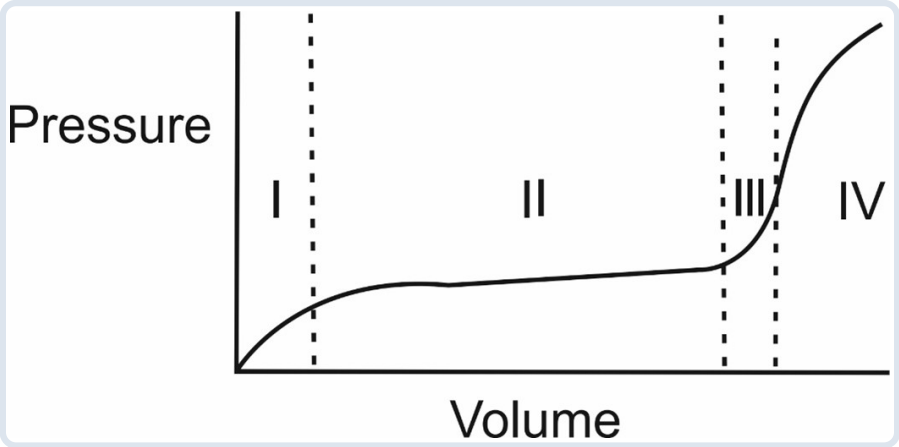
QUESTION

Which of the following statements is true regarding the given cystometrogram?



A Segment Ia is due to residual urine

B **Segment II is due to Laplace law** CORRECT

C Micturition fails to happen in segment II

D The dotted line represents that micturition has occurred

OK RIGHT ANSWER

B. Segment II is due to Laplace law

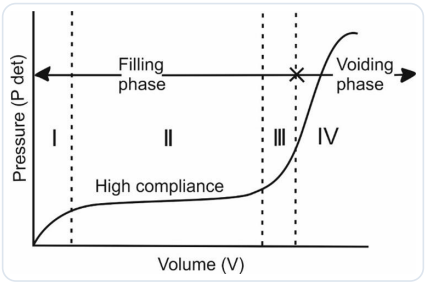
WHY THIS IS RIGHT

Segment II represents the high-compliance phase of the bladder, where volume increases with minimal rise in pressure.

This occurs due to Laplace law-as bladder radius increases, wall tension is distributed, limiting pressure rise.

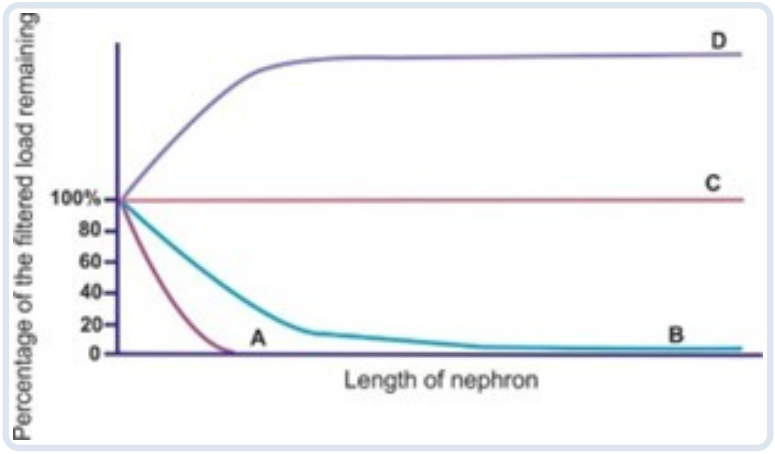
Micturition reflex is initiated later (segment III-IV), not in segment II.

Dotted lines indicate threshold points, not completion of micturition.



QUESTION

Identify the correctly matched pair of substances with their renal clearance from the graph given below?



A. A - Glucose, B - PAH, C - Bicarbonate and D - Inulin

B. **A - Glucose, B - Bicarbonate, C - Inulin and D - PAH** CORRECT

C. A - PAH, B - Inulin, C - Glucose and D - Bicarbonate

D. A - Inulin, B - Glucose, C - Bicarbonate and D - PAH

OK

RIGHT ANSWER

B. A - Glucose, B - Bicarbonate, C - Inulin and D - PAH

WHY THIS IS RIGHT

Glucose shows zero clearance as it is freely filtered and completely reabsorbed under normal plasma levels.

Bicarbonate has low but positive clearance due to near-complete reabsorption with regulated secretion.

Inulin clearance equals GFR as it is freely filtered with no reabsorption or secretion.

PAH has the highest clearance as it is filtered and actively secreted, approximating renal plasma flow.

Substance	Handling	Clearance	Key Concept
Glucose	Fully reabsorbed	0	Tm limited
Bicarbonate	Mostly reabsorbed	Low	pH regulation
Inulin	Filtered only	= GFR	Gold standard
PAH	Filtered + secreted	Highest	RPF marker

QUESTION

A patient has the following Starling forces at a capillary bed:

• Capillary hydrostatic pressure (Pc): 18 mmHg
• Capillary oncotic pressure (πc): 27 mmHg
• Interstitial oncotic pressure (πi): 7 mmHg

If there is no net filtration across the capillary, what is the value of the interstitial hydrostatic pressure (Pi)?

A 0 mmHg

B +1 mmHg

C +2 mmHg

D **-2 mmHg** CORRECT

OK RIGHT ANSWER

D. -2 mmHg

WHY THIS IS RIGHT

The Starling equation for net filtration pressure (NFP) is: NFP = (Pc - Pi) - (πc - πi)
Where:
• Pc = capillary hydrostatic pressure (pushes fluid OUT)
• Pi = interstitial hydrostatic pressure (pushes fluid IN)
• πc = capillary oncotic pressure (pulls fluid IN)
• πi = interstitial oncotic pressure (pulls fluid OUT)
• | Force | Direction | Function |
| ----- | --------- | -------- |
| Pc | Outward | Filtration |
| Pi | Inward (if positive) | Opposes filtration |
| πc | Inward | Reabsorption |
| πi | Outward | Filtration |

Net filtration pressure (NFP) = (Pc - Pi) - (πc - πi).
Given no net filtration, NFP = 0 => (18 - Pi) - (27 - 7) = 0.
So, 18 - Pi - 20 = 0 => Pi = -2 mmHg, making option D correct.

QUESTION

All of the following Decreases GFR except:

A Decreased glomerular coefficient Kf

B Increase glomerular oncotic pressure

C Reduced Glomerular Hydrostatic pressure

D **Increased Bowman Oncotic Pressure** CORRECT

RIGHT ANSWER

OK

D. Increased Bowman Oncotic Pressure

WHY THIS IS RIGHT

GFR decreases with reduced Kf, increased glomerular oncotic pressure, and reduced glomerular hydrostatic pressure. Bowman's space normally has negligible oncotic pressure because proteins do not filter across the glomerulus. Therefore, increased Bowman oncotic pressure does not occur physiologically and does not decrease GFR.

Option	Effect on GFR	Mechanism	Verdict
A	↓	↓ filtration capacity	□
B	↓	Opposes filtration	□
C	↓	↓ driving pressure	□
D	↑	Pulls fluid into Bowman space	□

$$GFR = K_f \times [(P_{GC} - P_{BS}) - (\pi_{GC} - \pi_{BS})]$$

QUESTION

Which of the following is not a mechanism by which antidiuretic hormone (ADH) facilitates the excretion of concentrated urine?

A. Increase absorption of urea in the medullary collecting duct

B. **Increase absorption of urea in the descending loop of Henle** — CORRECT

C. Decrease absorption of NaCl in the ascending loop of Henle

D. Increase water permeability in the collecting ducts

RIGHT ANSWER

OK

B. Increase absorption of urea in the descending loop of Henle

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

Which of the following best explains the development of hyponatremia in a hypovolemic patient presenting with diarrhea?

A Decreased reabsorption of sodium from kidney

B Decreased secretion of aldosterone

C **Non-osmotic release of antidiuretic hormone (ADH)** CORRECT

D Decreased sodium absorption from the intestine

OK RIGHT ANSWER

C. Non-osmotic release of antidiuretic hormone (ADH)

WHY THIS IS RIGHT

• Diarrhea -> hypovolemia
• Loss of:
• Na□
• Water

Effective circulating volume (ECV) \> Osmolality
Even if plasma osmolality is low or normal:
ADH is released via non-osmotic stimulus
• Triggered by:
• ↓ blood volume
• ↓ blood pressure

ADH:
• ↑ Water reabsorption (collecting duct)
• No proportional Na□ retention

Result:
• Dilutional hyponatremia

Hormonal response in hypovolemia:

System	Effect
ADH	↑ Water reabsorption
RAAS	↑ Na□ reabsorption
Sympathetic	↑ Renal vasoconstriction

Type	Volume Status	Mechanism
Hypovolemic	↓ volume	ADH due to volume loss
Euvolemic (SIADH)	Normal	ADH excess
Hypervolemic	↑ volume	CHF, cirrhosis

QUESTION

A 70-year-old man comes to the OPD due to increasing headaches, nausea, and vomiting. Medical history is significant for a transient ischemic attack that led to a right carotid endarterectomy 5 years ago. Blood pressure is 220/120 mm Hg and pulse is 70/min. Bilateral abdominal bruits are present. Blood testing in this patient would most likely show which of the following?

A Low renin, low aldosterone, high angiotensin II, low potassium

B **High renin, high aldosterone, high angiotensin II, low potassium** CORRECT

C Low renin, high aldosterone, high angiotensin II, low potassium

D Low renin, low aldosterone, low angiotensin II, high potassium

OK RIGHT ANSWER

B. High renin, high aldosterone, high angiotensin II, low potassium

WHY THIS IS RIGHT

Severe hypertension with bilateral abdominal bruits suggests bilateral renal artery stenosis, causing renovascular hypertension.

Renal hypoperfusion stimulates renin release, activating RAAS:
• ↑ Renin
• ↑ Angiotensin II
• ↑ Aldosterone -> Na□ retention & K□ loss (hypokalemia)

Thus, renovascular hypertension is characterized by high renin, high angiotensin II, high aldosterone, and low potassium.

QUESTION

54-year-old woman comes to the emergency department due to several hours of severe epigastric abdominal pain radiating to her back. She has also had nausea and several episodes of vomiting. The patient has a history of occasional upper abdominal pain after eating but has never had such severe symptoms. She has no other medical issues and takes no medications. Temperature is 37.6 C (99.6 F), blood pressure is 110/66 mm Hg, pulse is 118/min, and respirations are 24/min. The patient appears to be in moderate distress. Mucous membranes are dry. The abdomen is distended with marked epigastric tenderness. Bowel sounds are decreased.
Laboratory results are as follows:
Hematocrit: 48%
Leukocytes: 18,800/mm³
Total bilirubin: 2.2 mg/dL
Alkaline phosphatase: 370 U/L
Lipase: 2,192 U/L
Which of the following sets of renal findings are most expected in this patient?

	Renin Secretion	Efferend Arteriolar Resistance	Tubular Sodioum Reabsorption
A.	High	High	High
B.	High	Low	High
C.	Low	Low	Low
D.	Low	High	Low

A **Renin secretion high, Efferent arteriolar resistance high, Tubular sodium resporption high** CORRECT

B Renin secretion high, Efferent arteriolar resistance low, Tubular sodium resporption high

C Renin secretion low, Efferent arteriolar resistance low, Tubular sodium resporption low

D Renin secretion low, Efferent arteriolar resistance high, Tubular sodium resporption low

OK RIGHT ANSWER

A. Renin secretion high, Efferent arteriolar resistance high, Tubular sodium resporption high

WHY THIS IS RIGHT

Acute pancreatitis with vomiting and third-spacing leads to hypovolemia and decreased renal perfusion, causing prerenal azotemia.

Prerenal state activates RAAS, resulting in:
• ↑ Renin secretion
• ↑ Angiotensin II -> ↑ efferent arteriolar resistance (to maintain GFR)
• ↑ Aldosterone -> ↑ tubular sodium and water reabsorption

Thus, expected renal findings are high renin, high efferent arteriolar resistance, and high tubular sodium reabsorption.

QUESTION

Scientists studying the kidney's response to hypoperfusion apply a clip to a pig's right renal artery that reduces blood flow to the kidney by about 7 %. After 6 months, they perform a right nephrectomy and examine the glomeruli and tubules microscopically. Which of the following cell types would be most likely to undergo hyperplasia as a result of the clip placement?

A Cuboidal epithelial cells of the proximal tubules

B Endothelial cells of the efferent arteriole

C Intraglomerular mesangial cells

D **Modified smooth muscle cells of the afferent arteriole** CORRECT

OK RIGHT ANSWER

D. Modified smooth muscle cells of the afferent arteriole

WHY THIS IS RIGHT

Chronic renal hypoperfusion (as in renal artery stenosis/Goldblatt model) stimulates increased renin secretion.

Renin is produced by juxtaglomerular (JG) cells, which are modified smooth muscle cells of the afferent arteriole. Persistent decreased renal blood flow leads to hyperplasia and hypertrophy of JG cells due to sustained RAAS activation.

Thus, chronic hypoperfusion causes proliferation of modified smooth muscle cells of the afferent arteriole.

QUESTION

A decrease in GFR is associated with which of the following?

A Increased Renal Blood Flow

B Increased Glomerular hydrostatic pressure

C **Decreased Bowman's oncotic pressure** CORRECT

D Decreased Glomerular oncotic pressure

RIGHT ANSWER

OK

C. Decreased Bowman's oncotic pressure

WHY THIS IS RIGHT

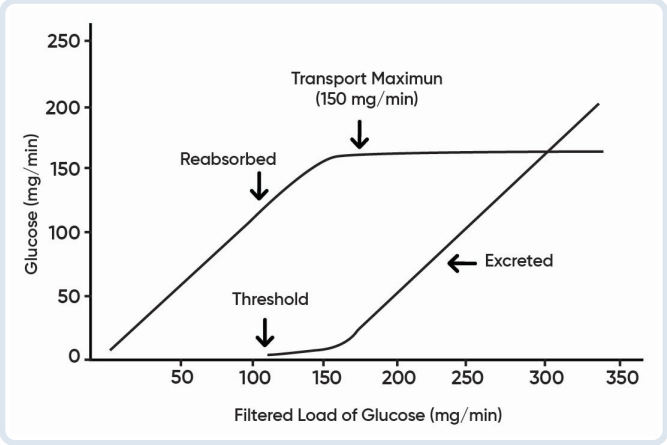
Glomerular filtration rate (GFR) depends on Starling forces across the glomerular membrane.

Oncotic pressure in Bowman’s space normally opposes filtration (though usually negligible). A decrease in Bowman’s space oncotic pressure reduces the driving force for filtration when proteins are present in Bowman’s space and then fall, leading to reduced effective filtration pressure and decreased GFR.

Thus, changes in hydrostatic and oncotic pressures across the glomerulus directly influence GFR.

QUESTION

A patient has a blood glucose level of 200 mg/dL and GFR of 90 ml/min. What is the amount of glucose excreted if the transport maximum of the patient is as shown below?



A 80 mg/min

B 50 mg/min

C 40 mg/min

D **30 mg/min** CORRECT

RIGHT ANSWER

OK **D. 30 mg/min**

WHY THIS IS RIGHT

The amount of glucose excreted in the above scenario is 30 mg/min.

Step 1: Total filtered glucose calculated by GFR x Plasma glucose concentration
Here,
• GFR = 90 ml/min
• Plasma glucose concentration = 200 mg/dL= 2 mg/ml (1 dL=100 ml).
• Total filtered glucose = 90 ml/min X 2 mg/ml = 180 mg/min.

Step 2: Check transport maximum:
Here, as shown in the graph transport maximum is 150 mg/min.

Step 3: Calculate the excretion
• Excretion = Total filtered glucose - Total reabsorbed glucose (Transport Maximum)
• As calculated above, total filtered glucose is 180, Transport maximum is 150
• = 180-150 mg/min = 30 mg/min is the excretion

Total filtered glucose=GFR × Plasma glucose concentration
Excretion=Total filtered glucose-Total reabsorbed glucose (Transport Max.)

QUESTION

Which of the following changes are most likely to occur in this patient's kidney function after starting therapy with NSAID?

A **Decreased renal perfusion, decreased intraglomerular pressure, and constant filtration fraction** CORRECT

B Increased renal perfusion, decreased intraglomerular pressure, and decreased filtration fraction

C Decreased renal perfusion, increased intraglomerular pressure, and decreased filtration fraction

D Decreased renal perfusion, decreased intraglomerular pressure, and constant filtration fraction

RIGHT ANSWER

OK **A. Decreased renal perfusion, decreased intraglomerular pressure, and constant filtration fraction**

WHY THIS IS RIGHT

NSAIDs inhibit renal prostaglandin synthesis (PGE□, PGI□), which normally dilate the afferent arteriole.
Blocking prostaglandins causes afferent arteriolar constriction, leading to:
• ↓ Renal perfusion (RPF)
• ↓ Intraglomerular pressure
• ↓ GFR

Since both RPF and GFR fall proportionally, the filtration fraction remains relatively constant.
Thus, NSAIDs can precipitate AKI, especially in patients with CHF, CKD, or volume depletion.

QUESTION

Goldblatt hypertension is best explained by which of the following mechanisms?

A Increased sodium retention due to primary hyperaldosteronism

B **Renal artery stenosis causing increased renin release and activation of the renin-angiotensin-aldosterone system** CORRECT

C Decreased renal perfusion causing pressure natriuresis

D Increased sympathetic discharge due to baroreceptor dysfunction

RIGHT ANSWER

OK **B. Renal artery stenosis causing increased renin release and activation of the renin-angiotensin-aldosterone system**

WHY THIS IS RIGHT

Goldblatt hypertension results from renal artery stenosis, which decreases renal perfusion pressure. This stimulates juxtaglomerular cells to release renin -> activates the renin-angiotensin-aldosterone system (RAAS) -> causes vasoconstriction, sodium and water retention -> systemic hypertension.
Thus, Goldblatt hypertension is a classic model of secondary hypertension due to renovascular activation of RAAS.

QUESTION

Which of the following correctly describes the nerve supply of the urinary bladder?

A Sympathetic fibers (T11-L2) cause detrusor contraction and internal sphincter relaxation

B **Parasympathetic fibers (S2-S4) cause detrusor contraction and internal sphincter relaxation** CORRECT

C Pudendal nerve (S2-S4) supplies the detrusor muscle

D Sympathetic fibers (S2-S4) mediate micturition reflex

RIGHT ANSWER

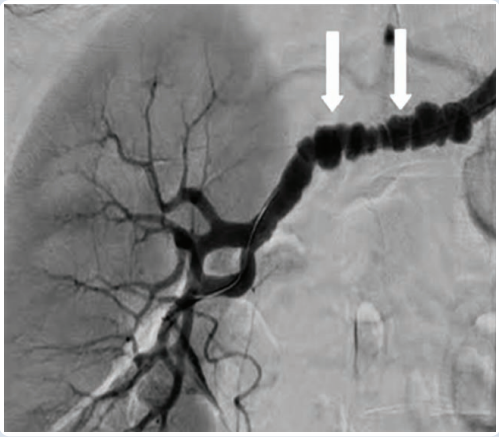
OK **B. Parasympathetic fibers (S2-S4) cause detrusor contraction and internal sphincter relaxation**

WHY THIS IS RIGHT

Normal micturition is mediated primarily by parasympathetic fibers (S2-S4) via pelvic splanchnic nerves. These fibers:
· Contract detrusor muscle
· Relax internal urethral sphincter
· Facilitating bladder emptying.
Sympathetic fibers (T11-L2) promote urine storage by relaxing the detrusor and contracting the internal sphincter, while the pudendal nerve (S2-S4) provides voluntary control of the external urethral sphincter.

QUESTION

A 32-year-old woman presents with refractory hypertension and recurrent headaches. There is no history of diabetes or dyslipidemia. Renal function tests are normal. A CT angiogram of the renal arteries image shown. Which of the following is the most likely diagnosis?



A. Atherosclerotic renal artery stenosis

B. Takayasu arteritis

C. **Fibromuscular dysplasia** — CORRECT

D. Polyarteritis nodosa

RIGHT ANSWER

OK

C. Fibromuscular dysplasia

WHY THIS IS RIGHT

Fibromuscular dysplasia (FMD) is a non-atherosclerotic, non-inflammatory disease-causing secondary hypertension in young women due to renal artery stenosis. Angiography classically shows a “string of beads” appearance caused by alternating areas of stenosis and aneurysmal dilatation in the renal artery.

It typically presents with refractory hypertension, normal renal function, and absence of atherosclerotic risk factors, and is treated with percutaneous transluminal angioplasty.

Why not other options:
A. Atherosclerosis: Elderly, proximal ostial stenosis, risk factors present.
B. Takayasu: Large-vessel disease with systemic features, not beaded renal artery.
D. PAN: Microaneurysms + systemic vasculitis signs.

QUESTION

A 30-year-old sexually active woman presented with dysuria, lower abdominal pain, and increased urinary frequency. Urine culture was done and it grew Staphylococcus saprophyticus with a colony count of 10^4 CFU/mL. What is the management?

A **It is a diagnosis of UTI** — CORRECT

B Sample contaminated. Take 2/3 more spontaneous fresh samples

C Reassure no treatment required

D Urgent CT scan

OK RIGHT ANSWER

A. It is a diagnosis of UTI

WHY THIS IS RIGHT

In symptomatic women, a colony count of ≥10^3-10^4 CFU/mL is sufficient to diagnose urinary tract infection.

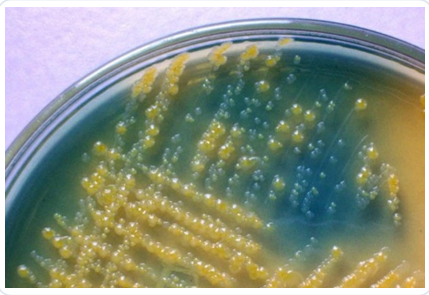
Staphylococcus saprophyticus is a common cause of UTI in sexually active young women.

Thus, this represents a true UTI requiring treatment, not contamination.

Situation	Significant bacteriuria
Symptomatic woman	≥10^3-10^4 CFU/mL □□
Asymptomatic	≥10^5 CFU/mL
Catheterized sample	≥10^2 CFU/mL

Mixed culture of fermenting (yellow colonies) and non-fermenting (blue colonies) bacteria on CLED agar.

Organism	Scenario
E. coli	Most common overall
Staph. saprophyticus	Young sexually active women
Klebsiella	Hospital / catheter
Proteus	Stones, alkaline urine



QUESTION

A 25-year-old male presented to the emergency department with head trauma due to a road traffic accident. In the hospital, the patient developed seizures. An emergency CT scan revealed widespread cerebral edema. Which of the following is the diuretic of choice for cerebral edema in this patient?

A **Mannitol** CORRECT

B Spironolactone

C Furosemide

D Hydrochlorothiazide

OK RIGHT ANSWER

A. Mannitol

WHY THIS IS RIGHT

Mannitol is the diuretic of choice for acute cerebral edema as it acts as an osmotic agent, drawing water out of brain parenchyma into the intravascular space.
IV Mannitol
↓
↑ Plasma osmolality
↓
Water shifts from brain -> blood
↓
↓ Brain volume
↓
↓ Intracranial pressure (ICP)

This rapidly reduces intracranial pressure and cerebral swelling in head injury.

Other diuretics do not provide rapid osmotic dehydration of brain tissue.

Drug	Mechanism	Role in Cerebral Edema
Mannitol	Osmotic	□ Drug of choice
Furosemide	Loop	Adjunct only
Spironolactone	Aldosterone antagonist	□ No role
Thiazide	DCT	□ No role

QUESTION

A patient presents with hypertension. He has a history of renal stones and has experienced a few episodes of renal colic. Which diuretic is the most appropriate to use?

A Furosemide

B **Hydrochlorothiazide** CORRECT

C Ethacrynic acid

D Spironolactone

RIGHT ANSWER

OK

B. Hydrochlorothiazide

WHY THIS IS RIGHT

Hydrochlorothiazide reduces urinary calcium excretion by increasing distal tubular calcium reabsorption. This helps prevent recurrent calcium renal stones while effectively treating hypertension.
Thiazide
↓
↓ NaCl reabsorption (DCT)
↓
↑ Ca²□ reabsorption (via Na□/Ca²□ exchanger)
↓
↓ Urinary Ca²□ (hypocalciuria)
↓
↓ Calcium stone formation

Loop diuretics increase calcium excretion, and spironolactone has no role in stone prevention.

Diuretic	Site	Effect on Ca²□	Stone Risk
Thiazide	DCT	↓ urinary Ca²□	□ Prevents stones
Loop (Furosemide)	TAL	↑ urinary Ca²□	□ Promotes stones
K□-sparing	Collecting duct	Neutral	No effect

Problem	Drug Benefit
Hypertension	Thiazide lowers BP
Recurrent Ca stones	↓ urinary Ca²□

QUESTION

A patient presented with a renal stone after a Proteus infection. What is the likely composition of the stone in him?

A Calcium phosphate

B **Magnesium ammonium phosphate** CORRECT

C Xanthine

D Cysteine

OK RIGHT ANSWER

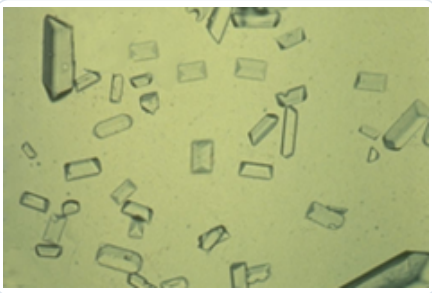
B. Magnesium ammonium phosphate

WHY THIS IS RIGHT

Urea
↓ (urease)
Ammonia (NH□) + CO□
↓
NH□ + H□O -> NH□□ + OH□
↓
Urine becomes ALKALINE (↑ pH)

Proteus species are urease-producing bacteria that alkalinize urine by splitting urea into ammonia.
Alkaline urine favors formation of struvite stones, composed of magnesium ammonium phosphate.

Stone Type	Composition	Urine pH	Cause
Struvite	MgNH□PO□	Alkaline	Proteus, Klebsiella
Calcium oxalate	CaC□O□	Acidic/neutral	Most common
Calcium phosphate	CaPO□	Alkaline	RTA type 1
Uric acid	Uric acid	Acidic	Gout
Cystine	Cystine	Acidic	Genetic defect



QUESTION

A 45-year-old man presents with flank pain, and imaging confirms the presence of a renal calculus. He was recently started on hydrochlorothiazide. What is the likely mechanism by which thiazides can cause this adverse effect?

A Increased excretion of calcium

B Decreased excretion of calcium

C Increased absorption of oxalate

D **Decreased excretion of citrate** CORRECT

RIGHT ANSWER

OK

D. Decreased excretion of citrate

WHY THIS IS RIGHT

Citrate is a natural stone inhibitor-↓ urinary citrate = stones (especially Ca stones), and thiazide-induced alkalosis promotes citrate reabsorption.

Role of Citrate	Effect
Binds Ca²□	Prevents Ca stone formation
Inhibits crystallization	Stops nucleation/aggregation

Thiazide diuretics can cause hypocitraturia by increasing proximal tubular citrate reabsorption. Citrate normally inhibits calcium stone formation by binding calcium in urine.

Reduced urinary citrate therefore predisposes to renal calculi, explaining this adverse effect.

Effect	Direction	Impact on Stones
Ca²□ excretion	↓	Protective
Citrate excretion	↓	Promotes stones
Volume	↓	Concentrates urine

QUESTION

First part of PCT has which of the following transport proteins?

1. Sodium potassium pump
2. Na- glucose co-transporter
3. Sodium hydrogen antiport
4. Na+ Bicarbonate cotransporter

A 1, 4

B **1, 2, 3** CORRECT

C 1, 3, 4

D 2, 3

OK

RIGHT ANSWER

B. 1, 2, 3

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

A 55-year-old patient with type 2 diabetes mellitus is being evaluated for medications to manage their condition and prevent renal complications. Which of the following drugs will not delay progression of renal disease?

A SGLT2 inhibitors

B Finerenone

C **Hydrochlorothiazide** CORRECT

D Angiotensin receptor blockers

RIGHT ANSWER

OK

C. Hydrochlorothiazide

WHY THIS IS RIGHT

Drug	Mechanism	Renal Protection
SGLT2 inhibitors	↓ hyperfiltration	□ Strong
Finerenone	↓ fibrosis/inflammation	□ Strong
Hydrochlorothiazide	↓ BP only	□ None
ARBs	↓ RAAS activity	□ Strong

Hydrochlorothiazide is an antihypertensive diuretic but has no proven renoprotective effect in diabetic kidney disease.

SGLT2 inhibitors, finerenone, and ARBs reduce intraglomerular pressure and albuminuria, thereby slowing CKD progression.

Hence, hydrochlorothiazide does not delay progression of renal disease.

QUESTION

A 58-year-old male presents with complaints of burning micturition and increased frequency. Prostatic examination is normal. Urinalysis shows >50 pus cells per high power field, but urine culture shows no growth. What is the most likely diagnosis?

A **Chronic bacterial prostatitis** CORRECT

B Acute bacterial prostatitis

C Chronic Granulomatous prostatitis

D Sterile pyuria

RIGHT ANSWER

OK

A. Chronic bacterial prostatitis

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1b” here and does not include a written explanation for this item.

QUESTION

Which of the following is correct about Finerenone?

1. It causes less hyperkalemia than spironolactone
2. It has a cardioprotective effect
3. It gets accumulated in the kidneys
4. It causes a significant reduction in blood pressure

A 1, 2 and 3

B **1 and 2** CORRECT

C 2, 3 and 4

D 1, 2 and 4

OK RIGHT ANSWER

B. 1 and 2

WHY THIS IS RIGHT

Solution - Finerenone:
• It is a non-steroidal mineralocorticoid receptor antagonist (MRA)
• Used mainly in:
• Chronic kidney disease (CKD) with diabetes
• Cardiovascular protection

Statement 3: Gets accumulated in kidneys
• Opposite is true
• Finerenone has Even distribution (heart + kidney)
• Unlike spironolactone -> heart accumulation
• Unlike eplerenone-> kidney accumulation

Statement 4: Significant BP reduction
• Only mild antihypertensive effect
• Not used as a primary antihypertensive

|
Feature	Finerenone	Spironolactone
Type	Non-steroidal MRA	Steroidal MRA
Tissue distribution	Balanced (heart + kidney)	Kidney predominant
Hyperkalemia	Less	More
Anti-androgen effects	None	Gynecomastia
Cardioprotection	Strong	Moderate
BP reduction	Mild	Moderate
CKD use	Preferred	Limited

QUESTION

What differentiates the distal convoluted tubule (DCT) from the proximal convoluted tubule (PCT)?

A Presence of carbonic anhydrase in luminal membrane

B Lack of Na□/K□ ATPase in the basolateral membrane

C Possession of brush border on luminal membrane

D **Presence of ‘tight’ tight junctions** CORRECT

OK

RIGHT ANSWER

D. Presence of ‘tight’ tight junctions

WHY THIS IS RIGHT

The distal convoluted tubule (DCT) has tight tight junctions between epithelial cells, making it relatively impermeable to water and allowing fine regulation of electrolyte transport.

In contrast, the proximal convoluted tubule (PCT) has leaky tight junctions and a prominent brush border for bulk reabsorption.

Thus, the presence of tight junctions is a key feature distinguishing the DCT from the PCT.

QUESTION

A 70-year-old man is brought to the emergency department by staff of the group home where he resides because of worsening confusion for the past week. He has a history of major depressive disorder and had an ischemic stroke 4 months ago. Current medications are aspirin and sertraline. His pulse is 78/min, and blood pressure is 135/88 mm Hg. Physical examination shows moist oral mucosa, normal skin turgor, and no peripheral edema. While in the waiting room, he has a generalized, tonic-clonic seizure. Laboratory studies show a serum sodium of 119 mEq/L and an elevated serum antidiuretic hormone concentration. Which of the following sets of additional laboratory is likely in the patient?

A Serum osmolarity increased, Urine sodium increased, Serum aldosterone increased

B **Serum osmolarity decreased, Urine sodium increased, Serum aldosterone decreased** CORRECT

C Serum osmolarity increased, Urine sodium decreased, Serum aldosterone increased

D Serum osmolarity decreased, Urine sodium increased, Serum aldosterone increased

RIGHT ANSWER

OK **B. Serum osmolarity decreased, Urine sodium increased, Serum aldosterone decreased**

WHY THIS IS RIGHT

This patient has SIADH (likely due to SSRI use or CNS disease), characterized by excess ADH causing water retention and dilutional hyponatremia.

Typical findings:
• ↓ Serum osmolality (dilutional)
• ↑ Urine sodium (euvolemic state with continued natriuresis)
• ↓ Aldosterone (suppressed due to mild volume expansion)
• Concentrated urine despite hyponatremia

Thus, SIADH presents with low serum osmolality, high urine sodium, and low aldosterone.

QUESTION

A 52-year-old male with Autosomal Dominant Polycystic Kidney Disease (ADPKD) was started on tolvaptan therapy. A few weeks later, he presents with symptoms of dry mouth and increased thirst. What is the likely mechanism behind these symptoms?

A **Increased free water clearance** — CORRECT

B V2 receptor agonism

C Increased renal cAMP levels

D Increase in urine osmolality

OK

RIGHT ANSWER

A. Increased free water clearance

WHY THIS IS RIGHT

Tolvaptan is a vasopressin V2 receptor antagonist used in ADPKD to slow cyst growth. Blocking V2 receptors in the collecting duct decreases aquaporin-2 insertion -> reduces water reabsorption -> increases free water clearance. This causes aquaresis (excretion of free water), leading to polyuria, thirst, and dry mouth.

Why not other options:
B. Tolvaptan is a V2 antagonist, not agonist.
C. Tolvaptan decreases cAMP, slowing cyst growth in ADPKD.
D. Causes dilute urine -> decreased urine osmolality.

QUESTION

A patient presents to the OPD with complaints of breathlessness on lying down and chest pain. He was diagnosed to have congestive cardiac failure and was administered torsemide. This drug inhibits which of the following type of transport mechanism?

A **Secondary active Cotransport** CORRECT

B Primary active transport

C Facilitated diffusion

D Secondary active Counter-transport

OK

RIGHT ANSWER

A. Secondary active Cotransport

WHY THIS IS RIGHT

Torsemide is a loop diuretic that inhibits the Na□-K□-2Cl□ cotransporter (NKCC2) in the thick ascending limb of the loop of Henle.

This transporter functions via secondary active cotransport, using the sodium gradient generated by Na□-K□ ATPase to reabsorb Na□, K□, and Cl□ together.

Inhibition leads to marked natriuresis and diuresis, reducing preload and relieving symptoms of congestive heart failure.

QUESTION

A 25 year old patient of bipolar disorder on lithium therapy presented with polydipsia and polyuria. Investigations revealed increased low urinary osmolality. Which of the following channels are involved here?

A AQP-1

B **AQP-2** CORRECT

C AQP-3

D AQP-4

RIGHT ANSWER

OK

B. AQP-2

WHY THIS IS RIGHT

Lithium therapy can cause nephrogenic diabetes insipidus by impairing ADH action in the collecting duct. Lithium reduces insertion and expression of Aquaporin-2 (AQP-2) channels on the luminal membrane of principal cells, leading to decreased water reabsorption.

This results in polyuria, polydipsia, and low urine osmolality due to inability to concentrate urine.

Why not other options:
A. AQP-1: PCT water channel, not ADH-dependent.
C. AQP-3 & D. AQP-4: Basolateral channels.

QUESTION

Identify the correct match out of the following diuretics and the respective site of action:

A Osmotic diuretics -> Collecting duct

B **Carbonic Anhydrase inhibitors -> Proximal convoluted tubule** CORRECT

C Thiazides -> Loop of Henle

D Aldosterone antagonists -> Distal Convoluted tubule

RIGHT ANSWER

OK **B. Carbonic Anhydrase inhibitors -> Proximal convoluted tubule**

WHY THIS IS RIGHT

Diuretics act at specific nephron sites:
• Carbonic anhydrase inhibitors (e.g., acetazolamide) act in the proximal convoluted tubule, inhibiting bicarbonate reabsorption and causing alkaline diuresis.

Other sites:
• Osmotic diuretics: mainly PCT & descending limb, not collecting duct
• Thiazides: distal convoluted tubule
• Aldosterone antagonists: collecting duct (principal cells)

Thus, carbonic anhydrase inhibitors correctly match with the proximal convoluted tubule.

QUESTION

Which of the following statements is true regarding clear cell carcinoma of the kidney?
1. It is the most common sporadic renal tumor
2. Associated with loss of long arm of chromosome 3
3. Arises from distal convoluted tubule
4. Cells show intracellular accumulation of PAS + Diastase sensitive substance

A 1, 2, 4

B 2, 3

C 1, 3

D **1, 4** CORRECT

OK RIGHT ANSWER

D. 1, 4

WHY THIS IS RIGHT

Clear cell renal cell carcinoma (ccRCC) is the most common sporadic renal tumor. It typically arises from the proximal convoluted tubule, not the distal tubule. Tumor cells appear “clear” due to intracellular accumulation of glycogen and lipids, which are PAS-positive and diastase-sensitive (glycogen).

Although ccRCC is classically associated with chromosome 3p deletion (VHL gene mutation), the key diagnostic features are:
• Most common RCC subtype
• Clear cytoplasm due to glycogen/lipid accumulation

Thus, statements 1 and 4 are correct.

QUESTION

A 22-year-old HIV/AIDS positive male has been on long term Amphotericin B therapy for mucormycosis. Laboratory studies reveal hypokalemia, and a normal anion gap metabolic acidosis. Which of the following structure/function combinations is most likely defective in this patient?

A Distal convoluted tubule - Secretion of hydrogen ions

B Distal convoluted tubule - Reabsorption of bicarbonate

C **Collecting duct - Secretion of hydrogen ions** CORRECT

D Proximal convoluted tubule - Reabsorption of bicarbonate

OK RIGHT ANSWER

C. Collecting duct - Secretion of hydrogen ions

WHY THIS IS RIGHT

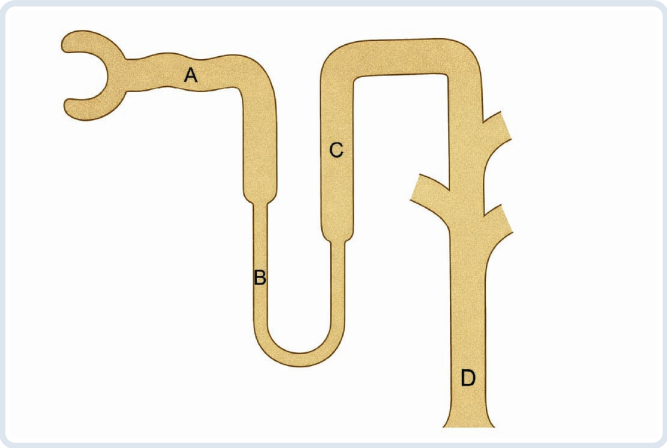
Amphotericin B can cause type 1 (distal) renal tubular acidosis by damaging the alpha-intercalated cells of the collecting duct, leading to impaired H secretion.

This results in:
• Normal anion gap metabolic acidosis
• Hypokalemia
• Inability to acidify urine

Thus, the primary defect is in the collecting duct - impaired hydrogen ion secretion.

QUESTION

Where in the tubule would a patient with severe central diabetes insipidus have the lowest tubular fluid osmolality?



A A

B B

C C

D **D** CORRECT

OK

RIGHT ANSWER

D. D

WHY THIS IS RIGHT

In severe central diabetes insipidus, there is absence of ADH, so the collecting duct remains impermeable to water.

Although the thick ascending limb and distal tubule dilute tubular fluid by reabsorbing solutes without water, in the absence of ADH the collecting duct continues to dilute the urine further, producing maximally dilute urine (\~50-100 mOsm/kg).

Therefore, the lowest tubular fluid osmolality is found in the collecting duct (D) in severe central DI.

QUESTION

Which of the following is false regarding Bartter's syndrome?

A Hypokalaemia and metabolic alkalosis

B Indomethacin may be used for treatment

C Associated with sensorineural deafness

D **Autosomal dominant disorder** CORRECT

RIGHT ANSWER

OK

D. Autosomal dominant disorder

WHY THIS IS RIGHT

Bartter syndrome is an autosomal recessive disorder due to defective Na□-K□-2Cl□ transport in the thick ascending limb, mimicking chronic loop diuretic use.

Key features include:
• Hypokalemia and metabolic alkalosis
• Normal/low BP with ↑ renin and aldosterone
• ↑ Prostaglandins -> treated with indomethacin
• Some variants associated with sensorineural deafness

Thus, it is not autosomal dominant, making that statement false.

QUESTION

Which of the following is most likely based on the lab values given?

Blood pH: 7.3
Na+: 134 mEq/L
K+: 2.8 mEq/L
Cl: 113mEq/L
HCO3-: 12mEq/L
BUN: 3 mg/dL
Creatinine: 0.6mg/dL
Urinary pH: 4.4

A Type 4 RTA

B Type 1 RTA

C **Type 2 RTA** CORRECT

D HAGMA

RIGHT ANSWER

OK **C. Type 2 RTA**

WHY THIS IS RIGHT

Low pH (7.3) with low HCO□□ (12) indicates metabolic acidosis. Normal anion gap with hyperchloremia (Cl□ 113) -> normal anion gap metabolic acidosis (RTA).
Presence of hypokalemia (K□ 2.8) and acidic urine pH <5.5 (4.4) indicates intact distal acidification but impaired proximal bicarbonate reabsorption -> Type 2 (proximal) renal tubular acidosis. Thus, Type 2 RTA is characterized by proximal HCO□□ wasting, hypokalemia, and urine that can be acidified (pH <5.5) once plasma bicarbonate falls.

Why not other options:
A. Type 4 RTA: Hyperkalemia present -> not here.
B. Type 1 RTA: Urine pH >5.5; here urine is acidic (4.4).
D. HAGMA: Anion gap normal.

QUESTION

Which of the following channels is NOT typically present on the luminal membrane of principal cells of the glomerular/renal collecting system?

A ENaC (Epithelial sodium channel)

B ROMK channels

C Aquaporin

D **Na-K +ATPase** CORRECT

OK RIGHT ANSWER

D. Na-K +ATPase

WHY THIS IS RIGHT

In principal cells of the collecting duct:
• Luminal (apical) membrane: ENaC (Na□ reabsorption), ROMK (K□ secretion), Aquaporin-2 (ADH-regulated water reabsorption)
• Basolateral membrane: Na□-K□ ATPase pump

The Na□-K□ ATPase is located on the basolateral surface, where it maintains the sodium gradient driving luminal sodium reabsorption; therefore, it is not present on the luminal membrane.

QUESTION

Which transport mechanism is primarily responsible for bicarbonate (HCO□□) reabsorption in the proximal convoluted tubule (PCT)?

A Sodium-potassium ATPase

B **Sodium-hydrogen antiporter** CORRECT

C Chloride-bicarbonate antiporter

D Potassium ATPase pump

OK RIGHT ANSWER

B. Sodium-hydrogen antiporter

WHY THIS IS RIGHT

In the proximal convoluted tubule, bicarbonate reabsorption occurs indirectly via the Na□-H□ antiporter (NHE).
· H□ is secreted into the tubular lumen in exchange for Na□.
· Secreted H□ combines with filtered HCO□□ -> forms CO□ and H□O (via carbonic anhydrase).
· CO□ diffuses into tubular cells and is converted back to HCO□□, which enters blood.
Thus, Na□-H□ antiporter is the primary mechanism driving bicarbonate reabsorption in PCT.

QUESTION

A newborn presents with proteinuria, low-molecular-weight proteins in urine, and vitamin D deficiency despite normal intake. Genetic analysis reveals a mutation in the LRP2 gene. Which of the following proteins is most likely dysfunctional in this patient?

A Cubilin

B Aquaporin-2

C **Megalin** CORRECT

D Na□-K□-ATPase

OK RIGHT ANSWER

C. Megalin

WHY THIS IS RIGHT

The LRP2 gene encodes megalin, a multiligand endocytic receptor located on the apical membrane of proximal convoluted tubule (PCT).
Megalin is responsible for reabsorption of:
· Low-molecular-weight proteins
· Vitamin D-binding protein (25-OH vitamin D complex)
· Other filtered proteins
· Retinol-binding protein
Mutation leads to impaired proximal tubular protein reabsorption -> low-molecular-weight proteinuria and vitamin D deficiency despite adequate intake.
Why not other options:
A. Cubilin: Different gene (CUBN), not LRP2.
B. Aquaporin-2: Water channel defect -> DI, not proteinuria.
D. Na□-K□-ATPase: General ion pump, not specific for protein reabsorption.

QUESTION

A 56-year-old man with newly diagnosed essential hypertension is started on hydrochlorothiazide as first-line therapy. After 4-6 weeks, his blood pressure is well controlled, but he complains of difficulty in achieving and maintaining an erection, which was not present earlier. He has no history of diabetes or vascular disease. Which of the following is the most likely cause of his new symptom?

A. Decreased testosterone levels due to aldosterone antagonism

B. **Reduced intravascular volume and altered vascular response caused by thiazide therapy** — CORRECT

C. Autonomic neuropathy secondary to hypertension

D. Increased prolactin levels

RIGHT ANSWER

OK

B. Reduced intravascular volume and altered vascular response caused by thiazide therapy

WHY THIS IS RIGHT

Thiazide diuretics (e.g., hydrochlorothiazide) can cause erectile dysfunction as a side effect due to reduced intravascular volume and decreased penile blood flow, leading to altered vascular response. They may also reduce zinc levels, indirectly affecting testosterone and sexual function.
Thus, erectile dysfunction is a recognized adverse effect of thiazide therapy and may require dose adjustment or change of antihypertensive drug.

QUESTION

All of the following statements regarding urea handling and function in the kidney are TRUE, EXCEPT:

A Urea contributes significantly to the corticomedullary osmotic gradient

B Urea permeability of the inner medullary collecting duct increases in the presence of ADH

C Urea recycling helps in maximal concentration of urine

D **Urea is actively secreted in the proximal convoluted tubule** CORRECT

OK RIGHT ANSWER

D. Urea is actively secreted in the proximal convoluted tubule

WHY THIS IS RIGHT

Urea plays a key role in urine concentration by contributing to the medullary osmotic gradient.
· It is passively reabsorbed in the proximal tubule.
· ADH increases urea permeability of the inner medullary collecting duct.
· Urea recycling between the collecting duct and loop of Henle helps maintain high medullary osmolality for maximal urine concentration.
Urea is not actively secreted in the proximal convoluted tubule; its movement is mainly passive.

QUESTION

A 60-year-old man with chronic congestive cardiac failure (CCF) presents with jugular venous distention, ascites, and bilateral pedal edema. Laboratory evaluation reveals elevated atrial natriuretic peptide (ANP) levels. ANP promotes natriuresis by decreasing sodium reabsorption predominantly at which of the following nephron segments?

A Proximal convoluted tubule

B Thick ascending limb of loop of Henle

C Distal convoluted tubule

D **Medullary collecting duct** CORRECT

RIGHT ANSWER

OK

D. Medullary collecting duct

WHY THIS IS RIGHT

Atrial natriuretic peptide (ANP) promotes natriuresis primarily by inhibiting sodium reabsorption in the medullary collecting duct.
ANP increases intracellular cGMP and:
· Inhibits epithelial sodium channels (ENaC)
· Reduces aldosterone secretion
· Increases GFR (via afferent dilation & efferent constriction)
The collecting duct is the final site of regulated sodium reabsorption, so ANP's action here produces significant sodium and water excretion, helping counteract volume overload in congestive heart failure.

QUESTION

Which of the following substances will have the highest concentration at the end of the proximal tubule compared to the beginning of the proximal tubule?

A Bicarbonate

B **Creatinine** CORRECT

C Glucose

D Sodium

RIGHT ANSWER

OK

B. Creatinine

WHY THIS IS RIGHT

Substances that are freely filtered but neither reabsorbed nor significantly secreted become more concentrated in tubular fluid along the proximal tubule because water is reabsorbed (\~65%).
Creatinine is minimally reabsorbed (and slightly secreted), so as water is reabsorbed, its tubular concentration increases.
Thus, creatinine concentration is highest at the end of the proximal tubule compared to the beginning.
In contrast:
· Glucose -> almost completely reabsorbed in PCT
· Bicarbonate -> largely reabsorbed
· Sodium -> reabsorbed proportionally with water (remains relatively unchanged in concentration)

QUESTION

A 6-year-old child with cystic fibrosis presents with recurrent pulmonary infections and thick, tenacious sputum. Genetic testing confirms a CFTR mutation. The pathophysiology involves increased sodium absorption across the airway epithelium, leading to dehydration of airway surface liquid. Which drug, when administered by inhalation, would most directly counteract this abnormal ion transport?

A Acetazolamide

B Furosemide

C **Amiloride** CORRECT

D Spironolactone

OK RIGHT ANSWER

C. Amiloride

WHY THIS IS RIGHT

In cystic fibrosis, defective CFTR channels lead to increased epithelial sodium (Na^+) absorption via ENaC and decreased chloride secretion, causing dehydration of airway surface liquid and thick mucus.
Inhaled amiloride, an epithelial sodium channel (ENaC) blocker, directly counteracts this abnormal ion transport by reducing sodium and water reabsorption from airway surfaces, thereby improving airway hydration and mucus clearance.
Why not others:
A. Acetazolamide: Acts in kidney (PCT); no airway ENaC effect.
B. Furosemide: Acts in loop of Henle; renal action only.
D. Spironolactone: Blocks aldosterone in kidney; not airway sodium channels.

QUESTION

A 56-year-old man has resistant hypertension despite adherence to four antihypertensive drugs, including a diuretic and mineralocorticoid receptor antagonist. Secondary causes have been excluded. He is being evaluated for novel non-pharmacological interventional therapies which is?

A Electrical inhibition of aortic baroreceptors

B Electrical stimulation of carotid baroreceptors

C Radiofrequency ablation of renal parasympathetic nerves

D **Endovascular radiofrequency ablation of renal sympathetic nerves** CORRECT

OK RIGHT ANSWER

D. Endovascular radiofrequency ablation of renal sympathetic nerves

WHY THIS IS RIGHT

Endovascular radiofrequency ablation of renal sympathetic nerves (renal denervation) is a novel interventional therapy for resistant hypertension. It works by disrupting renal sympathetic nerve activity, which reduces renin release, decreases sodium retention, and lowers systemic vascular resistance, ultimately reducing blood pressure.
Renal sympathetic overactivity plays a key role in resistant hypertension; therefore, catheter-based renal denervation is considered in patients with persistent hypertension despite optimal multidrug therapy and exclusion of secondary causes.

QUESTION

Lorundrostat lowers blood pressure primarily by inhibiting which of the following enzymes?

A 11β-hydroxylase (CYP11B1)

B **Aldosterone synthase (CYP11B2)** CORRECT

C Angiotensin-converting enzyme

D Mineralocorticoid receptor

OK RIGHT ANSWER

B. Aldosterone synthase (CYP11B2)

WHY THIS IS RIGHT

Lorundrostat is a selective aldosterone synthase (CYP11B2) inhibitor that lowers blood pressure by reducing aldosterone production in the adrenal zona glomerulosa. Decreased aldosterone leads to reduced sodium and water reabsorption and increased potassium retention, thereby lowering intravascular volume and blood pressure.
It represents a newer targeted therapy for resistant hypertension by directly blocking aldosterone synthesis rather than blocking its receptor or upstream RAAS components.

QUESTION

An elderly man presents with progressive swelling of the legs and frothy urine. Physical examination reveals bilateral pitting pedal edema. Urinalysis shows 4+ proteinuria and fatty casts. A renal biopsy is performed, and Congo red staining reveals apple-green birefringence under polarized light. Which of the following is the most likely underlying mechanism of his renal disease?

A Immune complex deposition due to membranous glomerulopathy

B Mesangial proliferation due to IgA deposition

C **Extracellular deposition of misfolded proteins in amyloidosis** CORRECT

D Autoantibody-mediated injury to podocytes

OK RIGHT ANSWER

C. Extracellular deposition of misfolded proteins in amyloidosis

WHY THIS IS RIGHT

Apple-green birefringence on Congo red staining is diagnostic of amyloidosis.

Renal amyloidosis causes nephrotic syndrome with heavy proteinuria, fatty casts, and edema. The underlying mechanism is extracellular deposition of misfolded amyloid proteins in the glomeruli.

Misfolded proteins (β-pleated sheets)
↓
Resistant to degradation
↓
Extracellular deposition in glomeruli
↓
Disrupts filtration barrier
↓
Massive proteinuria (nephrotic syndrome)

Type	Protein	Cause
AL	Light chains	Multiple myeloma
AA	Serum amyloid A	Chronic inflammation

Immune complex, IgA deposition, and podocyte injury describe other glomerulopathies, not amyloidosis.

QUESTION

A woman presents to you with fever, arthralgia, ulcers, fatigue for the past six months, and new-onset hematuria. Urine examination reveals RBC casts and proteinuria. What is the likely diagnosis?

A Acute interstitial nephritis

B Poststreptococcal glomerulonephritis

C **Lupus nephritis** CORRECT

D IgA nephropathy

RIGHT ANSWER

OK

C. Lupus nephritis

WHY THIS IS RIGHT

Chronic systemic symptoms with fever, arthralgia, and ulcers suggest systemic lupus erythematosus.
Autoantibodies (anti-dsDNA)
↓
Immune complex formation
↓
Deposition in glomerulus
↓
Complement activation
↓
Inflammation & injury
↓
Hematuria + proteinuria

Renal involvement with hematuria, proteinuria, and RBC casts indicates immune complex-mediated glomerulonephritis.
• “Full house” IF (IgG, IgA, IgM, C3, C1q)
• Wire loop lesions
• Immune complex deposition everywhere

This constellation is characteristic of lupus nephritis.

PSGN is post-infectious and acute, IgA nephropathy is episodic post-URI, and AIN lacks RBC casts.

Disease	Key Feature	Clue
Lupus nephritis	RBC casts + systemic symptoms	SLE
PSGN	Cola urine + infection	Child
IgA nephropathy	Hematuria after URTI	Episodic
AIN	Eosinophils	Drug-induced

QUESTION

A 10-year-old child presented with cola colored urine and had a history of skin infection few days back. His creatinine value was initially 2.5 mg/dl but even after 3 weeks of treatment it increased to 4.5 mg/dl. Which of the following is a likely kidney biopsy finding on electron microscopy?

A Subendothelial deposits

B Subepithelial deposits

C **Crescents in glomeruli** CORRECT

D Mesangial deposits

OK RIGHT ANSWER

C. Crescents in glomeruli

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

A 50-year-old patient has malaise for 2 weeks. His investigations revealed an elevated creatinine of 4.5mg/dl. His blood tests also showed the presence of HBs antigen. A renal biopsy was performed which demonstrated spike and dome on special staining. Which of the following conditions is the patient likely to be suffering from?

A Minimal change disease

B **Membranous glomerulopathy** CORRECT

C Focal segmental glomerulosclerosis

D Renal amyloidosis

OK RIGHT ANSWER

B. Membranous glomerulopathy

WHY THIS IS RIGHT

Hepatitis B infection
↓
Immune complex formation
↓
Deposition in subepithelial region (GBM)
↓
Complement activation (C5b-9)
↓
Podocyte injury
↓
Proteinuria (nephrotic syndrome)

Membranous glomerulopathy shows characteristic “spike and dome” appearance on silver stain due to subepithelial immune complex deposition.

It is classically associated with HBsAg positivity in secondary membranous nephropathy.

Patients present with nephrotic syndrome and elevated creatinine in adults.

Minimal change lacks spikes, FSGS shows segmental sclerosis, and amyloidosis shows Congo red positivity.

Disease	Key Feature	IF Pattern	Association
Membranous	Spike & dome	Granular	HBV, malignancy
Minimal change	Podocyte effacement	Negative	Children
FSGS	Segmental sclerosis	IgM/C3	HIV
Amyloidosis	Congo red +	Amorphous deposits	Chronic disease

QUESTION

A 47-year-old male presents with 400 ml urine output in 24 hours. On histology he had crescents with pauci immune glomerulonephritis. What is the main etiology?

A Anti GBM antibody

B **ANCA** CORRECT

C Anti phospholipase A2 receptor antibody

D Anti nculear antibody

OK RIGHT ANSWER

B. ANCA

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

Which of the following bands will increase in serum electrophoresis in Nephrotic syndrome?

A Gamma globulin

B **Alpha 2 globulin** CORRECT

C Beta globulin

D Albumin

OK RIGHT ANSWER

B. Alpha 2 globulin

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1b” here and does not include a written explanation for this item.

QUESTION

Which of the following findings are typically associated with glomerular hematuria?

A. Dysmorphic RBCs
B. Dysuria absent
C. Crystals in urine
D. Edema is rare

A **A, B** CORRECT

B A only

C A, B and C

D A, B, D

OK RIGHT ANSWER

A. A, B

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1c” here and does not include a written explanation for this item.

QUESTION

Which of the following renal histological features is characteristically seen in diabetic nephropathy but not typically seen in other glomerulonephritis?

A **Kimmelstiel-Wilson** CORRECT

B Mesangial sclerosis

C GBM thickening

D Foot process effacement

OK RIGHT ANSWER

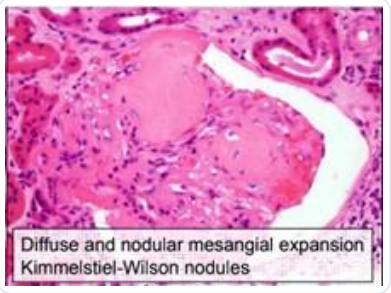
A. Kimmelstiel-Wilson

WHY THIS IS RIGHT

The “signature lesion”: Kimmelstiel-Wilson nodules
• Nodular, PAS-positive mesangial expansion
• Located in glomerular tuft
• Strongly associated with long-standing diabetes mellitus

These are pathognomonic of diabetic nephropathy, representing nodular mesangial expansion.
Mesangial sclerosis and GBM thickening can be seen in other chronic glomerulopathies, and foot process effacement is nonspecific.
Thus, nodular glomerulosclerosis uniquely points to diabetic nephropathy.

Feature	Description
GBM thickening	Early change
Mesangial expansion	Diffuse
K-W nodules	Nodular, pathognomonic
Hyaline arteriolosclerosis	Both afferent & efferent (important!)
Proteinuria	Nephrotic range



QUESTION

A child presents with periorbital oedema and pedal oedema. The laboratory investigations reveal serum creatinine = 2.1 mg/dL, urinary protein: 5.2 g/day, and Cholesterol: 310 mg/dL. Which of the following are included in the criteria of diagnosis of Nephrotic syndrome?

1. Proteinuria >3.5 g/day
2. Hyperlipidemia
3. Creatinine more than 1.5mg/dl
4. Hypoalbuminaemia

A 1 and 2

B **1, 2 and 4** CORRECT

C 1, 3 and 4

D 2, 3 and 4

RIGHT ANSWER

OK **B. 1, 2 and 4**

WHY THIS IS RIGHT

Core Diagnostic Criteria of Nephrotic Syndrome

Criterion	Mechanism
Proteinuria >3.5 g/day	Increased glomerular permeability
Hypoalbuminemia	Loss of albumin in urine
Hyperlipidemia	Liver compensatory synthesis
Edema	↓ oncotic pressure + Na retention

Nephrotic syndrome is defined by massive proteinuria (>3.5 g/day) leading to hypoalbuminemia, which in turn causes edema and secondary hyperlipidemia.
Serum creatinine is not part of the diagnostic criteria and may be normal or elevated depending on renal function.

Statement	Included in Criteria?	Explanation
1\. Proteinuria \>3\.5 g/day	□	Hallmark feature
2\. Hyperlipidemia	□	Liver compensation
3\. Creatinine	□	Reflects renal function, not definition
4\. Hypoalbuminemia	□	Due to urinary loss

QUESTION

A 5-year-old child with 4+ proteinuria is unresponsive to corticosteroid therapy. There is no history of hematuria and hypertension. Renal biopsy shows segmental sclerosis and hyalinosis, predominantly involving the juxtamedullary glomeruli. What is the most likely diagnosis?

A IgA nephropathy

B **Focal Segmental Glomerulosclerosis (FSGS)** CORRECT

C Membranous nephropathy

D Minimal change disease

OK RIGHT ANSWER

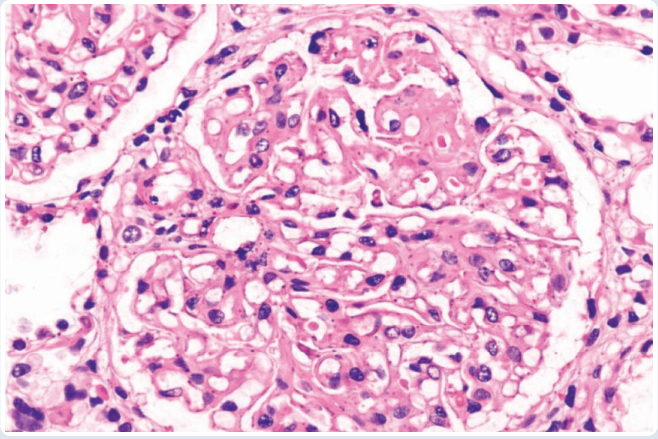
B. Focal Segmental Glomerulosclerosis (FSGS)

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1d” here and does not include a written explanation for this item.

QUESTION

A 50-year-old man on treatment for adenocarcinoma lung presents with complaints of facial puffiness and frothy urine. Given below is the histological image of the condition. Where will the deposits be seen?



A **Subepithelial** — CORRECT

B Intramembranous

C Subendothelial

D Mesangial

RIGHT ANSWER

OK **A. Subepithelial**

WHY THIS IS RIGHT

A patient with malignancy (lung adenocarcinoma) presenting with edema and frothy urine suggests secondary membranous nephropathy, a common paraneoplastic glomerular disease.

Membranous nephropathy is characterized by immune complex deposition along the subepithelial side of the glomerular basement membrane, leading to GBM thickening and nephrotic syndrome. Thus, deposits in membranous nephropathy are seen in the subepithelial region (“spike and dome” appearance on EM).

Why not other options:
B. Intramembranous: Seen in dense deposit disease (MPGN type II).
C. Subendothelial: Seen in lupus nephritis (class III/IV) and MPGN type I.
D. Mesangial: Seen in IgA nephropathy.

QUESTION

A 47-year-old woman comes to the physician because of a 2-week history of gradually worsening facial and lower extremity swelling. She has had a 4-kg weight gain during this time. Her blood pressure is 150/88 mm Hg. Examination shows periorbital edema and 2+ pretibial edema bilaterally. A 24-hour collection of urine shows 4.0 g of proteinuria. Microscopic examination of a kidney biopsy specimen shows thickening of the glomerular basement membrane. Electron microscopy shows dense subepithelial deposits. Further evaluation is most likely to show which of the following?

A **Anti-phospholipse A2 receptor antibodies** CORRECT

B Anti-myeloperoxidase antibodies

C Anti-streptolysin O antibodies

D Anti-proteinase 3 antibodies

RIGHT ANSWER

OK **A. Anti-phospholipse A2 receptor antibodies**

WHY THIS IS RIGHT

Nephrotic-range proteinuria (4 g/day), edema, and biopsy showing thickened glomerular basement membrane with subepithelial immune complex deposits are characteristic of membranous nephropathy.

Primary membranous nephropathy is strongly associated with anti-phospholipase A2 receptor (PLA2R) antibodies, directed against podocyte antigens. Thus, detection of anti-PLA2R antibodies supports the diagnosis of primary membranous nephropathy.

Why not other options:
B. Anti-myeloperoxidase (MPO) antibodies: Seen in p-ANCA-associated vasculitis -> pauci-immune crescentic GN, not membranous nephropathy.
C. Anti-streptolysin O (ASO) antibodies: Seen in post-streptococcal GN -> subepithelial “hump” deposits with nephritic syndrome.
D. Anti-proteinase 3 (PR3) antibodies: Seen in c-ANCA vasculitis (GPA) -> pauci-immune crescentic GN.

QUESTION

A 37-year-old woman comes to the OPD due to worsening leg swelling for the past 2 months. She has also felt excessively tired and has had achy pain in her hands. Serum creatinine level is 1.8 mg/dL. Urinalysis is positive for proteinuria and hematuria. Light microscopy of samples from a kidney biopsy shows diffuse, global endocapillary hypercellularity. Direct immunofluorescence demonstrates staining with diffuse, global, granular staining of glomerular capillary walls by IgG, IgM, IgA, C1q, and C3. Electron microscopy will demonstrate which of the following changes?

A **Subendothelial deposits** CORRECT

B Mesangial deposits

C Subepithelial deposits

D Intra-membranous deposits

OK RIGHT ANSWER

A. Subendothelial deposits

WHY THIS IS RIGHT

Diffuse endocapillary hypercellularity with “full house” immunofluorescence (IgG, IgA, IgM, C3, C1q) is characteristic of lupus nephritis, especially class III/IV.

In proliferative lupus nephritis, immune complexes deposit mainly in the subendothelial region, producing a “wire-loop” appearance on light microscopy and causing hematuria and proteinuria. Thus, subendothelial immune complex deposits are the key electron microscopy finding.

Why not other options:
B. Mesangial deposits: Seen in IgA nephropathy or class II lupus, not diffuse proliferative lupus.
C. Subepithelial deposits: Seen in membranous nephropathy (“spike and dome”).
D. Intramembranous deposits: Seen in dense deposit disease/MPGN type II.

QUESTION

A 44-year-old man comes to the physician because of a 2-week history of lower extremity swelling and frothy urine. He has a history of chronic hepatitis C infection. Physical examination shows 3+ pitting edema of the lower legs and ankles. Further evaluation of this patient is most likely to show which of the following?

A Decreased cholesterol

B **Increased lipoproteins** CORRECT

C Increased antithrombin III

D Increased immunoglobulins

RIGHT ANSWER

OK

B. Increased lipoproteins

WHY THIS IS RIGHT

Chronic hepatitis C can cause membranoproliferative glomerulonephritis, leading to nephrotic syndrome (edema, proteinuria, frothy urine). Nephrotic syndrome is characterized by:
• Heavy proteinuria
• Hypoalbuminemia
• Edema
• Hyperlipidemia with increased lipoproteins (compensatory hepatic synthesis)
• Lipiduria

There is also loss of antithrombin III and immunoglobulins in urine, causing hypercoagulability and increased infection risk.

QUESTION

A 47-year-old male presents with 400 ml urine output in 24 hours. On biopsy he had crescents with pauci immune glomerulonephritis. What is the next step?

A Anti GBM antibody

B **ANCA** CORRECT

C Anti phospholipase A2 receptor antibody

D Anti nculear antibody

OK RIGHT ANSWER

B. ANCA

WHY THIS IS RIGHT

Pauci-immune crescentic glomerulonephritis is most commonly associated with ANCA-associated vasculitis (e.g., granulomatosis with polyangiitis, microscopic polyangiitis). Biopsy shows crescents with minimal or no immune deposits on immunofluorescence (“pauci-immune”).

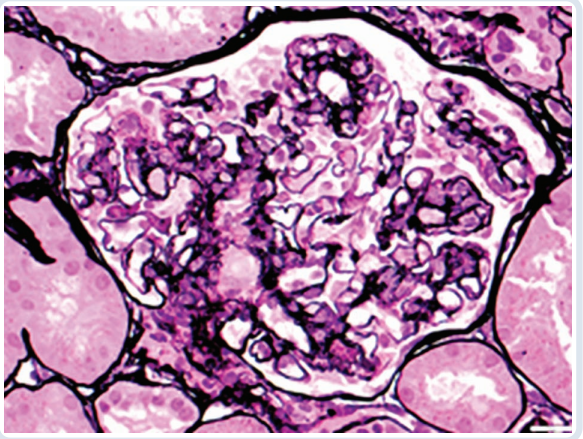
The next diagnostic step is testing for ANCA (c-ANCA/PR3 or p-ANCA/MPO) to confirm ANCA-associated vasculitis causing rapidly progressive glomerulonephritis.

Why not other options:

A. Anti-GBM: Linear IgG deposits (Goodpasture), not pauci-immune.
C. Anti-PLA2R: Membranous nephropathy (nephrotic).
D. ANA: Lupus nephritis with immune deposits.

QUESTION

A 48-year-old woman presents with progressive facial puffiness and lower extremity edema over the past 2 months. Laboratory studies reveal serum creatinine of 1.9 mg/dL, albumin level of 2.7 g/dL, and 3+ proteinuria on urinalysis. A kidney biopsy is performed, and light microscopy with hematoxylin and eosin staining is shown. Which of the following is the most likely cause of this patient's kidney findings?



A Diabetes mellitus

B Systemic lupus erythematosus

C **Hepatitis B infection** CORRECT

D Multiple myeloma

RIGHT ANSWER

OK

C. Hepatitis B infection

WHY THIS IS RIGHT

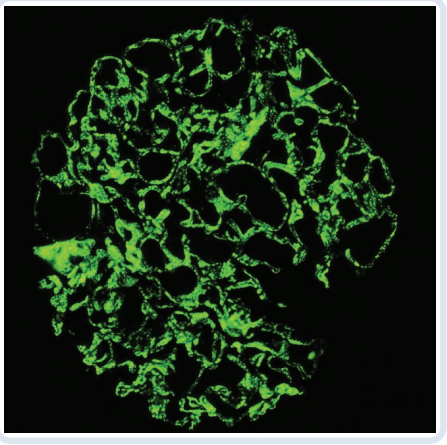
An adult with nephrotic syndrome (edema, hypoalbuminemia, heavy proteinuria) and biopsy showing thickened glomerular capillary walls on light microscopy suggests membranous nephropathy.

Secondary membranous nephropathy is classically associated with Hepatitis B infection, due to immune complex deposition along the subepithelial surface of the glomerular basement membrane.

Thus, chronic Hepatitis B infection is a well-known cause of membranous nephropathy presenting with nephrotic syndrome in adults.

QUESTION

A 13-year-old boy comes to the clinic due to shortness of breath and fatigue over the past 2 weeks. He reports significant weight gain, swelling around the ankles, and profound fatigue. Laboratory findings include red blood cell casts in urine and elevated creatinine. Immunofluorescence microscopy shown. Which of the following light microscopy findings is most likely to be seen in this patient's renal biopsy?



A Subepithelial deposits

B **Crescent formation** CORRECT

C Nodular glomerulosclerosis

D Normal glomeruli

RIGHT ANSWER

OK **B. Crescent formation**

WHY THIS IS RIGHT

Red blood cell casts, rising creatinine, and immunofluorescence showing linear/immune deposition along GBM suggest rapidly progressive glomerulonephritis (RPGN).

The characteristic light microscopy finding in RPGN is crescent formation, caused by proliferation of parietal epithelial cells and macrophage infiltration in Bowman's space. Crescents indicate severe glomerular injury and rapidly declining renal function requiring urgent treatment.

Why not other options:

A. Subepithelial deposits: PSGN, not RPGN.
C. Nodular glomerulosclerosis: Diabetic nephropathy.
D. Normal glomeruli: Minimal change disease (nephrotic).

QUESTION

All of the following conditions are known to be associated with chronic Hepatitis C virus (HCV) infection, EXCEPT:

A Mixed cryoglobulinemia

B Leukocytoclastic vasculitis

C Membranoproliferative glomerulonephritis (MPGN)

D **Gianotti-Crosti syndrome** CORRECT

OK

RIGHT ANSWER

D. Gianotti-Crosti syndrome

WHY THIS IS RIGHT

Chronic hepatitis C infection is associated with multiple immune-complex mediated extrahepatic manifestations, including:
· Mixed cryoglobulinemia
· Leukocytoclastic vasculitis
· Membranoproliferative glomerulonephritis (MPGN)
These occur due to circulating immune complex deposition.
Gianotti-Crosti syndrome is classically associated with hepatitis B infection (and some viral infections in children), not chronic hepatitis C.

QUESTION

Which of the following are considered poor prognostic factors in PSGN?
A. Younger age at presentation
B. Nephrotic syndrome at presentation
C. Presence of crescent formation on renal biopsy
D. Early penicillin therapy for streptococcal infection

A A,B

B A,B,C

C **B,C** CORRECT

D ALL

OK RIGHT ANSWER

C. B,C

WHY THIS IS RIGHT

Poor prognostic factors in post-streptococcal glomerulonephritis (PSGN) include features indicating severe glomerular injury.
These are:
· Nephrotic syndrome at presentation -> suggests severe glomerular damage
· Crescent formation on renal biopsy -> indicates rapidly progressive GN and worse renal outcome
Younger age generally has an excellent prognosis, and early penicillin therapy prevents spread of infection but does not worsen prognosis.

QUESTION

A 45-year-old male with septic shock and acute kidney injury (AKI) is hemodynamically unstable. Which of the following is the most appropriate modality of dialysis in this situation?

A Intermittent Hemodialysis (IHD)

B Extended Daily Dialysis (EDD)

C **Continuous Renal Replacement Therapy (CRRT)** CORRECT

D Peritoneal Dialysis (PD)

RIGHT ANSWER

OK

C. Continuous Renal Replacement Therapy (CRRT)

WHY THIS IS RIGHT

CRRT (24-hour continuous therapy)
↓
Slow solute removal
↓
Gradual fluid removal
↓
Minimal BP fluctuations
↓
Safe in unstable patients

Correct answer is C because hemodynamically unstable patients cannot tolerate rapid fluid and solute shifts seen with intermittent dialysis.

Continuous renal replacement therapy provides slow, steady fluid removal and solute clearance, maintaining cardiovascular stability.

IHD and EDD can worsen hypotension. Peritoneal dialysis is less effective in severe septic AKI.

Feature	CRRT	IHD	PD
Duration	Continuous	3-4 hrs	Continuous
Fluid removal	Slow	Rapid	Slow
Hemodynamic effect	Stable □□	Unstable □	Stable
ICU use	Best	Limited	Limited
Solute clearance	Moderate	High	Low

QUESTION

A patient with hyperkalemia and elevated urea levels underwent dialysis. Towards the end of the session, she became drowsy and had a sudden seizure episode. On examination, the patient was hypotensive. What is the treatment for this condition?

A Bumetanide

B Ethacrynic acid

C Nesiritide

D **Mannitol** CORRECT

RIGHT ANSWER

OK

D. Mannitol

WHY THIS IS RIGHT

This presentation is dialysis disequilibrium syndrome, caused by rapid urea removal leading to cerebral edema.

Neurological symptoms (drowsiness, seizures) with hypotension occur towards the end of dialysis.

Mannitol is an osmotic agent that reduces cerebral edema and is the treatment of choice.

Mannitol infusion
↓
↑ Plasma osmolality
↓
Water moves OUT of brain
↓
↓ Cerebral edema
↓
Relieves symptoms

Complication	Cause	Feature
DDS	Rapid urea removal	CNS symptoms
Hypotension	Fluid removal	Dizziness
Diselectrolytemia	Rapid shifts	Arrhythmias
Infection	Access site	Fever

Loop diuretics and nesiritide have no role in acute DDS management.

QUESTION

A patient with CKD with refractory hypertension is brought to the casualty with dizziness. His BP was found to be 200/120 mmHg. His BUN was 50 mg/dl and creatinine was 3 mg/dl. Potassium was 6 mEq/L. Which of the following is an indication of emergency hemodialysis?

A **Severe acidosis with elevated BUN and creatinine** CORRECT

B Hypertension

C Potassium level of 6 mEq/L

D Metabolic alkalosis

OK RIGHT ANSWER

A. Severe acidosis with elevated BUN and creatinine

WHY THIS IS RIGHT

Emergency Hemodialysis-

Letter	Indication	Explanation
A	Acidosis	Severe metabolic acidosis (pH < 7.1) not responding to treatment
E	Electrolytes	Severe hyperkalemia (usually >6.5 mEq/L or with ECG changes)
I	Intoxication	Dialyzable toxins (methanol, lithium, etc.)
O	Overload	Fluid overload refractory to diuretics
U	Uremia	Symptoms like encephalopathy, pericarditis

Emergency hemodialysis is indicated in CKD patients with severe metabolic acidosis associated with azotemia, reflecting life-threatening internal milieu disturbance.

Hypertension alone is managed medically, and K□ = 6 mEq/L without ECG changes is not an absolute indication.

QUESTION

A 10-year-old child presented with follicular tonsillitis, for which antibiotics were prescribed. In a few days, the child developed flank pain and rash. Peripheral blood demonstrated eosinophilia, and urinalysis showed WBC casts. What is the likely diagnosis?

A **Allergic Interstitial Nephritis** — CORRECT

B Pyelonephritis

C Berger Disease

D Urinary Tract Infection

RIGHT ANSWER

OK

A. Allergic Interstitial Nephritis

WHY THIS IS RIGHT

Recent antibiotic exposure followed by flank pain, rash, and eosinophilia is classic for drug-induced allergic interstitial nephritis.
Urinalysis showing WBC casts reflects interstitial inflammation rather than glomerular disease.
Drug exposure (e.g., antibiotics)
↓
Type IV hypersensitivity reaction
↓
Interstitial inflammation + edema
↓
Eosinophil infiltration
↓
Tubular injury
↓
WBC casts in urine
↓
Acute Interstitial Nephritis

Pyelonephritis causes neutrophilia and fever, Berger disease presents with hematuria, and uncomplicated UTI lacks eosinophilia and casts.

Option	Key Features	Why Right/Wrong
A) AIN	Drug + rash + eosinophilia + WBC casts	□ Classic
B) Pyelonephritis	Fever, bacteria, neutrophils	□ No eosinophilia
C) Berger disease (IgA)	Hematuria post-URI	□ No rash/eosinophilia
D) UTI	Dysuria, bacteria	□ No systemic hypersensitivity

QUESTION

According to KDIGO staging of chronic kidney disease (CKD), what is the GFR range for stage 3b?

A 60-90 mL/min/1.73m²

B 15-30 mL/min/1.73m²

C 45-59 mL/min/1.73m²

D **30-44 mL/min/1.73m²** CORRECT

OK RIGHT ANSWER

D. 30-44 mL/min/1.73m²

WHY THIS IS RIGHT

KDIGO classifies CKD stage 3b as an eGFR of 30-44 mL/min/1.73 m².
Stage 3 is subdivided into 3a (45-59) and 3b (30-44) due to differing risks of progression and complications.
Higher values represent earlier stages, while 15-30 corresponds to stage 4.

GFR ranges for CKD staging:
• Stage 1: >90 mL/min/1.73m²
• Stage 2: 60-89 mL/min/1.73m²
• Stage 3a: 45-59 mL/min/1.73m²
• Stage 3b: 30-44 mL/min/1.73m²
• Stage 4: 15-29 mL/min/1.73m²
• Stage 5: <15 mL/min/1.73m²

QUESTION

Which of the following conditions is the most appropriate indication for erythropoietin therapy?

A Aplastic anemia

B Nutritional deficiency anemia

C **Anemia with kidney disease** CORRECT

D Anemia in myelodysplastic syndrome

OK RIGHT ANSWER

C. Anemia with kidney disease

WHY THIS IS RIGHT

Erythropoietin (EPO)
• Hormone produced by peritubular interstitial cells in the kidney
• Stimulates erythropoiesis in bone marrow

Anemia in chronic kidney disease is due to deficient erythropoietin production by damaged renal peritubular cells. Exogenous erythropoietin therapy directly corrects the underlying cause, improving hemoglobin levels.

Condition	Problem	EPO Role	Verdict
Aplastic anemia	Marrow failure	Ineffective	□
Nutritional anemia	Deficiency	Ineffective	□
CKD anemia	↓ EPO	Highly effective	□
MDS	Marrow dysplasia	Limited	□

QUESTION

What is a patient's urine analysis most likely to show if they have hematuria and hypercalciuria?

A **Isomorphic RBCs** — CORRECT

B RBC casts

C Nephrotic range proteinuria

D Eosinophiluria

OK

RIGHT ANSWER

A. Isomorphic RBCs

WHY THIS IS RIGHT

Hematuria with hypercalciuria typically suggests a non-glomerular (urologic) source, such as nephrolithiasis or tubular irritation from calcium crystals. In non-glomerular hematuria, urine microscopy shows isomorphic (normal-shaped) RBCs because the bleeding occurs distal to the glomerulus, so RBCs are not distorted during passage.

In contrast:
• RBC casts indicate glomerular bleeding
• Nephrotic-range proteinuria suggests nephrotic syndrome
• Eosinophiluria suggests acute interstitial nephritis

Thus, hypercalciuria-associated hematuria shows isomorphic RBCs.

QUESTION

A 70-year-old man is hospitalized for acute chest pain and undergoes PCI. His serum creatinine is unchanged 3 days post-procedure, but from day 4 slowly rises to a peak of 2.4 mg/dL. Urinalysis demonstrates no proteinuria or red blood cells, and a few granular casts are present. Serum complement is low. Blood counts show eosinophilia. Which one of the following is likely?

A Allergic interstitial nephritis

B CIN

C **Atheroembolic disease** CORRECT

D RPGN

OK

RIGHT ANSWER

C. Atheroembolic disease

WHY THIS IS RIGHT

Subacute rise in creatinine several days after PCI, with:
• Minimal urinary findings (few granular casts)
• Low complement levels
• Peripheral eosinophilia

is characteristic of atheroembolic renal disease due to cholesterol crystal embolization from disrupted atherosclerotic plaques during vascular procedures. It typically presents with delayed AKI (days to weeks), systemic inflammatory features, and complement consumption.

Why not other options:
A. Allergic interstitial nephritis: Drug-induced; shows fever, rash, eosinophiluria, WBC casts - complements usually normal.
B. Contrast-induced nephropathy: Occurs within 24-72 hrs after contrast, no eosinophilia or low complement.
D. RPGN: Has hematuria, proteinuria, RBC casts and rapid severe renal failure.

QUESTION

38-year-old man is brought to the emergency department due to vomiting blood. After appropriate resuscitation measures, he undergoes upper gastrointestinal endoscopy, which reveals a bleeding duodenal ulcer. During hospital day 2, the patient develops decreased urine output. Serum creatinine rises to 3.0 mg/dL from a baseline of 1.2 mg/dL. Renal biopsy shows patchy epithelial necrosis of the tubules, intratubular casts. Supportive care is provided. Several days later, his urine output significantly increases, and serum creatinine levels decline. Over the next few days, this patient is at highest risk for which of the following complications?

A Hyperphosphatemia

B **Hypokalemia** CORRECT

C Metabolic acidosis

D Volume overload

OK RIGHT ANSWER

B. Hypokalemia

WHY THIS IS RIGHT

This patient developed acute tubular necrosis (ATN) due to hypovolemia from GI bleeding.

ATN progresses through phases:

1. Initiation phase - rise in creatinine
2. Maintenance (oliguric) phase - low urine output
3. Recovery (polyuric) phase - ↑ urine output due to tubular dysfunction

During the polyuric recovery phase, impaired tubular reabsorption leads to excessive loss of electrolytes, especially potassium, placing the patient at risk for hypokalemia (and dehydration). Thus, the major complication during recovery from ATN is hypokalemia.

QUESTION

21-year-old man is brought to the emergency department due to diffuse muscle aches and weakness. He has also noticed darkening of his urine. he patient recently joined the military and was participating in rigorous training exercises in hot weather earlier in the day.
Laboratory results are as follows:
Sodium: 136 mEq/L
Potassium: 5.6 mEq/L
Bicarbonate: 18 mEq/L
Creatinine: 2.0 mg/dL
CK: 22,000 U/L (normal: 30-170)
Which of the following urine microscopy is most likely present in this patient?

A Dysmorphic red blood cells

B Eosinophils

C **Granular casts** CORRECT

D Waxy casts

RIGHT ANSWER

OK

C. Granular casts

WHY THIS IS RIGHT

Severe muscle injury with markedly elevated CK and dark urine after strenuous exercise indicates rhabdomyolysis. Myoglobin released from muscle causes acute tubular necrosis (ATN) and acute kidney injury.

Urine microscopy in ATN shows muddy brown granular casts formed from necrotic tubular epithelial cells and protein debris. Thus, rhabdomyolysis-associated AKI is characterized by granular casts on urine microscopy.

Why not other options:
A. Dysmorphic RBCs: Glomerulonephritis.
B. Eosinophils: Acute interstitial nephritis.
D. Waxy casts: Chronic kidney disease.

QUESTION

71-year-old man has had decreased urine output <500 mL per day for the past 3 days. Physical examination shows vital signs with temperature 37° C, pulse 88/min, respiratory rate 18/min, and blood pressure 85/60 mm Hg. He has peripheral edema and diffuse rales on auscultation of the chest. Urinalysis shows specific gravity 1.019 and no protein, blood, glucose, ketones, WBCs, RBCs, or casts. His serum creatinine is 3.3 mg/dL, and urea nitrogen is 62 mg/dL. The fractional excretion of sodium (FENa) is <1%. Which of the following underlying conditions is he most likely to have?

A **Dilated cardiomyopathy** CORRECT

B Membranous nephropathy

C ATN

D Urothelial carcinoma

OK

RIGHT ANSWER

A. Dilated cardiomyopathy

WHY THIS IS RIGHT

Low urine output, hypotension (85/60 mm Hg), edema, pulmonary rales, elevated BUN/creatinine, and FENa <1% indicate prerenal azotemia due to decreased renal perfusion.

The presence of volume overload signs (edema, rales) with hypotension suggests low cardiac output, most consistent with dilated cardiomyopathy causing congestive heart failure.

In prerenal AKI:
• FENa <1%
• Concentrated urine (normal/high specific gravity)
• No active urinary sediment

Thus, decreased effective arterial blood volume from cardiac failure is the likely cause.

QUESTION

Which of the following medications is incorrect about microalbuminuria?

A Urine protein levels range from 30 mg/d to 300 mg/d

B It is an independent risk factor for cardiovascular morbidity in diabetic patients

C It is the earliest marker of diabetic nephropathy

D **It is detected by routine dipstick method** CORRECT

OK

RIGHT ANSWER

D. It is detected by routine dipstick method

WHY THIS IS RIGHT

Microalbuminuria (30-300 mg/day) is the earliest marker of diabetic nephropathy and an independent risk factor for cardiovascular morbidity and mortality.

It cannot be detected by routine urine dipstick testing because dipsticks detect only overt proteinuria (>300 mg/day).

Detection requires special tests such as urine albumin-creatinine ratio (ACR).

QUESTION

Dialysis will be effective for which of the following?
1. Seizures
2. Metabolic acidosis
3. Peripheral neuropathy
4. Uremic pericarditis
5. Hyperkalemia

A 1, 2, 3, 4, 5

B 2, 3

C **1, 2, 4, 5** CORRECT

D 1, 3, 5

OK RIGHT ANSWER

C. 1, 2, 4, 5

WHY THIS IS RIGHT

Dialysis is effective in treating acute, life-threatening complications of uremia, including:
• Seizures (uremic encephalopathy)
• Severe metabolic acidosis
• Uremic pericarditis
• Refractory hyperkalemia

However, peripheral neuropathy is a chronic complication of uremia and does not improve immediately with dialysis. Thus, dialysis is effective for 1, 2, 4, and 5, but not for chronic uremic neuropathy.

QUESTION

A 74-year-old man with congestive heart failure (CHF) on intravenous furosemide develops acute kidney injury (AKI) with serum creatinine rising from 1.0 to 2.1 mg/dL.
Laboratory findings show:
Urine Na□: 45 mEq/L
Urine creatinine: 55 mg/dL
Which of the following tests best distinguishes prerenal azotemia from acute tubular necrosis (ATN) in the setting of diuretic use?

A Fractional excretion of sodium (FeNa)

B **Fractional excretion of urea (FeUrea)** CORRECT

C Urine eosinophils

D Granular casts on dipstick alone

RIGHT ANSWER

OK **B. Fractional excretion of urea (FeUrea)**

WHY THIS IS RIGHT

In patients receiving loop diuretics (e.g., furosemide), the fractional excretion of sodium (FeNa) becomes unreliable because diuretics increase urinary sodium excretion.
In this setting, fractional excretion of urea (FeUrea) is preferred to distinguish:
· Prerenal azotemia -> FeUrea < 35%
· Acute tubular necrosis (ATN) -> FeUrea > 50%
Urea reabsorption occurs mainly in the proximal tubule and is less affected by diuretics, making FeUrea the best test to differentiate prerenal AKI from ATN when diuretics are used.

QUESTION

All of the following are indications for initiating dialysis in a patient with renal failure, EXCEPT:

A Hyperkalemia >6.5

B Severe metabolic acidosis not responding to medical therapy

C Pulmonary edema unresponsive to diuretics

D **Asymptomatic elevation of serum creatinine to 5 mg/dL** CORRECT

OK RIGHT ANSWER

D. Asymptomatic elevation of serum creatinine to 5 mg/dL

WHY THIS IS RIGHT

Indications for dialysis are based on clinical complications, not serum creatinine level alone.
Classic indications can be remembered by AEIOU:
· Acidosis: severe metabolic acidosis (pH <7.1-7.2) refractory to therapy
· Electrolyte imbalance: refractory hyperkalemia (>6.5 mEq/L)
· Intoxication: certain drug/toxin overdoses
· Overload: pulmonary edema unresponsive to diuretics
· Uremic symptoms: encephalopathy, pericarditis, bleeding
An asymptomatic rise in creatinine alone is not an indication for dialysis.

QUESTION

According to the KDIGO criteria, acute kidney injury (AKI) can be diagnosed based on urine output when it is:

A < 0.5 mL/kg/hour for 4 hours

B **< 0.5 mL/kg/hour for 6 hours** CORRECT

C < 0.3 mL/kg/hour for 12 hours

D Anuria for 48 hours

OK RIGHT ANSWER

B. < 0.5 mL/kg/hour for 6 hours

WHY THIS IS RIGHT

KDIGO AKI definition includes:
· ↑ Serum creatinine ≥0.3 mg/dL in 48 hours, or
· ↑ Serum creatinine ≥1.5× baseline within 7 days, or
· Urine output <0.5 mL/kg/hour for 6 hours
KDIGO Staging of AKI
Stage 1
• Creatinine: 1.5-1.9 × baseline OR ↑ ≥0.3 mg/dL
• Urine output: <0.5 mL/kg/hr for 6-12 hrs

Stage 2
• Creatinine: 2.0-2.9 × baseline
• Urine output: <0.5 mL/kg/hr ≥12 hrs

Stage 3
• Creatinine: ≥3 × baseline OR ≥4 mg/dL OR dialysis initiation

Urine output: <0.3 mL/kg/hr ≥24 hrs OR anuria ≥12 hrs

QUESTION

Peritoneal dialysis is associated with all of the following complications EXCEPT:

A Increased risk of peritonitis

B Poor filtration rate

C Hyperglycemia

D **Weight loss** CORRECT

RIGHT ANSWER

OK

D. Weight loss

WHY THIS IS RIGHT

Peritoneal dialysis (PD) complications include:
· Peritonitis -> most common and serious complication
· Poor filtration / inadequate clearance -> especially in large or long-term patients
· Hyperglycemia -> due to absorption of glucose-containing dialysate
· Weight loss is NOT typical
PD commonly causes weight gain, not loss, due to:
o Glucose absorption
o Increased caloric load
o Fluid retention

QUESTION

A 65-year-old male with a history of hypertension and diabetes, recently diagnosed with end-stage renal disease (stage 5) and on hemodialysis, seeks dietary advice. Which of the following characterizes the ideal feed for him?

A **Low volume, high calories** CORRECT

B High volume, low calories

C High volume, high calories

D Low volume, low calories

RIGHT ANSWER

OK

A. Low volume, high calories

WHY THIS IS RIGHT

In patients with end-stage renal disease (ESRD) on hemodialysis, the ideal diet is low volume and high calorie. Fluid intake must be restricted to prevent volume overload, hypertension, and pulmonary edema, while adequate calories are required to prevent protein-energy malnutrition and maintain nutritional status.
Thus, feeds should be calorie-dense with restricted fluid volume and controlled sodium, potassium, and phosphorus intake.

QUESTION

A woman traveling from Delhi to Leh develops discomfort due to high altitude. What is the underlying physiological mechanism?

A Metabolic acidosis

B Hypoventilation

C **Respiratory alkalosis** CORRECT

D Hypercapnia

OK RIGHT ANSWER

C. Respiratory alkalosis

WHY THIS IS RIGHT

High altitude causes hypobaric hypoxia, which stimulates peripheral chemoreceptors leading to hyperventilation.

Hyperventilation results in excessive CO□ washout, producing respiratory alkalosis.

This acute alkalosis is responsible for early symptoms of altitude sickness.

Hypercapnia and hypoventilation do not occur at high altitude, and metabolic acidosis develops later as compensation.

QUESTION

A male patient presents to the emergency department. The arterial blood gas report is as follows: pH 7.2, pCO2 81mm Hg, and HCO3 40 mEq/L. Which of the following is the most likely diagnosis?

A Respiratory alkalosis

B Metabolic acidosis

C **Respiratory acidosis** CORRECT

D Metabolic alkalosis

OK RIGHT ANSWER

C. Respiratory acidosis

WHY THIS IS RIGHT

Low pH (7.2) indcates acidemia with markedly elevated pCO□ (81 mm Hg) pointing to a primary respiratory process.

Elevated HCO□□ (40 mEq/L) reflects renal compensation, suggesting a chronic or partially compensated state.

Thus, the primary disorder is respiratory acidosis.

Metabolic causes would show primary HCO□□ changes with opposite CO□ response.

QUESTION

A young female is brought to the emergency department. Attendants inform that she has consumed 100 tablets of aspirin. What should be the next step in management?

A N-acetyl cysteine to replenish glutathione stores

B Pralidoxime

C Glucagon to control bradycardia and hypoglycemia

D **Give sodium bicarbonate to alkalize urine** CORRECT

RIGHT ANSWER

OK

D. Give sodium bicarbonate to alkalize urine

WHY THIS IS RIGHT

Acute aspirin (salicylate) poisoning causes respiratory alkalosis with high anion gap metabolic acidosis.

Urine alkalinization with sodium bicarbonate enhances salicylate ionization and renal excretion.

This is the key next step in management in significant salicylate overdose.

Goal	Method
Reduce toxicity	Activated charcoal (early)
Enhance elimination	Urine alkalinization (NaHCO□)
Severe cases	Hemodialysis

N-acetyl cysteine is for paracetamol, pralidoxime for organophosphates, and glucagon for beta-blocker toxicity.

Poison	Antidote
Paracetamol	NAC
Organophosphate	Atropine + Pralidoxime
Beta-blocker	Glucagon
Salicylate	NaHCO□ (urine alkalinization)

QUESTION

A 40-year-old male presents with altered mental status, tachycardia, tachypnea, and hypertension after consuming an unknown substance. On evaluation, he has high anion gap metabolic acidosis and hypocalcemia. Which of the following is likely to be the reason for his presentation?

A Iron

B Methanol

C **Ethylene glycol** CORRECT

D Digoxin

OK RIGHT ANSWER

C. Ethylene glycol

WHY THIS IS RIGHT

Ethylene glycol poisoning causes high anion gap metabolic acidosis due to accumulation of glycolic and oxalic acids.

Oxalic acid chelates calcium, leading to hypocalcemia and calcium oxalate crystal deposition.

This explains altered mental status, tachypnea (Kussmaul breathing), and cardiovascular instability.

Finding	Mechanism
Altered mental status	CNS toxicity
Tachypnea	Compensation for metabolic acidosis
Tachycardia	Stress response
Hypertension	Early sympathetic response
Hypocalcemia	Ca^{2+} bound by oxalate
High AG acidosis	Organic acids accumulate

Methanol causes visual symptoms, iron causes GI hemorrhage and acidosis without hypocalcemia, and digoxin causes arrhythmias without metabolic acidosis.

QUESTION

Which of the following conditions is most likely based on the lab values shown - Blood pH 7.28, Na 138 mEq/L, K 2.8 mEq/L, Cl 113 mEq/L, HCO3 12 mEq/L, BUN 3 mg/dL, and creatinine 0.2 mg/dL. Urinalysis showed a urinary pH of 9. Which of the following is the likely diagnosis?

A Type 4 RTA

B **Type 1 RTA** CORRECT

C Type 2 RTA

D Respiratory acidosis

OK RIGHT ANSWER

B. Type 1 RTA

WHY THIS IS RIGHT

Low pH with low HCO□□ (12 mEq/L) indicates metabolic acidosis with normal renal function.

Marked hyperchloremia, hypokalemia, and inappropriately alkaline urine (pH 9) indicate failure of distal H□ secretion.

Metabolic acidosis
↓
Normal AG -> RTA
↓
Check K□ -> LOW
↓
Check urine pH -> HIGH (>5.5)
↓
Type 1 (Distal) RTA

This is characteristic of distal (Type 1) renal tubular acidosis, which is associated with hypokalemia and high urine pH.

Feature	Type 1 (Distal)	Type 2 (Proximal)	Type 4
Defect	↓ H□ secretion	↓ HCO□□ reabsorption	↓ Aldosterone
Urine pH	\>5\.5 (alkaline)	<5.5 (later)	<5.5
K□	Low	Low	More
Stones	Common	Rare	No
AG	Normal	Normal	Normal

QUESTION

Evaluate the arterial blood gas with the following findings: pH 7.26, PaCO□ - 16 mm Hg, HCO□ - 8, Na□ 135, Cl□ - 108, K□ - 4.2.

A **Metabolic acidosis with compensatory respiratory alkalosis with high anion gap** CORRECT

B Respiratory acidosis with compensatory metabolic alkalosis with low anion gap

C Metabolic alkalosis

D Respiratory alkalosis

OK RIGHT ANSWER

A. Metabolic acidosis with compensatory respiratory alkalosis with high anion gap

WHY THIS IS RIGHT

Low pH with markedly reduced HCO□□ (8 mEq/L) indicates primary metabolic acidosis.

PaCO□ is appropriately low (16 mmHg), showing respiratory compensation.

Anion gap = Na□ - (Cl□ + HCO□□) = 135 - (108 + 8) = 19, confirming high anion gap metabolic acidosis.

Value	Meaning
Normal	8-12
This case: 19	□ High anion gap

Cause	Example
Lactic acidosis	Shock, sepsis
Ketoacidosis	DKA
Renal failure	Uremia
Toxins	Methanol, ethylene glycol

QUESTION

A 55-year-old patient presents with shortness of breath. Arterial blood gas analysis reveals the following- pH: 7.22, PaCO□: 66 mmHg PaO□: 45 mmHg HCO□□: 26 mEq/L. What is the most likely acid-base disorder?

A Metabolic alkalosis

B **Respiratory acidosis** CORRECT

C Respiratory alkalosis

D Metabolic acidosis

RIGHT ANSWER

OK

B. Respiratory acidosis

WHY THIS IS RIGHT

Low pH (7.22) with markedly elevated PaCO□ (66 mmHg) indicates a primary respiratory acidosis.

Near-normal bicarbonate (26 mEq/L) suggests minimal or no metabolic compensation, consistent with an acute process.

Acidemia (↓ pH)
+
↑ PaCO□ (primary abnormality)
+
Normal HCO□□ (no compensation)

Severe hypoxemia further supports hypoventilation as the underlying cause.

QUESTION

An elderly patient presents with increased frequency of breathing and weakness. ABG is given. What is the likely diagnosis?

- **pH = 7.3**
- **pCO□ = 44 mmHg**
- **pO□ = 85 mmHg**
- **HCO□□ = 16 mEq/L**
- **Na□ = 132 mEq/L**
- **Cl□ = 86 mEq/L**
- **Base excess = -12 mEq/L**

A Acute metabolic alkalosis with AG 16 mEq/L

B Acute respiratory acidosis with AG 16 mEq/L

C **Acute metabolic acidosis with AG 30 mEq/L** CORRECT

D Chronic Respiratory acidosis with AG 30 mEq/L

RIGHT ANSWER

OK **C. Acute metabolic acidosis with AG 30 mEq/L**

WHY THIS IS RIGHT

Low pH with markedly reduced HCO□□ (16 mEq/L) indicates primary metabolic acidosis.
Anion gap = Na□ - (Cl□ + HCO□□) = 132 - (86 + 16) = 30 mEq/L, which is elevated.
Near-normal pCO□ reflects early/partial respiratory compensation, confirming acute high anion gap metabolic acidosis.

Parameter	Finding
pH	↓ (acidemia)
HCO□□	↓ -> metabolic cause
pCO□	not appropriately ↓
AG	30 (high)

Acidemia (↓ pH)
↓
Check HCO□□ -> LOW -> Metabolic acidosis
↓
Calculate AG -> HIGH (30)
↓
Check compensation (Winter's formula)
↓
pCO□ higher than expected -> Acute / inadequate compensation

QUESTION

Which of the following crystalloid infusions, when given in large quantities, is likely to cause hyperchloremic metabolic acidosis?

A D5W

B **NS** CORRECT

C RL

D DNS

OK RIGHT ANSWER

B. NS

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

A 60-year-old man with a history of chronic obstructive pulmonary disease (COPD) presents with progressive dyspnea. Arterial blood gas analysis reveals:

- **pH: 7.32**
- **PaCO□: 60 mmHg**
- **HCO□□: 30 mmol/L**

Which of the following represents the primary acid-base disorder in this patient?

A Acute respiratory acidosis

B **Chronic respiratory acidosis** CORRECT

C Metabolic alkalosis

D Metabolic acidosis

OK RIGHT ANSWER

B. Chronic respiratory acidosis

WHY THIS IS RIGHT

Condition	PaCO□ ↑	HCO□□ Change	Mechanism
Acute respiratory acidosis	↑	↑ *slightly* (\~+1 per 10 CO□)	Buffer systems
Chronic respiratory acidosis	↑	↑↑ *more* (\~+4 per 10 CO□)	Renal compensation

Low pH with elevated PaCO□ indicates primary respiratory acidosis.

The elevated bicarbonate (30 mmol/L) reflects renal compensation, which develops over time.

This pattern is typical of chronic respiratory acidosis seen in long-standing COPD.

1\. COPD = classic chronic respiratory acidosis
• “CO□ retainers”
• Often have compensated pH (\~near normal)

2\. Oxygen therapy caution
In COPD:
• High O□ -> ↓ respiratory drive -> ↑ CO□ retention

QUESTION

A 45-year-old man has the following serum electrolyte values during evaluation:

- **Na□: 130 mmol/L**
- **Cl□: 84 mmol/L**
- **HCO□□: 16 mmol/L**

What is the calculated anion gap for this patient?

A 26

B **30** CORRECT

C 18

D 22

OK RIGHT ANSWER

B. 30

WHY THIS IS RIGHT

Anion gap is calculated as Na□ - (Cl□ + HCO□□).
Substituting values: 130 - (84 + 16) = 130 - 100 = 30.
Thus, the calculated anion gap is 30, indicating a high anion gap metabolic acidosis.

Anion Gap	Value	Meaning
Normal	8-12	No excess acids
Mild ↑	12-20	Early acidosis
High ↑	\>20	Accumulation of acids

High Anion Gap-

Letter	Cause
G	Glycols (ethylene glycol, propylene glycol)
O	Oxoproline (chronic paracetamol use)
L	L-lactate (shock, sepsis)
D	D-lactate
M	Methanol
A	Aspirin
R	Renal failure (uremia)
K	Ketoacidosis (DKA, starvation)

QUESTION

A man goes trekking at a high altitude. Due to the physiological response to decreased atmospheric oxygen tension, which of the following primary acid-base disturbances is most likely to develop in this individual?

A Metabolic acidosis

B Metabolic alkalosis

C Respiratory acidosis

D **Respiratory alkalosis** CORRECT

RIGHT ANSWER

OK

D. Respiratory alkalosis

WHY THIS IS RIGHT

At high altitude, reduced atmospheric oxygen tension causes hypoxemia, which stimulates peripheral chemoreceptors. At High Altitude

Parameter	Change
pH	↑ (alkalosis)
pCO□	↓
HCO□□	Initially normal -> later ↓ (renal compensation)

This leads to hyperventilation, resulting in excessive CO□ washout.
The primary acid-base disturbance is therefore respiratory alkalosis.

Phase	Mechanism	ABG
Acute (hours)	Hyperventilation	Respiratory alkalosis
Chronic (days)	Kidney excretes HCO□□	Compensated alkalosis

QUESTION

A 60-year-old with a history of COPD is brought to the ER with worsening dyspnea. ABG was done and pH was 7.3, pCO2 was 60 mmHg with HCO3- being 28 mEq/L. Which of the following is seen in this patient?

A Respiratory acidosis, hyperventilation, inadequate metabolic compensation

B Respiratory alkalosis, hypoventilation, inadequate metabolic compensation

C Respiratory acidosis, hypoventilation, inadequate metabolic compensation

D **Respiratory acidosis, hypoventilation, adequate metabolic compensation** CORRECT

RIGHT ANSWER

OK

D. Respiratory acidosis, hypoventilation, adequate metabolic compensation

WHY THIS IS RIGHT

In COPD:
• Air trapping + ↓ alveolar ventilation
• CO□ cannot be expelled -> hypercapnia
• Leads to respiratory acidosis

Low pH (7.3) with elevated pCO□ (60 mmHg) indicates primary respiratory acidosis due to hypoventilation, typical in COPD exacerbation.

Expected Compensation Rules:

Condition	Expected HCO□□ change
Acute respiratory acidosis	↑ 1 mEq/L per 10 mmHg CO□ rise
Chronic respiratory acidosis	↑ 4 mEq/L per 10 mmHg CO□ rise

Bicarbonate is elevated (28 mEq/L; should be 24 + 2=26meq/l considering acute on chronic COPD changes), showing inadequate metabolic compensation for the degree of hypercapnia.
Thus, this represents acute-on-chronic respiratory acidosis with hypoventilation.

QUESTION

Choose the incorrect statement about Renal Tubular Acidosis (RTA):

A Fanconi syndrome presents with RTA 2

B Bicarbonaturia is a feature of RTA 2

C **RTA 4 is associated with hypokalemia** CORRECT

D RTA 4 is due to aldosterone resistance

OK RIGHT ANSWER

C. RTA 4 is associated with hypokalemia

WHY THIS IS RIGHT

RTA Type	Site	Key defect	Potassium
Type 1 (Distal)	Distal tubule	↓ H□ secretion	↓ K□
Type 2 (Proximal)	PCT	↓ HCO□□ reabsorption	↓ K□
Type 4	Collecting duct	↓ Aldosterone effect	↑ K□

RTA type 4 is due to aldosterone deficiency or resistance and is characteristically associated with hyperkalemia, not hypokalemia.
Fanconi syndrome causes proximal (type 2) RTA with bicarbonaturia.
Hence, the statement that RTA 4 is associated with hypokalemia is incorrect.

Feature	Type 1 (Distal)	Type 2 (Proximal)	Type 4
Defect	↓ H□ secretion	↓ HCO□□ reabsorption	↓ Aldosterone
K□	↓	↓	↑
Urine pH	\>5\.5	Variable	<5.5
Stones	□ Common	Rare	□
Cause	Autoimmune	Fanconi	Diabetes

QUESTION

A patient arrives at the emergency department presenting with symptoms of abdominal pain, vomiting, tachypnea, and tachycardia. Laboratory results are as follows: pH: 7.34, Sodium: 135 mmol/L, Potassium: 5 mmol/L, Bicarbonate: 12 mmol/L, Chloride: 92 mmol/L, RBS: 450 mg/dL. Given these laboratory findings, which of the following statements best describes the patient's acid-base status?

A Non-Anion Gap Metabolic Acidosis (NAGMA)

B **High Anion Gap Metabolic Acidosis (HAGMA)** CORRECT

C Compensated Metabolic Alkalosis

D Respiratory Acidosis

OK

RIGHT ANSWER

B. High Anion Gap Metabolic Acidosis (HAGMA)

WHY THIS IS RIGHT

pH 7.34 with low bicarbonate (12 mmol/L) indicates a primary metabolic acidosis.
Anion gap = Na - (Cl + HCO□) = 135 - (92 + 12) = 31, which is markedly elevated.
Severe hyperglycemia with abdominal pain and tachypnea is classic for diabetic ketoacidosis, a cause of high anion gap metabolic acidosis.

1. anion gap increases:
• Ketone bodies:
• β-hydroxybutyrate
• Acetoacetate

These are unmeasured anions

1. tachypnea occurs because:

Respiratory compensation
• Blows off CO□
• Tries to increase pH

Kussmaul respiration

1. Potassium paradox:
• Serum K□ = 5 (normal/high)
• But:
• Total body potassium = LOW

Due to:
• Insulin deficiency
• Shift out of cells

QUESTION

A 35-year-old male presents to the clinic with complaints of nausea, diarrhea, and vomiting. Upon further investigation, arterial blood gas (ABG) analysis reveals the following: pH: 7.55, PCO2: 50 mmHg, HCO3-: 44 mmol/L. Given these findings, which of the following conditions is the most likely diagnosis?

A Respiratory Acidosis

B Respiratory Alkalosis

C Metabolic Acidosis

D **Metabolic Alkalosis** CORRECT

OK RIGHT ANSWER

D. Metabolic Alkalosis

WHY THIS IS RIGHT

ABG shows alkalemia (pH 7.55) with markedly elevated bicarbonate (HCO□□ 44 mmol/L), indicating a primary metabolic process.
The raised PCO□ (50 mmHg) represents appropriate respiratory compensation via hypoventilation.
• pH = 7.55 -> Alkalemia
• HCO□□ = 44 mmol/L (↑) -> pushes pH up
• PCO□ = 50 mmHg (↑) -> would push pH down (compensation)

HCO□□ = 44 -> (44-24)=20
• 0.7 × 20 = 14
• Expected PCO□ ≈ 54 ± 5 -> 49-59 mmHg

Observed PCO□ = 50 mmHg
Appropriate respiratory compensation

Thus, the diagnosis is metabolic alkalosis, commonly due to gastric acid loss from vomiting

$$PCO_2 \approx 0.7 \times (HCO_3^- - 24) + 40 \pm 5$$

QUESTION

What causes decrease in GFR?

A Decrease in Bowman's hydrostatic pressure

B Increase in glomerular hydrostatic pressure

C **Decrease in glomerular filtration coefficient** CORRECT

D Decrease in glomerular oncotic pressure

RIGHT ANSWER

OK

C. Decrease in glomerular filtration coefficient

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1b” here and does not include a written explanation for this item.

QUESTION

High Anion Gap Metabolic Acidosis (HAGMA) is not caused by:

A **Diarrhea** CORRECT

B Diabetic ketoacidosis (DKA)

C Acute renal failure

D Inborn errors of metabolism

OK RIGHT ANSWER

A. Diarrhea

WHY THIS IS RIGHT

If new acids enter the blood:
• H□ consumes HCO□□
• Unmeasured anions rise

-> Anion gap increases

High anion gap metabolic acidosis results from accumulation of unmeasured acids such as ketones, lactate, or retained uremic toxins.
DKA, acute renal failure, and inborn errors of metabolism all cause HAGMA.
Diarrhea causes normal anion gap (hyperchloremic) metabolic acidosis due to bicarbonate loss.

Feature	HAGMA	NAGMA
Cause	Acid accumulation	HCO□□ loss
Cl□	Normal	↑
Examples	DKA, AKI, toxins	Diarrhea, RTA

$$\text{Anion Gap} = Na^{+} - (Cl^{-} + HCO_3^{-})$$

QUESTION

Which of the following is seen during rapid ascent to high altitude?

A **Respiratory alkalosis** CORRECT

B Respiratory acidosis

C Metabolic alkalosis

D Metabolic acidosis

OK RIGHT ANSWER

A. Respiratory alkalosis

WHY THIS IS RIGHT

High altitude -> ↓ O□ (Hypoxia)
↓
Peripheral chemoreceptor stimulation
↓
Hyperventilation
↓
↓ CO□ (Hypocapnia)
↓
↑ pH
↓
Respiratory Alkalosis
Rapid ascent to high altitude causes hypoxemia, which stimulates peripheral chemoreceptors.
This leads to hyperventilation, causing excessive CO□ washout and resulting in respiratory alkalosis.
Renal compensation with bicarbonate loss occurs later.

Phase	Change
Immediate (hours)	Respiratory alkalosis
After 2-3 days	Renal compensation -> ↓ HCO□□
Long-term	↑ RBC, ↑ 2,3-BPG

QUESTION

The statements below mention about the anion gap in different conditions. Select the true statements.

1. Starvation: Increase
2. Diabetic ketoacidosis: Increase
3. Severe vomiting: Increase

A 1

B **1, and 2** CORRECT

C 2

D 2, and 3

OK RIGHT ANSWER

B. 1, and 2

WHY THIS IS RIGHT

Normal ≈ 8-12 mEq/L
Anion gap increases when unmeasured acids accumulate
These acids donate H□ -> consume HCO□□ -> gap widens
Starvation leads to ketosis with accumulation of organic acids, causing an increased anion gap metabolic acidosis.
Diabetic ketoacidosis is a classic cause of high anion gap metabolic acidosis due to ketone bodies.
Severe vomiting causes metabolic alkalosis with normal anion gap, not an increased anion gap.

Condition	Primary Disorder	Anion Gap	Mechanism
Starvation	Metabolic acidosis	↑	Ketone bodies
DKA	Metabolic acidosis	↑	Ketoacids
Vomiting	Metabolic alkalosis	Normal	Loss of H□ (not acid accumulation)

$$\text{Anion Gap} = Na^{+} - (Cl^{-} + HCO_3^{-})$$

QUESTION

PTH is increased in:

A Immobility

B **Familial hypocalciuric hypercalcemia** CORRECT

C Vitamin D toxicity

D Thiazide diuretics

RIGHT ANSWER

OK

B. Familial hypocalciuric hypercalcemia

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1c” here and does not include a written explanation for this item.

QUESTION

Which of the following is the most common regulatory mechanism of magnesium?

A Dietary intake

B Intestinal absorption

C **Renal reabsorption** CORRECT

D Bone resorption

RIGHT ANSWER

OK

C. Renal reabsorption

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1d” here and does not include a written explanation for this item.

QUESTION

Identify the acid base disorder in a patient with the following values: pH- 7.2 and pCO2 - 80 mm Hg?

A Metabolic acidosis

B Metabolic alkalosis

C **Respiratory acidosis** CORRECT

D Respiratory alkalosis

RIGHT ANSWER

OK

C. Respiratory acidosis

WHY THIS IS RIGHT

Interpretation of acid-base disorders is based on pH and pCO□:
• Normal pH: 7.35-7.45
• Normal pCO□: 35-45 mm Hg

In this case:
• pH = 7.2 -> Acidemia
• pCO□ = 80 mm Hg -> Markedly elevated (hypercapnia)

An increased pCO□ indicates CO□ retention, which shifts the equilibrium toward carbonic acid formation, lowering pH. Common causes of respiratory acidosis: Hypoventilation (e.g., COPD, airway obstruction, CNS depression)

Why other options are incorrect:
• Metabolic acidosis: ↓ pH with ↓ HCO□□ (not given; pCO□ usually compensatorily low)
• Metabolic alkalosis: ↑ pH with ↑ HCO□□
• Respiratory alkalosis: ↑ pH with ↓ pCO□

QUESTION

A 32-year-old man is brought to the emergency department due to progressive confusion and lethargy over the past several hours. The patient has no significant medical history but he has been depressed since a recent breakup with his girlfriend. He works at an automotive repair shop, and one of his coworkers reported seeing the patient consume antifreeze prior to symptom onset. Blood pressure is 110/66 mm Hg, pulse is 110/min, and respirations are 24/min. Bilateral costovertebral angles are tender to percussion. Bladder catheterization yields a small amount of bloody urine. Which of the following is most likely to be seen on this patient's arterial blood gas analysis?

A **pH 7.3, pO2 95, pCO2 24, HCO3 19** CORRECT

B pH 7.1, pO2 47, pCO2 45, HCO3 19

C pH 7.6, pO2 95, pCO2 18, HCO3 40

D pH 7.4, pO2 95, pCO2 24, HCO3 29

RIGHT ANSWER

OK **A. pH 7.3, pO2 95, pCO2 24, HCO3 19**

WHY THIS IS RIGHT

Ethylene glycol (antifreeze) poisoning causes high anion gap metabolic acidosis due to metabolism into glycolic and oxalic acids. This leads to:
• ↓ pH (acidemia)
• ↓ HCO□□ (metabolic acidosis)
• Compensatory ↓ pCO□ from hyperventilation
• Normal PaO□

Oxalate crystal deposition in kidneys causes flank pain, hematuria, and acute kidney injury.
Thus, ABG shows metabolic acidosis with respiratory compensation.

QUESTION

A 54-year-old man is brought to the emergency department by his wife because of high fever and confusion for the past 10 hours. His wife reports that 1 week ago he underwent an emergency appendectomy. His temperature is 40.1°C (104.2°F), pulse is 132/min, and blood pressure is 74/46 mm Hg. He is oriented only to person. Physical examination shows a surgical wound in the right lower quadrant with purulent discharge. The skin is warm and dry. Serum studies show a sodium concentration of 138 mEq/L, potassium concentration of 3.7 mEq/L, and lactate concentration of 3.5 mEq/L (N = 0.5-2.2 mEq/L). Arterial blood gas analysis on room air shows:
Which of the following is the most likely explanation for these laboratory changes?

pH	7.21
pCO_2	36
HCO_3^-	12
O_2 Saturation	87%

A Hyperventilation

B Primary adrenal insufficiency

C Salicylate toxicity

D **Respiratory fatigue** CORRECT

RIGHT ANSWER

OK

D. Respiratory fatigue

WHY THIS IS RIGHT

This patient has septic shock (fever, hypotension, purulent wound, elevated lactate) leading to lactic acidosis -> metabolic acidosis (pH 7.21, HCO□□ 12).

Appropriate compensation for metabolic acidosis should cause low pCO□ (hyperventilation). However, pCO□ is 36 mmHg (inadequately low), indicating superimposed respiratory acidosis due to respiratory fatigue.

Thus, failure of appropriate respiratory compensation in severe sepsis results in mixed metabolic and respiratory acidosis, most consistent with respiratory fatigue.

QUESTION

A 4-week-old, full-term boy is brought to the emergency department due to vomiting. His parents describe the emesis as undigested formula without blood or bile. The vomiting occurs after feeds and has increased in frequency and force over the past 3 days. Examination shows a sunken anterior fontanelle and dry mucous membranes. Arterial blood gas analysis is most likely to reveal which of the following sets of values?

A pH 7.29, PaCo2 30, HCO3 14, Anion gap Elevated

B pH 7.30, PaCo2 50, HCO3 28, Anion gap normal

C **pH 7.48, PaCo2 46, HCO3 34, Anion gap normal** CORRECT

D pH 7.53, PaCo2 22, HCO3 22, Anion gap normal

OK RIGHT ANSWER

C. pH 7.48, PaCo2 46, HCO3 34, Anion gap normal

WHY THIS IS RIGHT

Projectile, non-bilious vomiting in a 4-week-old infant suggests hypertrophic pyloric stenosis.

Loss of gastric HCl leads to metabolic alkalosis with:
• ↑ pH
• ↑ HCO□□
• Compensatory ↑ PaCO□ (respiratory hypoventilation)
• Normal anion gap
• Often associated with hypokalemia and hypochloremia

Thus, persistent vomiting of gastric contents causes hypochloremic metabolic alkalosis with respiratory compensation.

QUESTION

Interpret the following arterial blood gas (ABG) values:
pH: 7.20
PaO□: 69 mmHg
PaCO□: 30 mmHg
HCO□□: 15 mEq/L
What is the most appropriate interpretation?

A **Metabolic acidosis with compensatory respiratory alkalosis** CORRECT

B Metabolic acidosis with additional respiratory acidosis

C Metabolic acidosis with additional respiratory alkalosis

D Respiratory alkalosis with compensatory metabolic acidosis

OK RIGHT ANSWER

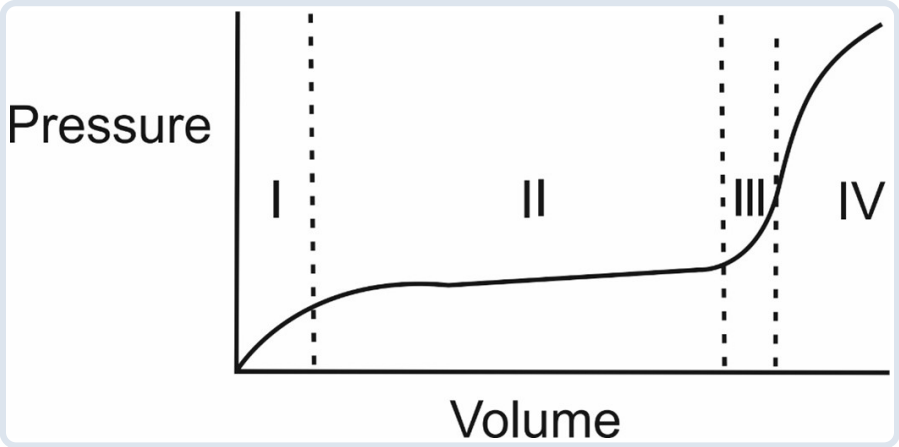
A. Metabolic acidosis with compensatory respiratory alkalosis

WHY THIS IS RIGHT

Stepwise ABG interpretation:
· pH 7.20 -> acidemia
· HCO□□ 15 (low) -> primary metabolic acidosis
· PaCO□ 30 (low) -> lungs are blowing off CO□ -> respiratory compensation
There is no evidence of an additional respiratory disorder. Since PaCO□ is reduced in the appropriate direction for compensation, the correct interpretation is Metabolic acidosis with compensatory respiratory alkalosis.
Low PaO□ (69 mmHg) indicates mild hypoxemia but does not change the primary acid-base diagnosis. Expected PaCO□ by Winter's formula:
(1.5 × HCO□□) + 8 ± 2 = 30.5 ± 2, which matches the measured PaCO□ (30)

QUESTION

Which of the following statements is true regarding the given cystometrogram?



A Segment Ia is due to residual urine

B **Segment II is due to Laplace law** CORRECT

C Micturition fails to happen in segment II

D The dotted line represents that micturition has occurred

OK RIGHT ANSWER

B. Segment II is due to Laplace law

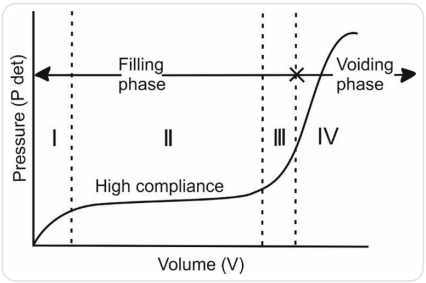
WHY THIS IS RIGHT

Segment II represents the high-compliance phase of the bladder, where volume increases with minimal rise in pressure.

This occurs due to Laplace law-as bladder radius increases, wall tension is distributed, limiting pressure rise.

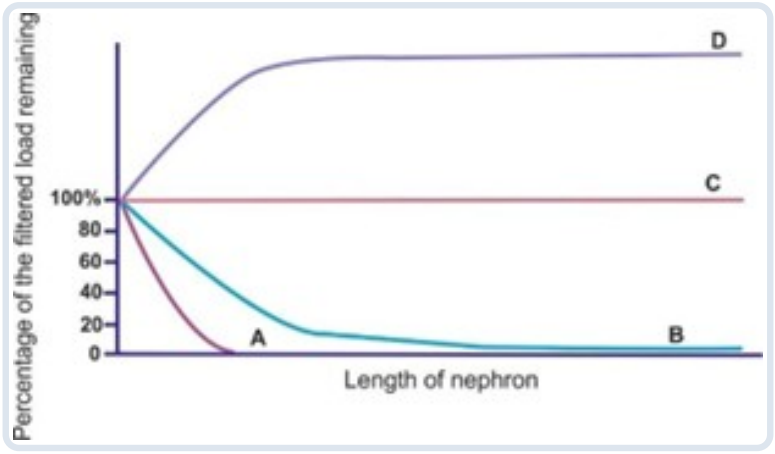
Micturition reflex is initiated later (segment III-IV), not in segment II.

Dotted lines indicate threshold points, not completion of micturition.



QUESTION

Identify the correctly matched pair of substances with their renal clearance from the graph given below?



A. A - Glucose, B - PAH, C - Bicarbonate and D - Inulin

B. **A - Glucose, B - Bicarbonate, C - Inulin and D - PAH** CORRECT

C. A - PAH, B - Inulin, C - Glucose and D - Bicarbonate

D. A - Inulin, B - Glucose, C - Bicarbonate and D - PAH

OK RIGHT ANSWER

B. A - Glucose, B - Bicarbonate, C - Inulin and D - PAH

WHY THIS IS RIGHT

Glucose shows zero clearance as it is freely filtered and completely reabsorbed under normal plasma levels.

Bicarbonate has low but positive clearance due to near-complete reabsorption with regulated secretion.

Inulin clearance equals GFR as it is freely filtered with no reabsorption or secretion.

PAH has the highest clearance as it is filtered and actively secreted, approximating renal plasma flow.

Substance	Handling	Clearance	Key Concept
Glucose	Fully reabsorbed	0	Tm limited
Bicarbonate	Mostly reabsorbed	Low	pH regulation
Inulin	Filtered only	= GFR	Gold standard
PAH	Filtered + secreted	Highest	RPF marker

QUESTION

A patient has the following Starling forces at a capillary bed:

• Capillary hydrostatic pressure (Pc): 18 mmHg
• Capillary oncotic pressure (πc): 27 mmHg
• Interstitial oncotic pressure (πi): 7 mmHg

If there is no net filtration across the capillary, what is the value of the interstitial hydrostatic pressure (Pi)?

A 0 mmHg

B +1 mmHg

C +2 mmHg

D **-2 mmHg** CORRECT

RIGHT ANSWER

OK

D. -2 mmHg

WHY THIS IS RIGHT

The Starling equation for net filtration pressure (NFP) is: NFP = (Pc - Pi) - (πc - πi)
Where:
• Pc = capillary hydrostatic pressure (pushes fluid OUT)
• Pi = interstitial hydrostatic pressure (pushes fluid IN)
• πc = capillary oncotic pressure (pulls fluid IN)
• πi = interstitial oncotic pressure (pulls fluid OUT)
• | Force | Direction | Function |
| ----- | --------- | -------- |
| Pc | Outward | Filtration |
| Pi | Inward (if positive) | Opposes filtration |
| πc | Inward | Reabsorption |
| πi | Outward | Filtration |

Net filtration pressure (NFP) = (Pc - Pi) - (πc - πi).
Given no net filtration, NFP = 0 => (18 - Pi) - (27 - 7) = 0.
So, 18 - Pi - 20 = 0 => Pi = -2 mmHg, making option D correct.

QUESTION

All of the following Decreases GFR except:

A Decreased glomerular coefficient Kf

B Increase glomerular oncotic pressure

C Reduced Glomerular Hydrostatic pressure

D **Increased Bowman Oncotic Pressure** CORRECT

RIGHT ANSWER

OK

D. Increased Bowman Oncotic Pressure

WHY THIS IS RIGHT

GFR decreases with reduced Kf, increased glomerular oncotic pressure, and reduced glomerular hydrostatic pressure. Bowman's space normally has negligible oncotic pressure because proteins do not filter across the glomerulus. Therefore, increased Bowman oncotic pressure does not occur physiologically and does not decrease GFR.

Option	Effect on GFR	Mechanism	Verdict
A	↓	↓ filtration capacity	□
B	↓	Opposes filtration	□
C	↓	↓ driving pressure	□
D	↑	Pulls fluid into Bowman space	□

$$GFR = K_f \times [(P_{GC} - P_{BS}) - (\pi_{GC} - \pi_{BS})]$$

QUESTION

Which of the following is not a mechanism by which antidiuretic hormone (ADH) facilitates the excretion of concentrated urine?

A Increase absorption of urea in the medullary collecting duct

B **Increase absorption of urea in the descending loop of Henle** CORRECT

C Decrease absorption of NaCl in the ascending loop of Henle

D Increase water permeability in the collecting ducts

OK RIGHT ANSWER

B. Increase absorption of urea in the descending loop of Henle

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

Which of the following best explains the development of hyponatremia in a hypovolemic patient presenting with diarrhea?

A Decreased reabsorption of sodium from kidney

B Decreased secretion of aldosterone

C **Non-osmotic release of antidiuretic hormone (ADH)** CORRECT

D Decreased sodium absorption from the intestine

RIGHT ANSWER

OK **C. Non-osmotic release of antidiuretic hormone (ADH)**

WHY THIS IS RIGHT

• Diarrhea -> hypovolemia
• Loss of:
• Na□
• Water

Effective circulating volume (ECV) \> Osmolality
Even if plasma osmolality is low or normal:
ADH is released via non-osmotic stimulus
• Triggered by:
• ↓ blood volume
• ↓ blood pressure

ADH:
• ↑ Water reabsorption (collecting duct)
• No proportional Na□ retention

Result:
• Dilutional hyponatremia

Hormonal response in hypovolemia:

System	Effect
ADH	↑ Water reabsorption
RAAS	↑ Na□ reabsorption
Sympathetic	↑ Renal vasoconstriction

Type	Volume Status	Mechanism
Hypovolemic	↓ volume	ADH due to volume loss
Euvolemic (SIADH)	Normal	ADH excess
Hypervolemic	↑ volume	CHF, cirrhosis

QUESTION

A 30-year-old sexually active woman presented with dysuria, lower abdominal pain, and increased urinary frequency. Urine culture was done and it grew Staphylococcus saprophyticus with a colony count of 10^4 CFU/mL. What is the management?

A **It is a diagnosis of UTI** — CORRECT

B Sample contaminated. Take 2/3 more spontaneous fresh samples

C Reassure no treatment required

D Urgent CT scan

OK RIGHT ANSWER

A. It is a diagnosis of UTI

WHY THIS IS RIGHT

In symptomatic women, a colony count of ≥10^3-10^4 CFU/mL is sufficient to diagnose urinary tract infection.

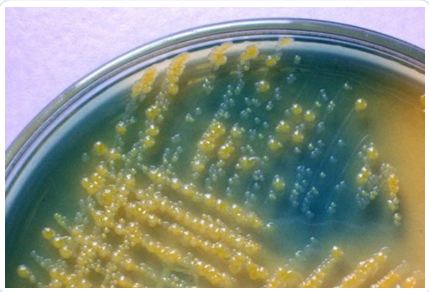
Staphylococcus saprophyticus is a common cause of UTI in sexually active young women.

Thus, this represents a true UTI requiring treatment, not contamination.

Situation	Significant bacteriuria
Symptomatic woman	≥10^3-10^4 CFU/mL □□
Asymptomatic	≥10^5 CFU/mL
Catheterized sample	≥10^2 CFU/mL

Mixed culture of fermenting (yellow colonies) and non-fermenting (blue colonies) bacteria on CLED agar.

Organism	Scenario
E. coli	Most common overall
Staph. saprophyticus	Young sexually active women
Klebsiella	Hospital / catheter
Proteus	Stones, alkaline urine



QUESTION

A 25-year-old male presented to the emergency department with head trauma due to a road traffic accident. In the hospital, the patient developed seizures. An emergency CT scan revealed widespread cerebral edema. Which of the following is the diuretic of choice for cerebral edema in this patient?

A **Mannitol** CORRECT

B Spironolactone

C Furosemide

D Hydrochlorothiazide

OK RIGHT ANSWER

A. Mannitol

WHY THIS IS RIGHT

Mannitol is the diuretic of choice for acute cerebral edema as it acts as an osmotic agent, drawing water out of brain parenchyma into the intravascular space.
IV Mannitol
↓
↑ Plasma osmolality
↓
Water shifts from brain -> blood
↓
↓ Brain volume
↓
↓ Intracranial pressure (ICP)

This rapidly reduces intracranial pressure and cerebral swelling in head injury.

Other diuretics do not provide rapid osmotic dehydration of brain tissue.

Drug	Mechanism	Role in Cerebral Edema
Mannitol	Osmotic	□ Drug of choice
Furosemide	Loop	Adjunct only
Spironolactone	Aldosterone antagonist	□ No role
Thiazide	DCT	□ No role

QUESTION

A patient presents with hypertension. He has a history of renal stones and has experienced a few episodes of renal colic. Which diuretic is the most appropriate to use?

A Furosemide

B **Hydrochlorothiazide** CORRECT

C Ethacrynic acid

D Spironolactone

OK RIGHT ANSWER

B. Hydrochlorothiazide

WHY THIS IS RIGHT

Hydrochlorothiazide reduces urinary calcium excretion by increasing distal tubular calcium reabsorption. This helps prevent recurrent calcium renal stones while effectively treating hypertension.
Thiazide
↓
↓ NaCl reabsorption (DCT)
↓
↑ Ca²□ reabsorption (via Na□/Ca²□ exchanger)
↓
↓ Urinary Ca²□ (hypocalciuria)
↓
↓ Calcium stone formation

Loop diuretics increase calcium excretion, and spironolactone has no role in stone prevention.

Diuretic	Site	Effect on Ca²□	Stone Risk
Thiazide	DCT	↓ urinary Ca²□	□ Prevents stones
Loop (Furosemide)	TAL	↑ urinary Ca²□	□ Promotes stones
K□-sparing	Collecting duct	Neutral	No effect

Problem	Drug Benefit
Hypertension	Thiazide lowers BP
Recurrent Ca stones	↓ urinary Ca²□

QUESTION

A patient presented with a renal stone after a Proteus infection. What is the likely composition of the stone in him?

A Calcium phosphate

B **Magnesium ammonium phosphate** CORRECT

C Xanthine

D Cysteine

OK RIGHT ANSWER

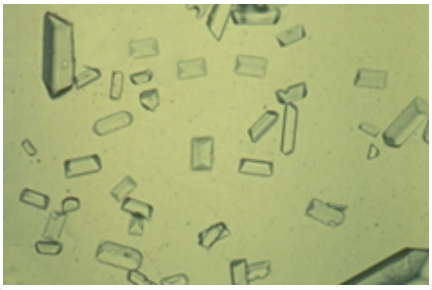
B. Magnesium ammonium phosphate

WHY THIS IS RIGHT

Urea
↓ (urease)
Ammonia (NH_3) + CO_2
↓
$NH_3 + H_2O \rightarrow NH_4^+ + OH^-$
↓
Urine becomes ALKALINE (↑ pH)

Proteus species are urease-producing bacteria that alkalinize urine by splitting urea into ammonia.
Alkaline urine favors formation of struvite stones, composed of magnesium ammonium phosphate.

Stone Type	Composition	Urine pH	Cause
Struvite	$MgNH_4PO_4$	Alkaline	Proteus, Klebsiella
Calcium oxalate	CaC_2O_4	Acidic/neutral	Most common
Calcium phosphate	$CaPO_4$	Alkaline	RTA type 1
Uric acid	Uric acid	Acidic	Gout
Cystine	Cystine	Acidic	Genetic defect



QUESTION

A 45-year-old man presents with flank pain, and imaging confirms the presence of a renal calculus. He was recently started on hydrochlorothiazide. What is the likely mechanism by which thiazides can cause this adverse effect?

A Increased excretion of calcium

B Decreased excretion of calcium

C Increased absorption of oxalate

D **Decreased excretion of citrate** CORRECT

RIGHT ANSWER

OK

D. Decreased excretion of citrate

WHY THIS IS RIGHT

Citrate is a natural stone inhibitor-↓ urinary citrate = stones (especially Ca stones), and thiazide-induced alkalosis promotes citrate reabsorption.

Role of Citrate	Effect
Binds Ca^{2}□	Prevents Ca stone formation
Inhibits crystallization	Stops nucleation/aggregation

Thiazide diuretics can cause hypocitraturia by increasing proximal tubular citrate reabsorption. Citrate normally inhibits calcium stone formation by binding calcium in urine.

Reduced urinary citrate therefore predisposes to renal calculi, explaining this adverse effect.

Effect	Direction	Impact on Stones
Ca^{2}□ excretion	↓	Protective
Citrate excretion	↓	Promotes stones
Volume	↓	Concentrates urine

QUESTION

First part of PCT has which of the following transport proteins?

1. Sodium potassium pump
2. Na- glucose co-transporter
3. Sodium hydrogen antiport
4. Na+ Bicarbonate cotransporter

A 1, 4

B **1, 2, 3** CORRECT

C 1, 3, 4

D 2, 3

OK RIGHT ANSWER

B. 1, 2, 3

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

A 55-year-old patient with type 2 diabetes mellitus is being evaluated for medications to manage their condition and prevent renal complications. Which of the following drugs will not delay progression of renal disease?

A SGLT2 inhibitors

B Finerenone

C **Hydrochlorothiazide** CORRECT

D Angiotensin receptor blockers

RIGHT ANSWER

OK

C. Hydrochlorothiazide

WHY THIS IS RIGHT

Drug	Mechanism	Renal Protection
SGLT2 inhibitors	↓ hyperfiltration	□ Strong
Finerenone	↓ fibrosis/inflammation	□ Strong
Hydrochlorothiazide	↓ BP only	□ None
ARBs	↓ RAAS activity	□ Strong

Hydrochlorothiazide is an antihypertensive diuretic but has no proven renoprotective effect in diabetic kidney disease.

SGLT2 inhibitors, finerenone, and ARBs reduce intraglomerular pressure and albuminuria, thereby slowing CKD progression.

Hence, hydrochlorothiazide does not delay progression of renal disease.

QUESTION

A 58-year-old male presents with complaints of burning micturition and increased frequency. Prostatic examination is normal. Urinalysis shows >50 pus cells per high power field, but urine culture shows no growth. What is the most likely diagnosis?

A **Chronic bacterial prostatitis** — CORRECT

B Acute bacterial prostatitis

C Chronic Granulomatous prostatitis

D Sterile pyuria

RIGHT ANSWER

OK

A. Chronic bacterial prostatitis

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1b” here and does not include a written explanation for this item.

QUESTION

Which of the following is correct about Finerenone?

1. It causes less hyperkalemia than spironolactone
2. It has a cardioprotective effect
3. It gets accumulated in the kidneys
4. It causes a significant reduction in blood pressure

A 1, 2 and 3

B **1 and 2** CORRECT

C 2, 3 and 4

D 1, 2 and 4

OK RIGHT ANSWER

B. 1 and 2

WHY THIS IS RIGHT

Solution - Finerenone:
• It is a non-steroidal mineralocorticoid receptor antagonist (MRA)
• Used mainly in:
• Chronic kidney disease (CKD) with diabetes
• Cardiovascular protection

Statement 3: Gets accumulated in kidneys
• Opposite is true
• Finerenone has Even distribution (heart + kidney)
• Unlike spironolactone -> heart accumulation
• Unlike eplerenone-> kidney accumulation

Statement 4: Significant BP reduction
• Only mild antihypertensive effect
• Not used as a primary antihypertensive

|
Feature	Finerenone	Spironolactone
Type	Non-steroidal MRA	Steroidal MRA
Tissue distribution	Balanced (heart + kidney)	Kidney predominant
Hyperkalemia	Less	More
Anti-androgen effects	None	Gynecomastia
Cardioprotection	Strong	Moderate
BP reduction	Mild	Moderate
CKD use	Preferred	Limited

QUESTION

An elderly man presents with progressive swelling of the legs and frothy urine. Physical examination reveals bilateral pitting pedal edema. Urinalysis shows 4+ proteinuria and fatty casts. A renal biopsy is performed, and Congo red staining reveals apple-green birefringence under polarized light. Which of the following is the most likely underlying mechanism of his renal disease?

A Immune complex deposition due to membranous glomerulopathy

B Mesangial proliferation due to IgA deposition

C **Extracellular deposition of misfolded proteins in amyloidosis** CORRECT

D Autoantibody-mediated injury to podocytes

OK RIGHT ANSWER

C. Extracellular deposition of misfolded proteins in amyloidosis

WHY THIS IS RIGHT

Apple-green birefringence on Congo red staining is diagnostic of amyloidosis.

Renal amyloidosis causes nephrotic syndrome with heavy proteinuria, fatty casts, and edema. The underlying mechanism is extracellular deposition of misfolded amyloid proteins in the glomeruli.

Misfolded proteins (β-pleated sheets)
↓
Resistant to degradation
↓
Extracellular deposition in glomeruli
↓
Disrupts filtration barrier
↓
Massive proteinuria (nephrotic syndrome)

Type	Protein	Cause
AL	Light chains	Multiple myeloma
AA	Serum amyloid A	Chronic inflammation

Immune complex, IgA deposition, and podocyte injury describe other glomerulopathies, not amyloidosis.

QUESTION

A woman presents to you with fever, arthralgia, ulcers, fatigue for the past six months, and new-onset hematuria. Urine examination reveals RBC casts and proteinuria. What is the likely diagnosis?

A Acute interstitial nephritis

B Poststreptococcal glomerulonephritis

C **Lupus nephritis** CORRECT

D IgA nephropathy

RIGHT ANSWER

OK

C. Lupus nephritis

WHY THIS IS RIGHT

Chronic systemic symptoms with fever, arthralgia, and ulcers suggest systemic lupus erythematosus.
Autoantibodies (anti-dsDNA)
↓
Immune complex formation
↓
Deposition in glomerulus
↓
Complement activation
↓
Inflammation & injury
↓
Hematuria + proteinuria

Renal involvement with hematuria, proteinuria, and RBC casts indicates immune complex-mediated glomerulonephritis.
• “Full house” IF (IgG, IgA, IgM, C3, C1q)
• Wire loop lesions
• Immune complex deposition everywhere

This constellation is characteristic of lupus nephritis.

PSGN is post-infectious and acute, IgA nephropathy is episodic post-URI, and AIN lacks RBC casts.

Disease	Key Feature	Clue
Lupus nephritis	RBC casts + systemic symptoms	SLE
PSGN	Cola urine + infection	Child
IgA nephropathy	Hematuria after URTI	Episodic
AIN	Eosinophils	Drug-induced

QUESTION

A 10-year-old child presented with cola colored urine and had a history of skin infection few days back. His creatinine value was initially 2.5 mg/dl but even after 3 weeks of treatment it increased to 4.5 mg/dl. Which of the following is a likely kidney biopsy finding on electron microscopy?

A Subendothelial deposits

B Subepithelial deposits

C **Crescents in glomeruli** CORRECT

D Mesangial deposits

RIGHT ANSWER

OK

C. Crescents in glomeruli

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1c” here and does not include a written explanation for this item.

QUESTION

A 50-year-old patient has malaise for 2 weeks. His investigations revealed an elevated creatinine of 4.5mg/dl. His blood tests also showed the presence of HBs antigen. A renal biopsy was performed which demonstrated spike and dome on special staining. Which of the following conditions is the patient likely to be suffering from?

A Minimal change disease

B **Membranous glomerulopathy** CORRECT

C Focal segmental glomerulosclerosis

D Renal amyloidosis

OK RIGHT ANSWER

B. Membranous glomerulopathy

WHY THIS IS RIGHT

Hepatitis B infection
↓
Immune complex formation
↓
Deposition in subepithelial region (GBM)
↓
Complement activation (C5b-9)
↓
Podocyte injury
↓
Proteinuria (nephrotic syndrome)

Membranous glomerulopathy shows characteristic “spike and dome” appearance on silver stain due to subepithelial immune complex deposition.

It is classically associated with HBsAg positivity in secondary membranous nephropathy.

Patients present with nephrotic syndrome and elevated creatinine in adults.

Minimal change lacks spikes, FSGS shows segmental sclerosis, and amyloidosis shows Congo red positivity.

Disease	Key Feature	IF Pattern	Association
Membranous	Spike & dome	Granular	HBV, malignancy
Minimal change	Podocyte effacement	Negative	Children
FSGS	Segmental sclerosis	IgM/C3	HIV
Amyloidosis	Congo red +	Amorphous deposits	Chronic disease

QUESTION

A 47-year-old male presents with 400 ml urine output in 24 hours. On histology he had crescents with pauci immune glomerulonephritis. What is the main etiology?

A Anti GBM antibody

B **ANCA** CORRECT

C Anti phospholipase A2 receptor antibody

D Anti nculear antibody

OK RIGHT ANSWER

B. ANCA

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1d” here and does not include a written explanation for this item.

QUESTION

Which of the following bands will increase in serum electrophoresis in Nephrotic syndrome?

A Gamma globulin

B **Alpha 2 globulin** CORRECT

C Beta globulin

D Albumin

RIGHT ANSWER

OK **B. Alpha 2 globulin**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1e” here and does not include a written explanation for this item.

QUESTION

Which of the following findings are typically associated with glomerular hematuria?

A. Dysmorphic RBCs
B. Dysuria absent
C. Crystals in urine
D. Edema is rare

A **A, B** CORRECT

B A only

C A, B and C

D A, B, D

OK RIGHT ANSWER

A. A, B

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1f” here and does not include a written explanation for this item.

QUESTION

Which of the following renal histological features is characteristically seen in diabetic nephropathy but not typically seen in other glomerulonephritis?

A **Kimmelstiel-Wilson** CORRECT

B Mesangial sclerosis

C GBM thickening

D Foot process effacement

OK RIGHT ANSWER

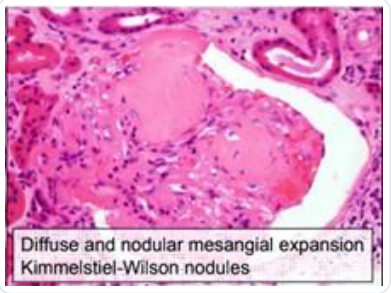
A. Kimmelstiel-Wilson

WHY THIS IS RIGHT

The “signature lesion”: Kimmelstiel-Wilson nodules
• Nodular, PAS-positive mesangial expansion
• Located in glomerular tuft
• Strongly associated with long-standing diabetes mellitus

These are pathognomonic of diabetic nephropathy, representing nodular mesangial expansion.
Mesangial sclerosis and GBM thickening can be seen in other chronic glomerulopathies, and foot process effacement is nonspecific.
Thus, nodular glomerulosclerosis uniquely points to diabetic nephropathy.

Feature	Description
GBM thickening	Early change
Mesangial expansion	Diffuse
K-W nodules	Nodular, pathognomonic
Hyaline arteriolosclerosis	Both afferent & efferent (important!)
Proteinuria	Nephrotic range



QUESTION

A child presents with periorbital oedema and pedal oedema. The laboratory investigations reveal serum creatinine = 2.1 mg/dL, urinary protein: 5.2 g/day, and Cholesterol: 310 mg/dL. Which of the following are included in the criteria of diagnosis of Nephrotic syndrome?

1. Proteinuria >3.5 g/day
2. Hyperlipidemia
3. Creatinine more than 1.5mg/dl
4. Hypoalbuminaemia

A 1 and 2

B **1, 2 and 4** CORRECT

C 1, 3 and 4

D 2, 3 and 4

RIGHT ANSWER

OK **B. 1, 2 and 4**

WHY THIS IS RIGHT

Core Diagnostic Criteria of Nephrotic Syndrome

Criterion	Mechanism
Proteinuria >3.5 g/day	Increased glomerular permeability
Hypoalbuminemia	Loss of albumin in urine
Hyperlipidemia	Liver compensatory synthesis
Edema	↓ oncotic pressure + Na retention

Nephrotic syndrome is defined by massive proteinuria (>3.5 g/day) leading to hypoalbuminemia, which in turn causes edema and secondary hyperlipidemia.
Serum creatinine is not part of the diagnostic criteria and may be normal or elevated depending on renal function.

Statement	Included in Criteria?	Explanation
1\. Proteinuria \>3\.5 g/day	□	Hallmark feature
2\. Hyperlipidemia	□	Liver compensation
3\. Creatinine	□	Reflects renal function, not definition
4\. Hypoalbuminemia	□	Due to urinary loss

QUESTION

A 5-year-old child with 4+ proteinuria is unresponsive to corticosteroid therapy. There is no history of hematuria and hypertension. Renal biopsy shows segmental sclerosis and hyalinosis, predominantly involving the juxtamedullary glomeruli. What is the most likely diagnosis?

A IgA nephropathy

B **Focal Segmental Glomerulosclerosis (FSGS)** CORRECT

C Membranous nephropathy

D Minimal change disease

OK RIGHT ANSWER

B. Focal Segmental Glomerulosclerosis (FSGS)

WHY THIS IS RIGHT

The source quiz contains the placeholder “$20” here and does not include a written explanation for this item.

QUESTION

A 45-year-old male with septic shock and acute kidney injury (AKI) is hemodynamically unstable. Which of the following is the most appropriate modality of dialysis in this situation?

A Intermittent Hemodialysis (IHD)

B Extended Daily Dialysis (EDD)

C **Continuous Renal Replacement Therapy (CRRT)** CORRECT

D Peritoneal Dialysis (PD)

RIGHT ANSWER

OK **C. Continuous Renal Replacement Therapy (CRRT)**

WHY THIS IS RIGHT

CRRT (24-hour continuous therapy)
↓
Slow solute removal
↓
Gradual fluid removal
↓
Minimal BP fluctuations
↓
Safe in unstable patients

Correct answer is C because hemodynamically unstable patients cannot tolerate rapid fluid and solute shifts seen with intermittent dialysis.

Continuous renal replacement therapy provides slow, steady fluid removal and solute clearance, maintaining cardiovascular stability.

IHD and EDD can worsen hypotension. Peritoneal dialysis is less effective in severe septic AKI.

Feature	CRRT	IHD	PD
Duration	Continuous	3-4 hrs	Continuous
Fluid removal	Slow	Rapid	Slow
Hemodynamic effect	Stable □□	Unstable □	Stable
ICU use	Best	Limited	Limited
Solute clearance	Moderate	High	Low

QUESTION

A patient with hyperkalemia and elevated urea levels underwent dialysis. Towards the end of the session, she became drowsy and had a sudden seizure episode. On examination, the patient was hypotensive. What is the treatment for this condition?

A Bumetanide

B Ethacrynic acid

C Nesiritide

D **Mannitol** CORRECT

RIGHT ANSWER

OK

D. Mannitol

WHY THIS IS RIGHT

This presentation is dialysis disequilibrium syndrome, caused by rapid urea removal leading to cerebral edema.

Neurological symptoms (drowsiness, seizures) with hypotension occur towards the end of dialysis.

Mannitol is an osmotic agent that reduces cerebral edema and is the treatment of choice.

Mannitol infusion
↓
↑ Plasma osmolality
↓
Water moves OUT of brain
↓
↓ Cerebral edema
↓
Relieves symptoms

Complication	Cause	Feature
DDS	Rapid urea removal	CNS symptoms
Hypotension	Fluid removal	Dizziness
Diselectrolytemia	Rapid shifts	Arrhythmias
Infection	Access site	Fever

Loop diuretics and nesiritide have no role in acute DDS management.

QUESTION

A patient with CKD with refractory hypertension is brought to the casualty with dizziness. His BP was found to be 200/120 mmHg. His BUN was 50 mg/dl and creatinine was 3 mg/dl. Potassium was 6 mEq/L. Which of the following is an indication of emergency hemodialysis?

A **Severe acidosis with elevated BUN and creatinine** CORRECT

B Hypertension

C Potassium level of 6 mEq/L

D Metabolic alkalosis

OK RIGHT ANSWER

A. Severe acidosis with elevated BUN and creatinine

WHY THIS IS RIGHT

Emergency Hemodialysis-

Letter	Indication	Explanation
A	Acidosis	Severe metabolic acidosis (pH < 7.1) not responding to treatment
E	Electrolytes	Severe hyperkalemia (usually >6.5 mEq/L or with ECG changes)
I	Intoxication	Dialyzable toxins (methanol, lithium, etc.)
O	Overload	Fluid overload refractory to diuretics
U	Uremia	Symptoms like encephalopathy, pericarditis

Emergency hemodialysis is indicated in CKD patients with severe metabolic acidosis associated with azotemia, reflecting life-threatening internal milieu disturbance.

Hypertension alone is managed medically, and K□ = 6 mEq/L without ECG changes is not an absolute indication.

QUESTION

A 10-year-old child presented with follicular tonsillitis, for which antibiotics were prescribed. In a few days, the child developed flank pain and rash. Peripheral blood demonstrated eosinophilia, and urinalysis showed WBC casts. What is the likely diagnosis?

A **Allergic Interstitial Nephritis** CORRECT

B Pyelonephritis

C Berger Disease

D Urinary Tract Infection

OK RIGHT ANSWER

A. Allergic Interstitial Nephritis

WHY THIS IS RIGHT

Recent antibiotic exposure followed by flank pain, rash, and eosinophilia is classic for drug-induced allergic interstitial nephritis.
Urinalysis showing WBC casts reflects interstitial inflammation rather than glomerular disease.
Drug exposure (e.g., antibiotics)
↓
Type IV hypersensitivity reaction
↓
Interstitial inflammation + edema
↓
Eosinophil infiltration
↓
Tubular injury
↓
WBC casts in urine
↓
Acute Interstitial Nephritis

Pyelonephritis causes neutrophilia and fever, Berger disease presents with hematuria, and uncomplicated UTI lacks eosinophilia and casts.

Option	Key Features	Why Right/Wrong
A) AIN	Drug + rash + eosinophilia + WBC casts	□ Classic
B) Pyelonephritis	Fever, bacteria, neutrophils	□ No eosinophilia
C) Berger disease (IgA)	Hematuria post-URI	□ No rash/eosinophilia
D) UTI	Dysuria, bacteria	□ No systemic hypersensitivity

QUESTION

According to KDIGO staging of chronic kidney disease (CKD), what is the GFR range for stage 3b?

A 60-90 mL/min/1.73m²

B 15-30 mL/min/1.73m²

C 45-59 mL/min/1.73m²

D **30-44 mL/min/1.73m²** CORRECT

OK RIGHT ANSWER

D. 30-44 mL/min/1.73m²

WHY THIS IS RIGHT

KDIGO classifies CKD stage 3b as an eGFR of 30-44 mL/min/1.73 m².
Stage 3 is subdivided into 3a (45-59) and 3b (30-44) due to differing risks of progression and complications.
Higher values represent earlier stages, while 15-30 corresponds to stage 4.

GFR ranges for CKD staging:
• Stage 1: >90 mL/min/1.73m²
• Stage 2: 60-89 mL/min/1.73m²
• Stage 3a: 45-59 mL/min/1.73m²
• Stage 3b: 30-44 mL/min/1.73m²
• Stage 4: 15-29 mL/min/1.73m²
• Stage 5: <15 mL/min/1.73m²

QUESTION

Which of the following conditions is the most appropriate indication for erythropoietin therapy?

A Aplastic anemia

B Nutritional deficiency anemia

C **Anemia with kidney disease** CORRECT

D Anemia in myelodysplastic syndrome

RIGHT ANSWER

OK

C. Anemia with kidney disease

WHY THIS IS RIGHT

Erythropoietin (EPO)
• Hormone produced by peritubular interstitial cells in the kidney
• Stimulates erythropoiesis in bone marrow

Anemia in chronic kidney disease is due to deficient erythropoietin production by damaged renal peritubular cells. Exogenous erythropoietin therapy directly corrects the underlying cause, improving hemoglobin levels.

Condition	Problem	EPO Role	Verdict
Aplastic anemia	Marrow failure	Ineffective	□
Nutritional anemia	Deficiency	Ineffective	□
CKD anemia	↓ EPO	Highly effective	□
MDS	Marrow dysplasia	Limited	□

QUESTION

A woman traveling from Delhi to Leh develops discomfort due to high altitude. What is the underlying physiological mechanism?

A Metabolic acidosis

B Hypoventilation

C **Respiratory alkalosis** CORRECT

D Hypercapnia

RIGHT ANSWER

OK

C. Respiratory alkalosis

WHY THIS IS RIGHT

High altitude causes hypobaric hypoxia, which stimulates peripheral chemoreceptors leading to hyperventilation.

Hyperventilation results in excessive CO_2 washout, producing respiratory alkalosis.

This acute alkalosis is responsible for early symptoms of altitude sickness.

Hypercapnia and hypoventilation do not occur at high altitude, and metabolic acidosis develops later as compensation.

QUESTION

A male patient presents to the emergency department. The arterial blood gas report is as follows: pH 7.2, pCO2 81mm Hg, and HCO3 40 mEq/L. Which of the following is the most likely diagnosis?

A Respiratory alkalosis

B Metabolic acidosis

C **Respiratory acidosis** CORRECT

D Metabolic alkalosis

RIGHT ANSWER

OK

C. Respiratory acidosis

WHY THIS IS RIGHT

Low pH (7.2) indcates acidemia with markedly elevated pCO□ (81 mm Hg) pointing to a primary respiratory process.

Elevated HCO□□ (40 mEq/L) reflects renal compensation, suggesting a chronic or partially compensated state.

Thus, the primary disorder is respiratory acidosis.

Metabolic causes would show primary HCO□□ changes with opposite CO□ response.

QUESTION

A young female is brought to the emergency department. Attendants inform that she has consumed 100 tablets of aspirin. What should be the next step in management?

A N-acetyl cysteine to replenish glutathione stores

B Pralidoxime

C Glucagon to control bradycardia and hypoglycemia

D **Give sodium bicarbonate to alkalize urine** CORRECT

RIGHT ANSWER

OK

D. Give sodium bicarbonate to alkalize urine

WHY THIS IS RIGHT

Acute aspirin (salicylate) poisoning causes respiratory alkalosis with high anion gap metabolic acidosis.

Urine alkalinization with sodium bicarbonate enhances salicylate ionization and renal excretion.

This is the key next step in management in significant salicylate overdose.

Goal	Method
Reduce toxicity	Activated charcoal (early)
Enhance elimination	Urine alkalinization (NaHCO□)
Severe cases	Hemodialysis

N-acetyl cysteine is for paracetamol, pralidoxime for organophosphates, and glucagon for beta-blocker toxicity.

Poison	Antidote
Paracetamol	NAC
Organophosphate	Atropine + Pralidoxime
Beta-blocker	Glucagon
Salicylate	NaHCO□ (urine alkalinization)

QUESTION

A 40-year-old male presents with altered mental status, tachycardia, tachypnea, and hypertension after consuming an unknown substance. On evaluation, he has high anion gap metabolic acidosis and hypocalcemia. Which of the following is likely to be the reason for his presentation?

A Iron

B Methanol

C **Ethylene glycol** CORRECT

D Digoxin

OK RIGHT ANSWER

C. Ethylene glycol

WHY THIS IS RIGHT

Ethylene glycol poisoning causes high anion gap metabolic acidosis due to accumulation of glycolic and oxalic acids.

Oxalic acid chelates calcium, leading to hypocalcemia and calcium oxalate crystal deposition.

This explains altered mental status, tachypnea (Kussmaul breathing), and cardiovascular instability.

Finding	Mechanism
Altered mental status	CNS toxicity
Tachypnea	Compensation for metabolic acidosis
Tachycardia	Stress response
Hypertension	Early sympathetic response
Hypocalcemia	Ca^{2+} bound by oxalate
High AG acidosis	Organic acids accumulate

Methanol causes visual symptoms, iron causes GI hemorrhage and acidosis without hypocalcemia, and digoxin causes arrhythmias without metabolic acidosis.

QUESTION

Which of the following conditions is most likely based on the lab values shown - Blood pH 7.28, Na 138 mEq/L, K 2.8 mEq/L, Cl 113 mEq/L, HCO3 12 mEq/L, BUN 3 mg/dL, and creatinine 0.2 mg/dL. Urinalysis showed a urinary pH of 9. Which of the following is the likely diagnosis?

A Type 4 RTA

B **Type 1 RTA** CORRECT

C Type 2 RTA

D Respiratory acidosis

OK RIGHT ANSWER

B. Type 1 RTA

WHY THIS IS RIGHT

Low pH with low HCO□□ (12 mEq/L) indicates metabolic acidosis with normal renal function.

Marked hyperchloremia, hypokalemia, and inappropriately alkaline urine (pH 9) indicate failure of distal H□ secretion.

Metabolic acidosis
↓
Normal AG -> RTA
↓
Check K□ -> LOW
↓
Check urine pH -> HIGH (>5.5)
↓
Type 1 (Distal) RTA

This is characteristic of distal (Type 1) renal tubular acidosis, which is associated with hypokalemia and high urine pH.

Feature	Type 1 (Distal)	Type 2 (Proximal)	Type 4
Defect	↓ H□ secretion	↓ HCO□□ reabsorption	↓ Aldosterone
Urine pH	\>5\.5 (alkaline)	<5.5 (later)	<5.5
K□	Low	Low	More
Stones	Common	Rare	No
AG	Normal	Normal	Normal

QUESTION

Evaluate the arterial blood gas with the following findings: pH 7.26, PaCO□ - 16 mm Hg, HCO□ - 8, Na□ 135, Cl□ - 108, K□ - 4.2.

A **Metabolic acidosis with compensatory respiratory alkalosis with high anion gap** CORRECT

B Respiratory acidosis with compensatory metabolic alkalosis with low anion gap

C Metabolic alkalosis

D Respiratory alkalosis

OK RIGHT ANSWER

A. Metabolic acidosis with compensatory respiratory alkalosis with high anion gap

WHY THIS IS RIGHT

Low pH with markedly reduced HCO□□ (8 mEq/L) indicates primary metabolic acidosis.

PaCO□ is appropriately low (16 mmHg), showing respiratory compensation.

Anion gap = Na□ - (Cl□ + HCO□□) = 135 - (108 + 8) = 19, confirming high anion gap metabolic acidosis.

Value	Meaning
Normal	8-12
This case: 19	□ High anion gap

Cause	Example
Lactic acidosis	Shock, sepsis
Ketoacidosis	DKA
Renal failure	Uremia
Toxins	Methanol, ethylene glycol

QUESTION

A 55-year-old patient presents with shortness of breath. Arterial blood gas analysis reveals the following- pH: 7.22, PaCO□: 66 mmHg PaO□: 45 mmHg HCO□□: 26 mEq/L. What is the most likely acid-base disorder?

A Metabolic alkalosis

B **Respiratory acidosis** CORRECT

C Respiratory alkalosis

D Metabolic acidosis

RIGHT ANSWER

OK **B. Respiratory acidosis**

WHY THIS IS RIGHT

Low pH (7.22) with markedly elevated PaCO□ (66 mmHg) indicates a primary respiratory acidosis.

Near-normal bicarbonate (26 mEq/L) suggests minimal or no metabolic compensation, consistent with an acute process.

Acidemia (↓ pH)
+
↑ PaCO□ (primary abnormality)
+
Normal HCO□□ (no compensation)

Severe hypoxemia further supports hypoventilation as the underlying cause.

QUESTION

An elderly patient presents with increased frequency of breathing and weakness. ABG is given. What is the likely diagnosis?

- **pH = 7.3**
- **pCO□ = 44 mmHg**
- **pO□ = 85 mmHg**
- **HCO□□ = 16 mEq/L**
- **Na□ = 132 mEq/L**
- **Cl□ = 86 mEq/L**
- **Base excess = -12 mEq/L**

A Acute metabolic alkalosis with AG 16 mEq/L

B Acute respiratory acidosis with AG 16 mEq/L

C **Acute metabolic acidosis with AG 30 mEq/L** CORRECT

D Chronic Respiratory acidosis with AG 30 mEq/L

RIGHT ANSWER

OK **C. Acute metabolic acidosis with AG 30 mEq/L**

WHY THIS IS RIGHT

Low pH with markedly reduced HCO□□ (16 mEq/L) indicates primary metabolic acidosis.
Anion gap = Na□ - (Cl□ + HCO□□) = 132 - (86 + 16) = 30 mEq/L, which is elevated.
Near-normal pCO□ reflects early/partial respiratory compensation, confirming acute high anion gap metabolic acidosis.

Parameter	Finding
pH	↓ (acidemia)
HCO□□	↓ -> metabolic cause
pCO□	not appropriately ↓
AG	30 (high)

Acidemia (↓ pH)
↓
Check HCO□□ -> LOW -> Metabolic acidosis
↓
Calculate AG -> HIGH (30)
↓
Check compensation (Winter's formula)
↓
pCO□ higher than expected -> Acute / inadequate compensation

QUESTION

Which of the following crystalloid infusions, when given in large quantities, is likely to cause hyperchloremic metabolic acidosis?

A D5W

B **NS** CORRECT

C RL

D DNS

OK RIGHT ANSWER

B. NS

WHY THIS IS RIGHT

The source quiz contains the placeholder “$21” here and does not include a written explanation for this item.

QUESTION

A 60-year-old man with a history of chronic obstructive pulmonary disease (COPD) presents with progressive dyspnea. Arterial blood gas analysis reveals:

• pH: 7.32
• PaCO□: 60 mmHg
• HCO□□: 30 mmol/L

Which of the following represents the primary acid-base disorder in this patient?

A Acute respiratory acidosis

B **Chronic respiratory acidosis** CORRECT

C Metabolic alkalosis

D Metabolic acidosis

OK RIGHT ANSWER

B. Chronic respiratory acidosis

WHY THIS IS RIGHT

Condition	PaCO□ ↑	HCO□□ Change	Mechanism
Acute respiratory acidosis	↑	↑ *slightly* (\~+1 per 10 CO□)	Buffer systems
Chronic respiratory acidosis	↑	↑↑ *more* (\~+4 per 10 CO□)	Renal compensation

Low pH with elevated PaCO□ indicates primary respiratory acidosis.

The elevated bicarbonate (30 mmol/L) reflects renal compensation, which develops over time.

This pattern is typical of chronic respiratory acidosis seen in long-standing COPD.

1\. COPD = classic chronic respiratory acidosis
• “CO□ retainers”
• Often have compensated pH (\~near normal)

2\. Oxygen therapy caution
In COPD:
• High O□ -> ↓ respiratory drive -> ↑ CO□ retention

QUESTION

A 45-year-old man has the following serum electrolyte values during evaluation:

- **Na□: 130 mmol/L**
- **Cl□: 84 mmol/L**
- **HCO□□: 16 mmol/L**

What is the calculated anion gap for this patient?

A 26

B **30** CORRECT

C 18

D 22

OK RIGHT ANSWER

B. 30

WHY THIS IS RIGHT

Anion gap is calculated as Na□ - (Cl□ + HCO□□).
Substituting values: 130 - (84 + 16) = 130 - 100 = 30.
Thus, the calculated anion gap is 30, indicating a high anion gap metabolic acidosis.

Anion Gap	Value	Meaning
Normal	8-12	No excess acids
Mild ↑	12-20	Early acidosis
High ↑	\>20	Accumulation of acids

High Anion Gap-

Letter	Cause
G	Glycols (ethylene glycol, propylene glycol)
O	Oxoproline (chronic paracetamol use)
L	L-lactate (shock, sepsis)
D	D-lactate
M	Methanol
A	Aspirin
R	Renal failure (uremia)
K	Ketoacidosis (DKA, starvation)

QUESTION

A man goes trekking at a high altitude. Due to the physiological response to decreased atmospheric oxygen tension, which of the following primary acid-base disturbances is most likely to develop in this individual?

A Metabolic acidosis

B Metabolic alkalosis

C Respiratory acidosis

D **Respiratory alkalosis** CORRECT

OK RIGHT ANSWER

D. Respiratory alkalosis

WHY THIS IS RIGHT

At high altitude, reduced atmospheric oxygen tension causes hypoxemia, which stimulates peripheral chemoreceptors. At High Altitude

Parameter	Change
pH	↑ (alkalosis)
pCO□	↓
HCO□□	Initially normal -> later ↓ (renal compensation)

This leads to hyperventilation, resulting in excessive CO□ washout.
The primary acid-base disturbance is therefore respiratory alkalosis.

Phase	Mechanism	ABG
Acute (hours)	Hyperventilation	Respiratory alkalosis
Chronic (days)	Kidney excretes HCO□□	Compensated alkalosis

QUESTION

A 60-year-old with a history of COPD is brought to the ER with worsening dyspnea. ABG was done and pH was 7.3, pCO2 was 60 mmHg with HCO3- being 28 mEq/L. Which of the following is seen in this patient?

A Respiratory acidosis, hyperventilation, inadequate metabolic compensation

B Respiratory alkalosis, hypoventilation, inadequate metabolic compensation

C Respiratory acidosis, hypoventilation, inadequate metabolic compensation

D **Respiratory acidosis, hypoventilation, adequate metabolic compensation** CORRECT

RIGHT ANSWER

OK

D. Respiratory acidosis, hypoventilation, adequate metabolic compensation

WHY THIS IS RIGHT

In COPD:
• Air trapping + ↓ alveolar ventilation
• CO□ cannot be expelled -> hypercapnia
• Leads to respiratory acidosis

Low pH (7.3) with elevated pCO□ (60 mmHg) indicates primary respiratory acidosis due to hypoventilation, typical in COPD exacerbation.

Expected Compensation Rules:

Condition	Expected HCO□□ change
Acute respiratory acidosis	↑ 1 mEq/L per 10 mmHg CO□ rise
Chronic respiratory acidosis	↑ 4 mEq/L per 10 mmHg CO□ rise

Bicarbonate is elevated (28 mEq/L; should be 24 + 2=26meq/l considering acute on chronic COPD changes), showing inadequate metabolic compensation for the degree of hypercapnia.
Thus, this represents acute-on-chronic respiratory acidosis with hypoventilation.

QUESTION

Choose the incorrect statement about Renal Tubular Acidosis (RTA):

A Fanconi syndrome presents with RTA 2

B Bicarbonaturia is a feature of RTA 2

C **RTA 4 is associated with hypokalemia** CORRECT

D RTA 4 is due to aldosterone resistance

RIGHT ANSWER

OK

C. RTA 4 is associated with hypokalemia

WHY THIS IS RIGHT

RTA Type	Site	Key defect	Potassium
Type 1 (Distal)	Distal tubule	↓ H□ secretion	↓ K□
Type 2 (Proximal)	PCT	↓ HCO□□ reabsorption	↓ K□
Type 4	Collecting duct	↓ Aldosterone effect	↑ K□

RTA type 4 is due to aldosterone deficiency or resistance and is characteristically associated with hyperkalemia, not hypokalemia.
Fanconi syndrome causes proximal (type 2) RTA with bicarbonaturia.
Hence, the statement that RTA 4 is associated with hypokalemia is incorrect.

Feature	Type 1 (Distal)	Type 2 (Proximal)	Type 4
Defect	↓ H□ secretion	↓ HCO□□ reabsorption	↓ Aldosterone
K□	↓	↓	↑
Urine pH	\>5\.5	Variable	<5.5
Stones	□ Common	Rare	□
Cause	Autoimmune	Fanconi	Diabetes

QUESTION

A patient arrives at the emergency department presenting with symptoms of abdominal pain, vomiting, tachypnea, and tachycardia. Laboratory results are as follows: pH: 7.34, Sodium: 135 mmol/L, Potassium: 5 mmol/L, Bicarbonate: 12 mmol/L, Chloride: 92 mmol/L, RBS: 450 mg/dL. Given these laboratory findings, which of the following statements best describes the patient's acid-base status?

A Non-Anion Gap Metabolic Acidosis (NAGMA)

B **High Anion Gap Metabolic Acidosis (HAGMA)** CORRECT

C Compensated Metabolic Alkalosis

D Respiratory Acidosis

OK RIGHT ANSWER

B. High Anion Gap Metabolic Acidosis (HAGMA)

WHY THIS IS RIGHT

pH 7.34 with low bicarbonate (12 mmol/L) indicates a primary metabolic acidosis.
Anion gap = Na - (Cl + HCO□) = 135 - (92 + 12) = 31, which is markedly elevated.
Severe hyperglycemia with abdominal pain and tachypnea is classic for diabetic ketoacidosis, a cause of high anion gap metabolic acidosis.

1. anion gap increases:
• Ketone bodies:
• β-hydroxybutyrate
• Acetoacetate

These are unmeasured anions

1. tachypnea occurs because:

Respiratory compensation
• Blows off CO□
• Tries to increase pH

Kussmaul respiration

1. Potassium paradox:
• Serum K□ = 5 (normal/high)
• But:
• Total body potassium = LOW

Due to:
• Insulin deficiency
• Shift out of cells

QUESTION

A 35-year-old male presents to the clinic with complaints of nausea, diarrhea, and vomiting. Upon further investigation, arterial blood gas (ABG) analysis reveals the following: pH: 7.55, PCO2: 50 mmHg, HCO3-: 44 mmol/L. Given these findings, which of the following conditions is the most likely diagnosis?

A Respiratory Acidosis

B Respiratory Alkalosis

C Metabolic Acidosis

D **Metabolic Alkalosis** CORRECT

RIGHT ANSWER

OK **D. Metabolic Alkalosis**

WHY THIS IS RIGHT

ABG shows alkalemia (pH 7.55) with markedly elevated bicarbonate (HCO□□ 44 mmol/L), indicating a primary metabolic process.
The raised PCO□ (50 mmHg) represents appropriate respiratory compensation via hypoventilation.
• pH = 7.55 -> Alkalemia
• HCO□□ = 44 mmol/L (↑) -> pushes pH up
• PCO□ = 50 mmHg (↑) -> would push pH down (compensation)

HCO□□ = 44 -> (44-24)=20
• 0.7 × 20 = 14
• Expected PCO□ ≈ 54 ± 5 -> 49-59 mmHg

Observed PCO□ = 50 mmHg
Appropriate respiratory compensation

Thus, the diagnosis is metabolic alkalosis, commonly due to gastric acid loss from vomiting

$$PCO_2 \approx 0.7 \times (HCO_3^- - 24) + 40 \pm 5$$

QUESTION

What causes decrease in GFR?

A Decrease in Bowman's hydrostatic pressure

B Increase in glomerular hydrostatic pressure

C **Decrease in glomerular filtration coefficient** CORRECT

D Decrease in glomerular oncotic pressure

RIGHT ANSWER

OK

C. Decrease in glomerular filtration coefficient

WHY THIS IS RIGHT

The source quiz contains the placeholder “$22” here and does not include a written explanation for this item.

QUESTION

High Anion Gap Metabolic Acidosis (HAGMA) is not caused by:

A Diarrhea — CORRECT

B Diabetic ketoacidosis (DKA)

C Acute renal failure

D Inborn errors of metabolism

RIGHT ANSWER

OK

A. Diarrhea

WHY THIS IS RIGHT

If new acids enter the blood:
• H□ consumes HCO□□
• Unmeasured anions rise

-> Anion gap increases

High anion gap metabolic acidosis results from accumulation of unmeasured acids such as ketones, lactate, or retained uremic toxins.
DKA, acute renal failure, and inborn errors of metabolism all cause HAGMA.
Diarrhea causes normal anion gap (hyperchloremic) metabolic acidosis due to bicarbonate loss.

Feature	HAGMA	NAGMA
Cause	Acid accumulation	HCO□□ loss
Cl□	Normal	↑
Examples	DKA, AKI, toxins	Diarrhea, RTA

$$\text{Anion Gap} = Na^{+} - (Cl^{-} + HCO_3^{-})$$

QUESTION

Which of the following is seen during rapid ascent to high altitude?

A **Respiratory alkalosis** CORRECT

B Respiratory acidosis

C Metabolic alkalosis

D Metabolic acidosis

RIGHT ANSWER

OK **A. Respiratory alkalosis**

WHY THIS IS RIGHT

High altitude -> ↓ O□ (Hypoxia)
↓
Peripheral chemoreceptor stimulation
↓
Hyperventilation
↓
↓ CO□ (Hypocapnia)
↓
↑ pH
↓
Respiratory Alkalosis
Rapid ascent to high altitude causes hypoxemia, which stimulates peripheral chemoreceptors.
This leads to hyperventilation, causing excessive CO□ washout and resulting in respiratory alkalosis.
Renal compensation with bicarbonate loss occurs later.

Phase	Change
Immediate (hours)	Respiratory alkalosis
After 2-3 days	Renal compensation -> ↓ HCO□□
Long-term	↑ RBC, ↑ 2,3-BPG

QUESTION

The statements below mention about the anion gap in different conditions. Select the true statements.

1. Starvation: Increase
2. Diabetic ketoacidosis: Increase
3. Severe vomiting: Increase

A 1

B **1, and 2** CORRECT

C 2

D 2, and 3

OK RIGHT ANSWER

B. 1, and 2

WHY THIS IS RIGHT

Normal ≈ 8-12 mEq/L
Anion gap increases when unmeasured acids accumulate
These acids donate H□ -> consume HCO□□ -> gap widens
Starvation leads to ketosis with accumulation of organic acids, causing an increased anion gap metabolic acidosis.
Diabetic ketoacidosis is a classic cause of high anion gap metabolic acidosis due to ketone bodies.
Severe vomiting causes metabolic alkalosis with normal anion gap, not an increased anion gap.

Condition	Primary Disorder	Anion Gap	Mechanism
Starvation	Metabolic acidosis	↑	Ketone bodies
DKA	Metabolic acidosis	↑	Ketoacids
Vomiting	Metabolic alkalosis	Normal	Loss of H□ (not acid accumulation)

$$\text{Anion Gap} = Na^{+} - (Cl^{-} + HCO_3^{-})$$

QUESTION

PTH is increased in:

A Immobility

B **Familial hypocalciuric hypercalcemia** CORRECT

C Vitamin D toxicity

D Thiazide diuretics

RIGHT ANSWER

OK **B. Familial hypocalciuric hypercalcemia**

WHY THIS IS RIGHT

The source quiz contains the placeholder “$19” here and does not include a written explanation for this item.

QUESTION

Which of the following is the most common regulatory mechanism of magnesium?

A Dietary intake

B Intestinal absorption

C **Renal reabsorption** CORRECT

D Bone resorption

RIGHT ANSWER

OK

C. Renal reabsorption

WHY THIS IS RIGHT

The source quiz contains the placeholder “$1a” here and does not include a written explanation for this item.

QUESTION

Identify the acid base disorder in a patient with the following values: pH- 7.2 and pCO2 - 80 mm Hg?

A Metabolic acidosis

B Metabolic alkalosis

C **Respiratory acidosis** CORRECT

D Respiratory alkalosis

OK RIGHT ANSWER

C. Respiratory acidosis

WHY THIS IS RIGHT

Interpretation of acid-base disorders is based on pH and pCO□:
• Normal pH: 7.35-7.45
• Normal pCO□: 35-45 mm Hg

In this case:
• pH = 7.2 -> Acidemia
• pCO□ = 80 mm Hg -> Markedly elevated (hypercapnia)

An increased pCO□ indicates CO□ retention, which shifts the equilibrium toward carbonic acid formation, lowering pH. Common causes of respiratory acidosis: Hypoventilation (e.g., COPD, airway obstruction, CNS depression)

Why other options are incorrect:
• Metabolic acidosis: ↓ pH with ↓ HCO□□ (not given; pCO□ usually compensatorily low)
• Metabolic alkalosis: ↑ pH with ↑ HCO□□
• Respiratory alkalosis: ↑ pH with ↓ pCO□