Renal System for the USMLE Step 2 CK/3

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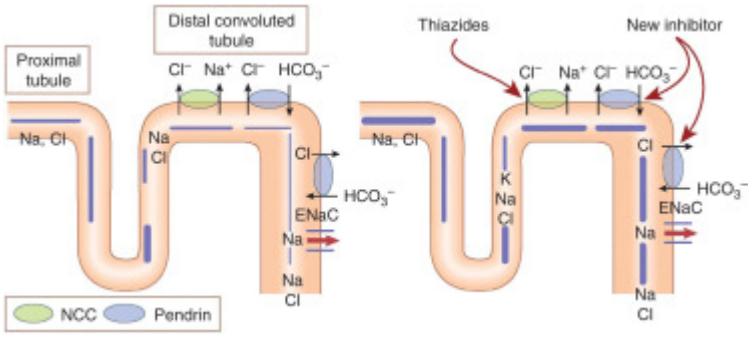
Condition	Cause	Current pH	Current Co2 or H20
respiratory acidosis	hypoventilation	low	high co2
respiratory alkalosis	hyperventilation	high	low co2
metabolic acidosis	too much acid or too little base	low	low HCO3
metabolic Alkalosis	too little acid or too much base	high	High Hco3

Low Ph

- **High Co2**
    - Respiratory Acidosis Hypoventilation = SOOLAR
    - Sedation
        - Common sedatives like benzodiazepines (e.g., diazepam) and barbiturates (e.g., phenobarbital) can decrease respiration through the mechanisms:
        - These sedatives act on the gamma-aminobutyric acid (GABA) receptors in the brain. This leads to a general depressant effect on the CNS, which can include respiratory centers in the brainstem
    - Opioids
        - Opioids bind to specific receptors known as opioid receptors (e.g., mu, delta, and kappa receptors) that are located in various parts of the brain, including the brainstem and the medulla oblongata. The mu-opioid receptor, in particular, is heavily involved in modulating pain and respiratory function.
    - Obesity hypoventilation
    - Lung disease
    - Acute Lung obstruction
    - Respiratory muscle weakness
        - In MG, the immune system mistakenly produces antibodies against acetylcholine receptors (AChRs) at the neuromuscular junction.
        - Amyotrophic lateral sclerosis: Amyotrophic: This term comes from the Greek words "a" (without), "myo" (muscle), and "trophic" (nourishment). Lateral: This refers to the areas in the spinal cord where the motor neurons are located. Sclerosis: hardening of the spinal cord and other affected regions, due to scar tissue formation. ALS affects the motor neurons in the brain and spinal cord, leading to the weakening and atrophy of the muscles responsible for breathing, such as the diaphragm, intercostal muscles, and accessory muscles

3 Reason why it is important to study HAGMA

1. Often serious/toxic causes,Often GI or renal losses
2. Needs urgent treatment (e.g. DKA),May need fluid or electrolyte correction
3. Clues you into “hidden acids”,Suggests direct bicarb loss or replacement

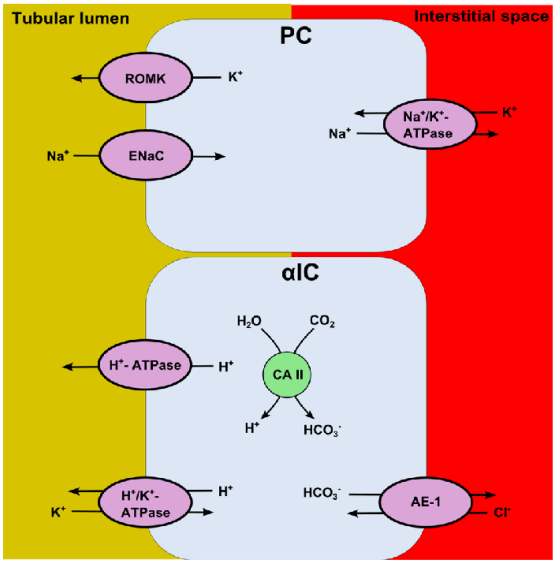
- **Low Bicarbonate**:
- Normal Anion Gap, (Anion gap is calculated as (Na+Cl-+ HCO3-)) Normal anion gap should be between 1 to 12 above 13 meq/l is considered Elevated anion gap
- Normal Anion gap metabolic Acidosis: Unmeasured anions can significantly affect the interpretation of sodium chloride (NaCl) and bicarbonate (HCO_3^-) levels. Unmeasured anions are negatively charged ions in the blood that are not typically included in routine electrolyte panels. Examples include lactate, phosphate, sulfate, and certain proteins and organic acids. The presence of these anions can influence the calculation of the anion gap.
    - Hyperalimentation
        - Excessive Chloride Intake: TPN solutions often contain a significant amount of chloride, and excessive chloride intake can lead to a condition called hyperchloremic metabolic acidosis.
        - Decreased Bicarbonate Production: In patients receiving TPN, especially if they have underlying conditions that impair kidney function, the production of bicarbonate may decrease. This can occur due to factors such as reduced metabolic activity in the liver due to prolonged fasting, or altered metabolism due to underlying diseases.
    - hyper-cholermia
        - hyperchloremic metabolic acidosis results from the loss of bicarbonate (HCO_3^-), whether through gastrointestinal losses (diarrhea) or renal losses.
    - Addison disease
        - In the case of Addison's disease, the retention of hydrogen ions does not significantly impact unmeasured anions, hence the anion gap remaining within the normal range.
        - 

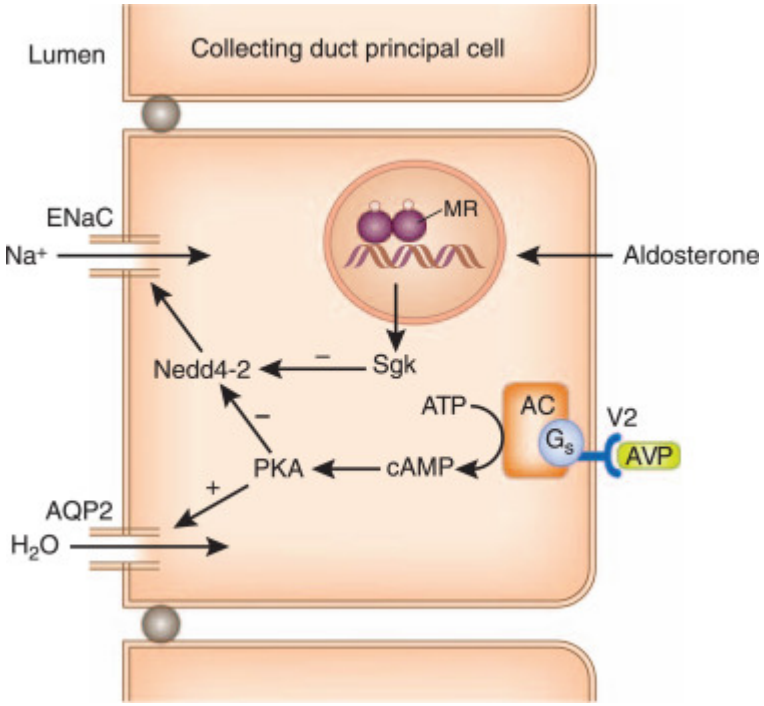
        - Aldosterone normally reabsorbs Sodium and excretes K+ and H+

Bicarbonate loss

- The retained **H^+** binds with bicarbonate (HCO_3^-), **consuming it** → bicarb levels **drop**.

- Also, **Na^+ reabsorption** is impaired, and Na^+ normally brings HCO_3^- with it. So you lose even more bicarbonate in urine.

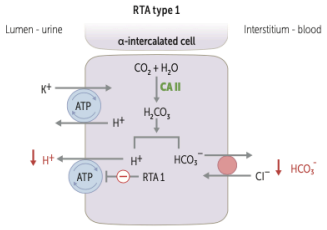
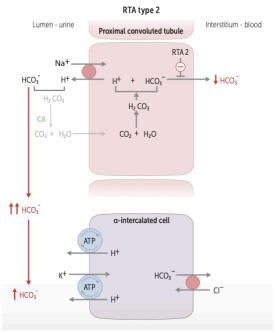
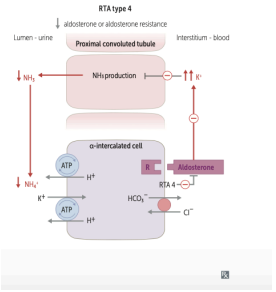
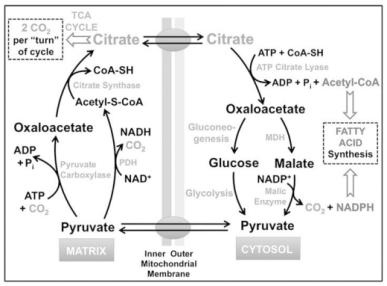
- 

- 

- Why are they called the Alpha Intercalated cells ?
  - The term "intercalated" highlights that these cells are interspersed within the other cells of the collecting duct.
  - **Alpha:**
    The "alpha" (or A-type) classification is based on their ability to secrete protons (H+) into the urine, contributing to the excretion of excess acid in the body.

- **RTA acidosis**

	RTA type 1	**RTA type 2**	**RTA type 4 (HyperKalemic tubular acidosis)**
Location:	Collecting duct	Proximal	Collecting Duct

		Convoluted tubes	
Pathology:	Impaired H+ secretion in the alpha intercalated cell 		
Serum Potassium	Low to Normal Increased Sodium Delivery to the Distal Nephron: Impaired H^+ secretion disrupts sodium reabsorption via the sodium-hydrogen exchanger. More sodium reaches the collecting duct, where it is reabsorbed in exchange for potassium via the ENaC channels. This increases potassium excretion.	Decreases Increase bicarbonate in the urine causes a osmotic diuresis and brings Sodium to the Distal nephron which leads to reabsorption of sodium and loss of potassium	Increase Low Aldo leads to high serum potassium
Urinary pH	> pH 5.5	<pH 5.5	<ph 5.5
Treatment:	Alkali Sodium Bicarbonate Sodium Citrate  When sodium citrate (Na3C6H5O7) is consumed, it dissolves in the body fluids and dissociates into sodium ions (Na+) and citrate ions (C6H5O7^3-).	Sodium citrate and potassium citrate are both metabolized in the liver to bicarbonate (HCO_3^-). Alkali therapy and Potassium Citrate.	Furosemide and Mineralocorticoid replacement. Loop diuretics cause mild **volume depletion** → stimulates **RAAS** → ↑ aldosterone
Mnemonic:	ONE = stONES in the kidney	2 = BIcarbonate	FOUR = HYPO

	Increase risk of calcium phosphate stones due to increase urine pH and increase bone turnover		ALDO

	Distal renal tubular acidosis (RTA type 1)	Proximal renal tubular acidosis (RTA type 2)	Hyperkalemic tubular acidosis (RTA type 4)
DEFECT	Inability of α-intercalated cells to secrete H^+ → no new HCO_3^- is generated → metabolic acidosis	Defect in PCT HCO_3^- reabsorption → ↑ excretion of HCO_3^- in urine → metabolic acidosis Urine can be acidified by α-intercalated cells in collecting duct, but not enough to overcome ↑ HCO_3^- excretion	Hypoaldosteronism or aldosterone resistance; hyperkalemia → ↓ NH_3 synthesis in PCT → ↓ NH_4^+ excretion
URINE pH	> 5.5	< 5.5 when plasma HCO_3^- below reduced resorption threshold > 5.5 when filtered HCO_3^- exceeds resorptive threshold	< 5.5 (or variable)
SERUM K^+	↓	↓	↑
CAUSES	Amphotericin B toxicity, analgesic nephropathy, congenital anomalies (obstruction) of urinary tract, autoimmune diseases (eg, SLE)	Fanconi syndrome, multiple myeloma, carbonic anhydrase inhibitors	↓ aldosterone production (eg, diabetic hyporeninism, ACE inhibitors, ARB, NSAIDs, heparin, cyclosporine, adrenal insufficiency) or aldosterone resistance (eg, K^+-sparing diuretics, nephropathy due to obstruction, TMP-SMX)
ASSOCIATIONS	↑ risk for calcium phosphate kidney stones (due to ↑ urine pH and ↑ bone turnover related to buffering)	↑ risk for hypophosphatemic rickets (in Fanconi syndrome)	

NMBE questions

Normal ANion gap
High Chloride
and Low HCO30

u got wrong

FCM

decreased HCO3 reabsorption causing increased flow rate to distal tubule and causing increased K excretion.

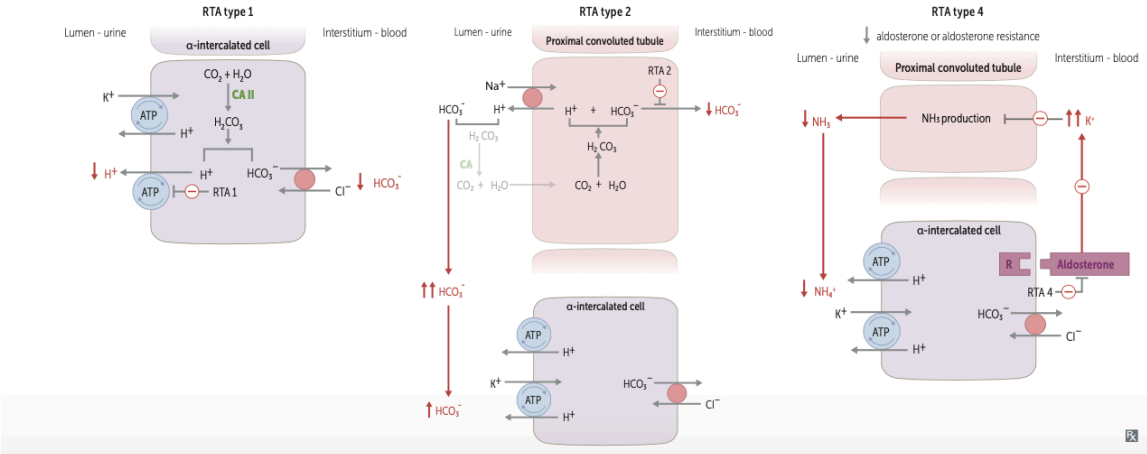
In type 4 RTA, the key defect is impaired ammoniagenesis. The ability to acidify the urine (that is, to secrete protons) remains intact. Since H+ATPase pumps function normally to excrete acid and since there is less buffer in the urine, urinary acidification in response to acidosis is intact and urine pH is low (<5.5).

AAC

Amphotercin
Analgesic
AI
Congenital Anomalies

Ace
Arb
Nsaids
Heparin
Cyclosporine
Adrenal Insuffeincy
Aldosterone resitance

HAAN AA



Version 2

Increased Sodium and Bicarbonate Delivery to the Distal Nephron:

Loss of bicarbonate in the urine causes osmotic diuresis, which increases the delivery of sodium and water to the distal nephron.

Increased Sodium Delivery to the Distal Nephron:

Impaired H^+ secretion disrupts sodium reabsorption via the sodium-hydrogen exchanger.
More sodium reaches the collecting duct, where it is reabsorbed in exchange for potassium via the ENaC channels.
This increases potassium excretion

Renal tubular acidosis			
Type	**1 (Distal)**	**2 (Proximal)**	**4**
Primary defect	Poor hydrogen secretion into urine	Poor bicarbonate resorption	Aldosterone resistance
Urine pH	≥5.5	<5.5	<5.5
Serum potassium	Low-normal	Low-normal	High
Causes	• Genetic disorders • Medication toxicity • Autoimmune disorders (eg, Sjögren syndrome, rheumatoid arthritis)	Fanconi syndrome (glucosuria, phosphaturia, aminoaciduria)	• Obstructive uropathy • Congenital adrenal hyperplasia

decreased HCO3 reabsorption causing increased flow rate to distal tubule and causing increased K excretion.

Ammonium (NH_4^+) plays a key role in buffering urine by binding hydrogen ions to form ammonium, which is then excreted in the urine. With low ammonium levels, there's less buffering capacity, so the kidney can't effectively increase urine pH even though systemic acidosis is present.

GAM

Genetirc

< 5.5 when plasma HCO – 3 below reduced resorption threshold

> 5.5 when filtired HCO3- exceeeds resprotive threshold

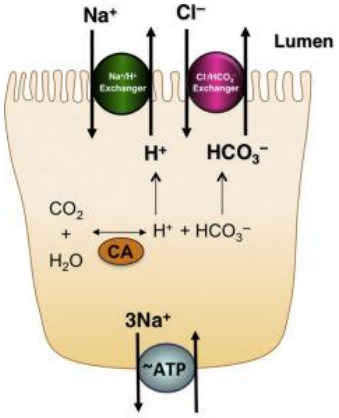
Possible low ph because of the amioaciduria

T/t:

- Type 1: **Alkali Therapy : Sodium Bicarbonate, Sodium Citrate**
- Type 2: **Alkali Therapy: Potassium citrate**
- Type 4: **Furosemide**, **Mineralocorticoid replacement**

Sodium citrate and potassium citrate are both metabolized in the liver to bicarbonate (HCO_3^-),

- Diarrhea



  - Diarrhea causes decrease in bicarbonate and the retention of chloride keeps the anion gap within a normal range. “Bicarbonate Bids Bye, Chloride Clings"
- Acetazolamide
  - Normally, carbonic anhydrase facilitates the conversion of **HCO_3^- + H^+ → H_2O + CO_2**, allowing bicarbonate reabsorption. Acetazolamide **blocks this enzyme**, preventing HCO_3^- reabsorption in the PCT. The body compensates for the lost bicarbonate by **reabsorbing chloride (Cl^-)** to maintain electroneutrality.
- Saline infusions
  - This excess volume can dilute bicarbonate levels in the plasma, leading to a decrease in the bicarbonate concentration, which is a key component of the buffer system that helps to neutralize acids. Increased Chloride Levels: The infusion of saline increases chloride levels in the serum.
- Spironolactone

        - In the nephron, specifically in the collecting duct, spironolactone reduces the secretion of hydrogen ions. This effect can lead to impaired acid excretion, resulting in retention of hydrogen ions and a corresponding reduction in serum bicarbonate, contributing to acidosis. In response to low reduction of bicarbonate chloride increases.

Anion gap Metabolic acidosis

- GOLD MARK CAT MUDPILES
- Cyanide CO and CHD
- Aminoglycosides
- Glycol
- Oxoproline
- Methanol
- A
- Renal
- Ketones
- Cyanide
- A
- T

High PH

- Metabolic Alkalosis → High Bicarbonate
    -
- Respiratory Alkalosis
- Low Co2
    - Respiratory Alkalosis Hyperventilation HA PPTS
    - Hypoxemia (high altitudes)
    - Anxiety/ Panic attack
    - Pregnancy
        - Increased Oxygen Demand: As the fetus grows, there is a higher demand for oxygen and progesterone secreted from the corpus luteum stimulates the respiratory center. (Syncytiotrophoblast secrete HCG stimulates the Corpus luteum and that stimulates the progesterone to maintain the uterine lining)
    - Pulmonary EMbolism
        - V/Q Mismatch: In PE, a blockage in a pulmonary artery reduces blood flow to parts of the lung. This creates a ventilation-perfusion (V/Q) mismatch, where certain areas of the lung are ventilated but not adequately perfused. This leads to ineffective gas exchange and decreased oxygen levels in the blood.
    - Tumor
        - Pheochromocytoma: This tumor of the adrenal glands can produce catecholamines (like adrenaline), leading to symptoms including anxiety, palpitations, and hyperventilation.
    - Salicylate (early)
        - Salicylates can directly stimulate the medullary respiratory center in the brainstem.
    -

Metabolic acidosis and Metablock alkalosis no change in pH DKA and Vomiting

Resp acidosis and Resp alkalosis = doesn't exist
Metabolic acidosis + Resp alkalosis =

- salicylate

Metabolic alkalosis + Resp acidosis =

- lung failure plus vomiting

Meta block alkalosis + respiratory alkalosis =

- diuretics + HF

Metabolic acidosis + Resp Acidosis =

- dka + lung failure

🔍 Pre-Renal Failure – Made Simple (and Interesting!)

When your body doesn't get enough blood to the kidneys 💔🩺, they can't do their job properly. That's what we call **pre-renal failure** — and it's all about **low blood flow**.

💧 **Dehydration**
💩 **Diarrhea**
🤮 **Vomiting**
😓 **Sweating**
⚠️ **Any type of Shock** causes low volume

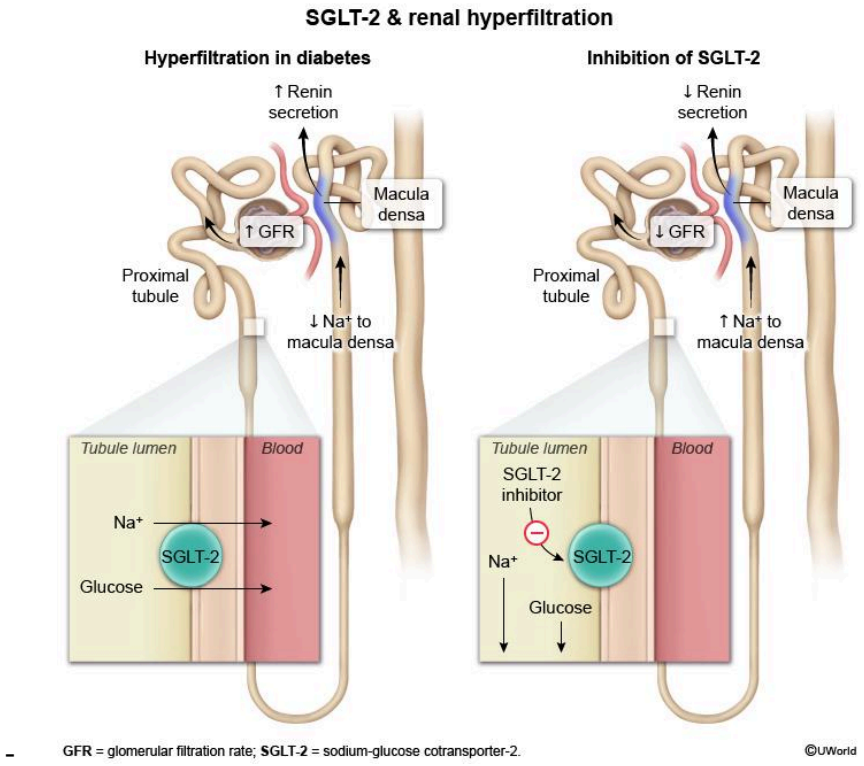
This leads to **3 key changes**:

1️⃣ Increase BUN:Cr > 20:1

- 🧪 Due to decreased renal perfusion, the kidneys activate various compensatory mechanisms to maintain homeostasis, including increased reabsorption of water 💦 and electrolytes ⚡.
- ↩️ This increased reabsorption also affects **urea**, leading to a higher BUN:Creatinine ratio.

2️⃣ Decrease in Sodium Excretion < 1%

- ⬇️ Low blood volume means less sodium reaches the distal tubule due to lower **glomerular filtration rate (GFR)**.
- 🔍 This decreased sodium concentration is sensed by the **macula densa**, which signals the **juxtaglomerular cells** to release **renin**.
- 🧬 Renin then catalyzes more **Angiotensinogen** → **Angiotensin I**, which is converted into **Angiotensin II** by the **ACE enzyme**.
- 🩸 **Angiotensin II** causes blood vessels to **constrict** ➡️ increasing systemic vascular resistance and raising **blood pressure**.
- 🧠 Also, Angiotensin II increases **Aldosterone** secretion, which promotes sodium and water retention.



3️⃣ Elevated Urine Osmolarity > 500 mOsm/kg

- 🚭 Less sodium in the urinary filtrate means less water follows it
- ➡️ Resulting in **elevated urine osmolarity**

Mixture of Acid Base disorder

Extreme pH

Combination	**pH**	**Example:**

PSGN

→ low serum C3 levels → immune complexes activate the **alternative complement pathway**

C1 Complex breaks C2b and C4b fragment → C3 convertase → C3 hydrolysis → C3b and C3a fragment → C3b cleaves C5 into C5a and b

A 60-year-old man comes to the emergency department due to severe abdominal pain and an inability to void urine for the past 18 hours. The patient has a year-long history of urinary hesitancy and a weak urinary stream but has always been able to urinate prior to today. He has not had fever, chills, dysuria, or recent genitourinary trauma or procedures. The patient also has a history of chronic arthritis of the lumbar spine and was recently prescribed baclofen, which has provided some pain relief. He does not use tobacco, alcohol, or illicit drugs. Temperature is 36.5 C (97.7 F), blood pressure is 145/90 mm Hg, pulse is 95/min, and respirations are 18/min. The patient appears restless and in moderate distress. Abdominal examination reveals suprapubic fullness and moderate tenderness to palpation with no guarding or rebound tenderness. An enlarged, smooth, nontender prostate is palpated by digital rectal examination; no masses or fecal impaction are present. There is no tenderness to palpation of the lower back. Abbreviated testing of strength and sensory function is normal. Laboratory results are as follows:

Complete blood count	
Hemoglobin	13.8 g/dL
Leukocytes	6,500/mm³
Serum chemistry	
Blood urea nitrogen	40 mg/dL
Creatinine	2.9 mg/dL

What is the best next step in management of this patient?

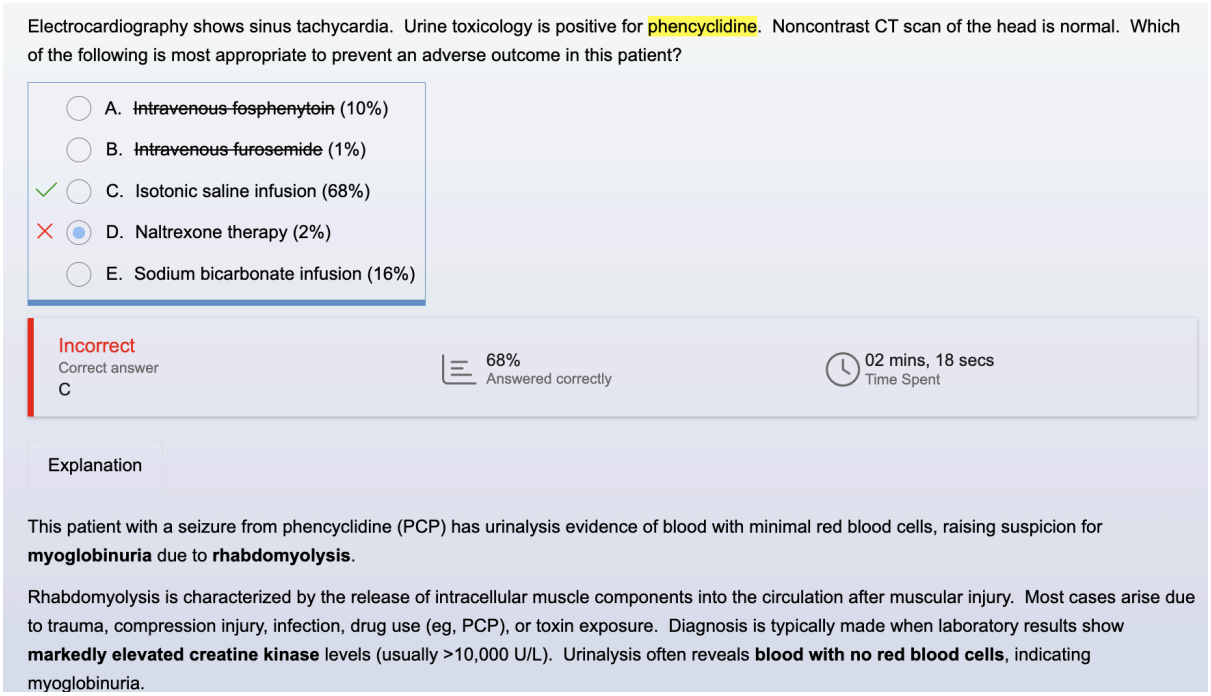
- A. ~~Infusion of intravenous fluid~~ (0%)
- B. Observation with repeated voiding trials (0%)
- C. ~~Suprapubic bladder aspiration for microscopy and culture~~ (0%)
- ✗ D. Suprapubic catheterization (15%)
- ✓ E. ~~Urethral catheterization~~ (82%)

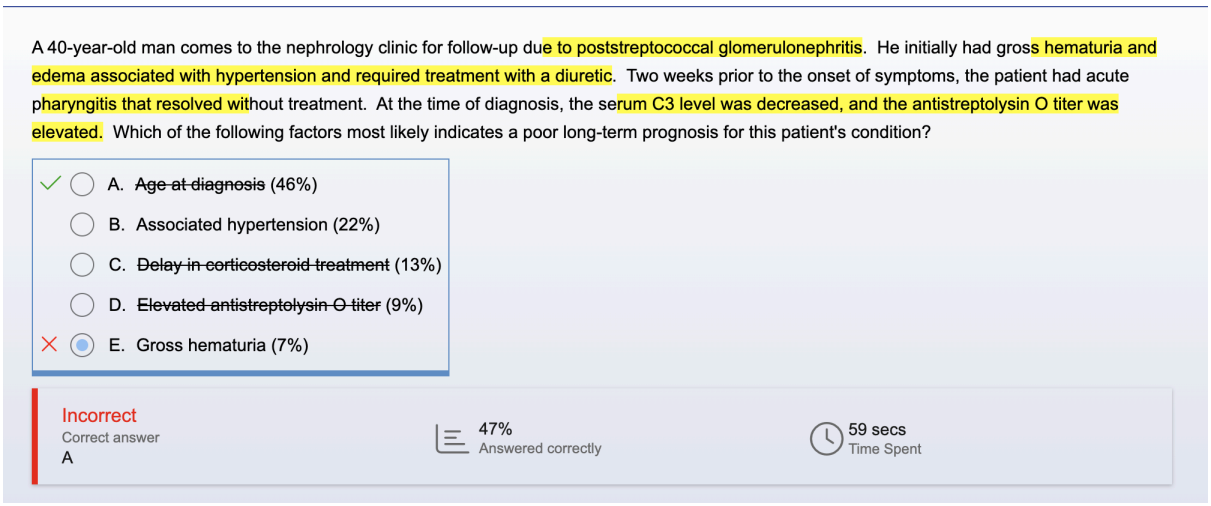
Step 3 wrongs

Yours = Suprapubic catheterization --> Suprapubic catheterization is usually reserved for patients with a recent history of genitourinary trauma/surgery or if urethral catheterization is unsuccessful

A 17-year-old male presents to the office because he had an episode of cola-colored urine. He appears scared and says that he ran 12 miles two days ago as a part of 'a run for heart disease awareness.' His past medical history is insignificant. He denies any recent upper respiratory or skin infections. He does not smoke or consume alcohol, and says that he has never used any recreational drugs. His father has hypertension and coronary artery disease. His temperature is 37.2 C (98.9 F), blood pressure is 118/76 mmHg and pulse is 78/min. His BMI is 23 kg/m^2. The physical examination is unremarkable. Urinalysis is positive for blood; there are numerous (100+) RBCs/hpf. Urine sediment did not show any RBC casts. Which of the following is the most appropriate next step in management?

- A. Cystoscopy (2%)
- B. Intravenous pyelography (0%)
- C. Renal biopsy (0%)
- ✓ D. Repeat urinalysis in 1 week (59%)
- ✗ E. Serum myoglobin level (36%)





step 3 wrong

What is a poor long term prognosis for this patient ?

{{c1::Age}}

Correct = Age of diagnosis
Yours = Gross hematuria -->
None of these findings significantly affects the prognosis, whereas the difference in prognosis between adults and children has been noted in multiple studie

Children: Typically experience a more benign and self-limited immune response to streptococcal infection. Adults: Often exhibit a more intense and prolonged immune response, which can cause more severe glomerular injury, leading to persistent or progressive renal damage.

What is the NBS in a patient w/ active bleeding and a bladder cancer after a cysotscopy for a bladder cancer ?

{{c1::Bladder irrigation}} --> first step to clear hematuria and prevent blood clot formation, blood clots cause blockage and urinary obsturction. bladder irrigation can also help w/ irritation or inflammation of the bladder epithelium.

Yours = Cystoscopy w/ cauterization

NBME 7 BLock 1
Patient w/ burns what sh9uld we check ?
Correct = Serum myoglobin
NBME 7 Block3
How long does it take for contrast associated AKI to recover ?

few days

Step 3 wrong

Yours = 2 to 4 weeks --> Contrast associated AKI takes 24 to 48 hours to develop and resolves within 3 to 7 days after contrast expsosure.
Fewer than a third of patient experience only partial recovery of renal fucntion following CA-AKI

1. Injury onset peaks around 2-3 days

1.a injury onset in is that there is direct toxicity to the tubules

2. Tubular cell turnover = repair and regen process takes a few day

9. **Item 2 of 2**
The most significant risk to this patient posed by the recommended arteriography and urokinase therapy is which of the following?

- A) Acute tubular necrosis
- B) Allergic reaction to radiopaque dye
- C) Hemorrhage
- D) Hepatic dysfunction
- E) Thrombosis

End of Set

NBME 7 B4

Correct Answer: C.

Acute limb ischemia refers to the sudden loss of blood flow to a limb in the setting of thromboembolism. This often affects the lower extremities. Risk factors include cardiac valvular disease, atrial fibrillation, hypercoagulability, diabetes mellitus, increased age, tobacco smoking, hypertension, and dyslipidemia. Once tissue perfusion becomes inadequate to meet metabolic demands, there is potential for ischemia and necrosis of the extremity. The affected limb is typically pulseless, pale, and cold. The patient may report paresthesia, pain, or paralysis of the limb. Arteriography with contrast is useful for assessing the patency of vessels in patients with suspected acute limb ischemia. Treatment is with emergent revascularization, which can be accomplished surgically with embolectomy or vascular bypass, or medically with thrombolytic agents such as alteplase or urokinase. Urokinase is a protease that cleaves plasminogen to active plasmin, resulting in thrombolysis. Such agents (plasminogen activators) are administered intravenously to achieve thrombolysis in the setting of acute limb ischemia, deep venous thrombosis, pulmonary embolism, acute ischemic stroke, or acute myocardial infarction. As with other thrombolytic agents, hemorrhage is the most significant risk associated with urokinase treatment.

Incorrect Answers: A, B, D, and E.

Acute tubular necrosis (ATN) (Choice A) describes necrosis of renal tubular cells and may occur due to ischemia or direct cytotoxic injury from chemicals such as contrast dye. Contrast-induced ATN may be preventable with oral or intravenous hydration, and typically carries a good prognosis. While the risks associated with administering contrast must be considered in all cases, the risk for potentially fatal hemorrhage from urokinase is more significant in this case.

1. **High Turnover Rate of Epithelial Cells:**
   The tubular epithelium, especially in the proximal tubules, consists of cells that have a high rate of cell division under normal conditions. This allows for rapid replacement of damaged or dead cells.
2. **Presence of Resident Stem/Progenitor Cells:**
   Some studies suggest that the kidneys have resident progenitor or stem-like cells capable of proliferating and differentiating to restore damaged tubules efficiently.

A 60-year-old man comes to the office for an initial evaluation of fatigue and dyspnea on exertion. Medical history is significant for hypertension and chronic kidney disease with a baseline serum creatinine of 2.5-3.0 mg/dL. Temperature is 36.1 C (97 F), blood pressure is 145/92 mm Hg, and pulse is 80/min. BMI is 28 kg/m^2. Physical examination shows displacement of the apical impulse to the left and downward and 1+ bilateral lower extremity edema. The remainder of the physical examination shows no abnormalities. ECG reveals left ventricular hypertrophy with repolarization abnormalities. Laboratory results are as follows:

Hemoglobin	8.3 g/dL
Mean corpuscular volume	88 µm^3
Ferritin, serum	320 ng/mL (normal: >100)
Transferrin saturation	38% (normal: 25%-50%)
Vitamin B_{12}, serum	normal
Estimated glomerular filtration rate	25 mL/min/1.73 m^2

Treatment with an erythropoietin-stimulating agent would most likely have a beneficial impact in terms of which of the following in this patient?

A. Chronic kidney disease progression
B. Hypertension contro
C. Left ventricular hypertrophy
D. Risk of ischemic stroke
E. Risk of venous thrombosi

Incorrect
Correct answer
C

39%
Answered correctly

01 min, 29 secs
Time Spent

- Unmet oxygen demand in the tissues leads to vasodilation and a reduction in systemic vascular resistance, which can encourage high-output heart failure (ie, volume overload) and left ventricular (LV) dilation.
- Myocardial hypoxia and loss of the cardioprotective effects of erythropoietin on the myocardium can facilitate myocardial apoptosis and necrosis, leading to myocardial fibrosis with reduced contractile function and compensatory LV dilation.

elderly male found on the streeet step 3 wrongs urine positive for ketones 167 mg/dl glucose WHat is the cause? {{c1::Alcoholic ketoacidosis }} Yours = DKA should be more than 250 mg/dl to show insulin defiencyAlcohol is metabolized primarily in the liver via alcohol dehydrogenase, producing acetaldehyde and then acetate, with a significant increase in the NADH/NAD+ ratio. This shift favors the conversion of pyruvate to lactate and oxaloacetate to

A 5-year-old girl is brought to the clinic due to 2 days of dark red urine. The patient was treated for facial impetigo a month ago. Blood pressure is 140/90 mm Hg. Urinalysis reveals hematuria, mild proteinuria, and occasional red blood cell casts. Which of the following glomerular changes are most likely to be present within this patient's kidneys?

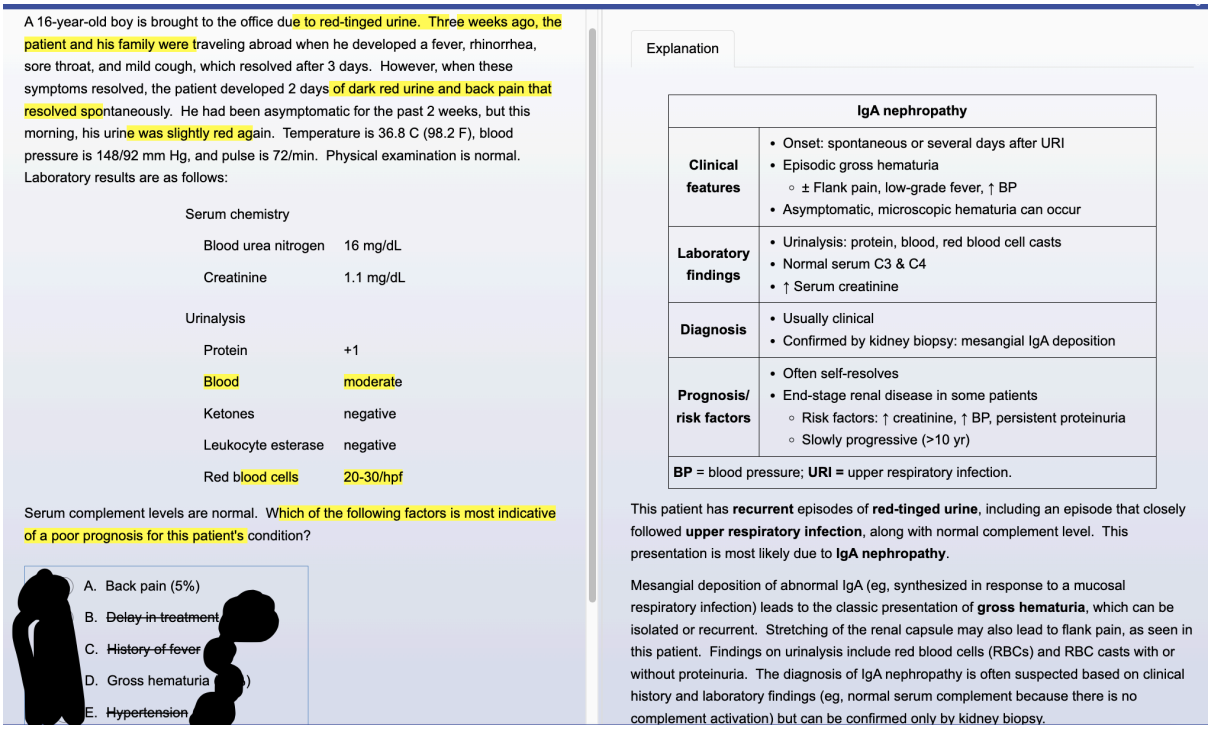
A. ~~Diffuse capillary wall thickening on light microscopy~~ (4%)
B. Discrete subepithelial humps on electron microscopy
C. ~~Glomerular basement membrane rupture on electron microscopy~~
D. Glomerular basement membrane splitting on light microscopy
E. ~~Linear IgG and C3 deposits on immunofluorescent microscopy~~

Incorrect
Correct answer
B

36%
Answered correctly

37 secs
Time Spent

malate, inhibiting gluconeogenesis.



A 16-year-old boy is brought to the office due to red-tinged urine. Three weeks ago, the patient and his family were traveling abroad when he developed a fever, rhinorrhea, sore throat, and mild cough, which resolved after 3 days. However, when these symptoms resolved, the patient developed 2 days of dark red urine and back pain that resolved spontaneously. He had been asymptomatic for the past 2 weeks, but this morning, his urine was slightly red again. Temperature is 36.8 C (98.2 F), blood pressure is 148/92 mm Hg, and pulse is 72/min. Physical examination is normal. Laboratory results are as follows:

Serum chemistry	
Blood urea nitrogen	16 mg/dL
Creatinine	1.1 mg/dL
Urinalysis	
Protein	+1
Blood	moderate
Ketones	negative
Leukocyte esterase	negative
Red blood cells	20-30/hpf

Serum complement levels are normal. Which of the following factors is most indicative of a poor prognosis for this patient's condition?

A. Back pain (5%)
B. ~~Delay in treatment~~
C. ~~History of fever~~
D. Gross hematuria
E. ~~Hypertension~~

Explanation

IgA nephropathy	
Clinical features	• Onset: spontaneous or several days after URI • Episodic gross hematuria ◦ ± Flank pain, low-grade fever, ↑ BP • Asymptomatic, microscopic hematuria can occur
Laboratory findings	• Urinalysis: protein, blood, red blood cell casts • Normal serum C3 & C4 • ↑ Serum creatinine
Diagnosis	• Usually clinical • Confirmed by kidney biopsy: mesangial IgA deposition
Prognosis/ risk factors	• Often self-resolves • End-stage renal disease in some patients ◦ Risk factors: ↑ creatinine, ↑ BP, persistent proteinuria ◦ Slowly progressive (>10 yr)
BP = blood pressure; **URI =** upper respiratory infection.	

This patient has **recurrent** episodes of **red-tinged urine**, including an episode that closely followed **upper respiratory infection**, along with normal complement level. This presentation is most likely due to **IgA nephropathy**.

Mesangial deposition of abnormal IgA (eg, synthesized in response to a mucosal respiratory infection) leads to the classic presentation of **gross hematuria**, which can be isolated or recurrent. Stretching of the renal capsule may also lead to flank pain, as seen in this patient. Findings on urinalysis include red blood cells (RBCs) and RBC casts with or without proteinuria. The diagnosis of IgA nephropathy is often suspected based on clinical history and laboratory findings (eg, normal serum complement because there is no complement activation) but can be confirmed only by kidney biopsy.

warfarrin + AMiodraone
What should be do the dosage of warfarrin ?

{{c2::reduce }}

Step 3 2 wrongs

Free 137 B1 How to dx kidney stones ?
Helical CT scan
Yours = x ray of the ureters
Free 137 B2 Q10
Add the screen shot here

Peds 6 Q 14
9F
(1) headache and Dizziness for past week
(2) at 2 years, she received diagnosis of VUR
(3) High BP
What is dx ?
{{c2::CKD}}
What is the nBS ?
Correct = Measure BUN and creatinine concentrations

Yours = CT scan of head —> FND or sudden onset of headache

How to dx kidney stones in pregnant women ?

Correct = US
Yours= Low dose helical CT scan