$$

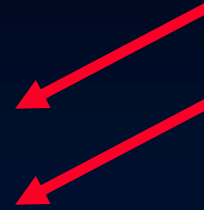
Anion and osmolar gap in diagnosis of intoxications

Anion gap acidosis	Osmolal gap	
+	Normal	Salicylates
+	High	Ethanol Ethylene glycol Propylene glycol Methanol
-	High	Isopropanol

Ethylene glycol



Alcohol
dehydrogenase



Ethanol

Fomepizole

Glycolic acid



Glyoxylic acid



Oxalic acid

Glycine

Pyridoxine

Thiamine

α -hydroxy- β -
ketoadipate

Management of (suspected) ethylene glycol or methanol poisoning

- GI decontamination
- Sodium bicarbonate
- Inhibit alcohol dehydrogenase
- Hemodialysis (level > 50 mg/dL, renal failure or severe acidosis)
- Thiamine
- Pyridoxine

A 78 year-old female has been in the ICU for 2 weeks because of cholecystitis, gram negative sepsis, hypotension, and ARDS requiring mechanical ventilation. A renal consult is called because of a persistently low serum bicarbonate. Her medications include imipenem, acetaminophen, dopamine, and noradrenaline.

Serum sodium	134 mEq/L
Serum potassium	3.8 mEq/L
Serum chloride	99 mEq/L
Serum bicarbonate	18 mEq/L
Blood urea nitrogen	14 mg/dL
Serum creatinine	1.4 mg/dL
Serum glucose	187 mg/dL
Serum lactate	1.6 mmol/L
Arterial pH	7.27
Arterial PCO ₂	41 mm Hg

Which of the following are appropriate in the management of this patient's acid-base disturbance:

- (A) CT scan to rule out ischemic bowel
- (B) Discontinue acetaminophen
- (C) Add colistin to cover carbapenem-resistant Enterobacteriaceae
- (D) Alkaline diuresis
- (E) A and C

Which of the following are appropriate in the management of this patient's acid-base disturbance:

- (A) CT scan to rule out ischemic bowel
- ☒ (B) Discontinue acetaminophen
- (C) Add colistin to cover carbapenem-resistant Enterobacteriaceae
- (D) Alkaline diuresis
- (E) A and C

pH 7.27, HCO_3^- 18, PCO_2 41

Actual PCO_2 = 41 (expected: $1.5(18)+8 = 35$)

Mixed metabolic and respiratory acidosis

$\text{AG} = 134 - 99 - 18 = 17$

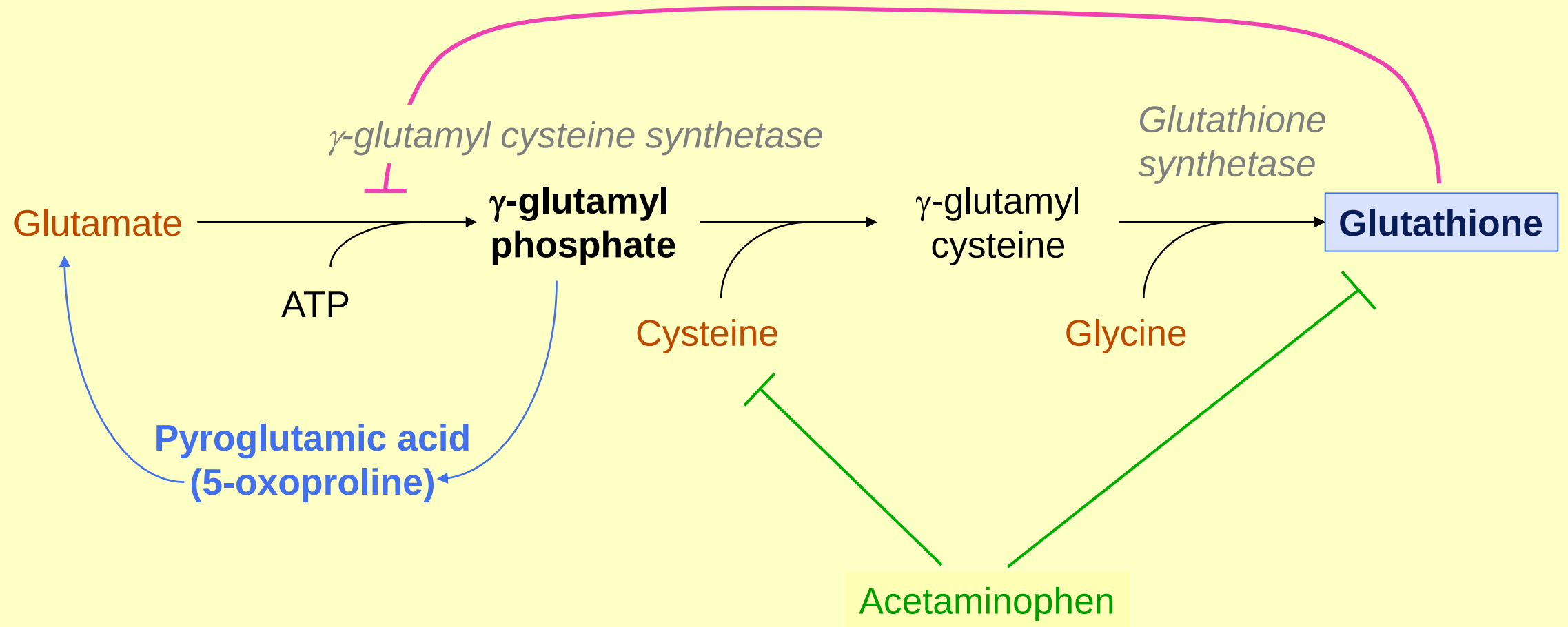
$\Delta\text{AG}/\Delta\text{HCO}_3^- = (17-10)/(24-18) \sim 1$

Pure anion gap acidosis

Pyroglutamic acidemia (5-oxoprolinemia)

- $\frac{3}{4}$ of cases are female, usually critically ill or malnourished
- Main risk factor is Rx with acetaminophen (also described with flucloxacillin)
- All have anion gap acidosis with elevated blood and urine pyroglutamic acid
- Discontinue acetaminophen, supportive care

Pyroglutamic acidemia (5-oxoprolinemia)



A 65 year-old woman developed a staphylococcal infection of a prosthetic knee and was treated with vancomycin. She developed vancomycin-resistant enterococcus infection and was switched to linezolid and treated for 6 weeks. She is now admitted for nausea, vomiting and abdominal discomfort. Vital signs and physical exam are unremarkable.

Serum sodium	132 mEq/L
Serum potassium	4.9 mEq/L
Serum chloride	99 mEq/L
Serum bicarbonate	14 mEq/L
Blood urea nitrogen	18 mg/dL
Serum creatinine	1.7 mg/dL
Serum glucose	74 mg/dL
Serum lactate	8.5 mmol/L
AST	37 U/L (NR 10-40)
ALT	25 U/L (NR 10-40)
Arterial pH	7.30
Arterial PCO ₂	29 mm Hg

Case A4

What is the most likely cause of the acid-base disturbance:

- (A) Seizure
- (B) Drug-induced lactic acidosis
- (C) Ischemic bowel
- (D) Occult sepsis
- (E) D-lactic acidosis

What is the most likely cause of the acid-base disturbance:

- (A) Seizure
- ☒ (B) Drug-induced lactic acidosis
- (C) Ischemic bowel
- (D) Occult sepsis
- (E) D-lactic acidosis

Type B lactic acidosis

Lactic acidosis that is not associated with impaired tissue oxygenation

- Seizures
- Malignancy (acute leukemia, lymphoma, solid tumor with liver metastases)
- Liver failure
- Vitamin deficiency (thiamine, riboflavin)
- Drugs/toxins (ethanol, methanol, ethylene glycol, propylene glycol, salicylates, metformin, NRTI, isoniazid, **linezolid**)

A 28-year-old Asian female with a photosensitive erythematous malar rash and arthralgias is referred to renal clinic because of abnormal laboratory electrolyte values. She denies vomiting or diarrhea, does not drink alcohol, and does not use laxatives or recreational drugs.

Laboratory studies:

Serum sodium	138 mEq/L
Serum potassium	3.2 mEq/L
Serum chloride	114 mEq/L
Serum bicarbonate	15 mEq/L
Blood urea nitrogen	7 mg/dL
Serum creatinine	0.5 mg/dL

Urine pH	6.5
Urine sodium	25 mEq/L
Urine potassium	35 mEq/L
Urine chloride	34 mEq/L

The most appropriate management for this patient is:

- (A) Loperamide
- (B) Hydrochlorothiazide
- (C) Calcium carbonate 1250 mg qd
- (D) Potassium citrate, 1-3 mEq/kg/day
- (E) Sodium bicarbonate, 5-15 mEq/kg/day

The most appropriate management for this patient is:

- (A) Loperamide
- (B) Hydrochlorothiazide
- (C) Calcium carbonate 1250 mg qd
- ☒ (D) Potassium citrate, 1-3 mEq/kg/day
- (E) Sodium bicarbonate, 5-15 mEq/kg/day

Non-gap (hyperchloremic) metabolic acidosis

1. Lower GI bicarbonate loss*
2. Renal tubular acidosis*
3. Dilutional acidosis
4. Urinary diversion
5. (Compensation for respiratory alkalosis)

*Common

Non-gap metabolic
acidosis



Urine anion gap

Negative
Diarrhea

Positive
RTA

Urine sodium	25 mEq/L
Urine potassium	35 mEq/L
Urine chloride	34 mEq/L

$UAG = +26$

Clinical features of RTA

	Diarrhea	Proximal RTA	Distal RTA	
			Type I	Type 4
Serum K ⁺	↓	↓		↑
Urine AG	Negative	Variable	Positive	
Urine pH	Variable	Variable	> 5.5	< 5.5
Other		Fanconi syndrome	Nephro-calcinosis	

Causes of RTA

Proximal RTA	Distal RTA	
	Type I	Type 4
1. Plasma cell dyscrasia (myeloma, amyloid, LCDD) 2. Ifosfamide 3. NRTI Heavy metals Cystinosis Wilson's	1. Sjogren's/SLE 2. Cirrhosis 3. Amphotericin 4. Medullary sponge kidney	1. Hyporeninemic hypoaldosteronism (DM) 2. Tubulointerstitial disease (sickle cell, SLE, obstruction, HIV) 3. Drugs (ACEI/ARB, NSAIDs, CNI, heparin)

Treatment of RTA

Proximal RTA	Distal RTA	
	Type I	Type 4
Na & K bicarbonate ≥ 4 mEq/kg/d	K bicarbonate or citrate 1-3 mEq/kg/d	Control of hyperkalemia (diuretics, polystyrene sulfate)

A 54-year-old male with a 12 year history of Type 2 diabetes mellitus maintained on insulin, and proliferative diabetic retinopathy, is referred because of proteinuria.

Laboratory studies:

Serum sodium	140 mEq/L
Serum potassium	6.0 mEq/L
Serum chloride	112 mEq/L
Serum bicarbonate	19 mEq/L
Blood urea nitrogen	27 mg/dL
Serum creatinine	1.6 mg/dL
Serum glucose	206 mg/dL

Urinalysis shows 3+ proteinuria.

The most likely cause of this patient's metabolic acidosis is:

- (A) Diarrhea due to diabetic autonomic neuropathy
- (B) Diabetic ketoacidosis
- (C) Solvent abuse
- (D) Type I renal tubular acidosis
- (E) Type IV renal tubular acidosis

The most likely cause of this patient's metabolic acidosis is:

(A) Diarrhea due to diabetic autonomic neuropathy

(B) Diabetic ketoacidosis

(C) Solvent abuse

(D) Type I renal tubular acidosis

(E) Type IV renal tubular acidosis

Hyporeninemic hypoaldosteronism

Hyperkalemia (disproportionate to level of GFR)

Type IV RTA

- Non-gap metabolic acidosis with positive urine anion gap and normal urine acidifying ability

Mild CKD

Often underlying tubulointerstitial disease:

- Diabetes mellitus
- SLE, obstruction, myeloma/amyloid, HIV etc.
- *NSAIDs*

A 22-year-old male with HIV infection was started on HAART three months ago. His medications are abacavir, tenofovir (TDF) and emtricitabine.

Serum sodium	141 mEq/L
Serum potassium	3.4 mEq/L
Serum chloride	115 mEq/L
Serum bicarbonate	18 mEq/L
Serum glucose	76 mg/dL
Blood urea nitrogen	7 mg/dL
Serum creatinine	0.4 mg/dL
Serum calcium	8.6 mg/dL
Serum phosphorus	0.9 mg/dL
Serum uric acid	1.7 mg/dL

Urinalysis: pH 6.0, specific gravity 1.015, 3+ glucose, trace protein

Urine phosphorus	25 mg/dL
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Urine creatinine	38 mg/dL
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Case A7

The most likely cause of this patient acid-base disturbance is:

- (A) HIV-related diarrhea
- (B) D-lactic acidosis
- (C) Respiratory alkalosis with metabolic compensation
- (D) Hepatic failure-associated renal tubular acidosis
- (E) Antiretroviral medication toxicity

The most likely cause of this patient acid-base disturbance is:

- (A) HIV-related diarrhea
- (B) D-lactic acidosis
- (C) Respiratory alkalosis with metabolic compensation
- (D) Hepatic failure-associated renal tubular acidosis
- (E) Antiretroviral medication toxicity

Serum bicarbonate is 18 mEq/L

Anion gap = $141 - 115 - 18 = 8$

Probably non-gap metabolic acidosis

Urine pH 6, serum K 3.4

Inappropriately alkaline urine and hypokalemia suggests Type I or II renal tubular acidosis

Clinical features of RTA

	Diarrhea	Proximal RTA	Distal RTA	
			Type I	Type 4
Serum K ⁺	↓	↓		↑
Urine AG	Negative	Variable	Positive	
Urine pH	Variable	Variable	> 5.5	< 5.5
Other		Fanconi syndrome	Nephro-calcinosis	

Diagnosis of renal phosphate wasting

$$F_E\text{PO}_4 = \frac{U_{\text{PO}_4} \times S_{\text{Cr}}}{S_{\text{PO}_4} \times U_{\text{Cr}}}$$

Serum phosphorus 0.9

$$F_E\text{PO}_4 = (25 \times 0.4) / (0.9 \times 38) = 29\% \text{ (normal 5-15\%)}$$

Hypophosphatemia and renal phosphate wasting

Glycosuria despite normoglycemia, hypouricemia

Fanconi syndrome

Cause:

Tenofovir

Fanconi syndrome & AKI from nucleotide reverse transcriptase inhibitors

- Acyclic nucleoside phosphonates: adefovir (22-50%), cidofovir (12% ARF, 1% Fanconi), tenofovir (2-4%)
- Transported into prox. tubule by organic anion transporter (OAT1), causing mitochondrial toxicity
- Both Fanconi syndrome and AKI (due to ATN)
- Can be induced by drug interaction (most pts with tenofovir toxicity were on ritonavir)
- Can be induced by renal insufficiency (all renally excreted)
- Can occur 3 wk to 18 mth after start of Rx

A 22-year-old female presents with polyuria, constipation and fatigue. She denies taking any prescription or over-the-counter medication. BP 96/54. Exam shows only mild bilateral parotid enlargement.

Serum sodium	133 mEq/L
Serum potassium	2.9 mEq/L
Serum chloride	82 mEq/L
Serum bicarbonate	38 mEq/L
Blood urea nitrogen	5 mg/dL
Serum creatinine	0.6 mg/dL
Urine sodium	15 mEq/L
Urine potassium	17 mEq/L
Urine chloride	< 5 mEq/L
Urinalysis: pH 4.5, 1+ ketones	

What is the most likely cause of this patient's hypokalemia and alkalosis:

- (A) Liddle's syndrome
- (B) Gitelman syndrome
- (C) Conn's syndrome
- (D) Diuretic abuse
- (E) Bulimia

What is the most likely cause of this patient's hypokalemia and alkalosis:

- (A) Liddle's syndrome
- (B) Gitelman syndrome
- (C) Conn's syndrome
- (D) Diuretic abuse
- ☒ (E) Bulimia

Time-dependent change in urine lytes in vomiting

	Na ⁺	K ⁺	Cl ⁻	HCO ₃ ⁻	pH
Early	↑	↑	↓	↑	> 6.5
Late	↓	↓	↓	↓	< 5.5

Urine chloride remains low throughout, hence
best diagnostic indicator of volume depletion

Cryptogenic metabolic alkalosis

	Volume status	Urine Cl ⁻	Urine diuretics
Hyperaldosteronism	↑	> 40 mEq/L	-
Surreptitious vomiting (or other extrarenal Cl loss)	NI or ↓	< 25 mEq/L	-
Diuretic abuse	NI or ↓	> 40 mEq/L*	+
Bartter/Gitelman syndrome	NI or ↓	> 40 mEq/L	-

*Can be low after post-diuretic effect

A 31 year-old male with a several month history of acid reflux and heartburn was admitted with nausea, vomiting, abdominal pain, and confusion. He avoids seeing doctors and takes only over-the-counter medications including ibuprofen, omeprazole, calcium carbonate and sodium bicarbonate. On exam he appears dehydrated and his abdomen is mildly tender.

Serum sodium	147 mEq/L
Serum potassium	5.1 mEq/L
Serum chloride	102 mEq/L
Serum bicarbonate	38 mEq/L
Blood urea nitrogen	68 mg/dL
Serum creatinine	4.1 mg/dL
Serum calcium	12.1 mg/dL
Serum albumin	4.5 g/dL

The most likely diagnosis in this patient is:

- (A) Vomiting-induced metabolic alkalosis
- (B) Hyporeninemic hypoaldosteronism
- (C) Primary hyperparathyroidism
- (D) Milk-alkali syndrome
- (E) Acute pancreatitis

The most likely diagnosis in this patient is:

- (A) Vomiting-induced metabolic alkalosis
- (B) Hyporeninemic hypoaldosteronism
- (C) Primary hyperparathyroidism
- ☒ (D) Milk-alkali syndrome
- (E) Acute pancreatitis

Milk-alkali syndrome

- Metabolic alkalosis + hypercalcemia + ARF
- Most commonly due to CaCO_3 ingestion

Synergistic effects:

- Alkalosis inhibits renal Ca^{2+} excretion
- Hypercalcemia inhibits renal bicarbonate excretion
- Hypercalcemia causes ARF
- Renal failure inhibits both Ca^{2+} and bicarbonate excretion

A 54-year-old smoker with chronic obstructive pulmonary disease, diabetes and CKD is admitted with pneumonia. Overnight he becomes progressively more obtunded; arterial blood gas shows a PCO_2 of 78 mm Hg and he is intubated and mechanically ventilated. A renal consult is called 3 days later because of persistent alkalemia and failure to wean from the ventilator.

Serum sodium	135 mEq/L
Serum potassium	4.6 mEq/L
Serum chloride	82 mEq/L
Serum bicarbonate	43 mEq/L
Blood urea nitrogen	38 mg/dL
Serum creatinine	2.4 mg/dL

Arterial pH	7.64
Arterial PCO_2	41 mm Hg

Which of the following would correct the metabolic alkalosis in this patient:

- (A) Acetazolamide
- (B) Intravenous saline
- (C) Hemodialysis
- (D) Arginine hydrochloride
- (E) All of the above

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- (A) Acetazolamide
- (B) Intravenous saline
- (C) Hemodialysis
- (D) Arginine hydrochloride
- ☒ (E) All of the above

Post-hypercapnic alkalosis

Chronic CO₂ retention



Compensatory renal HCO₃⁻ retention

Mechanical ventilation



Normalization of PCO₂ → Alkalemia



Excretion of excess HCO₃⁻ if

- Sufficient time
- Adequate renal function
- Euvolemia