



NBME EXPLANATIONS

NBME 21 Block 3

from CrackNBME

21-Block 3

Q1

A 28-year-old man develops a temperature of 39.9°C (103.8°F) and rigors after receiving high-dose chemotherapy for acute leukemia. Laboratory studies show:

Hemoglobin	8.8 g/dL
Leukocyte count	400/mm ³
Mean corpuscular volume	88 μm ³
Platelet count	60,000/mm ³

After appropriate cultures are done, antibiotic therapy is started. Administration of which of the following is most appropriate at this time?

- A. Erythropoietin
- B. Granulocyte colony-stimulating factor
- C. Interferon-γ
- D. Interleukin-8 (IL-8)
- E. Thrombopoietin

▼ Explanation

- Answer is B (G-CSF)

- IMP Clues:

- Temperature 39.9 & rigors (infection or febrile reaction?)
 - Hx of receiving chemotherapy for acute leukemia (chemotherapy will kill the neutrophils so he will probably be neutropenic)
 - Labs show Anemia, Thrombocytopenia, and neutropenia (okay bro is in pancytopenia)
 - Culture & antibiotics have been started (so infection confirmed)

- Dx → Febrile Neutropenia

- Q is asking what should be given next?
 - Given this patient's blood work, his neutrophils are **EXTREMELY** low.
 - He has an infection and was given antibiotics, so it would make sense to augment his immune system so that he can fight the infection. (If we don't, he is risking progression to *sepsis and death*)
 - **Granulocyte-Colony Stimulating Factor is what is given to stimulate granulocytes:**
 - It **shortens the cell cycle** of late granulocyte precursors (myeloblasts, promyelocytes, myelocytes) and **triggers release** of juvenile and mature neutrophils from bone marrow sinusoids to the bloodstream.
 - **Classically given to patients with neutropenic fever following cytotoxic chemotherapy** (like this patient here) **to reduce duration & severity**

? What second messenger does G-CSF use?

▼ Answer

Non-Receptor Tyrosine Kinase ⇒
Acidophils, Cytokines, AND Colony Stimulating Factors

- Examples are **GH, Prolactin, Interleukins & Interferons, EPO, TPO, and G-CSF**

▼ Other Choices + HY Related Notes

- **Choice A** → while EPO can be given, to treat the anemia, it is not our first priority here & this patient's Hb level is not that low anyways (WBCs are critical = priority)

On the USMLE, EPO is assessed in 5 ways:

1. Where is EPO (**a glycoprotein**) made?
 - **The kidney**
2. MOA of EPO?
 - **JAK-STAT (Non-Receptor Tyrosine Kinase)**
3. When do we give EPO?
 - **Anemia of chronic disease associated with renal insufficiency (HY)**
 - **Chemotherapy induced anemia (LY, but know it)**
4. When does the production of EPO Increase?
 - **Anemia**

- **Chronic hypoxia (COPD & High altitudes)**

5. When does the production of EPO decrease?

- **Polycythemia Vera**
- **Chronic Kidney Disease/Insufficiency**

- **Choice C** → Irrelevant here. Why would you give INF-gamma? To stimulate the immune cells? What immune cells dude.
- **INF- gamma is a very HY cytokine** on the USMLE & you will most likely get a Q or 2 on your test. So, let's master it together.

1. **The Cycle:**

- **INF-gamma** is produced by **Th1 cells**.
- **Macrophages** get stimulated by **INF-gamma**
- IL-12 produced by macrophages then stimulates back Th1 cells to produce more INF-gamma

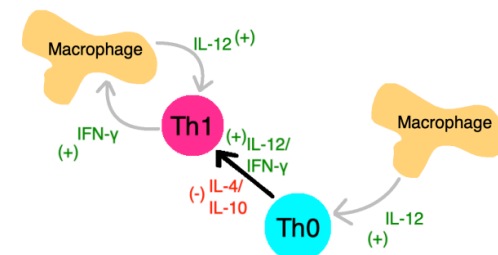
2. Makes **granulomas**

USMLE will show you a picture of a granuloma and will ask you what cell is responsible for what is seen in the picture below. So, you need to know 2 things:

1. **INF-gamma stimulates macrophages and activates them to form granulomas**
2. What a granuloma looks like. They can show you a **caseating granuloma (TB)** or a **noncaseating granuloma**

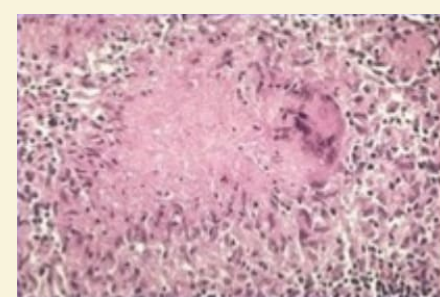
- **Choice D** → **IL-8** is a chemotactic molecule. Remember them?
- **LTB-4, C5a, IL-8, Platelet activating factor (PAF), Kallikrein. [asked on NBME]**

Qs about chemotactic molecules would be something like: In an experiment, there is a source of infection / inflammation & neutrophils were found in that site, what stimulated the arrival of the neutrophils? Then the answer would be one of these molecules. Easy.

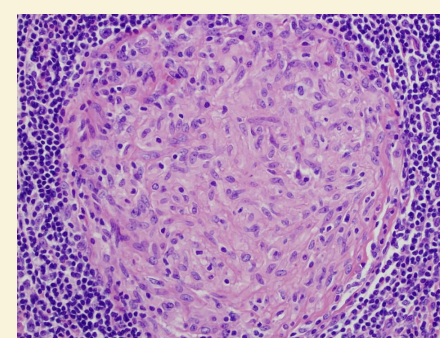


Granulomas

Caseating Granulomas:



Non-caseating Granuloma:



- **Choice E** → I have not seen thrombopoietin get assessed or asked about. It is like EPO but for platelets

Q2

A 65-year-old man comes to the physician because of a 1-month history of decreased urine output and difficulty starting and stopping urine flow. Physical examination shows an enlarged prostate. His serum prostate-specific antigen concentration is 12 ng/mL (N<4). A prostate biopsy specimen confirms prostate cancer. Screening shows metastatic disease to the L1 vertebra. The patient elects to have an orchiectomy. Which of the following changes in this patient's prostate is most likely to be increased secondary to the orchiectomy?

- A. Bromodeoxyuridine staining
- B. Cell size
- C. DNA fragmentation
- D. Mitotic rate
- E. Size of the prostate gland

▼ **Explanation**

- **Answer is C (DNA fragmentation)**
- **IMP Clues:**

- Decreased urine output & difficulty starting & stopping urine flow (prostate symptoms)
- Enlarged prostate (yes)
- PSA is 12 (super high & most likely cancer, biopsy is needed to confirm)
- Biopsy confirms prostate cancer with metastases to L1 vertebra
- Patient will have orchiectomy (removal of testes)

• **Dx → Metastatic Prostate Cancer**

- Q asks what will INCREASE secondary to the orchiectomy.
 - First, you need to know that **the prostate enlarges due to DHT**.
 - **Testosterone → DHT via 5-alpha-reductase**
 - Hence, **5-alpha reductase inhibitors (finasteride)** are used in the treatment of BPH.
 - Secondly, you need to know that **removing the testes = less testosterone production = less conversion to DHT = smaller prostate (apoptosis)**
 - So, what will increase? **Apoptosis**. which is characterized by **controlled DNA fragmentation** (the answer).

? Why does prostate cancer LOVE to metastasize to the vertebrae & pelvic bones?

▼ **Answer**

Prostate is drained by the prostatic venous plexus → connected to the vertebral venous plexus, which is valveless & drains the spine, pelvis, and connects to the brain.

Bidirectional Flow allows prostate cancer to flow **either to the spine, or down the spine to the pelvis, or up the spine to the brain.**



Prostate Cancer & BPH are very HY on the USMLE. Let's go through what we need to know.

1. BPH

- Prostate is made up of **glandular tissue & smooth muscles**.
- In the treatment of BPH, we give 2 classes of drugs:
 1. **Finasteride (5-alpha reductase inhibitor)** which decreases DHT so **glandular tissue SHRINKS**.
 2. **Tamsulosin (alpha-1 antagonist)** causes **relaxation of the prostate smooth muscle**.
 - It **DOESN'T** cause the prostate to shrink. Does help in urine retention though.

2. Prostate Cancer

- Most common cancer in men.
- Prostate cancer **LOVES to metastasize to the vertebra**. USMLE will test this concept by the following presentations:
 - i. Patient with prostate cancer + develops **acute back pain (stress fracture of vertebra)**
 - ii. Patient with prostate cancer + develops **pain of palpation of vertebra (metastasis)**
 - iii. Patient with prostate cancer + develops **acute neurological lower limb symptoms (metastasis + invasion of spinal cord / cauda equina)**

Questions could be tricky and give you an **old man with the above presentations & NO mention of prostate cancer**.

- If the question is somewhat easy, they will give you "spine shows **osteoblastic** lesions" → prostate cancer
- If the question is hard, they will give you nothing and you will have to infer that the old man most likely has prostate cancer

▼ **Other Choices**

- **Choice A** → Bromodeoxyuridine is a stain that stains proliferating cells. We have apoptosis here, so **decrease in proliferation NOT increase**.
- **Choice B** → Cell size would **decrease NOT increase** as Apoptosis = programmed cell death. Not enlargement of cells
- **Choice D** → Mitotic rate will **decrease NOT increase**. Again, Apoptosis.
- **Choice E** → The size of the prostate **would also decrease NOT increase**. Why? apoptosis. I think you get it now.

Q3

A 34-year-old man comes to the physician because of a 2-week history of daily episodes of severe, unilateral, periorbital headaches. The headaches, which develop rapidly and last 60 to 90 minutes, are often accompanied by lacrimation and nasal stuffiness on the same side as the headache. The patient had similar symptoms 2 years ago. He is currently asymptomatic. Vital signs are within normal limits. Physical examination shows no abnormalities. Which of the following is the most likely diagnosis?

- A. Cluster headache
- B. Common migraine
- C. Ophthalmoplegic migraine
- D. Sinus headache
- E. Trigeminal neuralgia

▼ Explanation

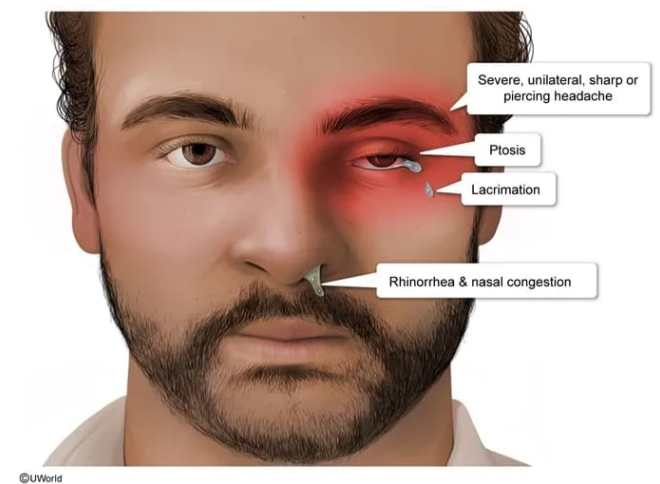
• Answer is A (Cluster Headache)

◦ IMP Clues:

- Daily episodes of severe unilateral periorbital headaches.
- Develop rapidly & lasts 60-90 mins
- Accompanied by lacrimation & nasal stuffiness

• Dx → Cluster Headache

- Unilateral Periorbital pain
- Associated **autonomic manifestations (lacrimation - stuffiness - miosis)** [characteristic]
- mins to hours in duration
- Treatment is **100% Oxygen (Most Effective) or Triptans (Serotonin Agonist)**
- Prophylaxis is **Verapamil**
 - **NOT** Beta Blockers [asked on NBME]



▼ Other Choices

• Choice B → Migraines

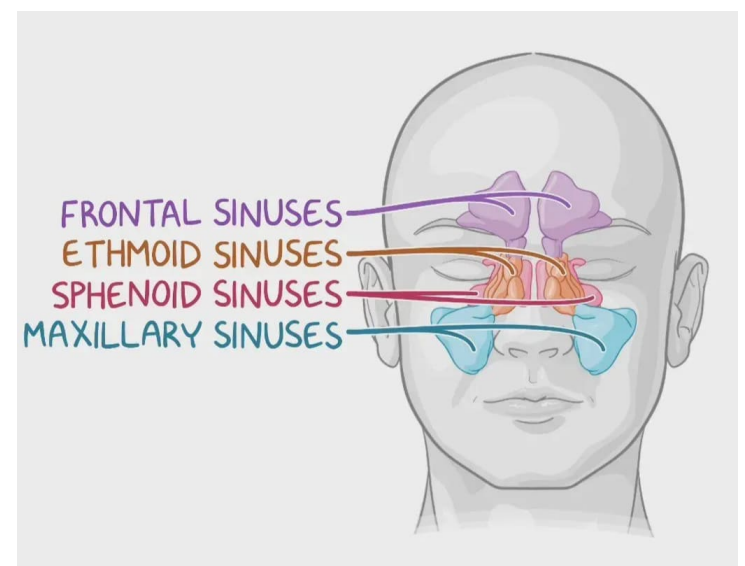
- Unilateral Pulsating pain
- Associated with **"auras" (attacks of visual / sensory manifestations before or after the migraine), nausea, photophobia, or phonophobia**
- Hours to days
- Treatment is **NSAIDs then Triptans, or Ergots**
- Prophylaxis is **Beta Blockers**



• Choice C → Ophthalmoplegic migraine is your normal migraine + visual aura

• Choice D → Sinus Headache

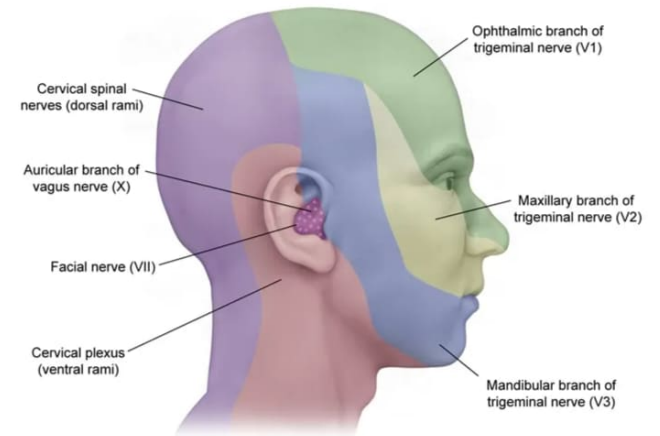
- Essentially the headache we all have when we get a cold or congestion or sinusitis
- Bilateral usually (can be unilateral sometimes)
- **Dull aching pain with fullness or congestion**
 - **increases** with bending forward or lying down
 - Days to Weeks in duration
- Location depends on sinus affected:
 - **Frontal sinus = Forehead pain**
 - **Maxillary sinus = Cheeks pain**
 - **Ethmoid sinus = periorbital / retroorbital pain**



- **Sphenoid sinus = Retroorbital pain**

• **Choice E → Trigeminal Neuralgia**

- Unilateral *Stabbing stitching pain*
 - **Precipitated by stimulation of affected segment** (for example, brushing teeth = cheek pain)
 - Seconds in duration
- Location depends on the affected division of trigeminal nerve
 - **V1 = Ophthalmic + Forehead distribution**
 - **V2 = Maxillary distribution (Most Common)**
 - **V3 = Mandibular distribution**
- Treatment is **carbamazepine** (anti-epileptic that can be used for tonic-clonic seizures & Trigeminal Neuralgia)



Trigeminal Neuralgia is a very common presentation of Multiple Sclerosis

- USMLE often give you a case of **MS + trigeminal neuralgia** & asks where the pathology is → **PONS**
 - They usually don't give any nerves in the choices
 - But we know that Trigeminal nerve originates from pons. Makes sense.



POP-QUIZ :)

Differentiate between Cluster Headaches, Migraine, Sinus Headache, and Trigeminal Neuralgia.

▼ **Answer**

Feature	Cluster Headache	Migraine	Sinus Headache	Trigeminal Neuralgia
Laterality	Unilateral	Unilateral	Usually bilateral (can be unilateral)	Unilateral
Pain Type	Stabbing / sharp	Pulsating / throbbing	Dull , aching, fullness	Stabbing , electric-shock-like
Triggers & Associations	Autonomic symptoms: lacrimation, nasal stuffiness, miosis	"Aura" (visual/sensory), nausea, photophobia, phonophobia	Congestion, worse with bending forward	Light touch (e.g., brushing teeth, wind, chewing)
Duration	Minutes to hours	Hours to days	Days to weeks	Seconds
Location	Around or behind one eye (periorbital)	Variable, usually hemi-cranial	Frontal (forehead), maxillary (cheeks), ethmoid / sphenoid (retroorbital)	Based on trigeminal branch: V1: Forehead V2: Cheeks (most common) V3: Jaw
Acute Treatment	Triptans , O ₂ is most effective	NSAIDs → Triptans / Ergots	Decongestants, antibiotics if bacterial	Carbamazepine
Prophylaxis	Verapamil	Beta blockers	Treat underlying sinus disease	Carbamazepine may be used chronically

Q4

A 25-year-old man develops shortness of breath after moving from sea level to a mining town at 3350 m (11,000 ft). Evaluation shows no abnormalities, but at rest, pulmonary hypertension with normal cardiac output is noted. Pulmonary function tests are normal. Which of the following best explains the pulmonary hypertension?

- Decreased alveolar Po₂
- Decreased mixed venous Po₂
- Decreased regional ventilation
- Increased pulmonary J receptor nerve traffic
- Increased pulmonary vagal activity
- Increased sympathetic nervous system activity

▼ Explanation

- **Answer is A (Decreased Alveolar PO₂)**

- **IMP Clues:**

- SOB after moving from sea level to mining town at 3350m (you know this is an altitude question)
 - Pulmonary HTN with normal cardiac output at rest
 - PFTs are normal (helps in exclusion)

- **Dx → Hypoxic-induced Pulmonary HTN**

- You will see this Q or a similar Q on every NBME. Mastering it is a must.
 - First, you need to understand that usually anywhere in the body, **CO₂ / hypoxia cause vasodilation**
 - **EXCEPT THE LUNGS ⇒ Hypoxia causes Vasoconstriction**
 - Secondly, you need to know that **vasoconstriction in the lungs cause increased pulmonary artery pressure** (aka Pulmonary HTN)
 - Thirdly, you need to know the following (SUPER ULTRA HY)
 - **What is the A-a gradient?**
 - The **A-a gradient** is the difference between the **oxygen concentration in the alveoli** (air sacs in your lungs) and the **oxygen concentration in your arterial blood**.
 - Normally it's **around 5-10 mmHg** (KNOW THIS NUMBER, IMP FOR ARROW Qs)
 - **Causes of Hypoxia are either:**
 - **Normal A-a gradient:**
 - ▮ **Nothing here is affecting the transfer of O₂ from alveoli to arteries. The hypoxia is a result of just low O₂ in alveoli so low O₂ in arteries**
 - 1. **Hypoventilation:** decreased washing out of CO₂
 - a. **Drugs: (opioids, benzos, barbiturates) [HY and asked on NBME]**
 - b. Obesity (Obesity Hypoventilation Syndrome)
 - c. Neuromuscular Weakness
 - 2. **Decreased Inspired Oxygen tension: (what we have here)**
 - a. Higher altitudes = less O₂ in air = hypoxia
 - **Increased A-a gradient:**
 - 1. **Diffusion affected:** Fibrosis
 - 2. **V/Q mismatch**
 - a. Ventilation affected (any lung pathology *except pulmonary embolism*)
 - b. **Perfusion affected** (Pulmonary Embolism)
 - c. Right-to-left shunt (Intracardial shunt)
 - After all of that, it should be simple.
 - **Patient + high altitude = decreased inspired oxygen tension (decreased O₂ in air) = decreased O₂ in alveoli = hypoxia = pulmonary vasoconstriction = Pulmonary HTN**

▼ Other Choices

- **Choice B** → mixed venous PO₂ is influenced by 2 things: CO (which is normal here) & O₂ consumption by tissues
 - Low CO = less O₂ to tissue = less mixed venous PO₂
 - high O₂ consumption by tissue = less mixed venous PO₂
- **Choice C** → decreased regional ventilation causes hypoxia which would cause vasoconstriction in the affected region. However, it is regional not widespread, so it doesn't BEST explain the pulmonary HTN
- **Choice D** → Pulmonary J receptors (Juxtacapillary receptors) are stimulated mainly by fluid accumulation in the lung → impulses to respiratory center via vagus nerve → dyspnea & SOB. NO fluid in the lung here so not cause of SOB + doesn't explain pulmonary HTN
- **Choice E** → Vagal activity would cause bronchoconstriction & vasodilation NOT vasoconstriction. Irrelevant.
- **Choice F** → Sympathetic stimulation would cause bronchodilation. Irrelevant.

Q5

A 75-year-old woman is brought to the emergency department comatose 16 hours after the onset of left arm and leg weakness. Physical examination shows left hemiplegia. A CT scan of the head shows an edematous right hemisphere. There is no evidence of hemorrhage, but there is sparing of the right occipital and midline frontal cortices. One hour later, she dies. At autopsy, microscopic examination of the right lateral frontal lobe is most likely to show which of the following?

- A. Numerous lymphocytes
- B. Numerous macrophages
- C. Numerous neutrophils
- D. Shrunken eosinophilic (red) neurons
- E. No abnormalities

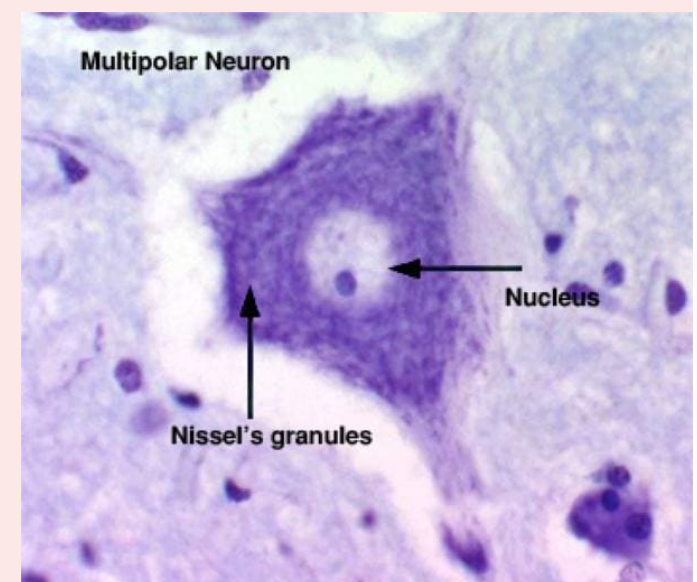
▼ Explanation

- Answer is D (Shrunken eosinophilic (red) neurons)
 - IMP Clues:
 - 16h history of left arm & leg weakness (aka 16h hx of stroke)
 - Left hemiplegia (yep, it's a stroke)
 - CT shows edema + no hemorrhage (so ischemic stroke)
 - One hour later she dies
- Dx → Ischemic Stroke
 - Q here asks what would be seen on autopsy of the ischemic part.
 - We have 4 things that can appear post-stroke on the USMLE, depending on the time frame:
 1. Within 12-24 hours: **Red Neurons**
 2. Within <1 weeks: Early Liquefactive necrosis - Microglia (Macrophages)
 3. Within 1-2 weeks: Reactive gliosis (Astrocytes are what form scars in the CNS) **[asked on NMBE]**
 4. >2 weeks: Scar formed
 - The Q here tell us that we are still within the first 24 hours (16hr hx after onset + 1 hour = 17 hours total) → **RED NEURONS**. EASY.

Ischemia, causes decreased oxygen supply to tissues, which in turn causes decreased ATP production

- Na/K ATPases fail as the ATP requirements are not met, which causes cellular swelling, pyknosis (nuclear shrinking), karyolysis (dissolution of the nucleus), and disappearance of the basophilic Nissl Bodies
 - **NO** Basophilic Nissl Bodies ⇒ Eosinophilic Staining "Red Neurons"

Normal Neuron:

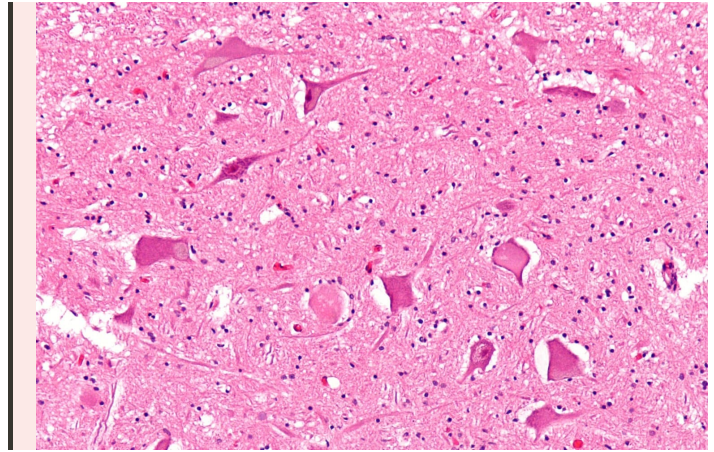


Red Neurons:



Types of Necrosis Rapid Review:

1. **Liquefactive Necrosis:** architecture is *NOT* maintained + dissolved by hydrolytic enzymes
 - HY examples are **Abscesses & CNS-related Injuries (Strokes)**
2. **Coagulative Necrosis:** Architecture is *MAINTAINED*
 - HY examples are **ATN & MI**
3. **Caseous Necrosis:** Cheese-like Necrosis
 - HY examples are **TB, Fungal Infections (Histoplasmosis is HY), and Bartonella henselae (stellate-granulomas)**
4. **Fat Necrosis**
 - a. **Enzymatic** Fat Necrosis: **Acute Pancreatitis** (Lipases + Ca → Saponification)
 - b. **Non-Enzymatic** Fat Necrosis: **Breast Trauma**
5. **Fibrinoid Necrosis: Polyarteritis Nodosa**
6. **Dry Gangrene:** **Tissue Death (Seen in DM) & Buerger Disease (Thromboangiitis Obliterans)**
7. **Wet Gangrene:** **Infection on top of a dry gangrene & Fournier gangrene (scrotum/perineal necrosis in DM)**
8. **Gas Gangrene:** **C.perferinges** causing **subcutaneous emphysema or Emphysematous Cholecystitis**
9. **Cystic Medial Necrosis: Aortic Dissection**
 - Seen in **Connective Tissue Disorders (Marfan Syndrome, Ehler Danlos) & Systemic Hypertension**



▼ Other Choices

- **Choice A** → Lymphocytes are cells of chronic inflammation. They would be seen in viral infection or toxoplasmosis for example
- **Choice B** → Macrophages would be seen from 3-5 days post-stroke. Remember, Macroglia = Macrophages when we are in the CNS.
- **Choice C** → Neutrophils would be seen in the first 1-2 days. They are the cells responsible for the liquefactive necrosis
- **Choice E** → Would never be the correct answer except if it was like for a TIA. NOT STROKE.
 - Before the development of red neurons (which appear 12-24hr post-stroke), in the first 12 hours, the cells become swelled.
 - Hypoxia causes failure of Na/K ATPase pump, which causes the Na to be retained inside = pulls water in = edematous cells.
 - Hypoxia also causes the disruption of the cytoskeleton of the cells.

Q6

A study is conducted to assess the normal mean serum urea nitrogen concentration in men ages 65 years and older to be used as reference values in a laboratory. Random selection of which of the following will provide an inaccurate but precise estimate of the mean serum urea nitrogen concentration in this group?

- A. 10 Men from a list of patients scheduled to be examined by a urologist
- B. 10 Men from a list of patients undergoing routine health screening at a local shopping center
- C. 500 Men from a list of patients scheduled to be examined by a urologist
- D. 500 Men from a list of patients undergoing routine health screening at a local shopping center

▼ **Explanation**

- **Answer is C (500 Men from a list of patients scheduled to be examined by a urologist)**
 - You need to know the difference between precise & accurate
 - **Accurate** = how close the value is from the **NORMAL** value
 - **Precise** = how close are all the values from **EACH** other
 - So, Q asks what which of these is inaccurate but precise
 - **Inaccurate** = patients to be examined by a urologist
 - They have some sort of disease that they got referred to a urologist.
 - So, they probably have a higher than normal serum urea (far from normal value so inaccurate)
 - **Precise** = 500 men
 - The greater the numbers, the more likely that they fall closer to each other

▼ **Other Choices**

- **Choice A → Inaccurate & not precise**
 - Just 10 men so not precise
 - Patients to be examined by a urologist so inaccurate
- **Choice B → Accurate but not precise**
 - Screening at a local shopping center so people most likely healthy so closer to normal value so accurate
 - Just 10 men so not precise
- **Choice D → Precise & Accurate**
 - 500 men so precise
 - Screening at a local shopping center so people most likely healthy so closer to normal value so accurate

Q7

Left radial arterial and venous blood samples are drawn from 10 chimpanzees breathing room air while resting quietly. The results indicate that the total carbon dioxide concentration average is 21.5 mM in the arterial samples and 24.3 mM in the venous samples. Which of the following accounts for the larger amount of carbon dioxide in these venous blood samples?

- A. CO₂ bound as carbaminohemoglobin
- B. Dissolved CO₂ in the plasma
- C. Dissolved CO₂ within erythrocytes
- D. HCO₃⁻ transported in the plasma
- E. HCO₃⁻ transported within erythrocytes

▼ **Explanation**

- **Answer is D (HCO₃ transported in the plasma)**
 - Q gives us CO₂ concentration average in arterial samples (21.5mM) and venous samples (24.3 mM) & asks what accounts for the larger concentration in the venous blood.
 - For you to answer this Q, you should know that **the greatest portion of CO₂ in the venous blood is carried as bicarbonate.**
 - This is caused by the **Chloride Shift phenomenon**:
 - CO₂ received as waste *from tissue* enters the RBC
 - CO₂ + H₂O are converted to H₂CO₃ by **Carbonic Anhydrase (CA)**
 - **H₂CO₃ dissociates into H + HCO₃**
 - HCO₃ is transported out of the RBC in exchanged with Chloride (negative ion for another negative ion)
 - This keeps the CA-induced reaction moving forward.
 - When the venous blood goes to the lung:
 - Chloride shifts out for bicarbonate in.
 - HCO₃ + H → H₂CO₃ → H₂O + CO₂ **(by CA)**
 - CO₂ is expired *out* by the lungs

Q8

A 48-year-old man is brought to the emergency department because of a 1-hour history of vomiting blood. One year ago, he was diagnosed with bleeding esophageal varices and cirrhosis. Variceal banding was ineffective in controlling the bleeding from the varices. Physical examination shows ascites. A surgical shunt between which of the following veins is most likely to relieve the esophageal bleeding in this patient?

- A. Hepatic and inferior phrenic
- B. Ileocolic and inferior mesenteric
- C. Splenic and left renal
- D. Superior epigastric and inferior epigastric
- E. Superior rectal and superior mesenteric

▼ Explanation

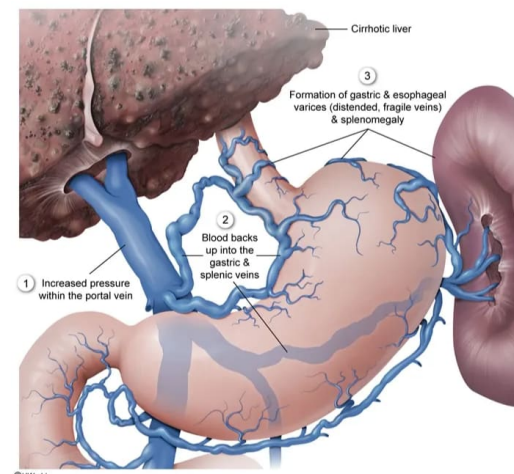
- Answer is C (Splenic & Left Renal)

- IMP Clues:

- 1hr hx of vomiting blood (okay so hematemesis)
 - Diagnosed with varices & cirrhosis (so most likely a recurrent attack)
 - Banding was ineffective (so probably a shunt will be used)

- Dx → Bleeding Esophageal Varices 2ry to decompensated liver failure

- Q asks what veins should be used for the shunting.
 - Okay, so we know patient is cirrhotic & hence the varices, he has **portal hypertension**.
 - So, what we need is a vein that is from the **systemic circulation** (NOT from the portal circulation) so that we can shunt the blood & decrease the pressure.
 - Splenic vein is part of the portal circulation while the left renal drains into the Inferior Vena Cava, so placing a shunt will cause blood to leave the high-pressure circulation (Portal) and escape into the low pressure circulation (systemic)
 - This will result in **less Portal HTN** and **decrease amount of congestion**, resulting in a **decrease in esophageal bleeding**.



▼ Other Choices

- Choice A → Both the hepatic vein & the inferior phrenic vein are part of the systemic circulation
- Choice B → Ileocolic drains into SMV, while IMV drains into splenic. Splenic & SMV join, forming the Portal vein
- Choice D → Both are part of systemic circulation
- Choice E → Superior rectal vein drains into IMV, and we already established the SMV as a part of the portal circulation

Q9

A 35-year-old man comes to the physician because of a 1-day history of pain and swelling of his right great toe. Examination of the toe shows erythema, swelling, warmth, and tenderness. Movement of the toe is limited by pain. Microscopic examination of aspirate from the affected joint shows numerous neutrophils and many needle-like crystals. Which of the following is the most likely cause of this patient's swelling?

- A. Complement activation
- B. Histamine release
- C. Increased vascular permeability
- D. Release of lipopolysaccharide

▼ Explanation

- **Answer is C (Increased vascular permeability)**

- **IMP Clues:**

- 1-day hx of pain & swelling of great toe (okay, gout or pseudogout mostly)
- Erythema, swelling, warmth, and tenderness + limitation of movement by pain (ok, noted)
- numerous neutrophils & many needle like-crystals (bingo, gout it is)

- **Dx → Gout**

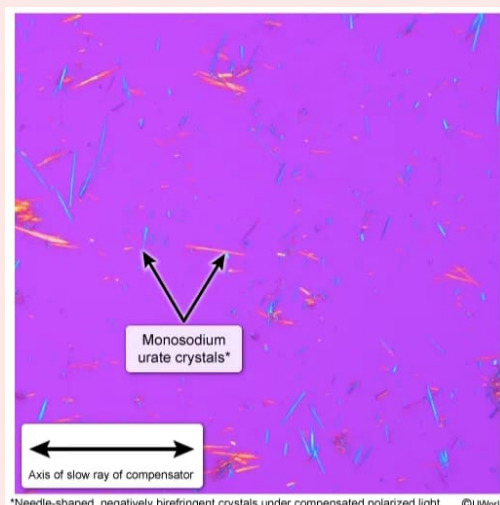
- Q asks what is cause of swelling.

- **Crystals deposition & mobilization from joints cause the release of inflammatory mediators**, which cause:

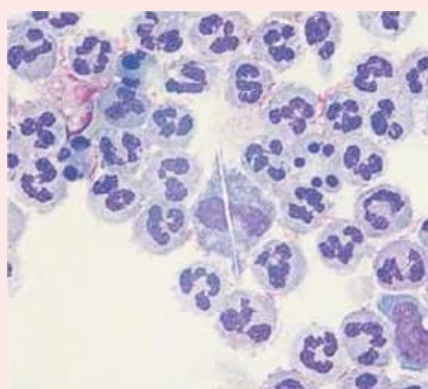
- **Vasodilation** (cause of swelling here)
- **Neutrophils migration** (remember the chemotactic molecules? see I told you it is HY)

How does the Crystals look like?

They are **Monosodium Urate Crystals**, needle-like in shape, and "Negatively Birefringent"



The below picture was on NBME 18, answer was Gout (due to presence of that singular needle-shaped crystal:



? What is the treatment of gout?

▼ Answer

Acute gout

- **NSAIDs** (Indomethacin preferably) [1st line, used if no C.I.]
- **Corticosteroids** [2nd line]
 - used if **hx of renal failure, Peptic ulcer, or renal transplant**
- **Colchicine** [3rd line]

Chronic Gout

- **Xanthine Oxidase inhibitors** (Allopurinol & Febuxostat) [1st line]
- **Uricosuric drugs** (Probenecid & Sulfapyrazone) [2nd line]
 - C.I. in **hx of renal / uric stones & uric acid overproduction**
- **Urate Oxidase analogues** (Rasburicase & Pegloticase) [3rd line]
 - used in **uric acid overproduction (Lesch-Nyhan Syndrome)**

This picture of Tophi was also used on NBME 19:



*** Tophi are chalky-white extraarticular urate crystal deposits that may form under the skin. They are **PAINLESS**.

▼ Other Choices

- **Choice A** → Not the cause of swelling here. Complement activation & deposition though is part of RA.
 - **IMP for Arrow Qs: patient with RA and asks for the arrows of complements.**
 - Answer is **DECREASED** (as they get activated and deposit around the joints)
- **Choice B** → **Histamine** is not involved in gout. It is involved in **type 1 HS reaction** & is the main cause of vasodilation & swelling there. Not here though.
- **Choice D** → **LPS** is involved in bacterial infections not gout
- **Choice E** → **T-lymphocytes** are involved more in RA, not gout.

Q10

An investigator is studying the efficacy of distinct vaccine formulations directed against the capsular polysaccharides of *Neisseria meningitidis*. The carbohydrates are chemically conjugated to various compounds and injected into laboratory mice. Titers of anticapsular IgG antibodies are then measured. Which of the following compounds is most likely to induce increased titers of these antibodies when conjugated to the polysaccharides?

- A. Dinitrophenol
- B. Flagellin
- C. Lactose
- D. Lipopolysaccharide
- E. Palmitic acid
- F. Polyinosinic acid-polycytidylic acid

▼ Explanation

- Answer is B (Flagellin)
 - Okay, let be simple here and not turn this into a Wikipedia page.
 - All you need to know is one concept:
 - polysaccharides (carbs) suck at inducing an immune response.
 - **Proteins are amazing at inducing an immune response.**
 - So, all you need to do in these types of Qs is choose the protein.
 - **Flagellin is the only sounding protein in here (they usually end with -in)**

▼ Other Choices

- All the other choices are not proteins

Q11

A 21-year-old man is brought to the emergency department 45 minutes after sustaining multiple injuries in a motor vehicle collision. His blood pressure is 90/50 mm Hg. Physical examination shows diffuse abdominal tenderness. He is diagnosed with laceration of the spleen and undergoes splenectomy. The splenectomy most likely predisposes this patient to develop future infection with which of the following causal organisms?

- A. *Candida albicans*
- B. Cytomegalovirus
- C. *Escherichia coli*
- D. Herpes simplex virus
- E. *Streptococcus pneumoniae*

▼ Explanation

- Answer is E (*Streptococcus pneumoniae*)
- Dx → OPSI (Overwhelming Post-Splenectomy Infection)
 - People who undergo **splenectomy (either elective - emergency - or even auto splenectomy in sickle cell anemia patients)** are at risk of something called **OPSI "Overwhelming Post-Splenectomy Infection"**.
 - This patient here had **emergency splenectomy after MVA**, so she should have been *vaccinated & she must be prescribed prophylactic antibiotics*
 - Otherwise, she is at **massive risk of developing sepsis → DIC → Death**
 - The Infection is caused by **encapsulated organisms**.
 - Most Commonly ***Streptococcus pneumoniae***

? What smear finding would be expected in a patient with sickle cell disease that warrants prophylactic antibiotics and vaccinations?

▼ Answer

Howell-Jolly bodies - DNA fragments that are normally removed from the RBCs by the spleen

- Presence of Howell-Jolly Bodies = **functional asplenia** due to repeated infarcts
 - The other HY cause is **post-splenectomy**.

- Other common ones include *H.influenza*, *N.meningitides*, and *E.coli*.

- Now in this Q, we have both *Streptococcus pneumoniae* & *E.coli*.
- Both are correct; however, if you had to choose, choose *Streptococcus pneumoniae*. It is the commonest by far.

Q12

A 17-year-old boy has the acute onset of lethargy, sore throat, and fever. Physical examination shows symmetrical, nontender cervical lymphadenopathy, an exudative erythematous pharynx, palatal petechiae, and splenomegaly. No rash is present. Serologic studies are positive for antibody to Epstein-Barr viral capsid antigen. In addition to a slightly decreased platelet count, the peripheral blood will most likely show an increased number of which of the following?

- A. Lymphocytes
- B. Monocytes
- C. Myeloblasts
- D. Nucleated erythrocytes
- E. Reticulocytes

▼ **Explanation**

• **Answer is A (Lymphocytes)**

◦ **IMP Clues:**

- Sore throat + erythematous pharynx + palatal petechiae
- Symmetrical nontender cervical lymphadenopathy
- Splenomegaly
- Serology positive for EBV capsid antigen

• **Dx → Infectious Mononucleosis**

- Caused by **Epstein-Barr Virus (EBV)**; which is a **dsDNA virus**
- **EBV binds to CD21 on B-cells → Infects them & causes B-cell proliferation (what is referred to by "monomorphous population of B lymphocytes")**
 - This **B-cell proliferation** is the cause of **hepatosplenomegaly & lymphadenopathy** (*Posterior Cervical Lymphadenopathy* is buzzy & HY for EBV infection)
- Smear Findings: **Reactive/Downy/Atypical T-cells**
 - EBV infects B-cells & causes their proliferation in lymph nodes & spleen
 - T-cells "react" to EBV by transforming their shape, named Reactive/Atypical T-cells in plasma

USMLE can give you a patient who had a graft rejection for a cadaveric renal she got. Doctors gave her maximal immunosuppression drugs, which caused **reactivation of EBV → enters the B-cells via CD21 [asked on NBMEs]** → causes B-cells to proliferate → HSM & lymphadenopathy.

? **What test is used to diagnose Infectious Mononucleosis?**

▼ **Answer**

Heterophil (or Monospot) test - If Q says these tests are negative, **IT IS NOT EBV**

? **21M with pharyngitis was given ampicillin & developed a maculopapular rash. What is the causative organism?**

▼ **Answer**

EBV - patients with Infectious Mononucleosis develop a maculopapular rash when given **ampicillin or amoxicillin**.



Q13

A 22-year-old man has had frequent episodes of cutaneous urticaria and occasional episodes of laryngospasm over the past year. An allergy evaluation fails to find any allergic cause for his urticaria. Based on this history, which of the following complement proteins is most likely to be deficient in his serum?

- A. C1 esterase inhibitor (binds activated C1r, C1s)

- B. C4 binding protein (binds C4b displacing C2b)
- C. Complement receptor 1 (binds C4b displacing C2b)
- D. Factor H (binds C3b displacing Bb)
- E. Factor I (serine protease cleaves C3b and C4b)

▼ Explanation

• Answer is A (C1 esterase inhibitor)

- Straight-up this Q is written poorly. I mentioned before the different types of angioedema:

1. Mast cell degranulation (ASSOCIATED with Pruritis & Urticaria)

- **IgE-mediated** mast cell degranulation (examples are *atopy* & *eczema*)
- **Direct-mast cell degranulation** (most common examples are *morphine* & *Vancomycin* & *IV Contrast*)

2. Bradykinin Mediated (**NOT** ASSOCIATED with Pruritis & Urticaria)

- **ACEi induced** - accumulation of Bradykinin due to inhibited ACE that normally degrades the Bradykinins
- **Hereditary Angioedema** (C1 esterase inhibitor deficiency)



Bradykinin accumulation caused by ACE inhibitors is **NOT caused by ARBs too.**



Hereditary Angioedema is treated with **Danazol**

- Danazol increases C1 esterase **inhibitor** production from liver



Patient got a vancomycin injection & started flushing + developed hypotension. What is the Diagnosis & Treatment?

▼ Answer

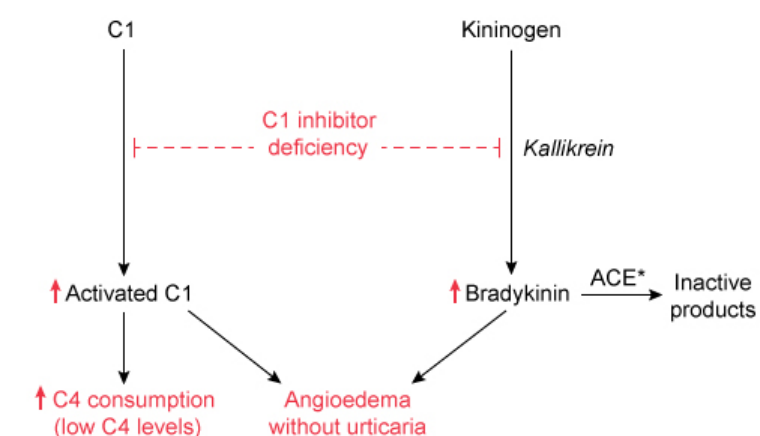
Diagnosis is **Red Man Syndrome**

- Caused by **Direct-mast cell degranulation**

Treatment is with **Antihistamines**

- Vancomycin causes direct-mast cell degranulation → histamine release
- Histamine is the molecule causing the VD (Flushing & hypotension)
- So treated with antihistamines

Hereditary angioedema



*ACE inhibitors exacerbate angioedema attacks.

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Q here says we do **HAVE** urticaria & laryngospasm + allergy evaluation **FAILS** to find a cause for his urticaria

- so “causes” can be excluded: IgE, direct degranulation, and ACE inhibitors
- What remains & is in the answer choices is **C1 esterase inhibitor deficiency**
- C1 esterase inhibitor deficiency **DOESN'T** present with urticaria. See what I mean?

Q14

A 4-year-old girl is conscious but unable to breathe spontaneously for 3 hours following uncomplicated removal of a branchial cleft cyst. The procedure was done under general anesthesia with sevoflurane. Succinylcholine was administered for intubation. Which of the following is the most likely cause of the prolonged apnea in this patient?

- A. Idiosyncratic reaction to sevoflurane anesthesia
- B. Intraoperative vagus nerve injury during the operation
- C. Overdose with midazolam used as preoperative sedation
- D. Pseudocholinesterase deficiency
- E. Ryanodine receptor defect

▼ Explanation

• Answer is D (Pseudocholinesterase deficiency)

- **IMP Clues:**

- Unable to breath spontaneously / Prolonged apnea (okay so a post-op complication)
- Succinylcholine was administered for intubation (once you see succinylcholine in a Q, 3/4 of the time it is the responsible agent)

• **Dx → Pseudocholinesterase deficiency**

- Basically, Succinylcholine is a **depolarizing neuromuscular blocker that is used to relax muscles**. Like here, they used it to relax the throat muscles for intubation.
- Succinylcholine is **short-acting agent** that is metabolized by **pseudocholinesterase**
- **If pseudocholinesterase is deficient, the action of succinylcholine is prolonged (like here, she cannot breathe because her throat is paralyzed basically)**

? **What's another HY potential side effect of succinylcholine? What is its treatment?**

▼ **Answer**

Malignant Hyperthermia

- **Fever, muscle rigidity, acidosis, and tachycardia**
- Caused by **persistent activation of Ryanodine calcium channels** on the Sarcoplasmic reticulum.
- Calcium keeps flooding into the cell → Muscle contraction & Rigidity
- Massive amounts of ATP are consumed
- Metabolic pathways in the body are pumped-up to keep with the increasing demand → Fever, Hypoxia, and Acidosis

Treatment → Dantrolene

- Binds to & inhibits Ryanodine Calcium channel Receptors

▼ **Other Choices**

- **Choices A & E** → refer to Malignant hyperthermia
 - caused by succinylcholine & volatile agents "-ane" (halothane, sevoflurane)
- **Choice B** → Vagus nerve injury would result in hoarseness / uvula deviation not apnea
- **Choice C** → Midazolam is a benzodiazepine. Not mentioned that it was used here.
 - It does cause respiratory depression though
 - Also causes CNS depression & pinpoint pupils

Q15

A 64-year-old man with bronchospastic pulmonary disease continues to have wheezing in spite of increasing doses of a β -adrenergic agonist inhaler. He is hospitalized and systemic prednisone is added to the regimen. Which of the following best explains the action of prednisone in enhancing the response to the β -adrenergic agonist?

- A. Enhanced absorption of agonist
- B. Enhanced action of the agonist at β -adrenergic receptors
- C. Inhibited excretion of the nonmetabolized agonist
- D. Inhibited metabolism of the agonist
- E. Inhibited metabolism of metabolites of the agonist

▼ **Explanation**

- **Answer is B (Enhanced action of the agonist at β -adrenergic receptors)**
 - This Q test the pharmacology concept of: **Permissiveness**
 - **Cortisol is permissive of the effects of catecholamines.**
 - Cortisol up-regulates adrenergic receptors. Now, the effects of catecholamines are much more pronounced.
 - In the dictionary, permissive means "allowing or characterized by great or excessive freedom of behavior." This sounds like what cortisol is doing here, right?

? **Patient in surgery gets severely hypotensive. The surgeon decided to administer a shot of steroids + Norepinephrine. Why did the surgeon do this?**

▼ **Answer**

Cortisol is permissive to the effects of catecholamines

- Cortisol upregulates alpha-1 receptors. This causes the effects of norepinephrine to be more pronounced.
- Alpha-1 activation = VC = increase in BP

Q16

A 67-year-old man comes to the emergency department because of a 2-day history of fever, sore throat, and difficulty swallowing. He has lived in various homeless shelters during the past year. His temperature is 38.9°C (102°F). Physical examination shows erythematous tonsils with a small area of gray exudate. A throat culture grows *Corynebacterium diphtheriae*. This pathogen produces diphtheria toxin, which blocks which of the following cellular processes?

- A. DNA repair
- B. Glucose uptake
- C. β -Oxidation
- D. Transcription
- E. Translation

▼ Explanation

• Answer is E (Translation)

◦ IMP Clues:

- Sore throat + erythematous tonsils with small area of grey exudate (pseudomembranes)
- Culture grows *Corynebacterium diphtheriae* (confirmed diagnosis)

• Dx → Diphtheria infection

- Q asks what is blocked by the diphtheria toxin?
 - It causes **Inhibition of Elongation factor 2**
 - Elongation Factor 2 is involved in **protein synthesis**.
 - Inhibition of EF-2 = Inhibition of protein synthesis (Translation)



Differentiate between those 2 confusing terms:

- **Transcription:** DNA → RNA
- **Translation:** RNA → Protein



Patient with sore throat + erythematous tonsils with small areas with grey exudate + culture shows c.diphtheriae. What other organism shares the same MOA with the diphtheria toxin?

▼ Answer

Pseudomonas

- Inhibits EF-2 → Inhibition of protein synthesis
- USMLE asks this.

Q17

A 7-year-old boy is brought to the physician by his mother because he has difficulty concentrating in school. The mother says that he feels tired all the time. Physical examination shows normal development. Laboratory studies show a mild hypochromic anemia and an increased serum creatinine concentration. Further laboratory studies are most likely to show which of the following additional abnormal concentrations in this patient?

- A. Decreased serum magnesium
- B. Decreased serum vitamin B₁₂ (cobalamin)
- C. Increased blood lead
- D. Increased serum vitamin D
- E. Increased serum zinc

▼ Explanation

• Answer is C (Increased blood lead)

◦ IMP Clues:

- Difficulty concentrating & tired all the time (nonspecific symptoms but could hint to anemia)
- Normal development (this excludes metabolic, neurological, endocrine causes & chronic diseases)
- Mild hypochromic anemia (okay, so our DD currently is: IDA, Anemia of chronic disease, lead poisoning)
- Increased serum creatinine (renal insufficiency)

- **Dx → Lead poisoning**

- Q is a little vague, but lead poisoning Qs are always this vague.
- Let's go through what we have:
 - DD of **mild hypochromic anemia** is:
 1. **Iron Deficiency Anemia:** nothing here supports IDA, some presentations would be:
 - a. A menstruating girl
 - b. **A child with celiac disease (malabsorption + IDA is buzzy)**
 - c. A child with parasites (notably *Ancylostoma duodenale* & *Necator americanus*)
 2. **Anemia of chronic disease:** tricky, because the high creatinine might trick you into thinking Renal Failure.
 - a. Q specifically says **normal development**.
 - b. If the child had some sort of chronic disease then his development would be affected. This is how children work.
 - c. This means that an acute/subacute condition is causing the renal failure
 - d. None of the choices support it.
 3. **Lead poisoning:**
 - a. **Fatigue?** Yes
 - b. **Renal affection?** Yes
 - c. **Difficulty concentrating?** Yes



Lead Clinical Presentation Rapid Review:

- **Asymptomatic**
- **Neurologic:** Behavioral/cognitive impairment, headache, irritability, peripheral neuropathy (resulting in foot or wrist drop), ataxia, acute encephalopathy
- **Renal:** Hypertension, gout
- **Hematologic:** Fatigue from anemia
- **Gastrointestinal:** Constipation, abdominal pain ("lead colic"), vomiting
- **Musculoskeletal:** Joint pains, muscle aches



Lead Poisoning Treatment is by *Chelation Therapy* (Succimer - Dimercaprol - EDTA "calcium disodium edetate")

▼ Other Choices + **HY Related Notes**

- **Choice A → hypomagnesemia** on the USMLE presents as ***hypocalcemia* or *hypokalemia resistant to treatment***.
 - Common in **alcoholics**
 - Magnesium is essential for the **function of PTH**.



HY Arrows Q that shows up: Alcoholic + asks for arrows of Mg, PTH, Ca, Phosphate:

▼ Answer

- **Mg → decreased**
- **PTH → decreased**
- **Ca → decreased**
- **Ph → increased**

Non-functioning PTH → Hypocalcemia & hyperphosphatemia

- **Choice B → Vitamin B12 deficiency**

- Most common causes on the USMLE are:
 - Old man on toast & tea diet
 - Vegetarian patient
 - Patient with pernicious anemia
 - Patient with Crohn's Disease
 - Patient underwent removal of ileum
- Features:
 - **Hematological complications** → Macrocytic anemia (MCV > 100)
 - **Neurological complications** → Subacute combined degeneration
 - **Psychological complications** → depression, psychiatric symptoms
 - **Biochemical complications** → **High Homocysteine & High Methylmalonic acid**



What are the affected components of Subacute combined degeneration?

▼ Answer

1. **Dorsal Column**
2. **Spinocerebellar Tract**
3. **Corticospinal Tract**



Homocysteine & Methylmalonic acid in vitamin B9 vs vitamin B12

▼ Answer

Vitamin B12 → **HIGH** Homocysteine & **HIGH** Methylmalonic acid
 Vitamin B9 → **HIGH** Homocysteine & **NORMAL** Methylmalonic acid

- **Choice D → Hypervitaminosis D** is commonly seen in **sarcoidosis**.
 - Sarcoidosis is characterized by **granuloma formation (non-caseating)**
 - The macrophages within these granulomas **overexpress 1-alpha hydroxylase** (yes, the same enzyme found in the kidney) causing uncontrolled formation of Vitamin D, hence hypervitaminosis D
 - Symptoms include: hypercalciuria, urinary stones, nephrogenic DI, weakness, bone and muscle pain, calcification of vessels.
- **Choice E →** Nothing on the USMLE is related to a high zinc concentration.



Source of Hypercalcemia in sarcoidosis is hypervitaminosis D due to **MACROPHAGES producing 1-alpha hydroxylase**

Q18

A 55-year-old man with hypertension comes to the physician because of severe headaches and confusion during the past week. He has smoked 1½ packs of cigarettes daily for 30 years. His blood pressure is 190/110 mm Hg. A faint bruit is heard over the left abdomen. A captopril renal radionuclide scan shows delayed function in the left kidney. Which of the following findings is most likely on abdominal aortography?

- A. Aortitis
- B. Left renal artery aneurysm
- C. Left renal artery atherosclerosis
- D. Left renal artery dysplasia
- E. Left renal cell carcinoma

▼ Explanation

- **Answer is C (Left renal artery atherosclerosis)**
 - **IMP Clues:**
 - 55-year-old man smoker (prime age + smoker + male, major risk factors for atherosclerosis)
 - Severe headache + confusion + BP is 190/110 (malignant HTN)
 - Faint bruit over left abdomen (we know it is renal artery pathology)
 - Captopril renal radionuclide scan shows delayed function (okay, so renal artery stenosis)
- **Dx → Renal Artery Stenosis**
 - Okay, so Renal artery stenosis in an old male smoker is most likely **atherosclerosis**
 - **Decreased RPF is detected by the JGA in the cortex → increased renin production → increased aldosterone & AT-2 production**
 - Increased Aldosterone → **hypokalemia & metabolic alkalosis**
 - Increased AT-2 → **hypertension**

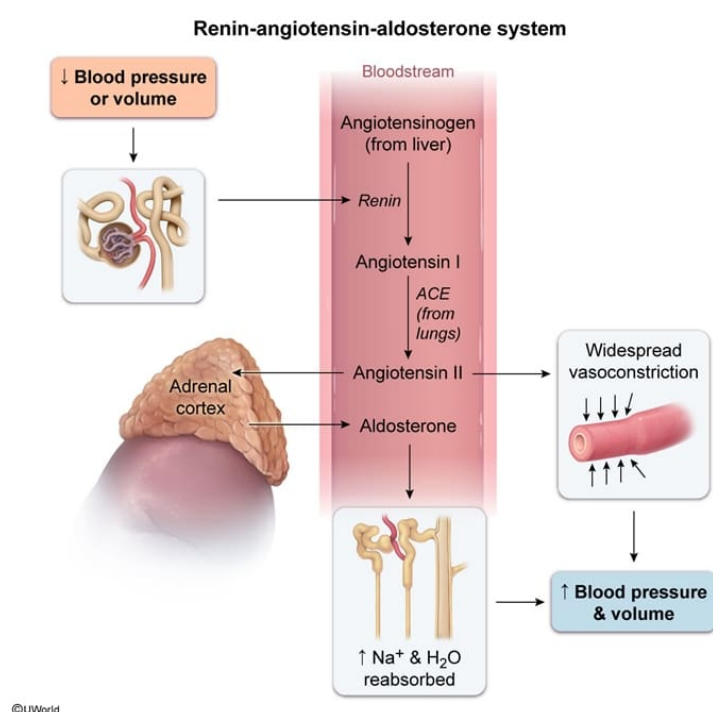


65-year-old man has blurry vision & headache. BP is 200/120. A bruit is heard over the neck. What is the cause of HTN?

▼ Answer

Renal Artery Stenosis (Renal Artery Atherosclerosis)

- Qs can be this tricky. Patient has carotid atherosclerosis (the neck bruit), so he definitely has atherosclerosis in other vessels.
- RAS causing decreased renal flow → increased RAAS activation → HTN

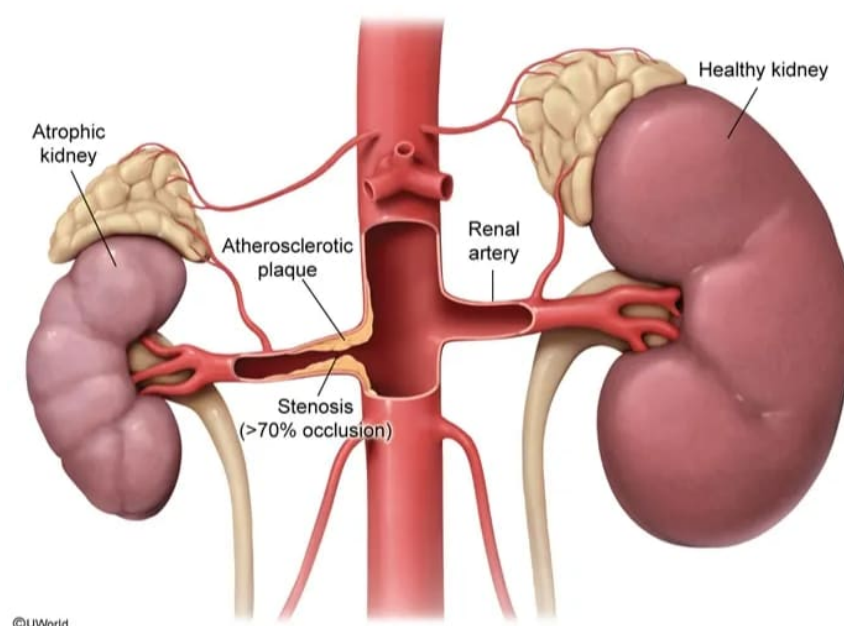


- **Captopril renal radionuclide scan** essentially assesses the **renal perfusion** before vs after captopril.
 - We know that if we have RAS + an ACEi is given, we get **worsening of renal function / decreased perfusion**. This is essentially what we have here.



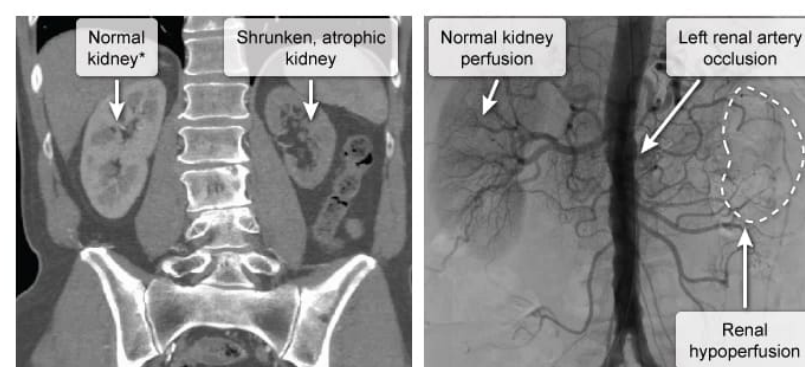
The intake of ACEi/ARBs causes decreased constriction of the efferent renal arterioles → decreased GFR, causing renin levels to rise.

- This sentence is super HY. USMLE will give yap nonsense to you, then mention that **creatinine/renin levels increased after giving ACEi/ARBs**, and the **answer will be RAS or FMD**



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Renal artery stenosis



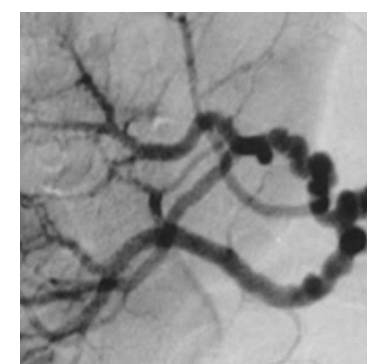
*Enlarged due to compensatory hypertrophy

Renal angiogram

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▼ Other Choices + HY Related Notes

- **Choice A → Aortitis** (inflammation of the aorta) is usually seen with:
 - **Syphilis**
 - “Tree-Bark” Aortitis
 - **Tertiary Syphilis** → **endarteritis obliterans** (obliteration of arteries) of vasa vasorum → ischemia of aortic walls → **Aneurysm formation with characteristic tree-bark appearance**
 - **Takayasu-Arteritis**
 - “Pulseless disease”
 - Usually affects middle-age Asian women
 - Inflammation of aorta & its branches causes weak upper limb pulse, myalgia, and sometimes visual disturbances
 - ESR is elevated
 - Biopsy shows: Transmural granulomatous thickening & narrowing of aortic arch and its proximal branches
- **Choice B → Left Renal Artery Aneurysm**
 - Tricky choice.
 - Atherosclerosis causes aneurysm
 - Aneurysms do cause HTN & an audible bruit
 - Why is it not the answer then? The captopril renal radionuclide.
 - As we said, it’s diagnostic for RAS not aneurysms.
 - CT Angiography is diagnostic for Aneurysms.
- **Choice D → Left Renal Artery Dysplasia**
 - Refers to **Fibromuscular dysplasia**
 - **Autoimmune disease that usually affects young women with no hx of cardiovascular disease**
 - Q present it as a young woman with no medical hx that got BP 180/120 & asks what the diagnosis is.
 - Dysplasia is actually as misnomer, it is hyperplasia.
 - Causes **“String of beads”** appearance on CT angiography.



“String of beads” appearance



Malignant HTN + renal bruit or elevated creatinine in response to ACEi/ARBs?

- 50-60s male + hx of cardiovascular disease / atherosclerosis / smoking?
 - **Atherosclerosis**
- 20-40 female + no hx of cardiovascular disease?
 - **Fibromuscular dysplasia**
- 50-60s female + hx of cardiovascular disease / atherosclerosis / smoking?
 - **Atherosclerosis**



Can you mention 3 HY differences between FMD & PAN?

▼ Answer

Fibromuscular Dysplasia

1. "String of beads" appearance
2. Tunica media hyperplasia
3. Proximal affection

Polyarteritis Nodosum

1. "String of pearls" appearance
2. Fibrinoid necrosis
3. Proximal sparing

- **Choice E → Left renal cell carcinoma**
 - Renal Cell Carcinoma
 - Most common subtype is clear cell carcinoma
 - **Weight loss + hematuria + flank pain in old smoker**
 - Associated paraneoplastic syndromes:
 1. **EPO → Polycythemia**
 2. **PTHrP → Hypercalcemia**

Q19

A 60-year-old woman with psoriasis comes to the physician because of progressive joint swelling and pain of her hands during the past 6 months. Physical examination shows joint tenderness and effusion. She asks for treatment with methoxsalen and ultraviolet A light (PUVA), which worked well for her similarly afflicted sister. This therapy would be contraindicated in this patient if she were to have which of the following conditions?

- A. Cataracts
- B. Congestive heart failure
- C. Porphyrria cutanea tarda
- D. T-cell lymphoma
- E. Type 2 diabetes mellitus
- F. Vitiligo

▼ Explanation

- **Answer is C (Porphyria cutanea tarda)**
 - Patient has psoriasis & asks the doctor to treat her with PUVA (UV A light).
 - PCT is one of the heme pathway disorders that is characterized by **photosensitivity**.
 - When you are photosensitive, UVA light treatment is contraindicated because it will worsen your condition & cause **severe skin damage & blisters**.



What do you know about Porphyria Cutanea Tarda?

▼ Answer

- **Enzyme deficiency** → **Uroporphyrinogen Decarboxylase**
- **C/P** → **Photosensitivity** that causes **blistering hyperpigmentation**
- **Exacerbating factors** → **Ethanol & UV light**
- **Associated with** → **HCV**

▼ Other Choices

- **Choice A** → PUVA can in fact worsen cataract, however, she will be using the PUVA therapy on her skin + will probably have eye protection on.
- **Choices B & E** → No relation whatsoever.
- **Choice F** → PUVA is actually one of the treatment options for vitiligo.

Q20

An 83-year-old man comes to the physician because of a 3-day history of painful blisters on his torso. His pulse is 72/min, and blood pressure is 125/85 mm Hg. Physical examination shows numerous 0.5- to 1-cm, clear-fluid-filled, tense blisters over the trunk. Histopathologic examination of a biopsy specimen from the affected area shows a subepidermal blister. Production of autoantibodies directed against which of the following structures best explains the findings in this patient?

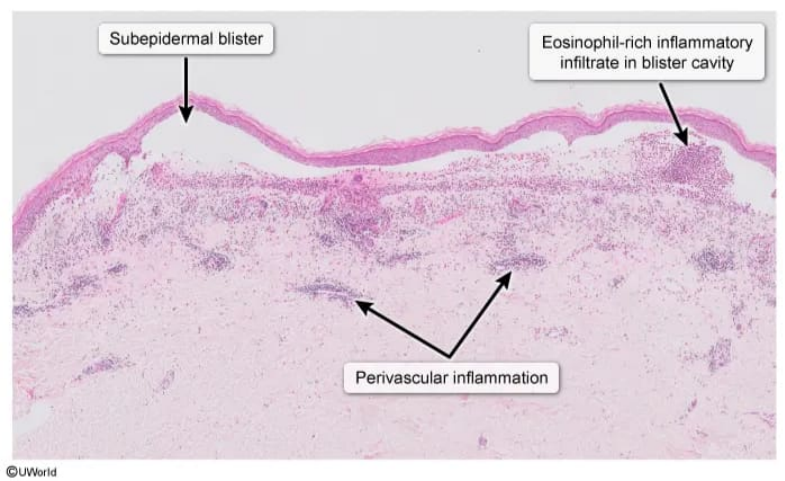
- A. Bullous pemphigoid antigen
- B. Collagen, type VII
- C. Cytokeratin
- D. Desmoplakin
- E. Plakoglobin

▼ Explanation

- Answer is A (Bullous pemphigoid antigen)
 - IMP Clues:
 - 83-year-old man (elderly patient)
 - blisters (okay, get the DD starting in mind)
 - numerous clear-fluid-filled TENSE blisters (okay elderly + tense blisters that Bullous pemphigoid)
 - Biopsy shows subepithelial blisters (indeed, confirms our diagnosis)
- Dx → Bullous pemphigoid
 - Q asks production of autoantibodies directed against what structure is the cause.
 - Well, we know it is **hemi-desmosomes**, but looking at the answer choices, **Bullous pemphigoid antigen** makes the most sense.

Hemi-desmosomes bind the basement membrane to the epidermis, so autoimmune targeting of the hemi-desmosomes results in:

Subepithelial (below the epithelium) blistering/inflammatory infiltration:



How to confirm the diagnosis for sure?

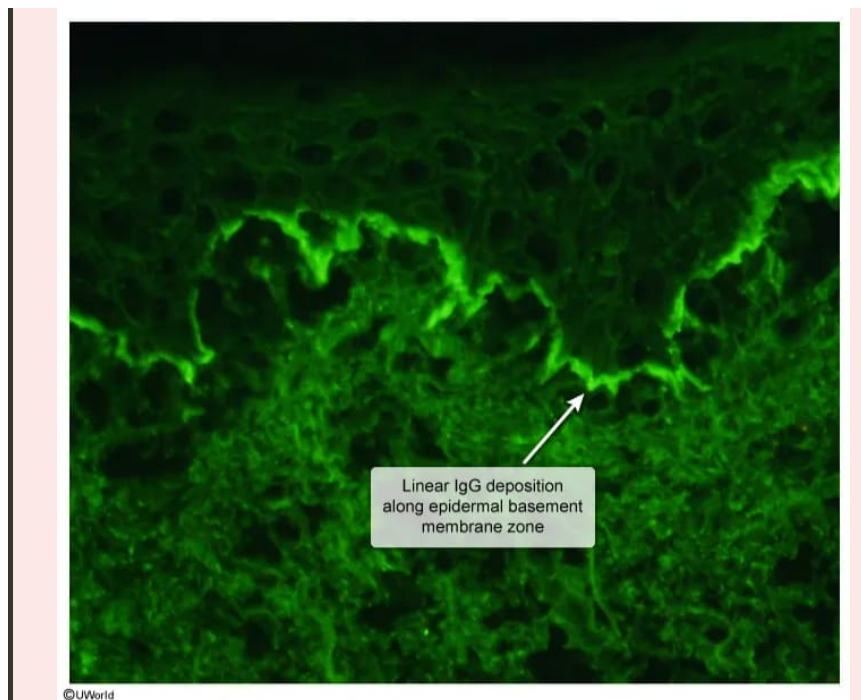
- Immunofluorescence shows **LINEAR** Deposits along basement membrane (as hemidesmosomes spans the length of the basement membrane)

? Can you mention 6 differences between Bullous Pemphigoid & Pemphigus Vulgaris?

▼ Answer

Features	Bullous Pemphigoid	Pemphigus Vulgaris
Oral Involvement	No	Yes
Nikolsky Sign	Negative	Positive
Autoantibodies Against	Hemidesmosomes	Desmosomes (Desmoglein)
Immunofluorescence	Linear deposition along basement membrane	Net-like (reticular) intercellular pattern
Blisters	Tense, subepidermal blisters	Flaccid, intraepidermal blisters
Ages	Elderly	Middle-aged adults

Bullous Pemphigoid Blisters are Tense:



▼ Other Choices

I don't think any of these are HY. Let's try to decipher these weird proteins.

- **Choice B** → *Collagen type 7* refers to **epidermolysis bullosa**
 - It is one of the LY-er diseases on the USMLE (you might see a Q or 2 on ALL of NBMEs)
 - Qs might give you a child with **fragile skin that blisters** + Qs says that there is some sort of **"anchoring fibrils" defect**.
 - Causes **sub-lamina blisters**. That's all you need to know.
- **Choice C** → "Cyto" for cytoskeleton & "Keratin" for keratinocytes
 - So, a structural protein in keratinocytes.
 - Nothing to do with blisters here.
- **Choice D** → "Desmo" sounds like desmosomes. We know that elderly + tense subepidermal blisters are hemi-desmosomes so automatically eliminated.
 - Actually, has nothing to do with Pemphigus Vulgaris.
 - Apparently causes some sort of cardiomyopathy & sometimes a subtype of epidermolysis bullosa. Garbage. Forget about it.
- **Choice E** → Another garbage answer choice. I'm putting it here for completeness sake.
 - A protein that is part of desmosomes
 - Desmosomes are found in skin & heart. So, abnormalities in plakoglobin causes skin + heart disease. Garbage for USMLE.

Q21

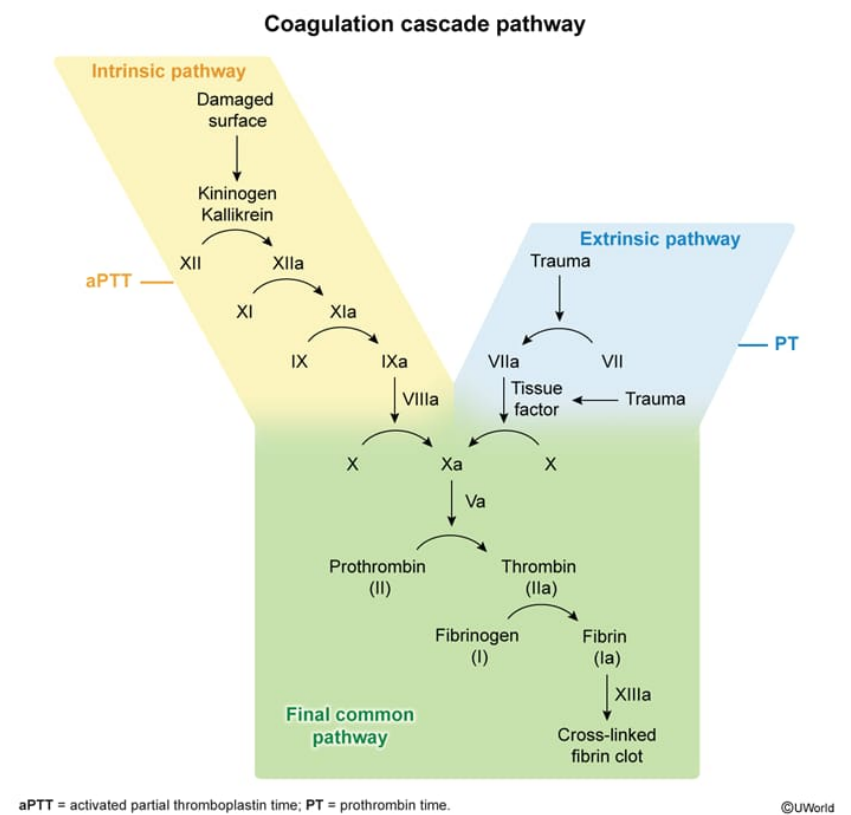
A 3-year-old boy is brought to the emergency department because of a nosebleed that cannot be stopped. Platelet count and thrombin time are within the reference range; partial thromboplastin and prothrombin times are prolonged. Which of the following coagulation processes is most likely to be affected?

- Activation of factor XII (Hageman factor)
- Hydrolysis of phosphatidylinositol 4,5-bisphosphate
- Synthesis of factor VIII (antihemophilic factor)
- Synthesis of factor IX (plasma thromboplastin component)
- Synthesis of factor X (Stuart factor)

▼ Explanation

- **Answer is E (Synthesis of factor X (Stuart factor))**
 - **IMP Clues:**
 - PLT count & Thrombin time are within reference range (okay so not a PLT disorder & Factor II is normal)
 - PTT & PT are prolonged (okay so both intrinsic & extrinsic pathways are affected. Must be something in the common pathways)

- **Dx → Factor X deficiency**
 - Common pathways include **factor X, II, and I.**
 - Q says **factor II is normal**
 - This leaves us with either **Factor I (not in choices)** or **Factor X (Choice E)**



▼ Other Choices

- **Choice A** → Affection of factor XII would cause prolonged PTT (intrinsic pathway) only.
- **Choice B** → Affection of PIP2 would cause PLT dysfunction (PLT are normal in the vignette)
- **Choice C** → Deficiency of factor VIII is **Hemophilia A**, which would cause prolonged PTT (intrinsic pathway) only.
- **Choice D** → Deficiency of factor IX is **Hemophilia B**, which would cause prolonged PTT (intrinsic pathway) only.

Q22

A 41-year-old man comes to the physician because of a 6-week history of numbness of his right hand. He works in construction. Sensation to pinprick is decreased in the thumb and over a portion of the anterior forearm. The physician suspects that there is compression of the C5 and C6 spinal nerves as they exit the spinal canal. Weakness of which of the following movements on the right is most likely to confirm the diagnosis?

- Abduction of the upper extremity
- Adduction of the index finger
- Elevation of the shoulder
- Extension of the index finger
- Flexion of the wrist

▼ Explanation

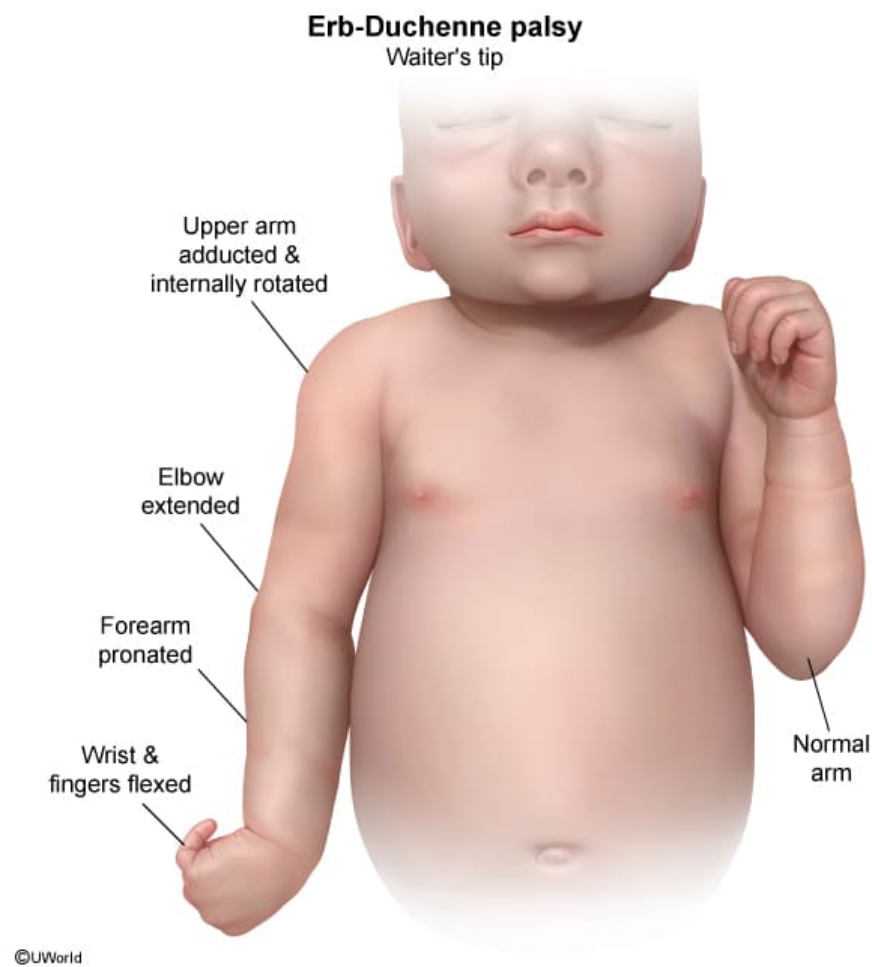
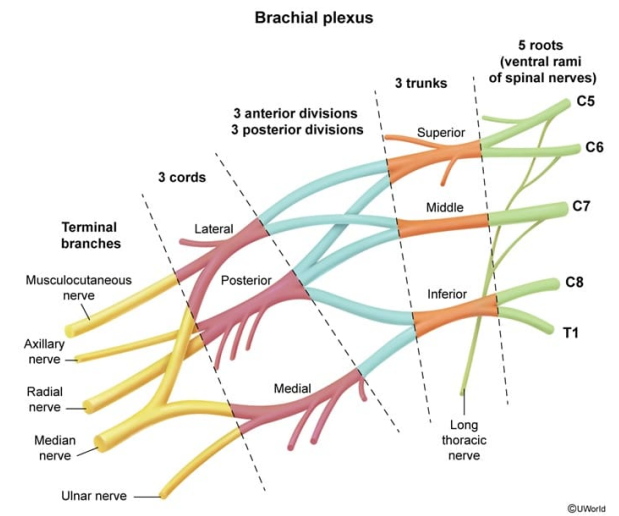
- **Answer is A (Abduction of the upper extremities)**
 - **IMP Clues:**
 - Numbness of right hand + decreased sensation to pinprick in the thumb & portion of anterior forearm (peripheral neuropathy affecting hands + arm)
 - Compression of C5 & C6 spinal nerves as they exit spinal canal (stenosis of spinal foramina)

- **Dx → C5-C6 Spinal Foramina Stenosis**

- Okay, knowing the brachial plexus anatomy is important (sadly) because you will see plenty of Qs testing this concept.

- **Brachial plexus is made up of C5-T1**

- C5-C6 injury = Upper brachial plexus injury = **Erb's palsy**
 - Arms are classically **adducted, internally rotated, and pronated**
 - So, you **CANNOT**:
 - **ABDUCT** your arm (loss of deltoids function → *axillary nerve*)
 - **EXTERNALLY rotate** your arm (loss of teres minor function → *axillary nerve*)
 - **SUPINATE** your hands (loss of biceps function → *musculocutaneous nerve*)



▼ **Other Choices**

- **Choice B** → Ulnar Nerve
- **Choice C** → Spinal Accessory Nerve
- **Choice D** → Radial nerve
- **Choice E** → Median Nerve

Q23

A 4-month-old boy is diagnosed with a rare autosomal recessive skeletal dysplasia involving abnormal endochondral bone formation. Genetic analysis shows null mutations in a gene for a protein that controls the traffic of vesicles into the Golgi complex. Electron microscopy of this patient's cells will most likely show which of the following findings?

- A. Decreased rough endoplasmic reticulum
- B. Decreased smooth endoplasmic reticulum
- C. Dilated rough endoplasmic reticulum
- D. Increased smooth endoplasmic reticulum
- E. Large lysosomes
- F. Small lysosomes

▼ **Explanation**

- **Answer is C (Dilated Rough Endoplasmic Reticulum)**

- Q asks what will most likely be seen in a patient with a mutation that traffics vesicles into Golgi complex.
- To tackle this Q, let's start by the basics
 - What's the function of the Golgi complex?
 - **Processes & packages proteins for delivery to other parts of the cells**
 - From where & how does these proteins reach the Golgi complex?
 - **From the Rough ER, proteins get carried by COP2 & sent off to Golgi.**
 - If this protein is mutated, what will happen?
 - **RER will retain all these protein vesicles & dilate.**
- So, answer is **Dilated Rough Endoplasmic Reticulum (Choice C)**



Rapid Fire Questions:

What is the protein responsible for delivery of vesicles from RER to Golgi?

▼ **Answer**
COP2

- Stupid trick I use to remember which is: Rough ER (2) to Golgi (1)
- So, COP 2
- Rough ER is 2 words, and Golgi is 1 word

What is the protein responsible for delivery of vesicle from Golgi to RER?

▼ **Answer**
COP1

- The second part of the stupid trick is: Golgi (1) to Rough ER (2)
- So, COP 1
- Golgi is 1 word, and Rough ER is 2 words

What mediates transport between Golgi & Endosomes?

▼ **Answer**
Clathrin-coated vesicles

Q24

An 80-year-old woman comes to the emergency department because of a 2-day history of "feeling funny." She says, "I have lost my pep." She has never been admitted to the hospital, but she has a history of poorly controlled hypertension. She just started a medication 2 weeks ago but does not recall the name. Her blood pressure is 130/85 mm Hg. Physical examination shows no abnormalities. Her serum potassium concentration is 3 mEq/L. Which of the following drugs is the most likely cause of this patient's condition?

- A. Clonidine
- B. Hydrochlorothiazide
- C. Lisinopril
- D. Metoprolol
- E. Triamterene

▼ **Explanation**

- **Answer is B (Hydrochlorothiazide)**
 - **IMP Clues:**
 - Hx of poorly controlled hypertension + started a medication 2 week ago (so probably her "feeling funny" is caused by a side effect of a HTN medication)
 - Serum potassium concentration is 3mEq/L (hypokalemia, very important clue)
- **Dx → Hypokalemia secondary to thiazide diuretics**
 - Thiazide & Loop diuretics (not in choices) both cause **hypokalemia**
 - Both cause **diuresis** → **less** blood volume → **Aldosterone** secretion **increases** → **Reabsorption** of **sodium** for **potassium & hydrogen**
 - Net effect:
 1. **Sodium & Water reabsorption**
 2. **Hypokalemia**
 3. **Metabolic alkalosis**



Patient took a diuretic + developed metabolic acidosis. What is the medication responsible?

▼ **Answer**
Acetazolamide (Carbonic Anhydrase Inhibitor)

- Hence the term “**contraction alkalosis**”. Contraction of blood volume (by diuretics) causes alkalosis.

The Side Effects of Thiazides are Super-HY, so let's review them “**hyperGLUC + others**”

1. Hyper**G**lycemia
2. Hyper**L**ipidemia
3. Hyper**U**ricemia
4. Hyper**C**alcemia
5. Others → **Hyperprolactinemia**, **Hyponatremia**, **Hypokalemia**, **Metabolic alkalosis**, **Lithium toxicity**

? Why should you worry when using thiazides with a patient with bipolar disorder?

▼ Answer

If thiazides are taken with lithium → **Lithium Toxicity**



Thiazides block Na/Cl channels in the Distal Convulated Tubules

▼ Other Choices

- **Choice A** → **Clonidine** is an *alpha 2 agonist* used for HTN
 - alpha-2 agonists cause decreased sympathetic stimulation
 - Decreased HR
 - Decreased BP
 - Decreased PVR
 - Side effects: Hypotension - Bradycardia
- **Choice C** → **Lisinopril** is an *ACE inhibitor*
 - Inhibits ACE (in lungs) → decreased AT-II activity
 - Decreased BP
 - Decreased Aldosterone secretion (high potassium, less salt-water retention)
 - Side effects: Dry cough - Angioedema - Hyperkalemia - Hypotension - Renal failure (if take in patients with bilateral RAS) - Teratogenic (fetal renal abnormalities)

? Patient taking ACEi + develops dry cough. What is the cause of the cough? To which medication should the patient be switched to?

▼ Answer

The cough is caused by bradykinin excess

- ACE breaks down bradykinin. If inhibited → bradykinin accumulates & causes the cough.

Patient must be switched to ARBs

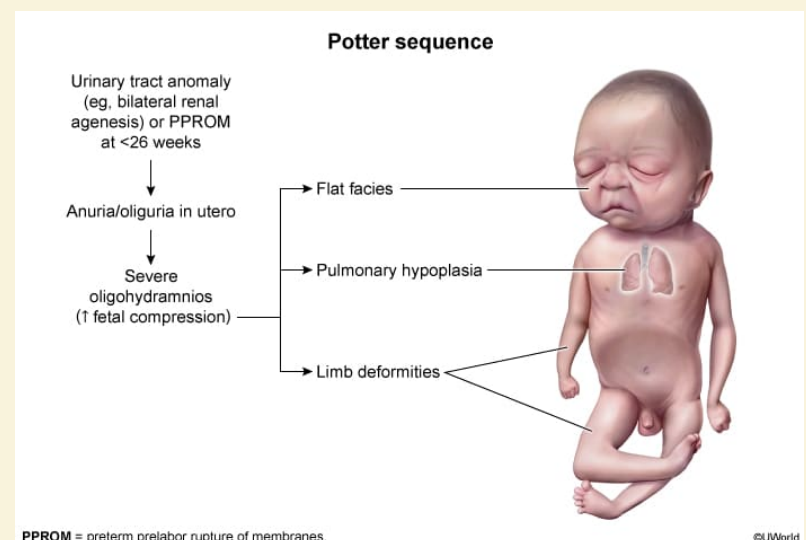
- ARBs just block the AT-II receptors without interfering with ACE

? A female with HTN managed with lisinopril discovers that she is 18 weeks pregnant. What would the expected anomaly be?

▼ Answer

As we know, ACEi / ARBs causes **fetal renal dysfunction**.

- Fetus cannot excrete urine → **Oligohydramnios**
- Oligohydramnios causes **Potter Sequence**
 - Flattened Face & Low-set ears
 - **Pulmonary hypoplasia (cause of death)**
 - Club feet & Limb contracture



- **Choice D** → **Metoprolol** is a *cardio-selective Beta Blocker*
 - Blocks Beta 1-adrenergic receptors in heart & kidney

- heart → Bradycardia & increases diastolic filling (which increases COP by Starling's law)
- Kidney → inhibits renin production → decreased RAAS activation → decreased BP
 - decreased RAAS = decreased Aldosterone = high potassium
- Side effects: Bradycardia & Heart block - hypotension - bronchospasm (if overdosed, losses selectively)



Beta Blockers:

starting with the letter **A → M** are **cardio-selective**

Starting from the letter **N → end are NOT cardio-selective**

- **Choice E → Triamterene** is a *potassium sparing diuretic* which causes hyperkalemia as a side effect.

Q25

The gene that codes for a protein normally found in the endoplasmic reticulum is mutated, and the protein produced by the mutated gene remains in the cytoplasm. Which of the following mutations is most likely to impair the transport of the protein into the endoplasmic reticulum?

- A. Conversion of a proline residue to a hydroxyproline residue
- B. Deletion of a hydrophobic amino acid sequence from the N terminus
- C. Disruption of a lysine-rich amino acid sequence near the middle of the protein
- D. Loss of a glycosylation site
- E. Premature termination of the polypeptide chain near the C terminus

▼ **Explanation**

- **Answer is B (Deletion of a hydrophobic amino acid sequence from the N terminus)**
 - Biochemistry garbage.
 - Know the following and move on:
 - **N-terminus** is what directs **intracellular translocation**.
 - N-terminus is detected by **Signal Recognition Particle (SRP)** and thus transported into the **ER**.
 - If deformed N-terminus = no recognition by SRP = cannot go to ER = retained in cytoplasm

▼ **Other Choices**

- **Choice A** → Involved in post-transcriptional modification AFTER the protein is inside the ER. Commonly asked in collagen disorders.
- **Choice C** → Involved in Intranuclear translation.
- **Choice D** → Glycosylation happens in ER. Protein must have entered already so not answer.
- **Choice E** → C-terminus is involved in trafficking proteins from ER to other organelles.

Q26

A study was performed that evaluated the relationship between television exposure and the development of learning disability in children. Children were divided into two groups based on the presence or absence of learning disabilities. The investigators asked many questions about the children's exposure to television. By comparing the likelihood of television exposure in these two groups, the investigators estimated the relationship between learning disability and television exposure using which of the following measures?

- A. Attack rate
- B. Incidence
- C. Odds ratio
- D. Prevalence
- E. Relative risk

▼ Explanation

- **Answer is C (Odd Ratio)**
 - Easy biostat Q. You should always get these correct. Easy points.
 - Q says a study evaluated relationship between tv exposure & development of learning disability in children, which were divided into 2 groups **based on presence or absence of learning disabilities**.
 - So basically, we divided the children based on whether they had the **"outcome/disease"** not based on the **"risk factor"**. This is a **case-control study**.
 - Case (children with learning disabilities) & control (normal children). Easy.
 - What is measured in case control studies? **Odd Ratio**.



Knowing these concepts are very HY:

Case-control studies = Odd Ratio

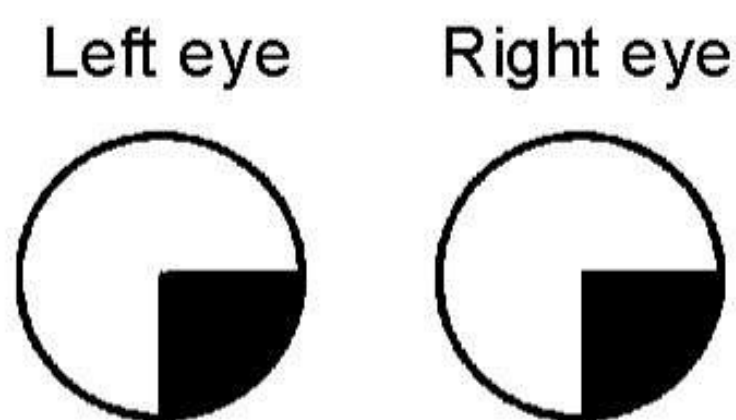
Cohort studies = Relative Risk

▼ Other Choices

- **Choice A** → Garbage.
- **Choice B** → **Incidence** is used **cross-sectional studies** not case-control
 - *Incidence = new cases / healthy population*
- **Choice D** → **Prevalence** is also used in **cross-sectional studies** not case-control
 - *Prevalence = all affected cases / total population*
- **Choice E** → **Relative Risk** is used in **cohort study**. It compares the relative risk of developing the diseases **based on the presence /absence of a specific risk factor**.

Q27

A 75-year-old man has the sudden onset of partial loss of vision. The visual fields are shown. The most likely explanation for the visual field defect is a lesion in which of the following?



- A. Occipital lobe, left
- B. Occipital lobe, right
- C. Optic chiasm
- D. Optic nerve, left
- E. Optic nerve, right
- F. Parietal lobe, left
- G. Parietal lobe, right
- H. Temporal lobe, left
- I. Temporal lobe, right

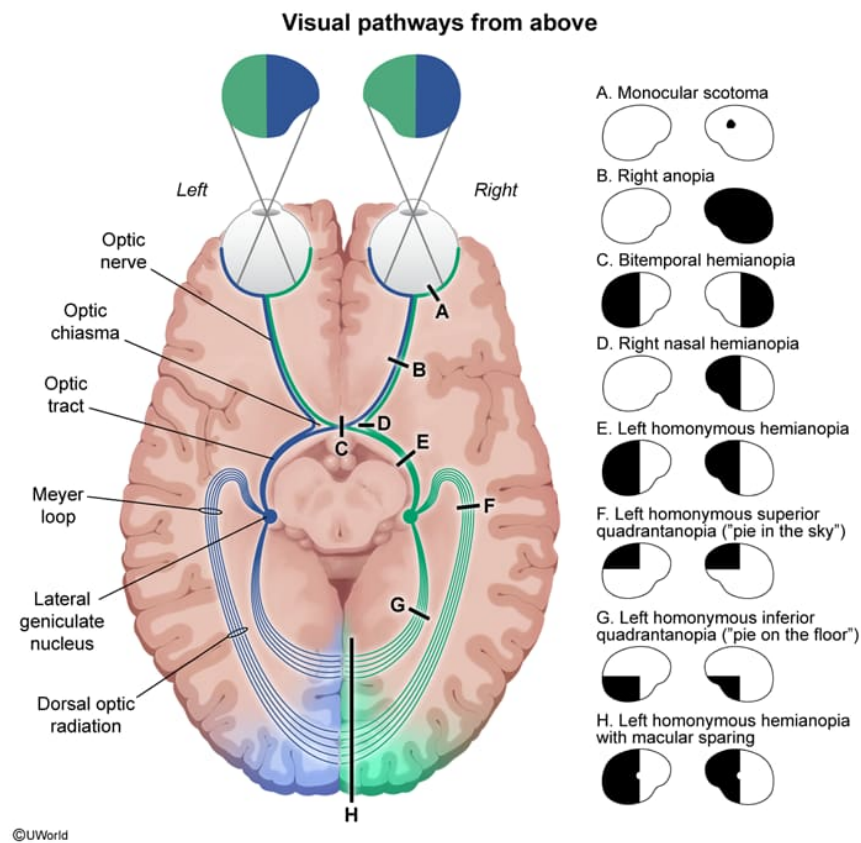
▼ Explanation

- **Answer is F (Parietal lobe, left)**



For Quadrantanopias, reverse everything.

- Right side affected = Left side of brain
- Lower side affected = upper side of brain (parietal)



▼ Other Choices

- **Choice A** → Right homonymous hemianopia with macular sparing (macula had dual supply)
- **Choice B** → Left homonymous hemianopia with macular sparing (macula had dual supply)
- **Choice C** → Bitemporal hemianopia (usually caused by pituitary pathology)
- **Choice D** → Left monocular vision loss
- **Choice E** → Right monocular vision loss
- **Choice G** → Left lower quadrantanopia
- **Choice H** → Right upper quadrantanopia
- **Choice I** → Left upper quadrantanopia

Q28

A 64-year-old man is evaluated for cough, dyspnea, and chest pain. He is afebrile. An x-ray of the chest shows an abnormal density in the hilar region. Lung tissue biopsy shows round cells with little cytoplasm that are about twice the size of lymphocytes. These cells are arranged in infiltrating sheets that have neither glandular nor squamous organization. Which of the following abnormalities in serum is most likely in this patient?

- A. Hyperkalemia
- B. Hyperlipidemia
- C. Hypermagnesemia
- D. Hypoalbuminemia
- E. Hypoglycemia
- F. Hyponatremia

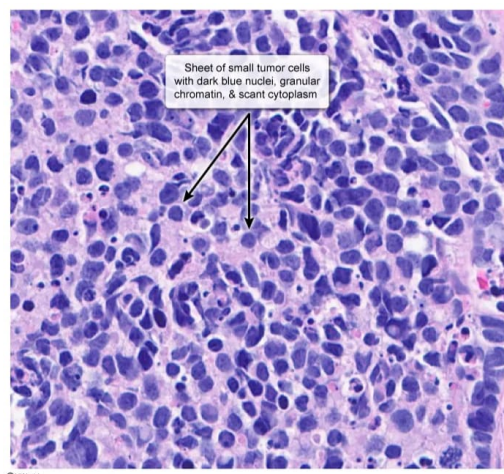
▼ Explanation

- **Answer is F (Hyponatremia)**
 - **IMP Clues:**

- Cough, Dyspnea, and chest pain (okay, so chest symptoms)
- CXR shows abnormal density in hilar region + Lung tissue biopsy shows round cells with little cytoplasm that are twice the size of lymphocytes (okay, sounds like cancer)
- Infiltrating sheets that are neither glandular nor squamous (excludes adenocarcinoma & SCC)

• **Dx → Small Cell Lung Cancer**

- Okay, “*round cells with little cytoplasm that are twice the size of lymphocytes*” is a description of **neuroendocrine tumors**.
- What is the neuroendocrine tumor of the lung? **Small Cell Lung Cancer**. We know it's not adenocarcinoma nor SCC.
- **Small Cell Lung Cancer is a Neuroendocrine tumor of the lung derived from Kulchitsky cells**
 - **Central mass** that most commonly affects **smokers**
 - HY paraneoplastic syndromes are: **SIADH, Cushing's syndrome, Lambert-Eaton Syndrome, and Neurological (Cerebellar degeneration + Encephalitis)**
 - **SIADH** → *Ectopic ADH secretion* → **hyponatremia (Choice F)**
 - **Cushing's syndrome** → *Ectopic ACTH secretion*
 - **Lambert-Eaton Syndrome** → *antibodies against presynaptic voltage-gated calcium channels*
 - **Neurological Syndromes** → *anti-Hu/Yo*
 - Biopsy → “**round cells with little cytoplasm that are twice the size of lymphocytes**” is a description used on NBME, “**small blue round cells**” can also be found (shown below)



Case Scenarios:

- Old man + smoker + muscle weakness + has to swing in chair multiple times before getting up.**
 - Dx** → **Lambert-Eaton Syndrome**
 - Etiology** → Antibodies against **pre-synaptic** voltage-gated **calcium** channels
 - Associated Tumor** → **SCLC**
 - Behavior** → Weakness **improves with repeated movements**
 - Response to Tensilon test (edrophonium)** → **NO** improvement
- Middle-aged female + muscle weakness + diplopia / ptosis / weakness at end of day.**
 - Dx** → **Myasthenia Gravis**
 - Etiology** → Antibodies against **post-synaptic Ach receptors**
 - Associated Tumor** → **Thymoma** “enlarged anterior mediastinal mass”
 - Behavior** → Weakness **improves with rest**
 - Response to Tensilon test (edrophonium)** → **Improvement**

Q29

Scientists have identified the flawed gene that causes about 1 in 6 cases of colon cancer. When the gene is working correctly, the gene product acts in the repair process that discriminates between the template and the newly synthesized DNA strand, correcting mistakes in the newly synthesized strand after replication. In which of the following processes is this gene product most likely to be involved?

- Base-excision repair
- Mismatch repair
- Nucleotide-excision repair
- Photo-induced repair
- Recombinational repair

▼ **Explanation**

- **Answer is B (Mismatch repair)**

- Flawed gene that causes 1 in 6 cases of colon cancer (colon cancer genetics, yuck)
- The gene's product acts in the repair process that discriminates between templates and the newly synthesized DNA strand, correcting the mistakes after replication (okay all this yapping = it compares the new strand to the template & fixes the "mismatching")

• **Dx → HNPCC (Lynch Syndrome)**

- Defects in **mismatch repair genes (MLH1, MSH2, MSH6, PMS1, and PMS2)** [memorize these genes]
- Causes a wide variety of cancers, but most commonly **colorectal carcinoma, endometrial carcinoma, and ovarian cancer**

? Remember the genes responsible for Lynch Syndrome? (DON'T CHEAT)

▼ **Answer**
MLH1, MSH2, MSH6, PMS1, and PMS2



FAP vs HNPCC

Feature	Familial Adenomatous Polyposis (FAP)	Lynch Syndrome (HNPCC)
Genetics	APC gene on chromosome 5 (tumor suppressor gene)	MLH1, MSH2, MSH6, PMS1, PMS2 (DNA mismatch repair)
Inheritance	AD	AD
Number of Polyps	Hundreds-thousands of polyps	Few polyps
Cancer Risk	100% risk of colorectal cancer by age 40	80% risk of colorectal cancer
Age of Onset	Teens	Usually after age 30
Location of CRC	Left-sided (rectosigmoid)	Right-sided (proximal colon)

▼ **Other Choices**

- **Choice A** → Choose **Base-excision** repair if Q mentions **glycosylases**
- **Choice C** → Choose **Nucleotide-excision repair** if Q mentions **pyrimidine-pyrimidine dimers, UV light, or Xeroderma pigmentosum**.
 - Q can give you a person with a skin cancer on a sun-exposed area & ask you what is the cause of the cancer?
 - Answer will be Nucleotide-excision repair defect
- **Choice D** → probably also refers to **pyrimidine-pyrimidine dimers & UV light**
- **Choice E** → **Recombinant dsDNA repair defects** refer to **BRCA & Fanconi anemia**

Q30

A 34-year-old woman comes to the physician because of a 6-week history of fatigue and shortness of breath. Physical examination shows mild cyanosis and clubbing. Cardiac examination shows fixed splitting of S₂. A midsystolic pulmonary ejection murmur is heard. Echocardiography shows a congenital heart defect, and the patient undergoes surgical repair. The patient is at greatest risk for intraoperative injury to which of the following?

- A. Atrioventricular bundle
- B. Coronary plexus
- C. Coronary sinus
- D. Purkinje fibers
- E. Sinoatrial node

▼ **Explanation**

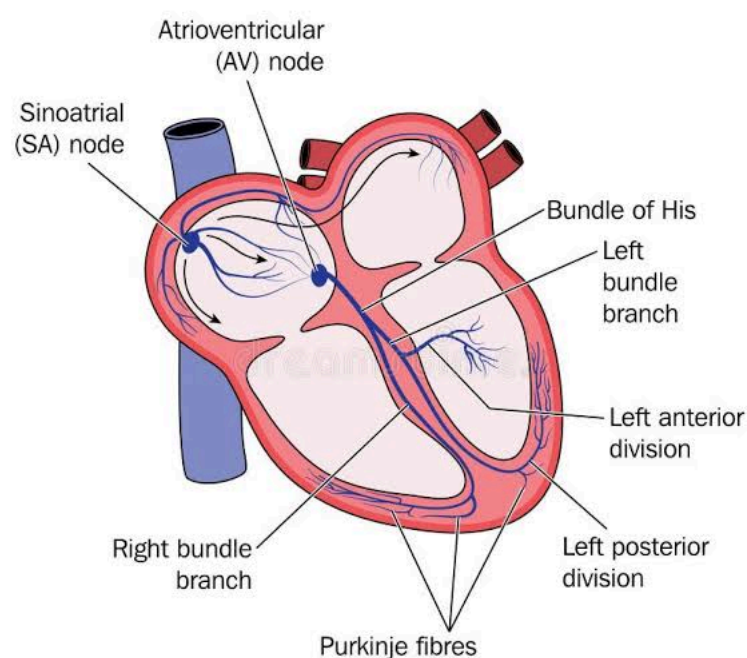
- **Answer is A (Atrioventricular Bundle)**
 - **IMP Clues:**
 - Fixed Splitting of S₂ (one you see this, you know it is ASD)
 - Midsystolic pulmonary ejection murmur (I will explain later)
 - ECHO shows congenital heart defect (ASD, as we said)
- **Dx → ASD**

- Q asks what is at greatest risk for intraoperative injury during surgical repair of the ASD. Okay what's in the location of the ASD?

▪ **The AV bundle.**

"The ejection systolic murmur is due to volume overload on the right-side of the heart → increased blood to RA → Increased blood to RV → Increased Blood through the pulmonary valve. This creates a "relative stenosis" of the pulmonary valve"

- It is not stenotic really, but the amount of blood increases & the pulmonary valve area is the same → same effect as if the blood amount was normal & the valve area was decrease (stenotic)



USMLE can give you a cardiac catheterization Q with O2 saturation pattern and ask for the diagnosis:

- The answer will be ASD if O2 sat increases from 70 in SVC → 70 in RA → 80 in RV.
 - The 70/80 are just for demonstration.
 - Increased of O2 sat before reaching the Lungs means there is a connection between the right and left side of the heart.

? If this patient were to develop a DVT for some reason, what is our biggest concern?

▼ Answer

Paradoxical Embolism, causing a stroke.

- DVT → Femoral Vein → Iliac Vein → IVC → RA → LA → LV → Aorta → Carotids → Cerebral Stroke

Normally, it would be RA → RV → Pulmonary artery → Pulmonary Embolism

! Splitting on the USMLE:

- Fixed Wide Splitting ⇒ **Atrial Septal Defect**
- Physiological Splitting ⇒ **Normal Finding** (inspiration causes increased VR, so more blood passes by pulmonary valve so delayed closure sound of P2)
- Wide Splitting ⇒ Delayed **RV** emptying (HY causes are **RBBB, RVH, Pulmonary Stenosis**)
- Paradoxical Splitting ⇒ Delayed **LV** emptying (HY causes are **LBBB, LVH, Aortic Stenosis**)

▼ Other Choices

- **Choice B** → When have you ever heard of coronary plexus injury?? Anyways the plexus runs on the coronary arteries, which are on the outside of the heart. So, No.
- **Choice C** → Runs in the AV groove of the heart & drains into Right atrium

? What vessel carries the most deoxygenated blood in the whole body?

▼ Answer

Coronary Sinus

- **Choice D** → **Purkinje fibers** are found within the ventricles & are the fastest conduction cells in the heart
- **Choice E** → **SA Node** is found in the right atrium near to the SVC

Let's Recap:

- SA Node → Right atrium near SVC
- AV Node → Interatrial septum
- Bundle of His → Interventricular septum (membranous part)

Q31

Which of the following best explains why the use of an anti-idiotypic antibody is inappropriate as a therapeutic reagent for the treatment of a patient with multiple myeloma?

- A. An insufficient quantity of the immunogen is available to generate the reagent
- B. Myeloma cells are not susceptible to antibody-dependent cell-mediated cytotoxicity
- C. Myeloma cells can be killed only in vivo by cytotoxic lymphocytes
- D. Myeloma cells do not have membrane-bound surface immunoglobulin
- E. Myeloma cells do not have tumor-specific antigens on their surfaces

▼ Explanation

- **Answer is D (Myeloma cells do not have membrane-bound surface immunoglobulin)**
 - Okay, this will be by far the hardest Q to understand on this block. Get a coffee & strap in.
 - First, what is multiple myeloma?
 - Essentially, it is cancer of **plasma cells**.
 - What are plasma cells and where do they come from?
 - Essentially, they are mature B-cells that secrete millions of **immunoglobulins**
 - How are immature B-cell different from mature plasma cells?
 - B-cells have this receptor bound to its membrane called **B-cell Receptor (BCR)**
 - When the BCR binds antigens, it stimulates the B-cell to mature into plasma cells that secrete immunoglobulins
 - The formed plasma cells have **NO** membrane receptor that binds to antigens. It just secretes immunoglobulins.
 - Now that we know what we are working with, let's look at the Q.
 - **What's anti-idiotypic antibodies?**
 - An idioype is a binding site on the receptor
 - Anti-idiotypic antibodies are antibodies that bind to that binding site. Easy.
 - **Q asks why the use of anti-idiotypic antibodies would *NOT WORK* with multiple myeloma.**
 - Well, as we said in multiple myeloma (cancer of plasma cells), the plasma cells have **NO** idioype binding site on its membrane (unlike the immature B-cells).
 - So, the answer is **"Myeloma cells do not have membrane-bound surface immunoglobulin"** I really hope you understood the concept.

▼ Other Choices

- **Choice A** → First this doesn't explain why the anti-idiotypic antibodies do not work for MM. Second, Plasma cells produce MILLIONS of immunoglobulins so saying "insufficient quantity" is just absolutely incorrect.
- **Choice B** → Antibody-dependent cell-mediated cytotoxicity is the method that NK cells use to kill cells. Antibodies bind to a cell receptor (Antibody-dependent) then NK cells come and kill the cell (cell-mediated cytotoxicity).
 - MM cells can be killed with Antibody-dependent cell-mediated cytotoxicity but not by the antibodies binding to the idiotypic binding site
 - This has nothing to do with anti-idiotypic antibodies
- **Choice C** → "killed ONLY in vivo by cytotoxic lymphocytes" is incorrect. There are multiple treatment regimens for MM. Some examples are proteasome inhibitors, chemotherapy, or even NK cells cytotoxicity
- **Choice E** → They do have tumor specific antigens on their surfaces that can be targeted by some monoclonal antibodies. It's just not the idioype binding site we are discussing here.

Q32

A 70-year-old woman has had recurrent pneumonia, fatigue, and weight loss for the past 3 months. Serum thyroxine (T_4) and thyroid-stimulating hormone (TSH) concentrations are within the reference range, and serum triiodothyronine (T_3) concentration is decreased. Serum TSH concentration increases after the administration of thyrotropin-releasing hormone. Which of the following is the most likely diagnosis?

- A. Euthyroid sick syndrome
- B. Hypopituitarism
- C. Hypothalamic abnormality
- D. Primary hypothyroidism
- E. Thyroid neoplasm

▼ **Explanation**

• **Answer is A (Euthyroid Sick Syndrome)**

◦ **IMP Clues:**

- recurrent pneumonia, fatigue, and weight loss (patient is sick)
- Serum T4 & TSH concentrations are within reference ranges (normal T4 & TSH)
- Serum T3 concentration is decreased (isolated decrease in T3)
- TSH concentration increases after administration of TRH (pituitary working fine, helps in exclusion)

◦ **Dx → Euthyroid Sick Syndrome**

- Essentially, when you get sick or your body is put under a lot of stress. As a result, **stress hormones (Cortisol - Catecholamines - Glucagon) & cytokines (IL-1, IL-6, TNF-alpha)** increase.
- The effects of these hormones & cytokines on thyroid homeostasis are:
 - **Suppression of 5-deiodinase enzyme which converts T4 into T3 peripherally.**
 - T4 is then shunted into **reverse T3 (rT3) production**, essentially an inactive form of T3.
 - The levels of T4 essentially stays the same, so no change in TSH levels.
- This explains the labs of euthyroid sick syndrome:
 - **LOW T3**
 - **HIGH rT3**
 - **NORMAL T4**
 - **NORMAL TSH**
- So, you see a sick patient with normal thyroid labs except a low T3 and/or high rT3 think euthyroid sick syndrome. Easy right?

▼ **Other Choices**

- **Choice B →** Hypopituitarism would result in decreased TSH (released from pituitary), which would cause a decrease in T4 & T3. Additionally, other pituitary-released hormones would be affected. Q also mentioned that giving TRH caused an increase in TSH levels, so the pituitary is just fine.
- **Choice C →** Hypothalamus produces TRH. If the hypothalamus is abnormal, then TRH release would be affected. This would cause a low TSH, Low T4, and Low T3.
- **Choice D →** Primary Hypothyroidism means the thyroid gland itself is not working. We would have Low T4 & T3. This would cause the pituitary to increase TSH release. None of these labs match what we have here.
- **Choice E →** Thyroid neoplasm? If you chose this answer based on “weight loss” and decided to ditch every other single detail in this vignette, then cmon man.

Q33

A 39-year-old woman with rheumatoid arthritis develops proteinuria following treatment with penicillamine. Examination of a specimen obtained on renal biopsy shows diffuse fine granular deposits of IgG and C3 in the glomerular basement membrane. Which of the following is the most likely diagnosis?

- A. Acute poststreptococcal glomerulonephritis
- B. Goodpasture syndrome
- C. IgA nephropathy
- D. Membranoproliferative glomerulonephritis
- E. Membranous glomerulonephritis
- F. Minimal change disease

▼ Explanation

• Answer is E (Membranous Glomerulonephritis)

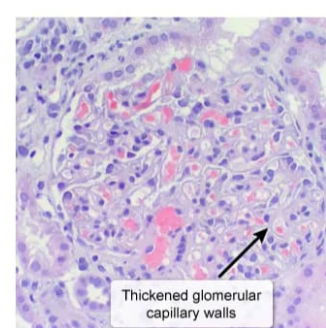
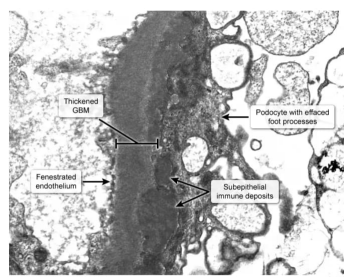
◦ IMP Clues:

- Proteinuria after treatment with penicillamine (HY drug known to cause Nephrotic syndrome (proteinuria) specifically Membranous GN)
- Biopsy shows diffuse fine granular deposits of IgG & C3 on GBM (immune complexes on GBM)

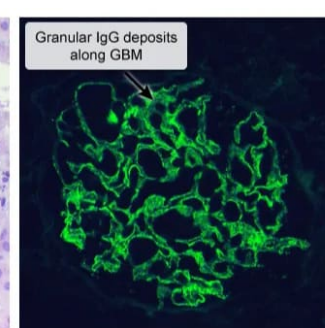
• Dx → Membranous Glomerulonephritis

◦ Causes:

1. Drugs: **Penicillamine - Dapsone - Gold Salts - Sulfonamides**
2. Autoimmune diseases: **PLA2 Receptor antibodies** (Primary Disease)
3. Infections: **HBV - HCV**
4. Malignancies: Breast - Pancreas



Hematoxylin & eosin stain



Immunofluorescence

- **EM: Spike & Dome Appearance of subepithelial deposits**
- **LM: Highly eosinophilic (pink) and inflamed / thickened capillary walls**

▼ Other Choices

• Choice A → PSGN or Proliferative GN or Acute GN

- Hematuria ~2 weeks after onset of **pharyngitis or cellulitis/impetigo** by **streptococcus pyogens**.
- **Biopsy** shows **subepithelial lumpy bumpy appearance**
- HY for arrow Qs: **C3 IS DECREASED** in PSGN

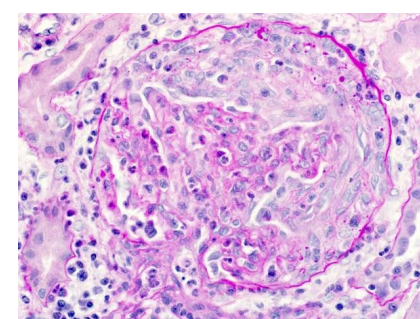
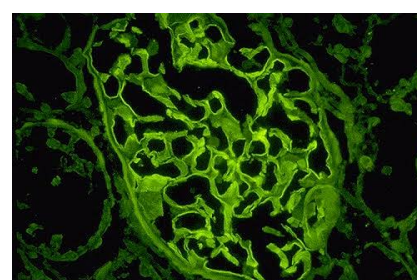
? What indicates bad prognosis long-term in patients with PSGN?

▼ Answer

Adult onset - they are more liable to chronic HTN & renal insufficiency

• Choice B → Goodpasture syndrome is a type 2 hypersensitivity reaction against alpha 3 chains of type 4 collagen ("**234**" mnemonic).

- It causes **linear deposits** on GBM (antibodies attack the whole GBM as it is type 4 collagen) causing **hematuria**
- Lung is also attacked (type 4 collagen) causing **hemoptysis**
- Can progress to **RPGN** (Fibrin crescent formation) → **Acute deterioration of renal function**



HY causes of RPGN include on the USMLE:

- Wegner Granulomatosis
- Microscopic Polyangiitis
- Eosinophilic granulomatosis with polyangiitis (Churg-Strauss)
- Goodpasture syndrome

• Choice C → IgA Nephropathy

- Hematuria ~2 days after onset of **URTI** (usually viral)

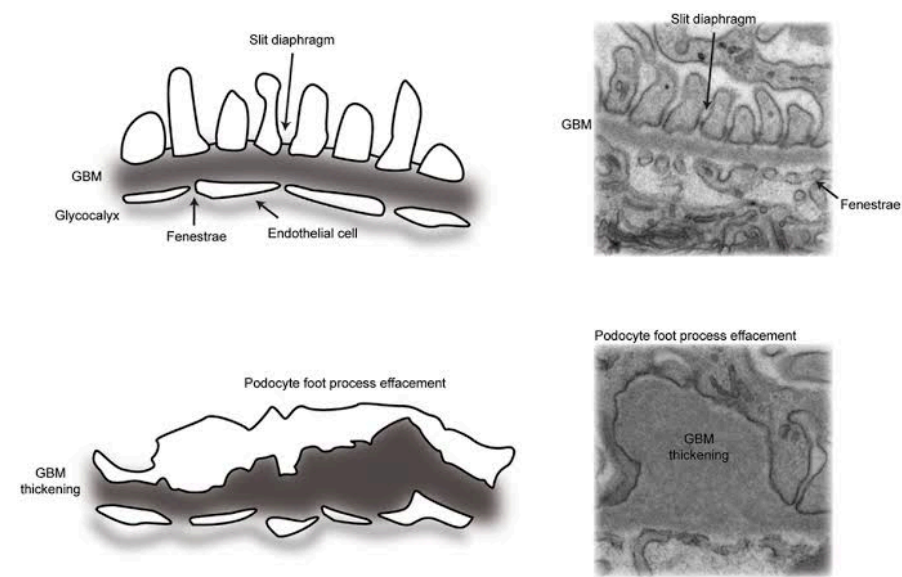
• Choice D → Membranoproliferative GN

- Hematuria in a patient with **Heroin use, HCV, Malignancies**
- **Splitting of GBM** → **Tram-Track appearance**

- **Choice F → Minimal Change Disease**
 - A Child with **proteinuria (NO hematuria here)**
 - Biopsy shows **no deposition- just effacement of podocytes**


Nephrotic & Nephritic Diseases:

Nephritic Syndrome	Nephrotic Syndrome
Post-Streptococcal Glomerulonephritis (PSGN)	Minimal Change Disease
IgA Nephropathy (Berger disease)	Membranous Glomerulonephritis (Membranous GN)
Goodpasture Syndrome	Diabetic Glomerulosclerosis
Alport Syndrome	Renal Amyloidosis
Membranoproliferative GN (MPGN)	Focal Segmental Glomerulosclerosis (FSGS)
Diffuse Proliferative GN (DPGN)	
Rapidly Progressive GN (RPGN)	



Q34

A 78-year-old woman is brought to the emergency department by her husband 1 hour after the sudden onset of severe pain in her left leg. She does not recall any trauma to the area. Physical examination shows the left lower extremity to be cool and pale, with absence of pulses. An ECG shows an irregularly irregular rhythm. Which of the following treatments is most appropriate to provide effective relief from these symptoms?

- A. Oral Aspirin therapy
- B. Oral Bosentan therapy
- C. Oral Clopidogrel therapy
- D. Embolectomy
- E. Fasciotomy

▼ Explanation

- **Answer is D (Embolectomy)**
 - **IMP Clues:**
 - 1hr sudden onset of severe pain in her left leg + cool and pale + absent pulse (okay, clearly this is acute ischemic limb)
 - No hx of trauma
 - ECG shows irregularly irregular rhythm (Atrial Fibrillation, okay so we know where the embolism came from)
- **Dx → Acute Limb Ischemia 2ry to AFib**
 - **AFib causes stasis in atria → mural thrombus formation → embolization**
 - The embolism can go anywhere:
 - Limb = **Acute Limb Ischemia** (what we have here)
 - SMA/IMA = **Acute Mesenteric Ischemia**
 - Retinal Artery = **Central Retinal Artery Occlusion**
 - Cerebral vessels = **Stroke**
 - How do we treat an embolism? we remove it. Either **surgically (Embolectomy)** or via **thrombolysis**


Mitral Stenosis is the biggest risk factor for AFib

▼ Other Choices

- **Choice A → Aspirin** is a **COX1/2 irreversible nonselective inhibitor** that inhibits PLT **aggregation** so decreases risk of thrombus formation
- **Choice B → Bosentan** is **Endothelin-1 Receptor antagonist** used for **Pulmonary HTN**
- **Choice C → Clopidogrel** is a **ADP2Y12 blocker** that *inhibits* activation of **glycoproteins 2a/3b** so decreases PLT **aggregation** so decreases risk of thrombus formation
- **Choice E → Fasciotomy** is used to treat **acute compartment syndrome**.

Q35

A 19-year-old woman comes to the office because of a 1-month history of abdominal pain and severe diarrhea; she has had a 5.4-kg (12-lb) weight loss during this period. She is 168 cm (5 ft 6 in) tall and now weighs 57 kg (126 lb); BMI is 20 kg/m². Physical examination shows no abnormalities. Colonoscopy shows a cobblestone appearance of the colonic mucosa. In addition, a stricture is found in a retroperitoneal portion of the bowel. Which of the following is the most likely location of this stricture?

- A. Cecum
- B. Descending colon
- C. Sigmoid colon
- D. Transverse colon

▼ **Explanation**

- **Answer is B (Descending Colon)**
 - Always start reading the last few lines in the Qs. Which of the following is the most likely location of this stricture? okay sentence before it says: Stricture is found in retroperitoneal portion of the bowel.
 - Okay so essentially the Q is: Which part of the bowel is retroperitoneal?
 - **Answer is Descending Colon.**



Retroperitoneal Structures

Use the mnemonic:

"SADPUCKERS"

- **S = Suprarenal**
- **A = Aorta & IVC**
- **D = Duodenum (except 1st part)**
- **P = Pancreas (except tail)**
- **U = Ureters**
- **C = Colon (Ascending & Descending)**
- **K = Kidney**
- **E = Esophagus**
- **R = Rectum**

Q36

A 10-year-old girl develops fever, malaise, and loss of appetite, followed by excess salivation 6 weeks after exploring a cave with her friends. Over the next week, she develops delirium, seizures, paralysis, and hyperexcitability. The most likely cause of her condition is a virus that has reached the brain by which of the following routes?

- A. Direct spread from olfactory system
- B. Retrograde transport through nerves
- C. Spread from subarachnoid space

- D. Transport via bloodstream
- E. Transport via lymphatics

▼ Explanation

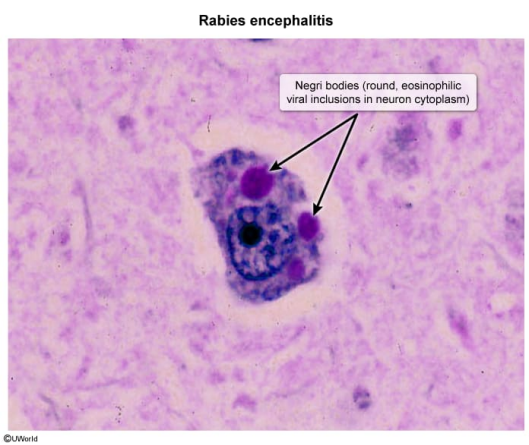
- Answer is B (Retrograde transport through nerves)
 - IMP Clues:
 - Excess Salivation, delirium, seizures, paralysis, and hyperexcitability (CNS symptoms + excess salivation, we know where this is going)
 - Exploring a cave (what is in caves? bats)
 - Dx → Rabies
 - Rhabdovirus:
 - Negative sense single stranded RNA Enveloped Bullet shaped virus with a Helical Capsid
 - Most common transmission is through Bats (in caves usually)
 - Other animals (although less frequent) include foxes, racoons, and skunks
 - Infects post-synaptic motor endplates → retrograde transport in nerve → replicates in motor neurons → travels to dorsal root ganglia → CNS
 - Presents as either:
 - Encephalitis:
 - Hydrophobia (Pathognomonic) [caused by laryngeal spasm]
 - Autonomic instability
 - Spasticity
 - Agitations & AMS
 - Paralysis: Ascending flaccid paralysis

?

What are Negri bodies & where are they seen?

▼ Answer

Eosinophilic Cytoplasmic inclusions in hippocampal pyramidal cells & cerebellar Purkinje cells



Rabies encephalitis

Negri bodies (round, eosinophilic viral inclusions in neuron cytoplasm)



Q37

A 27-year-old man comes to the physician for an examination before starting employment at a plastics factory. He has no history of major medical illness. He has never been sexually active. He has minimal contact with his parents and siblings and has no hobbies. When asked if he feels depressed, he says no. He shrugs in response to congratulations about his new job. Physical examination shows a flat affect. This patient most likely has which of the following types of personality disorders?

- A. Antisocial
- B. Avoidant
- C. Borderline
- D. Schizoid
- E. Schizotypal

▼ Explanation

- **Answer is D (Schizoid)**
 - **IMP Clues:**
 - Never been sexually active & 27-years old (something is suspicious)
 - Minimal contact with his parents and siblings & has as no hobbies (bro is a loner)
 - Not depressed (this shows that he is not annoyed about it / prefers it)
 - Shrugs in response to congratulations about his new job
- **Dx → Schizoid**
 - A person who prefers to be alone & not annoyed by it.
 - The distinction between schizoid & avoidant is very HY:
 - **Schizoid: is distant/alone & actually *prefers* it**
 - (He is happy / doesn't care that he is single)
 - **Avoidant personality disorder: is alone & *HATES* it.**
 - (He is upset & dreams of having a girlfriend)



Q that describes a person with Schizoid (what we have here) **or Schizotypal** (look at Other Choices section) **and ask what is he at risk of developing??**

▼ Answer
Schizophrenia

▼ Other Choices

- **Choice A →** > 18 years old person that commits crimes / starts fights / cheats on tests
 - Must have a history of similar actions before 15 years of age
- **Choice B →** Person desires social interactions but can't engage in it. He has this fear of rejection that stops him from meeting / socializing with people. He still wishes he can.
- **Choice C →** Patient will present with hx of unstable relationships & impulsivity. Will usually have suicidal actions that will not actually kill him/her
- **Choice E →** A person that dresses wildly & thinks wildly. Qs might say a patient that has "multiple layers of clothes" & a deck of card that can read the future. He is not delusional, he's just schizotypal.

Q38

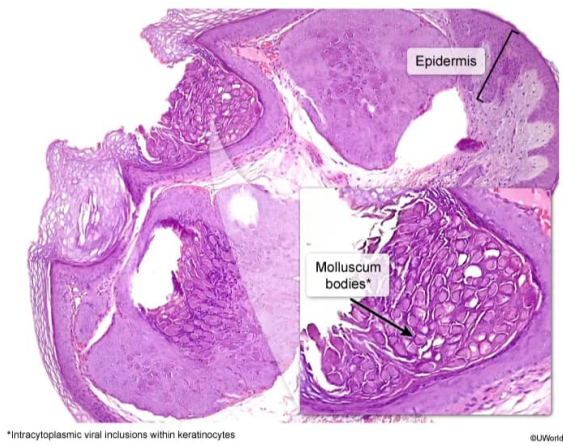
A 6-year-old girl is brought to the physician by her mother because of a 4-day history of round shiny bumps in areas where she has eczema. Her mother remembers seeing similar bumps on a playmate when they attended a pool party 3 weeks ago. The patient has no other symptoms. Physical examination shows firm, smooth, umbilicated papules 2 to 4 mm in diameter in clusters. The causal organism most likely belongs to which of the following viral groups?

- A. Adenovirus
- B. Flavivirus
- C. Herpesvirus
- D. Paramyxovirus
- E. Poxvirus

▼ Explanation

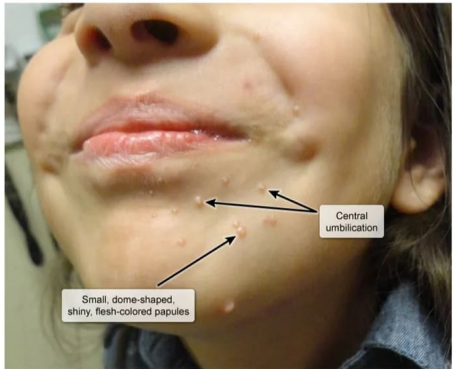
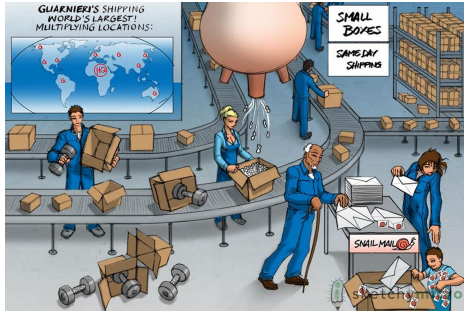
- **Answer is E (Poxvirus)**
 - **IMP Clues:**
 - Round Shiny bumps
 - Similar bumps on a playmate at pool party
 - Firm smooth umbilicated papules in clusters
- **Dx → Molluscum Contagiosum**
 - Pass-level in my opinion. Classic description of molluscum contagiosum: **Umbilicated papules that are shiny, firm, smooth, and round. easy.**
 - Transmits easily through touch (hence the name contagiosum)
 - **One of the Poxviruses.**

- **Largest Double-stranded DNA viruses with dumbbell shaped core**
- Makes their own envelope **(DNA-dependent RNA polymerase)**
- **Guarnieri Bodies** (intracytoplasmic inclusions due to poxvirus)



*Intracytoplasmic viral inclusions within keratinocytes

⚠ Don't confuse it with **Cowdry bodies** (Intranuclear inclusions in HSV)



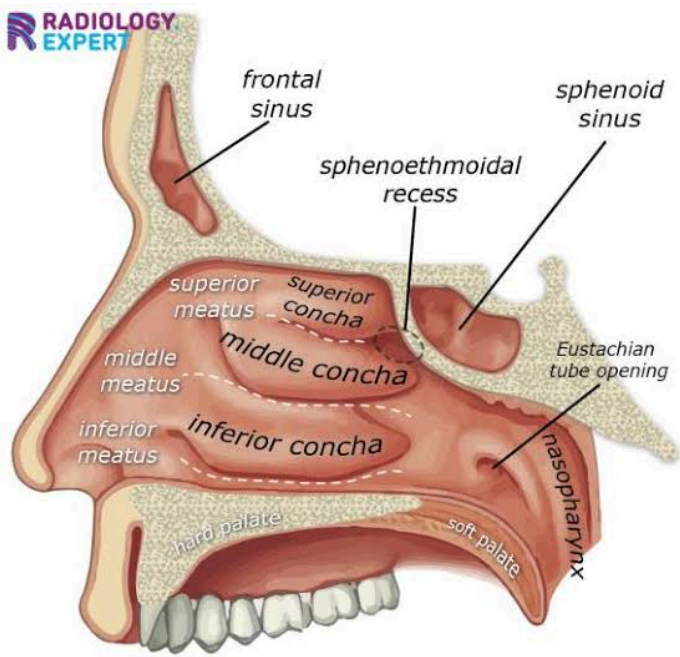
Q39

A 35-year-old woman undergoes flexible nasopharyngoscopy because of chronic episodes of bacterial sinusitis. Purulent discharge from the right sphenothmoidal recess is noted during the procedure. Which of the following best describes the location of this structure?

- A. Anterior to the nasolacrimal duct
- B. Distal to the vestibule
- C. Inferior to the hiatus semilunaris
- D. Posterior to the middle concha
- E. Proximal to the fusion of the hard and soft palates
- F. Superior to the superior concha

▼ Explanation

- Answer is F (Superior to the superior concha)
 - Simple anatomy. **Discharge from sphenothmoidal recess** & asks for its location. Easy.
 - **Superior to the superior concha**



Q40

A 30-year-old man comes to the physician because of a pruritic rash on his lower back for the past week. He underwent liver transplantation 1 year ago and has been maintained on immunosuppressive drugs since then. Physical examination shows vesicles containing clear fluid associated with ulcerated and crusted lesions. Which of the following is the most likely diagnosis?

- A. Bullous pemphigoid
- B. Dermatitis herpetiformis
- C. Erythema multiforme
- D. Secondary syphilis
- E. Varicella-zoster virus infection

▼ **Explanation**

• **Answer is E (Varicella-Zoster Virus Infection)**

◦ **IMP Clues:**

- pruritic rash on lower back for past week (okay distribution is important for DD)
- Underwent liver transplantation 1 year ago + maintained on immunosuppressive drugs (immunocompromised)
- Vesicles containing clear fluid + ulcerated and crusted lesions (also helps DD)

• **Dx → Shingles**

- Caused by **VZV reactivation**.
- Primary infection of VZV is **chickenpox**. The Virus then remains latent in the **dorsal root ganglion [asked on NBMEs]**. Once immunosuppressed, sick, or stressed, **VZV reactivates as shingles**
 - Rash → Painful, burning, itching **vesicular lesions** in **dermatomal distribution** that later crust & form ulcers
 - **Tzanck test** → Smear of vesicular fluid showing **multinucleated giant cells** (seen in VZV & HSV)
 - Treatment → **Acyclovir** (**HSV/VZV DNA Polymerase inhibitor**)

? **What is the cause of Acyclovir resistance?**

▼ **Answer**

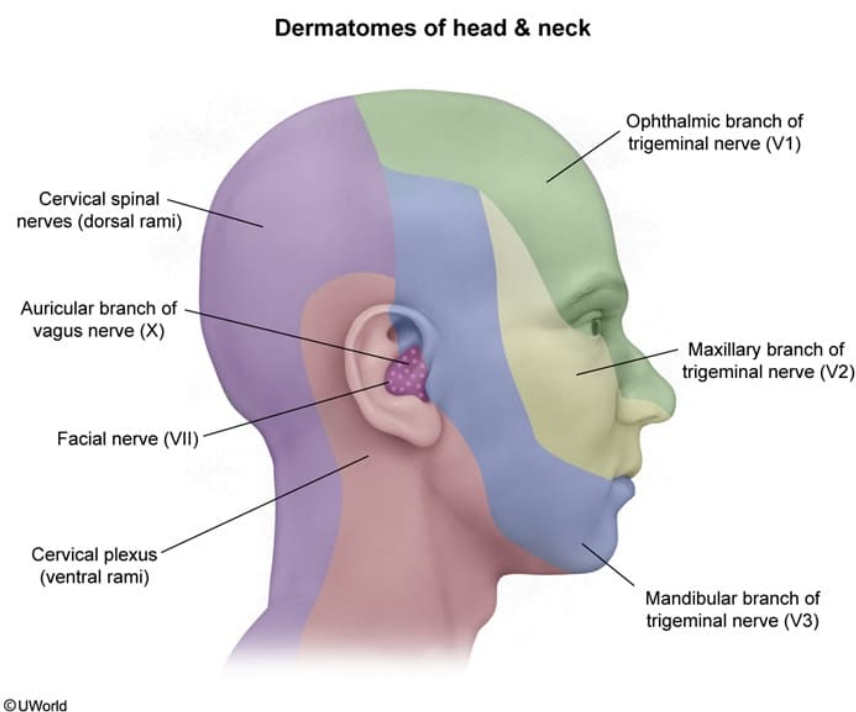
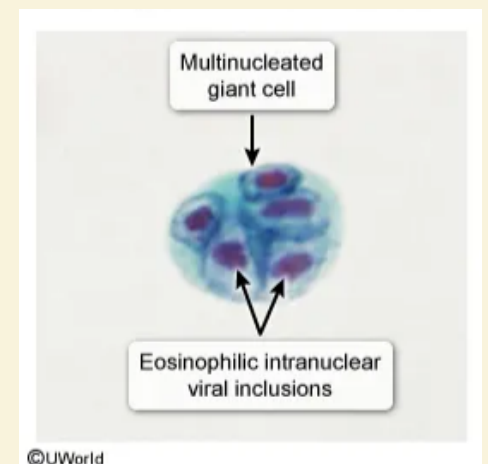
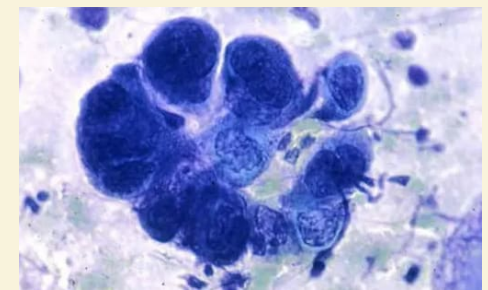
Altered/Absent Thymidine Kinase

? **Where does HSV1/2 remain latent at?**

▼ **Answer**

- HSV 1 → **Trigeminal Ganglia**
- HSV 2 → **Sacral Ganglia**

 Tzanck Smear:



 **VZV Reactivation** in the **ophthalmic division (V1)** can cause **VZV Keratitis (Herpes Zoster Ophthalmicus)**, which is characterized by **pseudodendritic corneal ulcer**, **vesicular lesions**, and **pain** in V1 dermatome.

- Presence of vesicular lesions on **tip of nose (Hutchinson's Sign)** indicated **nasociliary branch involvement** & is associated with **high risk of keratitis**.

 **Ramsay Hunt Syndrome (Herpes Zoster Oticus)** is reactivation of VZV in the **geniculate ganglion of Facial nerve**, resulting in **ipsilateral ⇒ facial pain, ear pain, and vesicular rash on the external ear canal, pinna, or ear drum**.

▼ Other Choices + HY Related Notes

- **Choice A → Bullous pemphigoid** would have **tense bullae / blisters not** vesicles. It is an autoimmune disease characterized by antibodies against **hemidesmosomes**. It also affects the elderly, while pemphigus vulgaris is the one that affects middle-aged adults.
 - Here is a simple HY table comparing the two:

Features	Bullous Pemphigoid	Pemphigus Vulgaris
Oral Involvement	No	Yes
Nikolsky Sign	Negative	Positive
Autoantibodies Against	Hemidesmosomes	Desmosomes
Immunofluorescence	Linear deposition along basement membrane	Net-like (reticular) intercellular pattern
Blisters	Tense, subepidermal blisters	Flaccid, intraepidermal blisters
Ages	Elderly	Middle-aged adults

- **Choice B → Dermatitis Herpetiform** is a purple-ish pruritic vesicular skin condition that affects the **extensors of the body** (usually elbows).
 - Associated with **celiac disease**
 - Biopsy shows **IgA deposits at dermal papillae**
- **Choice C → Erythema multiforme** are **target like lesions** caused most commonly by **HSV, Mycoplasma pneumoniae, or sometimes sulfa drugs**
 - It is a **type 4 hypersensitivity reaction**
- **Choice D → Secondary syphilis** rash is **nonpruritic & painless** in general
 - Appears weeks after the **painless chancre** has disappeared
 - **Diffuse maculopapular** not vesicles that **INVOLVES palms and soles**



What are other HY causes of rashes that involve the palms & soles on the USMLE?

▼ Answer

- **Secondary Syphilis** - *Treponema pallidum*
- **Hand-Foot-Mouth Disease** - *Coxsackievirus A*
- **Rocky Mountain Spotted Fever** - *Rickettsia Rickettsii*
- **Kawasaki Disease** - autoimmune disease
- **Meningococcemia** - *Neisseria meningitides*

Q41

A 25-year-old woman develops increasing shortness of breath with exertion 2 weeks after the birth of her first child. Physical examination shows jugular venous distention, evidence of edema in the lung bases, mild hepatomegaly, and mild pitting edema in the lower extremities. Which of the following cardiac abnormalities is most likely present at this time?

- A. Asymmetric septal hypertrophy
- B. Endocardial fibroelastosis
- C. Four-chamber dilation
- D. Lymphocytic infiltration of the myocardium
- E. Myocardial disarray

▼ Explanation

- **Answer is C (Four-chamber dilation)**
 - **IMP Clues:**
 - SOB 2 weeks after birth of her child ()
 - Jugular venous distension + mild hepatomegaly + mild pitting edema in lower extremities (Right side of the heart is affected)
 - Evidence of edema in lung bases (Left side of the heart is affected)

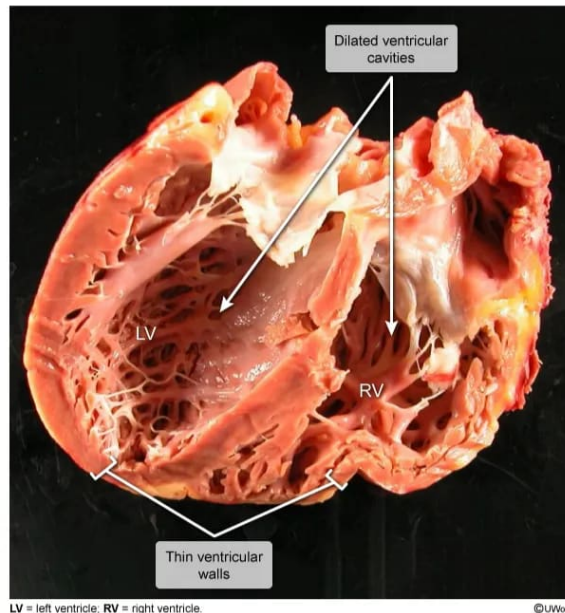
- Dx → Peripartum Cardiomyopathy

- Form of **Dilated Cardiomyopathy**, which is characterized by **systolic dysfunction secondary to eccentric hypertrophy of the heart**

- **Sarcomeres are added in series [asked on NBME] ⇒ Dilated Chambers (Four Chamber Dilation, choice C) with relatively thinner walls ⇒ Systolic Dysfunction**

- **Right-side HF ⇒ Jugular venous distension, Mild Hepatomegaly, Mild Pitting Edema in lower extremities**
- **Left-side HF ⇒ Edema in lung bases**
- **Basically ⇒ Four Chamber Dilation**

- **Peripartum cardiomyopathy** affects young women in their **last months of pregnancy until a few months after**.



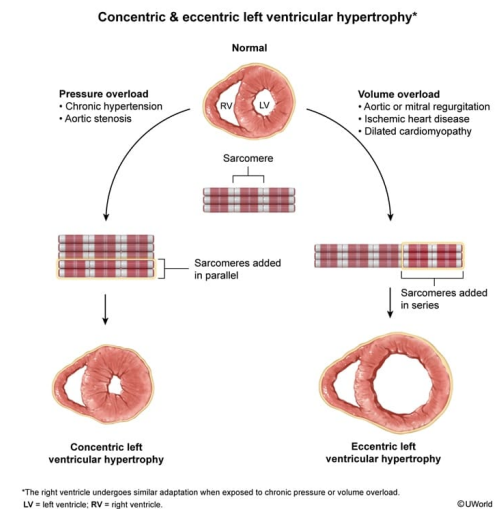
HY Findings:

- **S3 Sound** (Rapid Ventricular filling due to dilated ventricles)
- **ECHO shows dilated heart** (Grossly Enlarged)
- **CXR shows Balloon Appearance**

? What are other HY causes of DCM?

▼ Answer

- **Doxorubicin**
 - Anti-Cancer drug that causes increased ROS
 - **ttt → Dexrazoxane**
- **Coxsackievirus B**
- **Chagas Disease** (Trypanosoma Cruzi)
- **Peripartum Cardiomyopathy**
- **Wet Beri Beri** (Thiamine Deficiency)
- **Stress Cardiomyopathy** ("Broken Heart Syndrome" or "Takotsubo Cardiomyopathy")
 - **ECHO shows ventricular apical ballooning**
 - **Caused by "Catecholamine Excess" or "Increased Sympathetic Stimulation"**



Most common cause of DCM is familial due to **TTN gene mutation that encodes for the **titin sarcomere protein****

▼ Other Choices

- **Choices A & E** → refers to **HOCM**, in which there is **LV septal hypertrophy** that causes **aortic obstruction symptoms**.
 - Patients are usually kids or young adults that **get syncope on exertion / sudden death**
 - **Diagnosed by ECHO as septum being > 15mm** [you don't need to know the number, just saying]
 - Associated murmur is the same as **AS: Crescendo-decrescendo ejection systolic murmur**
- AD transmission: **Mutation in Beta-myosin heavy chain & Myosin binding protein C [asked on NBME]**
 - Muscle fibers added in **parallel (with septal favoring)**
 - Which essentially means: LV concentric hypertrophy + septum enlarged more (**Asymmetric hypertrophy**) = **Myofibrillar**

disarray.

- Treatment is **Beta-Blockers** [asked on NBME]
 - Longer diastole = more filling of RV = more blood to LV = more pressure = less septal bulge = less outflow obstruction



HOCM MURMUR UNIQUE FEATURES:

Increases in intensity with Valsalva or standing

- **Reason?**

- Valsalva & standing decrease VR to heart to less blood going to LV so less pressure so the septum bulges into the cavity causing turbulence, more turbulence = louder murmur

Decreases in intensity with squatting

- **Reason?**

- Squatting increases VR + increases Afterload.
 - Increased VR = increased blood to LV = more pressure = septum bulges less into cavity = less turbulence = softer murmur
 - Increased afterload = more pressure against LV = septum bulges less into cavity = less turbulence = softer murmur

- **Choice B → Endocardial fibroelastosis** is a cause of **restrictive cardiomyopathy**

- It is a pediatric condition in which they have thick fibroelastic tissue in their endocardium.
- Most common causes of **Restrictive CM on the USMLE: Amyloidosis, Post-radiation, Sarcoidosis, Hemochromatosis**
- ECHO shows **thickened ventricles + ECG shows low voltage**
- Examination shows **Kussmaul sign = increased JVD on inspiration**



Hemochromatosis can present with either *Restrictive* Cardiomyopathy or *Dilated* Cardiomyopathy.

- **Choice D →** most likely refers to **viral myocarditis**

- Myocarditis presents as: **Features of HF + high CK-MB / Troponin + ST-segment changes on ECG.**
- Differentiated from IHD by Coronary Angiography (which is normal in Myocarditis)

Q42

A 20-year-old primigravid woman at 12 weeks' gestation comes to the physician for genetic counseling. She underwent repair of a unilateral cleft lip as an infant; she had no other abnormalities. Her maternal great-uncle had a bilateral cleft lip and palate; he also had no other abnormalities. She is concerned that her child might have the same condition. Which of the following is the most likely mode of inheritance of this disorder in this patient's family?

- A. Autosomal dominant
- B. Autosomal recessive
- C. Mitochondrial
- D. Multifactorial
- E. X-linked

▼ Explanation

- **Answer is D (Multifactorial)**

- **IMP Clues:**

- She underwent repair of unilateral cleft lip as an infant + no other abnormalities (isolated cleft lip)
- Her maternal great-uncle had bilateral cleft lip and palate + no other abnormalities (isolated too)

- **Dx → Isolated non-syndromic cleft lip/palate**
 - Basically, cleft lip/palate most commonly presents alone (non-syndromic).
 - It does run in families and is thought to be due to a mixture of environmental + genetic causes.
 - Skip the garbage talk, what you need to know is: **Most cleft lips/palates are multifactorial & not associated with syndromes. [Q repeats on future NBMEs]**



USMLE asks a similar Q regarding isolated VSD. Patient had an isolated VSD & no other findings + family history of some sort of a similar isolated VSD. Patient came in for genetic counseling & asks what are the chances that my baby has a similar condition?

- Isolated VSD in newborns is estimated to be found in **2-3%** of the population
- Yes, USMLE asks for the %. Crazy I know.

Q43

A 53-year-old woman with gastroesophageal reflux disease comes to the physician for a follow-up examination. Dietary changes and stress reduction have had no effect on the frequency or intensity of her symptoms. Physical examination shows mild epigastric tenderness. Which of the following drugs is most likely to be effective in both relieving this patient's symptoms and promoting healing of her esophageal mucosa?

- A. Bethanechol
- B. Famotidine
- C. Metoclopramide
- D. Misoprostol
- E. Omeprazole

▼ Explanation

- **Answer is E (Omeprazole)**

◦ IMP Clues:

- GERD + diet changes & stress reduction had no effect (so failed lifestyle modification so time for pharmacological ttt)
- Epigastric tenderness (classic GERD)

- **Dx → GERD**

- Q asks what is best ttt for relieving this patient's symptoms AND promoting healing of her esophageal mucosa?
 - Now it is between either **Misoprostol (PG analogue) & Omeprazole (PPI)**
 - Now let's go through which is correct:
 - **Misoprostol:**
 - PG analogue
 - Function of PGs: protect mucosa from acid & promote healing
 - Mainly used for ulcer protection for people taking NSAIDs
 - **Omeprazole**
 - Proton Pump Inhibitor (Irreversible)
 - **Most effective drug at decreasing gastric acid production**
 - This causes less symptoms.
 - Less acid = less damage to mucosa = more time / ability to heal.
 - So, effective in BOTH relieving symptoms & promoting healing would be omeprazole. Tricky Q no doubt.

How do PPIs work?

- **PPIs irreversibly inhibit H⁺/K⁺ ATPase (the proton pumps) of the parietal cells of the stomach**
 - Due to its **irreversible inhibition**, acid secretion is inhibited until new pumps are synthesized, hence the effectiveness/potency and long duration of action
 - Used in all conditions associated with **acid hypersecretion like GERD, PUD, ZE Syndrome, and H.pylori infection**



H2 Blockers are inferior to PPIs in suppression of gastric acid, as H2 blockers reversibly block histamine (H2) receptors on gastric parietal cells.



What are the HY side effects of PPIs on the USMLE?

▼ Answer

- Increased **CI.difficile infection**
- **Decreased** absorption of **Iron, Vitamin B12, calcium, and magnesium**
- Increased risk of **Osteoporosis**
- **Rebound Hypersecretion**
- Increases risk of **acid labile** infections

▼ Other Choices

- **Choice A** → **Bethanechol** is a *muscarinic receptor agonist* used for **neurogenic (flaccid hypocontractile) bladder**
- **Choice B** → **Famotidine** is an *H2-Blocker* that is actually used for **GERD**. However, it is substantially weaker than PPIs & reversible too.
- **Choice C** → **Metoclopramide** is a *D2 antagonist*. Used mainly an **anti-emetic**.

? What are some HY Metoclopramide side-effects you need to know

▼ Answer

Extrapyramidal effects, hyperprolactinemia, and QT prolongation.

Q44

An 18-year-old woman comes to the office because of a 2-week history of pain in her right elbow. She is on the tennis team at her high school and has practiced for 5 hours daily during the past month. Physical examination shows tenderness over the lateral aspect of the right elbow. This patient most likely has inflammation at the origin of which of the following muscles?

- A. Abductor pollicis longus
- B. Extensor carpi radialis brevis
- C. Flexor digitorum superficialis
- D. Palmaris longus
- E. Pronator teres

▼ Explanation

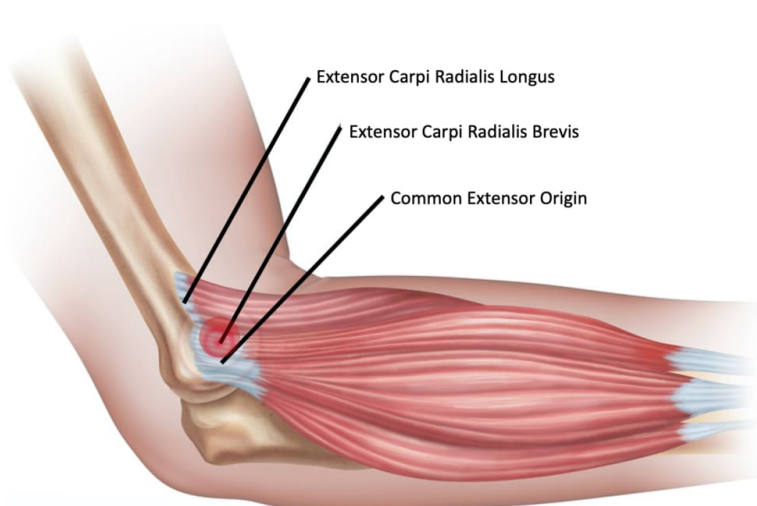
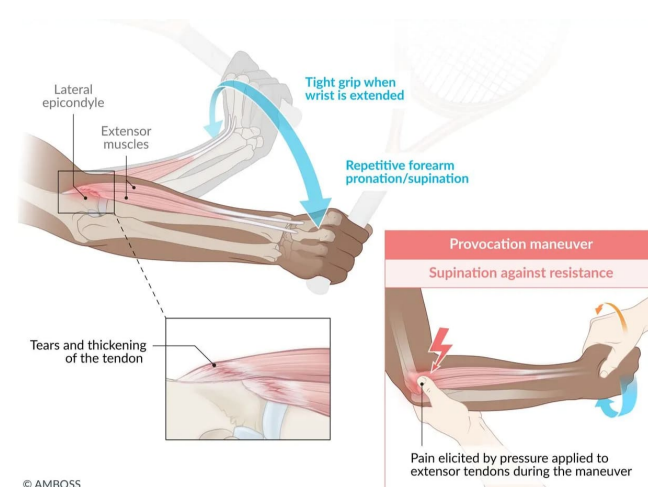
• **Answer B (Extensor Carpi Radialis Brevis)**

◦ **IMP Clues:**

- Pain in her elbow (Elbow pain DD: Golfer's elbow, Tennis elbow, bursitis, tendinitis)
- Tennis team + practices for 5 hours daily (repetitive usage of her elbow)
- Tenderness over lateral aspect of right elbow (Lateral elbow inflammation)

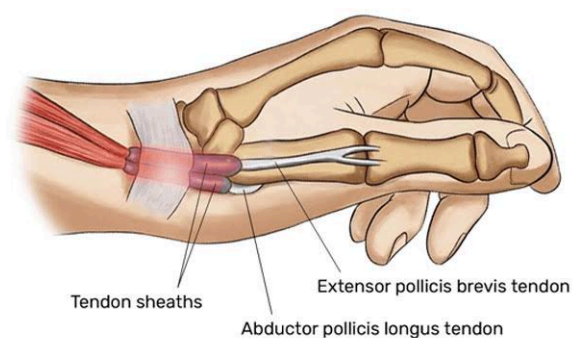
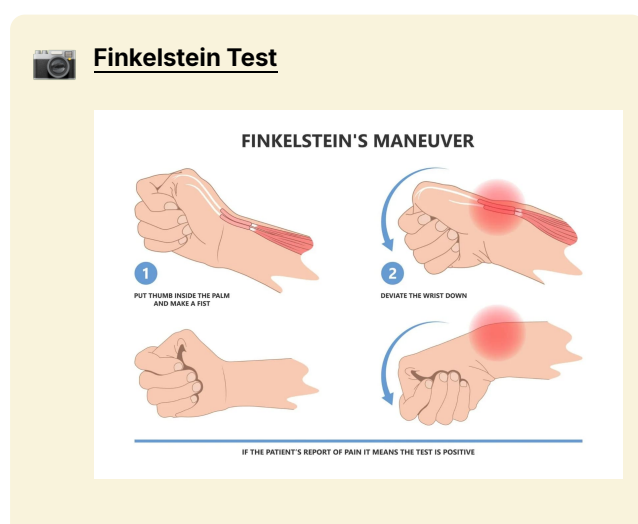
◦ **Dx → Tennis Elbow (Lateral epicondylitis)**

- Repetitive extension (like in tennis training) causes injury / inflammation of the **extensors** of the forearm.
- Most commonly it is the **Extensor Carpi Radialis Brevis**

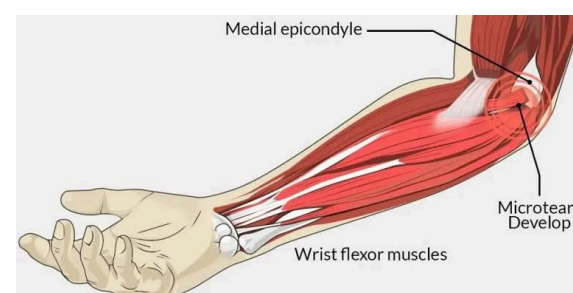


▼ Other Choices

- **Choice A → Abductor pollicis longus** inflammation causes **De Quervain tenosynovitis**
 - Pain over **posterior thumb base (radial wrist)**
- Special test = **Finkelstein test**



- **Choice C → Flexor Digitorum Superficialis** inflammation causes **Golfer's Elbow**
 - Pain over **medial epicondyle (Medial epicondylitis)**



- **Choice D → Palmaris longus** is irrelevant. Actually, a lot of people don't have this muscle
- **Choice E → Pronator Teres** inflammation would cause pain with?? **Pronation**. Aren't you a brilliant boy? Girl?

Q45

A 38-year-old woman comes to the physician because of a swollen, painful calf for 2 days. She gave birth to a healthy term newborn 5 days ago. Physical examination shows pitting edema and tenderness with dorsiflexion of the foot. One day later, she has a cerebral infarction with hemiplegia. Passage of an embolus into the systemic circulation is most likely to occur at which of the following locations?

- A. Bronchial artery
- B. Patent foramen ovale
- C. Pulmonary artery
- D. Pulmonary vein
- E. Uterine vein

▼ Explanation

- **Answer is B (Patent foramen ovale)**
 - **IMP Clues:**
 - swollen, painful calf for 2 days (we know DVT is on the DD list)
 - She gave birth to a newborn 5 days ago (hypercoagulable state)
 - Pitting edema & tenderness with dorsiflexion of foot (classic features of DVT)
 - Cerebral infarction with hemiplegia (okay DVT → Stroke, why? this is what is asked)

- **Dx → Patent Foramen Ovale**
 - Patient had **DVT**
 - Hx of **pregnancy (Hypercoagulability is a major risk factor)**
 - C/P of **Edema, tenderness (Homan's sign = tenderness on foot dorsiflexion)**, and **swelling** is classic DVT.
 - Usually, DVT → Right Side of Heart → Pulmonary circulation → Pulmonary Embolism
 - Patient had a stroke??
 - This means the embolus is in the **systemic circulation**. How did reach the left side circulation?
 - **DVT → Right Side of Heart → Patent Foramen Ovale → Left side of heart → Aorta → Cerebral arteries**

Q46

A 50-year-old man has had progressive dyspnea on exertion over the past 6 months. He now has dyspnea at rest. He has had a persistent dry cough since having an upper respiratory tract bacterial infection 1 year ago. Examination of lung tissue obtained on biopsy shows chronic inflammation and fibrous thickening of the alveolar septa. Which of the following is the most likely diagnosis?

- A. Chronic bronchitis
- B. Diffuse alveolar damage
- C. Emphysema
- D. Sarcoidosis
- E. Usual interstitial pneumonitis

▼ **Explanation**

- **Answer is E (Usual Interstitial pneumonitis)**
 - **IMP Clues:**
 - Dyspnea on exertion for 6 months + now at rest (okay so SOB, prepare your DD)
 - Persistent dry cough since bacterial URTI 1 year ago (okayyyy, dry cough helps the DD + hx of bacterial infection so usually severe damage is done)
 - Biopsy shows chronic inflammation & fibrous thickening of alveolar septa (okay we have fibrosis of the alveoli, which explains the symptoms)
 - **Dx → Pulmonary Fibrosis 2ry to an infection**
 - Usually, bacterial infections are severe & cause severe damage (**either directly or indirectly from the immune system's response / ROS**) **[asked on NBME]**
 - **Fibrosis = decreased diffusion = increased A-a gradient.** remember? (please say yes, if not go to Q4)
 - Decreased O₂ diffusion = more CO₂ & less O₂ which explains the dyspnea.
 - Another name for lung fibrosis is Interstitial Pulmonary Fibrosis or Usual Interstitial pneumonitis.

▼ **Other Choices + HY Related Notes**

- **Choice A → Chronic bronchitis** would most likely have a *productive* cough + history of **smoking**. Q can also give you PFTs with an **obstructive pattern**. Nothing here really supports it.
- Choice B → **Diffuse Alveolar Damage is essentially ARDS**
 - Injury to alveoli & endothelium = inflammation = increased vascular permeability = **protein-rich fluid leakage into alveoli [hyaline membranes = fibrin + necrosis]** = gas exchange impairment.
 - Causes: (these are the ones I commonly see as a cause of ARDS)
 - **Sepsis**
 - **Pneumonia**
 - **Pancreatitis**
 - **Aspiration (of gastric contents / acid)**
 - **Drowning**
 - **Transfusion reactions**

- **HY Random facts:**

- **Surfactant Protein D** → lung injury marker & is *increased* in people with ARDS & people recovering from ARDS
- ARDS causes **restrictive lung diseases pattern**
- ARDS is a cause of **increased A-a gradient hypoxia**

? Patient had ARDS. What will cause the regeneration of the necrotic endothelium?

▼ **Answer**

Type 2 pneumocytes

- **Choice C → Emphysema** would be supported by a **hx of smoking / alpha 1 antitrypsin deficiency**.

- Remember, **Smoking causes Centrilobular / Centriacinar emphysema** while **alpha-1-antitrypsin deficiency causes Panacinar / Panlobular emphysema**.
 - MOAs:
 - Protease is an enzyme that breaks elastic fibers of alveoli
 - Alpha-1-antitrypsin inhibits protease, so its deficiency causes increase protease activity = more elastic fibers breakdown = emphysema
 - Smoking activates protease = increase protease activity = more elastic fibers breakdown = emphysema



USMLE loves to test emphysema with arrow Qs of chest examination findings:

- **LOW TVF**
- **Resonant / Hyper-resonant**
- **Decreased breath sounds**

- **Choice D → Sarcoidosis** vignettes usually give you a middle-aged African American patient with **bilateral hilar lymphadenopathy** +/- pulmonary infiltrates.
 - Can cause dry cough & fibrosis, however, there will be hx of constitutional symptoms.
 - Biopsy will show a **noncaseating granuloma**
 - Associated Uveitis - Erythema nodosum - Hypervitaminosis D
 - **High ACE - High Calcium - High CD4/CD8 ratio**
 - Sometimes, Qs will get tricky & they will omit most findings except that of hypercalcemia & expect you to reach the diagnosis



What causes the Hypervitaminosis D in sarcoidosis?

▼ **Answer**

1-alpha-hydroxylase released by macrophages

Q47

A screening program is instituted for detection of vaginal Chlamydia trachomatis infection among first-year women college students. At the initial screening, evidence of C. trachomatis infection is found in 500 of 2500 students. One year later, screening shows vaginal C. trachomatis infection in an additional 200 students. Which of the following is the annual incidence of C. trachomatis infection in this population of women students?

- A. 8%
- B. 10%
- C. 16%
- D. 20%
- E. 28%

▼ **Explanation**

- **Answer is B (10%)**

- Easy biostat Q here. Q asks for annual incidence of C.trachomatis infection in the given population
- *Incidence = new cases / healthy population*
 - $200 / 2000 = 0.1 = 10\%$
- Q tries to trick you by adding that at the initial screening, the infection was found in 500 of 2500 students. So, the unaffected students are how much?
 - 2000.
 - Now from the healthy unaffected students, 200 this year got the infection. So, incidence is 10%



If the Q asked about the prevalence of the disease instead, what would the answer be?

▼ **Answer**

28% (Choice E)

- *Prevalence = all cases / all population*
 - 500 old cases + 200 new cases this year = 700
 - The whole population is 2500
 - $700/2500 = 0.28 = 28\%$

Q48

A 75-year-old man has fever, back pain, and pain on urination. He has a long-standing history of prostatitis. Gram stain of urine shows gram-positive cocci in chains. Which of the following bacteria is the most likely cause of his illness?

- A. Enterococcus faecalis
- B. Escherichia coli
- C. Klebsiella pneumoniae
- D. Proteus mirabilis
- E. Staphylococcus aureus
- F. Streptococcus pyogenes (group A)

▼ Explanation

• Answer is A (Enterococcus faecalis)

◦ IMP Clues:

- Fever, back pain, and pain on urination (okay UTI)
- Hx of prostatitis (trying to throw you off, more on that later)
- Gram stain shows gram positive cocci in chains (okay so either A or F)

◦ Dx → UTI caused by Enterococcus faecalis

- Gram positive cocci in chains means either streptococcus pyogenes or Enterococcus.
- Enterococcus faecalis is associated with UTIs (remember sketchy?)
- Streptococcus pyogenes is associated with cellulitis, pharyngitis, erysipelas, PSGN.

Why is “Hx of prostatitis” trying to throw me off?

- Well, if the Q didn’t include the gram stain → UTI + Hx of prostatitis = E.coli.
- So, lack of knowledge of the gram stain or if you skipped over it then you would have gotten the Q wrong. CONCENTRATE.

• Causes of Prostatitis

- > 35 years = E.coli
- < 35 years = Gonorrhea or Chlamydia

• Treatment of Prostatitis on the USMLE

- Ciprofloxacin: a fluoroquinolone
 - Inhibits DNA gyrase “Prokaryotic topoisomerase 2/4”
 - Most HY side-effect is tendinopathy / cartilage damage
 - Can present as sore ankles or rupture tendon (most common is Achillis)

▼ Other Choices + HY Related Notes

- Choice B → gram positive rods. If gram stain was not included in the vignette, this would have been the right answer
- Choice C → gram positive rods. Actually, a very common causative organism of UTIs. USMLE likes it for pneumonia though.
 - Concurrent jelly red sputum [not to be confused with concurrent jelly red stools of intussusception]
 - Causes lung lesions similar to TB on CXR.
 - USMLE likes to ask about the difference between klebsiella & Enterobacter / Serratia:
 - Klebsiella = Immotile & Urease positive
 - Enterobacter / Serratia = BOTH motile & urease negative
- Choice D → gram positive rod. On the USMLE, if you get a Q about staghorn (struvite) kidney stone, choose Proteus. Easy.
- Choice E → gram positive cocci in CLUSTERS.

Q49

A 46-year-old man comes to the physician because of a 2-month history of prolonged bleeding from minor cuts. Physical examination shows hepatosplenomegaly. Laboratory studies show mild anemia, thrombocytopenia, and leukopenia. A bone marrow biopsy specimen shows accumulations of lipid-laden macrophages. If the incidence of this disease among Ashkenazi Jews is 1/900, which of the following is the approximate carrier frequency of this mendelian trait in this population?

- A. 1/15
- B. 1/30
- C. 1/60
- D. 1/120
- E. 1/240

▼ Explanation

- **Answer is A (1/15)**
 - **Hardy-Weinberg** equation garbage (I hate it so much)
 - First what are the equations that we must know?
 - $p^2 + 2pq + q^2$
 - $p + q = 1$
 - p is normal people [always calculate it as 1, trust me]
 - q is affected people
 - 2pq is carriers
 - Q asks for the carrier frequency. Okay so we need $2pq$
 - Q gives us the incidence of the disease with is 1/900, so $q^2 = 1/900$
 - So, $q = 1/30$
 - so $2pq = 2 \times (1) \times (1/30) = 2/30 = 1/15$

Q50

A normal-appearing 17-year-old girl has never had a menstrual period. The vagina ends blindly and no uterus or ovaries are palpated. A buccal smear shows chromatin-negative nuclei. Which of the following is the most likely diagnosis?

- A. Androgen insensitivity syndrome
- B. Congenital adrenal hyperplasia
- C. Gonadal dysgenesis 45,X (Turner syndrome)
- D. Prenatal exposure to excess androgen
- E. Prenatal exposure to excess estrogen

▼ Explanation

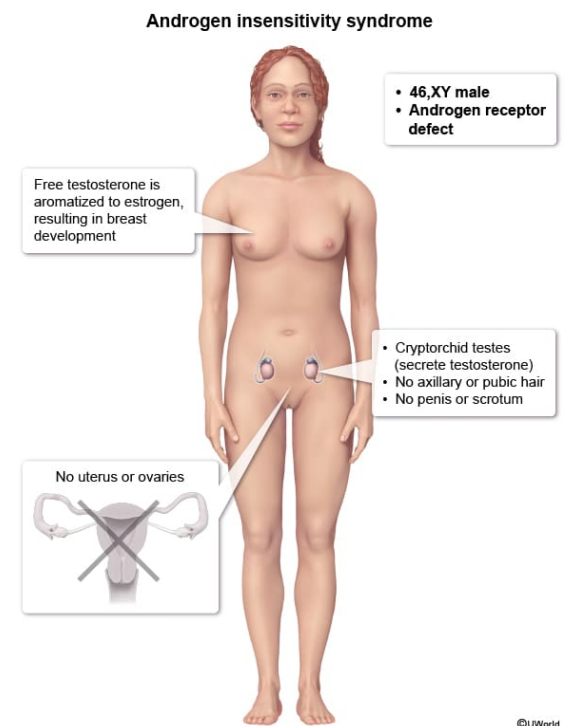
- **Answer is A (Androgen insensitivity syndrome)**
 - **IMP Clues:**
 - Normal appearing 17y old girl (saying normal appearing excludes Turner syndrome)
 - Never had a menstrual period (primary amenorrhea)
 - Vagina ends blindly & no uterus or ovaries are palpated (excludes CAH & prenatal exposure to excess estrogen & androgen)
 - Buccal smear shows chromatin-negative nuclei (1 Barr Body means only one X is present so either XY or XO)

- **Dx → Androgen insensitivity syndrome**

- Diseases characterized by **receptor insensitivity to androgen**.
- Patient is actually a boy with **XY genotype**
 - However, when the testes produce androgen, they **CANNOT EXERT** its effects on the **Wolffian duct** due to receptor insensitivity to androgen
 - hence the name “androgen insensitivity syndrome”

⚠ **Androgen normally enhances the growth of the Wolffian duct, which then produces the male sexual organs.**

- So, patients have:
 - **Undescended testes**
 - **NO** uterus or ovaries (hence the primary amenorrhea)
 - Female “**appearing**” external genitalia that ends with blind-ended pouch.
 - Fair breast development (**testosterone → estrogen via aromatization**)
 - **NO/SPARSE** pubic & axillary hair (androgen insensitivity)



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▼ **Other Choices**

- **Choice B → CAH**

- Usually presents as a XX newborn with ambiguous genitalia that is mistake for a boy. Increased androgen causes the clitoris to grow so gets mistake for a penis.
- Vagina will be normal (not blind-end pouch)
- Uterus & Ovaries will be palpated
- XX genotype of buccal smear shows chromatin-**positive** nuclei
- Will present with **virilization** (excess hair growth - increases muscle mass - clitoromegaly - deepening of voice)

? **What's the commonest cause of CAH?**

▼ **Answer**

21-alpha hydroxylase deficiency

- **Choice C → Turner Syndrome**

- **XO (Gonadal Dysgenesis)**
- **Short female with low set ears, flat “shield” chest, wide carrying angle, webbed neck, and lymphedema of hands & feet.**
- Associated anomalies: **Bicuspid Aortic valve, Coarctation of aorta, Horse-shoe kidney**
- Vagina will be normal (not blind-end pouch)
- Streak ovaries (non-functioning)
- XO genotype of buccal smear shows chromatin-**negative** nuclei



HY Factoids about horseshoe kidney

1. Patients with Horseshoe kidney are at **increased risk of Wilm's Tumor (Nephroblastoma)**
2. Horseshoe kidney **gets stuck below the IMA** as they ascend from the pelvis
3. Patients with horseshoe kidney normally have a lot of abnormal blood supply to that kidney from surrounding vascular structures (commonly aorta), which can cause difficulty in some surgeries **[asked on NBME]**

- **Choice D → Excess prenatal exposure to androgen will cause virilization of a baby female**

- **Choice E → Excess prenatal exposure to estrogen will cause slight feminization of a baby male.**



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