

w.Tommyk posts ( 1-147)

Q.1 Patient, young, with obesity, hypotonia, mental retardation, short stature, hypogonadotropic hypogonadism, strabismus, and small hands and feet. What disease and what is tx? [

A. Prader Willi Syndrome. Treat with GH ]

Q. 2 Pt w/ symptoms include tall stature, ectopia lentis, mitral valve prolapse, aortic root dilatation, and aortic dissection? What gene is missing and what is treatment of choice? Don't peek below w/o guessing.[

A. Marfan's Syndrome (This WILL be on your test). Defect in fibrillin gene. Treat the aortic dissection with B-Blockers. Warn them about pneumothorax and strenuous exercise. Tell patients that they are AD inheritance. Warn them about weird things like an elevator that travel up too fast or an airplane without decompression.

You have to know that many test takers said it really "helped" to do the NBME Step 2 questions and the NBME Step 3 questions that they have on the website. Please do not neglect them. Just ignore the "next step" questions, and do the diagnosis problems ]

Q.3 IF you are given a diagram with an LDL receptor molecule, and ... Then if you are asked what ion binds to it, what would you guess?

Choices: Na, Ca, Fe? [

A. The answer is Ca. You should look at the concept of diagrams of receptors. Remember, many of the writers of the questions are MD-PhDs and they specialize in their own receptor research. ]

Q.4 Uric acid stones (which are transLUCENT on x ray unlike Ca stones), are common in what three diseases? Bonus: what do uric acid stones cause symptom wise [

A. the three diseases are:  
HGPRT deficiency

PRPP synthetase overactivity

Glucose-6-phosphatase deficiency

The stones will present most commonly with hematuria, then fever/nausea/vomiting, then UTI!! ]

Q. 5 You HAVE to know this...

Sorry to be patronizing, but you will get this concept most likely...

What is the primary treatment for the uric acid stones? 2nd treatment if refractory? [

A. 1st thing is to alkalinize the urine and hydrate! Wait for the stone to pass. If that doesn't work, give allopurinol!

BUT, if the stone is more than .5cm, then use lithotripsy because the stone will not pass by itself!  
Stones are SO common and SO common stuff are all over the USMLE]

Q.6 A patient who had her gall bladder removed for stones STILL feels colicky pain, what could be the reason? This is a very HY concept.... [

A. loss of inhibitory enteric innervation (motor) ]

Q.7 YOU WILL definitely be asked to understand the concept that a person with an injury to the SURGICAL neck of the humerus/or the dislocation of anterior shoulder will have which nerve injury? [

A. AXILLARY nerve, not the radial nerve. ]

Q.8 You HAVE to know this crucial concept tested on most exams and in clinic!

A woman who diets and cuts out all fats but still eats carbohydrates. Will she lower her LDL? HDL [

A. Everyone will be tested on the concept that chylomicrons are blood lipoproteins produced from dietary fat.

It is the VLDLs that are produced mainly from dietary carbohydrate.

IDL and LDL are produced from VLDL.

Thus, HER LDL level will still BE HIGH. Crucial concept!!! ]

Q.9 Methinks that every single human taking USMLE had to know that a man with:

Diffuse demineralization of the bone associated with hypercalcemia, anemia, hypergammaglobulinemia, proteinuria, and normal serum alkaline phosphatase is most suggestive of? [

A. Multiple Myeloma. I CAN BET MY BOTTOM DOLLAR THAT YOU WILL SEE MULTIPLE MYELOMA ON YOUR TEST. I definitely did. ]

Q.10 A woman with sarcoidosis or with hypercalcemia (there are a thousand ways to ask this concept) enters your clinic, which is the diuretic of choice? [

A. Furosemide, NOT thiazides or mannitol, or acetazolamide ]

Q.11 A Super high yielder is Hardy Weinberg. If the number of homozygotes is 1/4900, can you tell me the number of heterozygotes[

A. use  $q^2$  and then use equation  $2pq$  ]

Q. 12 Everyone is reporting that they MUST master the concept of

transgenic mice. Here is one concept you must understand:  
Transgenic mouse with defect in B2 microglobulin gene. What is the immuno defect? [

A. The B2 microglobulin is part of the MHC Class I molecule. So, a defect here will cause a problem with CD8 + cells so cell mediated immunity is crushed!

The MHC includes a polymorphic set of genes encoding cell surface glycoproteins, designated class I and class II molecules, whose function is to present antigenic peptides to CD8+ and CD4+ T cells, respectively. Peptides generated in the cytosol from denatured proteins fragmented by proteasomes, some components of which are MHC-encoded are transported into the endoplasmic reticulum (ER) by peptide pumps or transporters associated with antigen processing (TAP) whose encoding genes are again located in the MHC. Peptide binding to the class I heavy chain facilitates association with b2-microglobulin (b2-M) and stabilizes the complex allowing it to migrate to the cell surface.

B2 microglobulin, a component of MHC I molecules, functions to transport MHC I to cell surface, ditto.

Lack B2 microglobulin, no MHC on cell surface. CD8+ cytotoxic T cell needs to bind to MHC I molecules.

result: defect on CD8+ cytotoxic T cells mediated immunity ]

Q.13 A wise man said that you cannot avoid understanding Biochem thoroughly. So, if I gave you a pic of cbiochem cycles and asked where is it inhibited by acyl CoA and enhanced by citrate, would you succeed [

A. During fatty acid synthesis in the CYTOSOL, Citrate will activate acetyl CoA into malonyl CoA, Acyl CoA will block this. (SORRY, in the question above I meant to say Acyl CoA, not Acetyl CoA)

Nevertheless, this is a crucial biochemical step underappreciated...by all ]

Q. 14 Aside from drug abuse and high exercise, which is a given, what is the next most common cause of lactic acidosis? There are a thousand poss. ways to ask this concept [

A. shock, like septic shock or hypoperfusion. ]

Q. 14 ubiquitous question in USMLE, clinic, life, and love is:

A child comes in with meconium ileus, other than Hirschsprung's, what is the other MAIN common disease you see?

Like on Family Feud, the game show, the best answer is [

A..Cystic Fibrosis.

It is too easy to merely ask about salty sweat and fatty stools, although some will invariably get the easy questions about this disease. ]

Q.15 Speaking of my previous question about Meconium ileus, there is a disease EVERYONE will get on their test, and in clinic...Meconium ileus is a block of the terminal ileum and is the most common cause of

obstruction and congenital GI anomaly. What is the other name of this that starts with the letter M? [

A. Meckel's diverticulum, persistence of the vitelline duct. This is SO HY

You will see this concept everywhere you turn: ]

Q. 16 A pt. comes in with overdose of scopolamine because she went on a roller coaster in Disneyland. The doctor in line slips her physostigmine instead of neostigmine, etc. why? and now > [

A. it is because of the cholinomimetics, physostigmine crosses the blood brain barrier to CNS.  
Neostigmine is better for urinary retention after plastic surgery (or any surgery).  
You will see this concept in your life.....soon... ]

Q.17 A med student grabs gentamicin for you to treat Bacteroides. You will hit him over the head because he is wrong. Why? (What is MOA of Aminoglycosides) [

A. Bacteroides is an anaerobe. Aminoglycosides do not work on anaerobes b/c they need O<sub>2</sub> for uptake, thus blocking formation of 30S initiation complex! ]

Q. 18 Young girl has early acne, you give tetracycline, then years later family comes back at her homecoming and kicks you. Why? (What is SE of TETRACycline) [

A Tetracycline has the famous concept of discoloring teeth and blocking bone growth in children, along with photosensitivity. ]

Q 19 .Anatomical common injuries are super duper HY.

A football player comes in with an injury in the shaft of his humerus. What nerve is crushed (choices: radial, median, ulnar, axillary) and what prob. does he have?

Radial nerve is damaged. He is lose his triceps, brachioradialis, and have wrist drop.  
This is a must know...

The aforementioned athlete breaks his supracondyle of humerus. What nerve is crushed?

(Radial, median, ulnar, or axillary)

(All of you will get a variation of the upper arm injuries) [

A. MEDIAN nerve is blown. He loses his finger flexing ability and some thumb movements and some loss of sensation over lateral palm and thumb and radial 2.5 digits ]

Q. 20 Everyone I hear is asked about brachial plexis injuries. But they are not easy direct questions. E.g.

A supermodel in a car crash looks at you with a "claw hand". What two cord segments contribute to the nerve which is damaged? [

A. Cord segments are C8 and T1! See, not so easy, right? ]

Q.21 Adenosine deaminase def can cause what problem immunologically? [

A. Adenosine deaminase def can cause SCID. ]

Q. 22 Most I spoke to got this on their test. Distinguish the Rinne and Weber TEST!?! [

A. absolutely HY. Weber test- tuning fork in midline of skull- localizes hearing loss to one side or the other- if it is a conductive loss, patient hears better on side of defect. If it is sensorineural hearing loss, hearing is better on opposite side of defect. Rinne test- place tuning fork on mastoid process until patient can no longer hear vibrations, then place tuning fork next to external auditory meatus- if patient cannot perceive vibrations- BC ( bone conduction) is better than AC(air) and patient has a conductive hearing loss on that side. If AC is better than BC, then that is the normal ear ]

Q.23 Everyone seems to need to understand that:

A Bicornuate uterus, which prevents a woman from fertility, is caused by what? [

A. it is due to the incomplete fusion of the PARAMESONEPHRIC ducts. Amen ]

Q.24 YOU WILL be asked this question:

There will be a person with a history of travel who goes to Mexico or thereabouts. Then he or she will return with bloody bloating crampy diarrhea. They will ask you either what is the bug and the disease, and the treatment. So what are the answers? Look below [

A. Ambiasis, dx is dysentery, and you treat with Metronidazole and the bug is Entamoeba histolytica. ]

Q.25 Since taking the test, I spoke to "a Lot" of people and the US licensing board wants everyone to know a certain fixed "universe" of diseases and txs, if you master those, you will at least PASS. That is what I am trying to help to do for all of us family VALUE MDs!  
I "got" this question, my "roommate" got this question, in 2030 probably, my sons/daughters will get this concept:

What is the MOA of Acyclovir [

A. Acyclovir blocks viral DNA polymerase when phosphorylated by viral thymidine kinase.

Some people will be asked to understand that Acyclovir is used for the HSV, Varicella, Epstein Barr Virus

The boards LOVE Acyclovir ]

Q.26 So common, definitely on everyone's test:

A baby come to your clinic with loud cough that resembles the barking of a seal, difficulty breathing, and a grunting noise or wheezing during breathing. What is the dx? And the secondary question they WILL ask is is it enveloped and what is the structure? [

A. Dx is Croup!  
Paramyxovirus,

It Has an envelope, has single strand, nonsegmented. ]

Q.27 Case: Cilia lack ability to move, so your patient is sterile, no sperm, and he has ongoing sinus inflammation. What is syndrome and the protein that is lacking? [

A. Kartagener's Syndrome, due to dynein arm defect! ]

Q.28 EVERYONE, seriously, EVERYONE I talked to needed to master this concept for the test:

Case: Child with multiple fractures and BLUE sclera. The two secondaries are

What is specific defect?

What is the inheritance pattern? [

A. Osteogenesis Imperfecta, with abnormal collagen type I, and inheritance pattern is autosomal dominant! Good Luck! ]

Q.29 Hard one, but def. a must!  
Here it is..

A child has defect in eustachian tube and middle ear, which pharyngeal pouch is defective? [

A. The first pouch... some of my friends last year got a whole slew of arches and pouches, a favorite of the NBME, KNOW IT ]

Q.30 YOUR SOUL MUST UNDERSTAND that if you have a patient with probs with his circadian rhythms and autonomic regulation and a DETAILED diagram with arrows of brain pops up, which nucleus is affected?!

[

A. The suprachiasmatic nucleus of the hypothalamus! ]

Peace to Everyone on Earth!

Yoda says rather asks you:

Q.31 Which one of the following is responsible for peripheral myelin production?

(Choices: Ependymal cells, Oligodendroglia, Astrocytes, Microglia,

Schwann cells) [

A. Schwann cells! Remember that Oligodendroglia are CENTRAL myelin production ]

Q.32 Even my grandmother I think had to understand this for USMLE:

Case: A patient with a defect in apo C-II and Lipoprotein Lipase. How will her labs look like? I.E. What is her disease

You WILL get questions PLURAL on the big three: Diabetes, Hypertension, Hyperlipidemia. so, ... [

A.The answer to my question was HYPERLIPIDEMIA

hyperlipidemia type I is associated with : uncontrolled diabetes mellitus, obesity, and sedentary habits, all of which are more prevalent in industrialized societies than in developing nations. In both epidemiologic and interventional studies, hTG is a risk factor for coronary disease.

Two rare genetic causes of hTG (lipoprotein lipase [LPL] deficiency and apolipoprotein [apo] C-II deficiency) lead to triglyceride (TG) elevations ]

Q.33 Consequence: cardio disease! They love porphyrias. Maybe they watched the movie "The Madness of King George" over and over, I dunno, but in order to pass the test, you have to understand that if you get a patient with bizarre symptoms like stomach pains with very

13

mild photosensitivity, delirium, and his urine darkens in the light, you are looking at ACUTE INTERMITTENT PORPHYRIA! So you have to know four things:

What is the deficient enzyme? AND, What substances accumulate in the urine? AND what two amino acid begin this synthesis of porphrin molecule? AND what metallic ion cofactor is required. YOU HAVE TO KNOW THIS TO PASS. [

A. Deficient enzyme: uroporphyrinogen 1 synthetase

Porphobilinogen and aminolevulinic acid accumulate in urine

Glycine and Succinyl CoA are precursors of porphrin

Metallic ion is Fe! ]

Q.34 Pt. who drinks his whole life, say the question describes to you he has Wernicke-Korsakoff syndrome (you know how to spot this right?), and say the question asks what vitamin is missing AND what DOES THIS VIT DO. Can you tell me? (It is not enough to know just the vitamin)[

A. Vitamine B1 (thiamine), it functions as a cofactor for OXIDATIVE DECARBOXYLATION OF PYRUVATE and is involved in the crucial HMP shunt!

REMEMBER...thiamine and the word DECARBOXYLATION RXN ]

Q.35 Ahh... the all important Folic Acid def. Everyone will see this, guaranteed since it is the most common vit deficiency.

YOU HAVE to understand that if you see a slide with macrocytic megaloblastic anemia, what is missing vitamin (I gave it away, Folic Acid, but it could also be Vit B12 but without Neuro sym)....anyways, I digress...What IS the EXACT function of it, and type of reaction? [

A. Methylation reactions ...

and it is an enzyme for the all important one carbon transfers.

Folic acid=METHYLATION reactions ]

Q.36 with meowing catlike cry and later is mentally retarded. But always it is the SECONDARY QUESTION, so what is the disease, the genetic defect, and the organ that is primarily affected and how? I sound like a broken record, but EVERY DOCTOR-TO-BE SHOULD KNOW THE CONCEPTS THAT ARE ON THESE POSTS! [

A. Cri-du chat syndrome...BUT did you know that... chromosome 5's short arm is deleted AND pt has cardiac defects primarily VSD and ASD!!!!!!! ]

Q.37 Guaranteed you have to know:

Case: A college student comes into your clinic with fever, hepatosplenomegaly, lymphadenopathy and + heterophil Ab test. What is the "bug" and most crucial, is it:

SS or DS? (single stand or double strand)

Envelope or no envelope?

linear or circular?

What is the family?

{Believe me, you will see this question} [

A. Pt has Mono, and it is Epstein Barr Virus. Most importantly, the NBME will not stop there!!!! You will have to answer it is a Herpesvirus family, DS, linear, and it has an envelope. Failure to master this concept will result in a veil of tears ]

Q.38 Older patient comes to you with bone pain, Visual inspection may reveal bony deformities, such as an enlarged skull, spinal kyphosis, and bowing of the long bones of the extremities. Localized pain and tenderness may be elicited with manual palpation. Labs: elevated alk phos.

What is this common disease and drug Rx? You have to catch this diagnosis b/c it can lead to cancer!!!! [

A. Paget's Disease, treat with bisphosphonates, physical therapy, could have viral etiology. Result in Secondary Osteo and Fibro Sarcoma ]

Q.39 Suppose you are a pathologist to be and are shown a pic of lymph node. Could you point to EXACTLY where the T-cells are housed on a histo slide? You have to know this [

A. Hey, look up on Webpath and pick out the PARACORTEX, where the T-cells are housed. You have to know this on a pic, not just on words...

]

Q.40 case: skin manifestations include peripheral nerve involvement with fibromas and plexiform neurofibromas; the iris, with Lisch nodules; optic nerve gliomas; pheochromocytomas in some patients; skeletal abnormalities, including craniofacial dysplasia. What is this disease that you are SURE to have on your USMLE? What is inheritance pattern? [

A. Neurofibromatosis, AD (Don't confuse with McCune Albright which is assoc with girls and precocious puberty) Cheers!!! ]

Q.41 Pt appears healthy at birth. Diagnosis is usually made in infants aged 6-24 months. Inguinal and umbilical hernias are commonly seen at birth. On physical examination, these patients are observed to have corneal clouding, hepatosplenomegaly, skeletal deformities (dysostosis multiplex), coarse facial features, large tongue. You will see this presentation likely on your test because it is so serious.

They will ask, "What is the missing enzyme?" [

A. Hurler's Syndrome and you are having a deficiency of alpha L iduronidase. Love for everyone!!! ]

Q.42 EVERYONE I CONSULTED SAID THEY HAD THE UREA CYCLE ON THEIR TEST AND IT IS SO IMPORTANT IN OUR CLINICS AND LIVES. Know the cycle COLD until you can draw it out from memory.

17

For example, we know that an ammonium ion comes in in the mitochondria with carbamoyl phosphate, BUT urea has TWO nitrogens, which compound provides the second nitrogen? KNOW THIS. IT is essential for life. [

A Answer is aspartate feeds it in!

NOBODY, but NONE of US will give up. We will ALL succeed and become doctors. Let's let none of us give up and be left behind with their dreams. ]

Q. Don't be surprised if you are asked to know this classic common concept:

Pt with right sided ataxia, loss of pain temp of right face and left upper and lower extremities, hoarseness, dysphagia, loss of taste of right tongue, with vertigo and nystagmus. This IS SO CLASSIC FOR WHAT LESION YOU WILL see in your clinic and a famous test?

[

A. PICA, posterior inferior cerebellar artery stroke! Be SURE you can identify it on a brainstem slide. ]

Q. Invariably, you will be asked:

Pt, older gentleman with visual field defects from a Circle of Willis (they'll give a pic) hemorrhage. Point to the artery in Webpath. for now, though, what is the name of this most famous artery? [

A This is a case of anterior communicating artery stroke, the most common circle of Willis aneurysm! Got it? Got Milk? ]

Q.LIVE to know that:

Niacin, Melatonin and serotonin are derived from what amino acid?

Think hard first before looking! [

A. answer is tryptophan! Don't forget.... ]

Q. Sorry to continue to bug you all, but the galaxy members informed me that all need to know that if:

Given a midsagittal section of the brain, there is an arrow pointing to the different structures, but the question is:

Case: a child come to your clinic with symptoms of hypopituitarism.

Where is the lesion? POINT TO IT! What is the dx? [

A. Pick the answer choice where the arrow is point to the pituitary (it is next to the hypothalamus, find it on your atlas). This is a classic question of a craniopharyngioma which is the most common cause of hypopituitarism in children and it compresses the optic chiasm and hypothalamus. ]

Q. This is a question that a 99%er told me he knew but for the rest of us we can be OK if we are clueless:

A man comes in with bilateral and multicentric retinal angiomas, central nervous system (CNS) hemangioblastomas; renal cell carcinomas; pheochromocytomas; islet cell tumors of the pancreas; endolymphatic sac tumors; and renal, pancreatic, and epididymal cysts. CNS hemangioblastoma is the most commonly recognized manifestation of and occurs in 40% of patients. What is the dx? No secondary here. Just the diagnosis is Hard enough! BUT common

19

enough for USMLE CONSIDERATION! [

A. Von Hippel Lindau Disease. There will be a MRI of a brain with a cyst in the cerebellum from a hemangioblastoma. Excellent work my brothers and sisters ]

Q. A patient presents with recurrent viral infections from T-cell deficiency and symptoms pointing to hypocalcemia. Can you tell me disease (dx) and what failed to develop? A USMLE glorious favorite!!! Kinda hard though, but popular. You HAVE to know this. [

A. Faulty development of 3rd and 4th POUCH caused DiGeorge's syndrome and thymic hypoplasia and hypocalcemia.

Warning, I heard a lot of students messed this with the arches, and put 3rd and 4th ARCH (so close and yet so far!) ]

Q. What is the precursor for heme, which aa? Know this concept like your mom's birthday [

A. glycine. don't forget! ]

Q. You WILL see a pic and case presentation of a woman with a picture of an atypical mole (big hint is dysplastic nevus). What is the associated neoplasm, is it benign or not? [

A. It predisposes to malignant melanoma. The NBME wants you to know the stuff that you CANNOT AFFORD to miss that are COMMON. ]

Q. Speaking of skin stuff, Suppose you are dreaming and you see a color photo of a hyperpigmented skin lesion in the axillary area on an

20

obese person that you have nailed as acanthosis nigricans (as an aside KNOW THAT THIS LESION IS MORE COMMON WITH DARKER SKINNED INDIVIDUALS). Say they ask you the most notable associated malignancy, what will you say? [

A. Commonly associated with cases with dark skinned obese individuals, you must be wary that they may get GASTRIC adenocarcinoma! You cannot miss this and the NBME won't let you off if you don't know this. ]

Q. Here we go:

There is a young person who comes in with mild tachypnea because of acidosis, he has enlarged liver, is slightly to moderately icteric; accompanying hypoglycemia (watch for seizures). What is the MISSING ENZYME? [

A. This is a classic presentation of Aldolase B deficiency. They may want you to know it is autosomal recessive inheritance and you must terminate BOTH fructose and sucrose in the diet ]

Q. will faint with disbelief if you don't get this on your test and also in clinic and in life:

Case: Visual field defect of homonymous hemianopsia, there will be a series of diagrams of the eye nerves (you guys know with pic I am talking about right?) with arrows everywhere. Where exactly is the lesion? [

A There are at least two dozen questions that can be asked from this crucial concept with those visual field defects. Master them all. an arrow point to the nerves behind the optic chiasm contralateral. ]

Q. You WILL see this on your test because in clinic you will prob see it everywhere:

Case: There is a older man with signs of LOWER (not upper) GI bleeding. What is the most common disease (hint, neoplasm is not the answer), secondaries are What area of the bowel is affected and what drug can be given if surgery is not indicted? [

A.This is classic diverticulosis/itis of the lower descending colon and sigmoid (all proximal to the ligament of Treitz). You can give vasopressin as a drug.

Watch for distractor answer choices like Meckel's Diverticulum and Intususception and IBD, these are found in children and adolescents more often. Always always first consider your age and gender and ethnicity and travel and meds of your patients! ]

Q. This USMLE FAVORITE is kinda easy but just in case: Patient is older gentleman and had a history of lytic lesions and M protein spike and now present w/ lesion in the kidney, lesion was stained w/Congo Red? What is the dx and the name of the tissue stained (condition)? [

A. Multiple Myeloma and the stain is amyloidosis. These two diseases are EVERYWHERE, like Britney Spears pictures on magazines ]

Q. The NBME declares that you must know your basic oncogenes, guaranteed. So...

Case: You are given a clinical case where the gene that is active is c-myc (this is a oncogene, not TSG), what is the related tumor and specific gene translocation? [

A. This is Burkitt's lymphoma, some of you will be asked that it is a t8;14 gene translocation ]

Q. Speaking of oncogenes, many will be forced to address this point (not in Error! Hyperlink reference not valid. but def. in NBME's brain: Case: A clinical presentation of MENI and or MENII (review this quick), then you have to pick the oncogene that is activated. What will you chose?

we are talking about the ret oncogene. repeat that in your mind ten times NOW

Again, you are given a blood smear photo (medium quality) that you know to be follicular lymphomas (review on Webpath). But of course, the answer is a secondary. So tell me, give a series of answer choices, what is the oncogene responsible [

A. It is bcl-2 which block apoptosis. YES! YOU GOT IT! ]

Q. So Classic, so repeated, so in vogue, so know it...

A clinical presentation is given where a pupil constricts with accommodation and is not reactive to light. What is the treatment? The bug? The name of the syndrome? AND give me the method to visualize the bug! [

A. Penicillin G = Tx

Bug = Syphillis, T. Pallidum

Syndrome = Argyll-Robertson pupil

Visualized by = dark field microscopy

THIS IS A NBME FAVORITE! And you should know it for life for your patients! ]

Q. Here is a hard one, but certain to appear:

Case: One of your patients is in childhood with hepatosplenomegaly, pancytopenia, and crippling skeletal disease. He is Jewish and a liver biopsy shows glycolipid laden cells. What is the disease name and the enzyme deficiency given 5 choices that are agonizingly difficult? [

A. This is Gaucher's Disease and the enzyme def. is B-glucocerebrosidase!

KNOW that Gaucher's like most other enzyme deficiencies are AUTOSOMAL RECESSIVE! YES! Go and kick TUSH on this test! ]

Q. This is an interesting and crucial case seen around the world in testing centers:

A baby patient of yours has loss of sensation around the jaw, and suppose the answer choices ask which brachial arch is defective? What will you answer? (NBME loves those arches) [

A. Answer is Brachial arch 1,

cranial nerve V3 is affected along with all the "m" muscles (e.g. Muscles of mastication, masseter, medial pterygoid), Malleus, and a couple of others ]

Q. On test day, you see a question which asks you for the mechanism of RESISTENCE of bacteria to norfloxacin or ciprofloxacin and then asks you also the side effects? Will you know? [

A. Resistance comes from a mutational change of the bacterial DNA gyrase. This drug is eliminated renally so don't give to renal compromised patients. A scary side effect of this is inflammation of

24

tendons and cartilage damage.

NOTE: These Quinolones have NO EFFECT on anaerobes! ]

Q. Quickly, you see that oh-so-familiar diagram of th Cardiac Cycle/EKG. And you are asked what valve corresponds with the END of the first heart sound (Arrow is pointing there) and is it closing or opening? What do you say? [

A. The Aortic Valve OPENS at the end of the first heart sound (KNOW THIS) ]

Q. While we are on the subject, everyone in the world will face the Cardiac cycle/EKG graphs. So, There is an arrow points to the place where the S2 STARTS. What valve is opening or closing? [

A. The Aortic Valve closes at the beginning of the 2nd heart sound (KNOW THIS) ]

Q. Simply, what is the MOA of Cyclosporine [

A. inhibits IL-2 ]

Q. Case: You are given a classic presentation of an older man with Benign Prostatic Hypertrophy (this disease is everywhere). What is the drug of choice and what is the mech of action [

A. You should choose finasteride, a 5 alpha reductase inhibitor. ]

25

Q. You will not get away from Step 1 without seeing a case of...

An obese woman with infertility, acne, alopecia, hirsute. Now, I must ask you what is the hormonal abnormality and the drug of choice? You could also be asked what cancer is she most at risk of?

(THIS CONCEPT IS A MUST KNOW [

A. This is a case of PCOS. There is elevated LH/FSH ratio, and the LH stimulates testosterone. The lack of progesterone predisposes the woman to endometrial cancer.

Treat with Oral Contraceptive Pills or an anti androgen like Spironolactone ]

Q. EVERY MAN EVERY SINGLE MAN who lives long enough will get this disease:

Case: Older gentleman with urinary control problems and complaints include back and hip pain as well as other symptoms such as fatigue, malaise, and weight loss. There may also be a history of bone fractures. What is the disease, and the drug of choice (2 NBME favorite choices)? [

A. This is sadly prostate cancer with mets to spinal cord. You need to aim to stop testosterone production. Although castration is best (seriously), the choice most men opt for is Lupron or generic name Leuprolide (A LHRH agonist) or Flutamide. ]

Q. You will get a case of a patient with ptosis and inability to turn the

26

eye up, down, or inward. At rest, the eye is deviated down and temporally, and the iris sphincter may be involved or spared. He has a history of an aneurysm, and his eye does not constrict. Two secondaries: What nerve is lesioned, AND if you are given a picture of the circle of Willis and a bunch of arrows, which artery will you pick?! [

A. This is an aneurysm of the posterior communicating artery which is causing CN III to be affected! ]

Q. Friends, this concept comes up I hear on every exam and hospital pimp session:

If you get a man with a history of atherosclerosis, and he dies very suddenly, and he had no thrombus to cause an MI, he died of a VENTRICULAR ARRHYTHMIA

Q. I present you with a patient who has angina at rest with atherosclerosis, is this:

Prinzmetal angina  
Stable angina  
or Unstable angina  
or MI  
UNstable angina, [

A. KNOW if you get a version asking Prinzmetal's, you see ST elevation on stress ECG and ST depression with exertional/stable angina ]

Q. Here is one that rings through eternity on USMLE (rhymes!):

Case: A 15 year old soccer player named Goober comes into your clinic because of acute, serious throbbing pain in the right knee and is limping. He was "clipped" on his lateral right side of the knee. What three structures are affected [

A. This super HYer is the triad of anterior cruciate ligament, medial meniscus, and medial collateral ligament. (Think in abbreviations, ACL, MM, MCL)

]

Q. If I give you a case with a lumbar puncture (w/ a pic), and ask with arrows where do I get CSF from, can you tell me?

(Choices: Dural, Subdural, Subarachnoid, Arachnoid)

Also asked is between what two spaces is CSF taken? [

A. IT is Subarachnoid, the most common wrong answer is arachnoid or pia mater.) between L4 and L5 ]

Q. Some patient comes with a history of arrhythmias and is on a med and she presents with antinuclear antibodies, arthralgias, rash. What med is she on [

Procainamide, KNOW that this and HYDRALAZINE gives SLE like symptoms (drug induced ]

You will be given a diagram with the Arachidonic acid products pathways with arrows everywhere. You have to know which arrow is pointing to where Zafirlukast acts. (Don't confuse with Zileuton) [

Zafirlukast acts on the arrow pointing at the end step where

Leukotrienes are inhibited. Zileuton acts before and the level of Lipoxygenase BEFORE HPETE. Don't forget! Review that classic diagram, it is in BRS and [FA](#) ]

A pt complains to you about his skin thinning and mild osteoporosis and saying his esophagus burns. What med is he on that causes this? (Very popular point) [

He is on a Glucocorticoid, notice that I did not say "buffalo hump", or central obesity. The boards avoids "clicker" words. ]

Case: If I present a sideways angiogram of the head, choose the arrow pointing exactly to the sigmoid sinus AND, can you point to the cavernous sinus? [

The cavernous sinus is right behind the eyes and the sigmoid floats along the back. LOOK at WEBPATH ]

Case: What is the proposed mech of action of Lithium, and does your patient have hyper or hypothyroidism? What about poly- or oligouria? A MUST KNOW [

You bipolar patient has hypothyroidism and polyuria, Li blocks PIP cascade. ]

YOU WILL KNOW THIS CONCEPT!:

Case: A 27 yo AID patient has pulmonary complications. Exam of tissue shows yeast-like with capsules. What does he have? Secondary seen is how do you treat? Very tricky. [

He has Cryptococcus Neoformans, NOT Pneumocystis carinii due to ID of the capsule. Treat Cryptococcus with Amphotericin B. KNOW Cryptococcus usually causes meningitis, BUT, it also easily hits the lungs.

While on the SUPER HY topic of AIDS: I remembered I have to tell you... ]

Case: 32 yo male has demonstrated AIDS and you see cysts containing sporozoites can be seen with silver-stained preparations in the lungs, and he is rather asymptomatic. X-ray shows interstitial infiltrates. What now are you thinking and what drug will you grab! [

He has PCP, the most common disease of the AIDS, treat with TMP-SMX!!!!

USMLE LOVES... ]

Case that you nailed as Influenza...secondaries seen are where does it replicate? Pick among answer choices does it have envelope? Linear or NOT? [

It along with HIV are the only RNA viruses to replicate in the NUCLEUS, and.... it has an envelope and is linear single stranded!!!!!!!!!!!!!!!!!!!! ]

BIGGIE CANDY KWESCHON

A thousand times you will see...

A pt or question defining the subject of DOPAMINE (A million dollar concept). Which dopamine receptors are excitatory, which are

inhibitory, and is the second messenger cAMP or Ca? This concept alone will let you answer a thousand questions, seriously... [ The oh so important Dopamine has:

D1 and D5 which are excitatory which rev up kidney perfusion in shock, AND

D2, 3, 4 are inhibitory. Most schizophrenic drugs work on the D2 receptor which is inhibitory!!!! Wow, I feel great!

Finally, dopamine works on G-protein coupled cAMP second messengers...

Easily one of the most missed because people THOUGHT they knew: ]

PIC: HISTO of muscle fiber. Can you do these if arrows are everywhere?

- 1) Point to myosin fibers
- 2) Point exactly where ATP works/acts in EM.
- 3) To what does Ca bind to (answer is diff for smooth and skeletal muscle)

ANSWER ME, PLEEEAASE! (Well, silently, I cannot actually hear you) [

- 1) Myosin are the middle lines/area (Look up Histo atlas)
- 2) ATP is bound to myosin on the Head
- 3) Ca binds to troponin in skeletal muscle and CALMODULIN (which activates MLCK)

See, isn't it easy to forget? So DON'T! ]  
HARD ONE:

Patient complain of gradually worsening shortness of breath, progressive exercise intolerance, and fatigue, and swollen feet. He is an older man with amyloid deposits everywhere? From 4-6 answer choices of -myopathies, what does he have? (Hint: Loud diastolic S3 heard) [ he has the rather rare but often quizzed Restrictive Cardiomyopathy (myocardium is stiff) ]

Case: (VERY COMMON)

Young child with clinical triad of mental retardation, epilepsy, and facial angiofibromas. What associated cancer is common CNS hamartomas and cardiac rhabdomyomas You will see skin lesions so don't pick neurofibromatosis as the answer choice for the pre cancerous condition or I will cry.

You are given a case and asked to quickly calculate the ejection fraction. What's the equation? [ Stroke vol/ EDV ]

You will be asked questions about Down Syn. Tell me:

What is the organ most commonly affected (although Down's hits all systems)?

What cancer is associated?

What hormone do you often treat them with?

Is alpha feto protein low or high at 14 week gest? [

Cardiac (e.g. VSD)

Cancer is ALL

Hormone is thyroid hormone

Alpha fetoprotein is low in testing ]

You will know Jedi Knight,

A pic with B1 receptor, which neurotransmitter acts here (Epi, norepi, Ach, Dopamine)?

Now you see a pic of Lung with B2 receptors. Does same neurotransmitter act there? [

**BIG CONCEPT:**

Norepinephrine acts on B1 receptors but NOT B2 receptors (epi does though) ]

Picture like on Webpath of LOBAR Pneumonia. Histo shows encapsulated orgs. Then you see myriads of bact/fungi/viruses as possibilities. What is your first choice [

Strep Pneumoniae! ]

Slide with megaloblastic anemia, pt looks like a B12 def. Intrinsic factor administered. Patient improves. What disease did he have? (Pick between terminal ileum deficiency and atrophic gastritis) Also, could there be a bug involved? Which one? [

He has atrophic gastritis fr. H. Pylori. ]

Quick! Can you tell me what is the term for the most appearing number amongst a given series of number values [

it is called the MODE. Came up before ]

Fast! Tell me the ABCs or name three anaerobes and what is name of enzyme lacking which makes them vulnerable to oxidative damage? [

Actinomyces

Bacteroides

Clostridium

They are missing catalase. Treat with Clinda above the diaphragm and Metronidazole below the diaphragm!!! ]

You are given a case with a druggie and he has Hepatitis C. Choose and tell me if it is RNA/DNA/SS/DS/Helical/Square [

RNA, SS, LINEAR (remember that all RNA viruses are single stranded

34

except Reovirus, AND the letter PCR denote the NON-ENVELOPED VIRUSES or P-Picorna, C-Calic, R-Reo) ]  
You will see this:

A man comes into your office acting very strange, sticking out his swollen tongue, and complaining of numbness and prickling. He is a vegetarian. What two crucial reactions cannot occur because of the missing diet cofactor [

This is classic triad for Vit B12 deficiency. Homocysteine METHylation and Methyl malonyl CoA step into TCA cycle is blocked! Ain't that awesome, I mean the knowledge, I feel sorry for the patient though ]  
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Here is a biggie:

Your patient goes for plastic surgery to look like Michael Jackson and he is given succinylcholine (muscle relaxant). He suffered prolonged respiratory paralysis and muscle paralysis afterwards! What enzyme or mineral is defective? (Hypomagnesium, Hypokalemia, Pseudocholinesterase def) [

It is pseudocholinesterase deficiency. Many causes, but pregnancy, neonates, elderly, burn victims, pesticide poisoning, can be presented by the Boards ]

Banana-split question! A patient presents with epigastric symptoms and melena.. You should pick PUD or peptic ulcer disease (this disease is everywhere, like air), BUT there is a secondary! Labs rule out H.Pylori (most common). What is the next HUGE cause? [

Chronic NSAID use. Man, I had to do so many anal exams for this (checking for bleeding with those little Heme cards). They call it the M-

3 student consult. ]

Wow this a biggie fry with a biggie drink question:

You have a patient with a description of allergic rhinitis (some 50 million Americans suffer this, you will see this tested), and he is taking steroids, antihistamines, and pseudoephedrine. He is depressed and wants anti depressants. You pick one from 5 choices and your attending knocks you silly. Which one did you pick that is a no-no? [

MAO inhibitors cause hypertensive crisis. You deserved the punch. ]

Every single person sitting for USMLE gets one of the Immune def questions, no exception I hear. So, you have a young patient with a gene defective in making myeloperoxidase, thus the cause of his recurrent infections. What cells are weakened, what is the MECHANISM LOST, what is the metal ion in MPO? [

(You will see this case, or DiGeorge's, SCID, etc.)

The ability of the immune cells to engage in respiratory burst is cut off. Myeloperoxidase, MPO, catalyzes the conversion of hydrogen peroxide and chloride ions (Cl) into hypochlorous acid. Hypochlorous acid is 50 times more potent in microbial killing than hydrogen peroxide.

Neutrophils are weakened which contain Fe

Hey, compare and contrast this oft seen lingering factoid! ]

Case: You get another child just like the previous case with bacterial infections. BUT, this time you discover there is a defect in microtubules and phagocytics. You see severe gingivitis and oral mucosal ulceration PLUS albinism on the skin. Secondaries: What is the disease, what two bugs eat at you, and what is the first drug you reach for? [

Here is Chediak-Higashi disease (not too common). But you get strep and staph infections and you treat with Acyclovir. The KEY to this diagnosis is the mouth stuff and hypopigmentation! You start with Acyclovir THEN give the missing globulins through IV because Chediak Higashi is an IMMUNE DISEASE and Acyclovir boosts the recovery while fighting the viruses. The globins you transfuse will address the Staph and Strep. OK? ]

IMPOSSIBLE ODDS, but, look...

Still in your peds rotation, your next patient comes in with recurrent bronchpulmonary, bacterial, neurologic disease, thymus aplasia, telangiectasias, growth retardation, and impaired organ mutation, and is walking funny and waddling. What are you looking at NOW? [

HERE,

you are looking at Ataxia telangiectasia, where both the T and B cells are busted. The alpha fetoprotein levels are always elevated, and they key finding is ATAXIA!

OOHMHIGOSH! ]

ANOTHER, would you believe, child, younger this time, 2 years old

walks in, again with recurrent bacterial, fungal, infections. His mom say he suffers often from candida. And you note he has IL-2 def, poss. reticular or ZAP-70 gene def. Your attending walks in and says he will suffer from PCP and Herpes. She (attending) asks you to write a prescription for .... ????. What disease? What med? [

This unfortunate child has Severe combined immunodeficiency or SCID. They usually die by age 2 from PCP. You must prophylaxis with TMP-Sulfmethoxazole. Consider IV globin transfusion if counts stay low. ]

YOU are starting to hear TWILIGHT ZONE MUSIC BECAUSE....

YET ANOTHER CHILD comes into your peds clinic with an immune def. But this time, the child is hyperreflexic on exam, has abnormal facies, congenital heart disease, hypocalcemia on labs, and increased susceptibility to infections. A radiograph shows he has no thymus! What do you tell your chief? What do you prescribe? [

This child has DiGeorge's Disease or thymic aplasia. His 3rd and 4th arch failed to develop. This concept is a favorite of NBME. Including considering marrow transfusion, you must prescribe Calcium salts and Vit D!!!! ]

106. Fiddlesticks, just when you thought you were free,

ANOTHER child walks in with his mom with another immune deficiency. (I keep on with rhymes, he-he-he). Here he is 4 years old, with recurrent otitis media, eczema, and thrombocytopenia from Strep pneumoniae. AND, he bleeds a lot. His IgM is low. Your attending and chief are wondering if you are able to distinguish all these immune def. diseases. Will you get an honors grade(Name disease)? What will you treat with? What is mech that is broken? [

He has X-LINKED Wiskott-Aldrich syndrome. This is often confused

with the others and Bruton's on exams..hint, hint. But remember the tendency to get attacks from capule bugs like Strep, with otitis, eczema , and BLEEDING. The key is LOW IgM, High IgA,and the bleeding. IgM response curtailed. He is not nearly as bad as SCID case, and you must give him amoxicillin (there are a lot of options here, like you can give ceftriaxone too) plus globins.  
FOR ALL OF THESE IMMUNO CASES STAY AWAY FROM LIVE VACCINES. The NBME will ask you this, if not now, then later, if not later, then someone will ask you..... ]

107. Oh no mate! Another ONE! BUT YOU HAVE TO KEEP GOING! EVERY TEST WILL HAVE ONE OR MORE OF THESE DISEASES BECAUSE YOU CANNOT AFFORD TO MISS THEM. IT AIN"T JUST ANOTHER COLD BABY.

This boy has low IgG and presents like WAS syndrome with continued bacterial infections, diarrhea. And you find out this is X-linked too! In the absence of functional Btk, mature B cells expressing surface immunoglobulin and the marker CD19 are few to absent. What disease? [

Here is the first immune def. described by Dr. Bruton. So similar to WAS syndrome, but WAS boys will BLEED. OK? Get them straight in your head!!! IT IS HARD! ]

108. This is just a day that won't end! Another child, this time let's make it a GIRL, comes in with OF COURSE, an immune deficiency with bacterial and fungal infections. HOWEVER, the NBME has to give up some info (er...I mean the girl's features do I mean..). {This knowledge is good to know for life of course, not just a test}.

Soo...you note that all the immune def. choices are mixing but you see her presenting with lymphadenopathy, hepatosplenomegaly, growth failure, and stigmata of chronic skin infections. Your fellow med student (star student) whispers something about def. w/ phagocytes. TWO distinct hints. AND culture comes back and she has Aspergillus. TELL YOUR ATTENDING WITH CONFIDENCE.....!????? [

This is Chronic Granulomatous Disease. This is marked by the granulomas (skin stuff) and key words phagocyte def. and Aspergillus infection. Are you getting it all down. YOU HAVE TO IN ORDER TO PASS. All the immune def. will be among answer choices, they differ so slightly. Master them! ]

The clock is approaching 5:30, AND the nurse squeezes in another patient and whaddaknow, he has immune def. with recurrent bacterial sinopulmonary infections. The NBME, er, I mean attending starts pimping you with choices...but you note that the patient is OLDER, LESS SYMPTOMATIC (i.e. less severe disease), and complains of GI symptoms too like diarrhea. What words are coming out of your mouth? [

This is the OH SO COMMON IgAD or Immunoglobulin A def. Many stay asymptomatic, IgG and Neutrophil levels could be normal. Give antibiotics....Confused yet? I hope not, I hope I gave you cues to distinguish the diseases

As an aside, I spoke to 100 people and they all scream back, KNOW ENDOCRINE!}

Soo.....

Now it is 5:00 pm. You are beat, but happily this time your patient is not an immune def. case. BUT, you rub your eyes because standing in front of you are 3 answer choices..errr, i mean fraternal triplets (listen I am tired, I have not slept yet)... ]

LISTEN CLOSE, THEY ALL HAVE systemic symptoms such as weakness, fatigue, malaise, and fever low-grade, two have neck pain, one does not. Physical exam shows hypothyroidism. But here is the concept that comes again again again again:

Child A has hypothyroidism, neck pain, and fever chills and dysphagia

Child B has hypothyroidism, neck pain, and sort of looks a little like he was hyperthyroid last week from history

Child C is shorter and his neck is NOT tender and gets constipation a lot

SUPER CONCEPT: Who has what???????????????????? A must know!! [

Child A has ACUTE THYROIDITIS (bacterial) so you must manage aggressively with antibiotics (penicillin G is DOC)

Child B has SUBACUTE THYROIDITIS (viral) so you just give aspirin and return visit. (KEY!!, HYPER, then HYPOTHYroid features)

Child C has AUTOIMMUNE THYROIDITIS. This is bad because it is a life-long condition. Treat with levothyroxine.

THIS QUESTION WAS WORDED VERY ODDLY, BUT YOU WILL REGRET IT IF YOU DON'T TAKE HOME THE CONCEPT!!!! ]

as to the HY Concept 110, consider that...

someone I knew said they had to distinguish the hypothyroiders (I did not say it, but you KNOW TSH is high right), and then, he was given a series of graphs pointing to thyroid levels. Recall Subacute thyroiditis can start with HYPER then HYPO thyroidism. The NBME likes to ask things in a scary way that makes you forget everything, even your own name during the exam. HOLD YOUR WITS. YOU KNOW MORE THAN YOU THINK

What MAJOR MAJOR drug other than trimethoprim blocks the loved enzyme dihydrofolate reductase? [

Methotrexate:

KNOW you often use it for rheumatoid arthritis, hydatiform mole, leukemias and it works its magic in the synthesis phase, stopping thymidine (thymidineless death) and blocks protein synthesis. As I mentioned, I AM NOT REPEATING "EXAM CONTENT" but know that the NBME will give you a picture and ask you to POINT to where methotrexate works its magic. They like doing that. Last year, I wish someone told me just how the NBME likes us to understand stuff. No one told me. Now I want to lift others up.

AGAIN BEING VAGUE AFTER AZSKEPTIC's warning...

I think that is ridiculous to say that I am disseminating material with all due respect to AZSKEPTIC. Like I said, there is a purpose to this very very hard test. There are trillions are pieces of info, and you HAVE to pick and choose. But the NBME needs to know that you are not going to come to the US and kill people, soooo..... I relay the concepts like "Don't give ACE inhibitors to a pregnant woman". This is SURE to be on the USMLE Step 1,2,3, but am I breaking a RULE to tell people this VITAL piece of info? I am giving out "exam content" in the sense that I am relaying that IL-5 revs UP IgA and IL-6 (like IL-1) revs up the acute phase response...BUT THESE ARE the BASICS that NBME wants US doctors to master. That is why if I recall from my test a case of a drug overdose and how to treat it, I FEEL COMPELLED to say it on this board in such a way that does not violate copyright laws or "giving out answers". Because....every doctor in the world SHOULD know what drug a person probably took based on his or her symptoms and how to treat them. I encourage everyone to share the concepts after their exams. The NBME should not mind unless I tell everyone that "if you get test version KX-115 then the answer to #1 is B, #2 is A, #3 is E, etc." But to share knowledge that the difference between ALS and multiple sclerosis is that ALS has no sensory deficits, well that is just making everyone wiser and better doctors. What do you guys believe?

Anyhow, let truth reign! Let's say a patient comes into your office at 6:00 pm, my my, and he has vertigo and remarks that he has difficulty with taste and swallowing. Before you give a prescription for antivert, is this a dysfunction of the vestibular apparatus of the inner ear? Or is it a brain stem issue? If it is a brain stem issue, what two nuclei and nerves are involved ]

Tricky case. Because vertigo has many causes, note the DIFFICULTY with taste and swallowing. This pushes up the suspicion of a lesion to the nucleus solitarius and ambiguous with nerves 7,9, and 10 also lesioned. AND for the cherry, we see that all the time with a

POSTERIOR INFERIOR CEREBELLAR ARTERY stroke which supplies that area! See? [

SO, don't just send them home with antivert and a reminder slip for a return 3 month visit!! (This IS USMLE MATERIAL, but a MUST KNOW FOR LIFE!) IF we avoid all discussion and thought of USMLE material, what is the USE ]

DRAT! AGAIN...to BE VAGUE....

KEY KEY KEY point. if a patient has no pupillary reaction to light shined on the right side but there is a reaction to light in both eyes, when light is shined on the left. The lesion is what? NOW I change the patient so there is pupil rxn to light on only the right side, when light is shined in either eye. NOW, where is the lesion? [

ABSOLUTELY USMLE BEGS FOR YOU TO UNDERSTAND THIS. IT WILL BE ON YOUR TEST, IN YOUR LIFE, IN YOUR PRACTICE, IN HUMANITY FOREVER....

For the first patient, the lesion is the right CN2. For the second, the lesion is left CN3. KNOW IT! ]

NEURO IS PRIZED LIKE A CHILD FOR THE NBME... so,

Say your pt comes in and you touch both her corneas one at a time with a q-tip, and you note that ONLY the LEFT eye blinks, then which cranial nerve is activated? [

KEY TO THE CITY point!

Right CN7 (NOT THE LEFT ONE, common mistake) ]

will try to be vague so I don't anger azskeptic or NBME, without peeking, what drug blocks out enzyme dihydrofolate reductase!!???  
(This is NBME's 10 ten list of favorite enzymes) [

Trimethoprim blocks it. NOW FOR THE NEXT QUESTION... ]

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Here is a King Kong Konzept!

Two patients walk into your office. Listen close.

Patient A has a stroke in motor cortex that lesions UMN tract to central facial n.

Patient B went on a camping trip and has a lesion to the LMN CN VII.

Tell me how each patient will present on physical exams... [

Patient A will have CONTRALATERAL, and LOWER QUADRANT paralysis.

Patient B will have same side Bell's Palsy features (can't smile and may drool on affected side)

PROMISE ME that you will know this for LIFE for your PATIENTS' HEALTH! because tx are distinct! Review neuro pictures, it will be clear. ]

Presenting the GODZILLA of concepts:

Now it is 6:30 in your peds clinic (and you are wondering if you are actually in a surgery rotation), and the nurse brings in a 15 year old boy with "fatty" thick calf muscles. The child trips on a toy and strangely uses his proximal muscles to assist in standing....

Ahh...you are thinking Duchenne's (gave it away), BUT WAIT, your attending says NO! What is the disease and what is the defective protein? And what are the labs? Crucial...crucial... What is the only drug with known effectiveness for his condition????? [

The disease is Becker's, a milder form of progressive muscular dystrophy.

The defective protein is DYSTROPHIN!

The labs show elevated CPK!

The ONLY drug with current known effectiveness is prednisolone.

BAD, BAD, disease. Treat well....and be sincere. ]

To avoid angering AZSKEPTIC, I will make my concepts less and less sharp and more vague and fuzzy so that I can still feel I am helping and yet not feel worried that powers greater than me will get upset...even though I cannot imagine why...but then then again, the world is MAD...sometimes...

OOOHHH!! What is bigger and stronger than King Kong or Godzilla. Maybe that new Transformers Robot. This concept is at least that big!

Anemias, SO COMMON, SO TESTED, SO SEEN, SO DIFFICULT ON EXAMS...SO DON'T NEGLECT...

You see a female with a blood smear with RBCs small n'round, physical is anemia, hyperbilirubinemia, and abnormal results on the osmotic fragility test. OK OK she has hereditary spherocytosis (so common in clinics). But of course, you need to know:

- 1) What protein is defective?
- 2) What is the inheritance pattern?
- 3) What are the main two complications?
- 4) Surgical treatment?
- 5) What do you, an intern prescribe to them? [

KNOW IT AS YOUR LIFE DEPENDED ON IT!

- 1) spectrin
- 2) AD inheritance
- 3) cholecystitis and aplastic anemia
- 4) Splenectomy
- 5) They need folic acid! ]

What's next, yes, the MECA-Godzilla or maybe Mothra of Concepts:

Another patient comes in weak with signs pointing to anemia. You take a blood smear and whoa! cytopenia...blast cells, reticulocytes, sparse RBCs. And you know this is not autoimmune because it is recent. Hold it...she mentions she had a gonorrheal infection and is on a med. OH YES! OK, so what is the disease, name of the med she is on AND what will be the name of the med you give her as you transfuse bone marrow!?!?! [

Chloramphenicol is the drug she is on that caused aplastic anemia. AND you can give cyclosporine or a steroid along with her transfusion. REMEMBER, aplastic anemia has many causes so be careful. Benzene, pregnancy, CMV, HIV, EBV, and autoimmune causes are all to be considered ]

Can we do it over Godzilla? Yes, here is the Pillsbury Dough Boy of Concepts:

An African American male comes into your office with signs of very very mild anemia, almost no symptoms, a little jaundice. His main complaint--a UTI. Your senior hints this is the most common enzyme pathology. A smear shows Heinz bodies (review please). Now your senior starts a pimping away.

1) What is his disease?

2) Why is it so prevalent?

3) What does the enzyme catalyze? What is the end product?

4) You grab some sulfamide and nitrofurantoin to treat his Urinary Tract Infection and your attending smacks you on the other side of the face that she missed before. Why was she so upset with you? [

1) G6PD Deficiency

2) It confers protection against malaria

3) The G6PD enzyme catalyzes the oxidation of glucose-6-phosphate to 6-phosphogluconate while concomitantly reducing the oxidized form of nicotinamide adenine dinucleotide phosphate (NADP+) to nicotinamide adenine dinucleotide phosphate (NADPH). NADPH, a required cofactor in many biosynthetic reactions, maintains glutathione in its reduced form. RBCs need NADPH to protect itself against oxidative stresses. (Long winded explanation, but you have to know it., sorry).

4) You cannot give an oxidizing agent like primaquine, choroquine, or a sulfa drug, or nitrofurantoin to a patient with G6PD def. Their RBC will hemolyse and you will lose your license and your attending will lose her's and your hospital will close and turn into an apartment complex. ]

Case: an elderly psych patient of yours complains of arrhythmias, what drug is she on? (amitriptyline or thioridazine or lithium or olanzapine?) [

she's on amitriptyline, a tri cyclic antidepressant. (OTHER tricyclics are imipramine and nortriptyline.

Bad side effect: arrhythmias. Review MOA. ]

Another elderly psych patient comes to your office with complaints of colds and a peripheral smear shows low WBCs, what drug caused this?

[ MOST LIKELY one..Secondaries: which two receptors does it block

Clozapine, blocks 5HT-2 and dopamine. Causes leukopenia. ]

Yet another elderly psych patient comes into your office this time with constipation and rigid muscles and (hint other antimuscarinic sym). He was given a med FOR an ACUTE psych episode where he shouted and hit others. What is the drug? 2nd: Receptor/MOA? And Name at least two other drugs in this family

[

Answer: He is on Haloperidol (used for Positive symp, in ACUTE cases), the drug blocks D2 receptors, and fluphenazine and thioridazine are within this family named NEUROLEPTICS,

assoc of course too tardive dyskinesia!

(these are different from the atypicals, make sure you know the atypicals are risperidone, but also clozapine and olanzapine--neg symptoms controlled more, diff receptors involved) ]

An M&M candy question:

Woman walks in with chronically sore right knee. She is neg. on labs for rheumatoid factor. Under microscope, you see crystals appearing shorter and often rhomboidal. Under a polarizing filter, crystals do not change color depending upon their alignment relative to the direction of the red compensator.

What on earth does she have and what is the name of the crystals deposited! [

Answer IS NOT rheumatoid arthritis or gout, BUT, the answer is pseudogout, and you see calcium pyrophosphate crystals as opposed to birefringent needle crystals in gout! P=Pseudo=Positively birefringent ]

an M&M peanut candy question:

Next a child enters your clinic with chronic diarrhea and fatty stools. A younger med student asks you if he has Cystic Fibrosis, Giardia, or Ulcerative Colitis, or Chron's. But, YOU go further and order labs. They come back with weird D-xylose test, anti-IgA antibodies, B-cells in the lamina propria

You go Hoorah because you know:

- 1) Disease
- 2) Etiology (viral/immune/etc)
- 3) is there a specific substance or drug he should take or avoid? [

- 1) He has Celiac sprue
- 2) Autoimmune/hereditary/Europe
- 3) Avoid gliadin wheat in diet ]

An OVERSIZED CANDY question (BIG FAVORITE)

Next, you have a older African American male who comes in with chronic CHF and began a new medication. But he suddenly one morning found his left foot joints swollen and so tender even the weight of the bedsheets are so painful! (BIG HINT COMING). Labs come back and you see crystals with needle shapes (shown a pic), (-) birefring...

But NOT SO FAST, THIS IS USMLE!

- 1) Tell me the likely med he was on and at least two other meds which could cause this condition.
- 2) What is the short term and LONG term treatment?
- 3) MOA (Mech of Action) of disease?
- 4) What compound builds up?
- 5) What foods should he avoid?
- 6) Bonus Biggie: He had a great grandfather who had similar symptoms but was mildly retarded and scratched himself like crazy!

52

Dx? [

- 1) Thiazide diuretics, Cyclosporine, Nicotinic Acid and a LONG LONG list can do this.
- 2) Colchicine short term/and Indomethacin and Allopurinol long term
- 3) Uric acid precipitates from supersaturated extracellular (ie, synovial) fluid. The resulting crystals stimulate phagocytosis by neutrophils and initiation of the inflammatory cascade. OUCH.
- 4) PRPP
- 5) (Purine rich foods (especially of anchovies, sardines, sweetbread, kidney, liver, meat extracts)
- 6) Lesch-Nyhan syndrome, (a NBME favorite) ]

NOT IN THE FORM OF A QUESTION BUT THIS IS A LIFE POINT:

People keep missing Goodpasture's and Wegner's, you know, the diseases with BOTH kidney damage and Lung damage. Can't discern.

POINT IS THAT WITH WEGNER'S GRANULOMATOSIS LOOK FOR UPPER RESPIRATORY SIGNS LIKE SINUSITIS TO DIFFERENTIATE ON THE BOARDS, ER, I MEAN CLINICS!!!!

So hard, but the reason so many friends of mine failed is because they could not differentiate the subtle differences of:

Case; Pt comes in and says she has: Inability to eat dry food, such as crackers, which sticks on the roof the mouth Tongue sticking to the roof of the mouth She always has to be putting a glass of water on

53

their bed stand to drink at night. She has difficulty speaking for long periods of time, and her eyes are dry and her right wrist is starting to hurt.

HERE'S THE MONEY:

- 1) Disease?
- 2) Which HLA is involved?
- 3) Drug of Choice (DOC)?
- 4) What dx, is she at increased risk for? [

Answers:

- 1) Sjogren's syndrome (they'll give choices like Reiter's, PSS, etc.)
- 2) HLA 3
- 3) Pilocarpine to stim. secretions! And eye drops!
- 4) a lymphoproliferative disorder ]

This one's is KEY:

Next, a male patient comes in with myalgias and low back pain. He also has reddish (infection like) tinge on his left eye. Your subordinate med student yells out! "Ankylosing spondylitis!, Rheum. Arthritis!. But not so fast! You note that labs came back positive for HLA B27, BUT so did chlamydia culture!!!!

You scold your med student.

1 Why? Because he had picked the wrong disease, the right one is? [

1.. Reiter's syndrome!

The KEY finding is the Chlamydia or could be Salmonella and urethral connections. The closing of the triad is the conjunctivitis. Don't be tricked my brothers and sisters! ]

YOU COULD BE ASKED BY YOUR ATTENDING/BOARDS WHICH BUG IS HE MOST SUSCEPTIBLE TO...(they have millions of ways to twist the questions but the concept remains the same!) MINOR ADDENDUM on hy concept 129, Reiter's= male Sjogren's=female

This one's is a MAGIC KEY:

Next, another male patient comes in with myalgias and low back pain! He also has reddish (infection like) tinge on his left eye. HLA-B27+ Unreal! You are about to say that you have another case of Reiter's, but you note his labs reveal cardiac anomalies....Your subordinate med student yells out....What? [

(This time your med student is RIGHT!)

This one is ankylosing spondylitis, compare carefully with Reiter's. One triad has the heart, the other has the urethra!!!! Got It? Got Milk? Got

55

Love? Got God?  
Oh boy.. ]

Another patient comes with lower back pain and the usual suspects. But she says her arthritis often comes with a fever and is WORSE IN THE MORNING! You know this dx of course, you know it is NOT osteoarthritis, which has osteophytes, but what if I presented a pic of the hands with arrows to all joints. WHICH ONE(S) OF THE THREE JOINTS ARE AFFECTED (DIP, MCP, PIP)? (See, you HAVE to know pictorially the secondaries.)

Besides NSAIDS, what other three drugs are often tried? [

She has rheumatoid arthritis, + rheumatoid factor. This autoimmune dx has systemic symptoms like her fever and malaise. The answer is:

MCP and PIP joints

OSTEOarthritis has DIP joint inflammation ]

Q. So depressing...a young girl comes into your office with a fever and history of weakness, infections, cardiac flow murmur and petechiae. You order a CBC and find that her smear shows what looks like immature leukocytes...but you cannot seem to distinguish between ALL and AML (THIS IS A MAJOR TEACHING POINT, BECAUSE THE SMEARS CAN LOOK VERY VERY SIMILAR AND THERE WILL BE BOTH

56

ON THE ANSWER CHOICES, SO LOOK IT UP IN A HISTO ATLAS!). You sud and more nucleoli. You think you nailed the diagnosis: but see this:

- 1) What is it?
- 2) What is the most common sub-classification?
- 3) What were those thin stick like bodies?
- 4) What enzymatic test NAILS the diff between ALL and AML? [

THIS IS A BIGGIE!

A. 1) This is AML, the myeloblasts have delicate nuclear chromatin, strong nuceoli, LOOK FOR GRANULES in cytoplasm

2) M2 is the largest incidence

3) Auer Bodies

4) Myeloperoxidase - positive test ]

I AM SCREAMING YOU NEED TO KNOW THIS!

forgot to mention also regarding HY Concept 133 the following: The test, or attending, could only tell you the markers: so you have to know that:

CD3 is T-cell so think

CD19 is B-cell so think [

CD3 is T-cell so think ALL

CD19 is B-cell so think AML

A lot of this stuff is NOT mentioned in [FA](#) but I KNOW is part of USMLE Step 1 understanding so I am making sure you are educated about it! ]

Q. A 40 yr old gentleman middle aged enters your clinic with a complaint of fatigue, weight loss, splenomegaly. You know that this is critical and SUPER HY because the NBME knows that this is something you cannot afford to miss. A lot of newspapers report that **doctors are missing leukemias thinking the disease is a common cold**. Big mistake and big problems. I know the NBME is stepping up its coverage of leukemias because it is a diagnosis you cannot afford to miss!

So back to the case...the gentleman's blood smear looks distinct with a lot of mature neutrophils (twisting nuclei) (CML, ok I gave it away) is very distinct from ALL, AML and CLL on slides, it has a lot of mature neutrophils. But of course you need the secondaries....

1) What is the chromosome that is definitive of CML? Definitely will be asked.

2) The translocation results in the formation of what gene? [  
A. Key is the Ph or Philadelphia chromosome

2) You will see bcr-c-abl fusion gene ]

Q. Now to complete, you see an elderly male come into the office with vague symptoms of anemia but is very stable over years. His peripheral smear is distinct and shows a whole bunch of lymphocytes

58

(this is CLL, OK sorry I gave it away but you need to see the slide, it is very distinct). But here is the questions you will encounter:

- 1) In this rather indolent disease, what is the CD marker?
- 2) Is CLL mostly B or T cells?
- 3) What main leukemia is CLL associated with? [

A. 1) CD5

2) B-cells most than 95%

3) small lymphocytic lymphoma

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Q. HARD QUESTION, but YOU NEED TO KNOW:

speaking on leukemias, you finally get to figuring out the NBME is talking about a myeloid leukemia where you will see a bunch of monoblasts on a smear (YOU HAVE TO KNOW THIS). But you are asked, which FAB classification is this? (This is NOT TOO MUCH DETAIL) [

A. You are looking at M5 type. This is crucial. ]

Q Did you know?? That many people told me they could not differentiate Multiple Myeloma (A ABSOLUTE MUST KNOW, KNOW EVERYTHING ABOUT THIS ONE, READ UP ON IT) and Waldenstrom's macroglobinemia on questions. Tell me what is the difference [

A. Multiple Myeloma has an M spike of IgG while..

Waldenstrom's macroglobinemia's M spike is IgM!!!!!!!!!!!!!!!!!!!!!! ]

Q UGLY UGLY, but my sources tell me after leukemias you HAVE to master LYMPHOMAS, and those are much harder to manage as concepts. I beg you to review [Error! Hyperlink reference not valid.](#) or [Kaplan](#). But at the very least, tell me which of the Hodgkin's lymphomas has the best prognosis? [

A. Nodular sclerosing type has the best prognosis and is also most common with few Reed Sternberg cells ]

Q. Key concept is this:

You have a case that confuses you about leukemias and you are lost, but you see DIC (Disseminated Intravascular Coag) as an answer choice. Now you know that is the answer because there is a clue that the patient is a woman giving birth...What key finding nails the diagnosis and is often asked? [

A. The key is they must and will give you lab findings where aPTT, PT is elevated for DIC, and KEY is low platlets and POSITIVE D-Dimer test ]

Q. Sorry to say, but you will be confused because you will see a case of anemia symptoms and you have to know the differences between:

HUS (Hemolytic Uremic Syndrome)

ITP

TTP

DIC

Hemophilias

von Willebrand's disease

They can all present so similar with the same signs like bleeding problems, microhemorrhages, etc. You need to know the keys to differentiate! Especially confusing is choosing between HUS and TTP (TTP and ITP are easier because TTP will have neuro findings). So my million dollar question is: How do you tell HUS and TTP and Hemophilias and von Willebrand's apart on a test? [

A. Hey listen, Thrombotic thrombocytopenic purpura (TTP) is a life-threatening multisystem disorder of unknown etiology, first described by Moschcowitz in 1924. TTP and hemolytic uremic syndrome (HUS) are both thrombotic microangiopathies characterized by microvascular lesions with platelet aggregation.

TTP and HUS share the same pathophysiological etiology and may be varied expressions of the same underlying disease process. They both have abnormal bleeding times due to low platelets.

BUT HEY GUYS, KNOW that HUS is more common in children and is characterized by prominent renal involvement. TTP is more common in adults and is associated with pregnancy; diseases such as HIV, cancer, bacterial infection, and vasculitis; bone marrow transplantation; and drugs.

KNOW THAT... TTP and HUS have abnormal bleeding times and NORMAL aPTT and PT. Contrast with Hemophilias which have labs with prolonged aPTT and PT and NORMAL bleeding times!!!! (Another popular secondary is Hemophilia A= factor VIII, Hemo B= factor IX).

61

YOU WILL KNOW to pick vonWillebrand's because it has abnormal aPTT AND prolonged bleeding times.  
between HUS and TTP, you may be asked to know that TTP has SCHISTOCYTES visible on a smear. ]

SORRY THIS IS SO CONFUSING, BUT YOU REALLY NEED TO KNOW THIS COLD!!!! READ THIS PARTICULAR POST MORE THAN ONCE!

OK, again, this is a BIGGIE FRY AND BIGGIE DRINK,

Q. ANEMIA presentation again, so I won't repeat myself, but NOW YOU DEFINED A CLEAR CASE OF SICKLE CELL ANEMIA. This is easier to diagnosis for obvious reasons. The important secondaries will be:

- 1) What is the newest drug for treatment (NOT PAIN MEDS)?
- 2) What is the mechanism of disease?
- 3) Which bug causes an osteomyelitis?
- 4) Very important: What can trigger an onset of the sickling? [ A.
  - 1) Hydroxyurea!
  - 2) There is a substitution of valine for glutamic acid at the sixth position of the Beta chain!!!! KNOW THIS!
  - 3) Often Salmonella, maybe Staph

4) Any kind of hypoxemia or disturbance of core body temperature!!!!!!!!!!!!!!!!!!!!!! Look for respiratory causes!

Anemias are a favorite, you will see so many smears...and you have to know the two different thalassemias cold!!!

I am getting sleepy, so can you read up on these yourself? Sorry!

BUT...Know that B-thalassemia heterozygote is common and you can survive, but A-thalassemia IS DEADLY and will result in fetal hydrops.

ALL these are reported MUST KNOWS. ]

Q. Finishing off the all important anemias, a lot of my pals said you must understand the basics of microcytic/macrocytic/normocytic anemias. There are a thousand HY concepts here, (like Vit B12 has neuro stuff and Folate doesn't but BOTH are macrocytic), so master this. I will ask one candy bar question though, just to quiz you...

A child comes into your office and his mom said he was eating paint chips in their OLD apartment building!

What does his smear look like? [

A. This is a classic favorite of lead poisoning and the smear is hypochromic, microcytic, JUST LIKE IRON DEF, the most common of them all. ]

Q. Let's move to the NBME's ALL TIME FAVORITE OF SUBJECTS...DRUG SIDE EFFECTS. I hear over and over they want you to understand this because they said they have a responsibility to Congress to make sure IMGs don't come into US Hospitals and mix

drugs that turn patients into zombies. So...

Pt with tinnitus, headache, can't hear a thing. Patient has a heart condition. What med did this? [

A. quinidine, a class IA anti arrhythmic. ]

Q. Just think... you are in OB GYN in NYC USA and a woman comes in with convulsions. She is on birth control pills!!!!!!!!!! BIG POINT!

What vitamin is she going to lack??? [

A. She is showing deficiency of vit B (specifically B6 or pyridoxine). This all important vitamin is used in

TRANSAMINATION REACTIONS, SULFUR RXNS, RACEMIC RXNS, AND TRANSAMINATION, and must be in active form of pyridoxal phosphate.

MAJOR MAJOR MAJOR!]

TOMMYPOSTS 2 ( 149-200)

Q. I present you with a LM image of the thyroid with arrows everywhere. Tell me the cell and the exact location on the image where calcitonin is secreted [

A. The parafollicular or C-cells secrete calcitonin. Make sure of it!!!! ]

Q. The parafollicular or C-cells secrete calcitonin. Make sure of it!!!! [

A. It binds TUBULIN AND BLOCKS POLYMERIZATION OF

MICROTUBULES, THUS BLOCKING MITOSIS. ]

PARASITE S

Q. NBME wants you to understand all the HELMITHS, one of my students said he got a whole block of them! (he was prob. exaggerating though)

So, one by one... [

A pt of yours comes in with abd pain after eating raw fish. He looks lk he has cholecystitis. What drug do you give? What is the bug? (PIC GIVEN)

A. This is a fluke (looked weird like a worm), Bug is CLONORCHIS SINENSIS, treat with PRAZIQUANTEL. ]

Q. A young boy comes to your clinic with diarrhea after eating "mud pies", what is the bug and the tx? [

A. But is the infamous Strongloides stercoralis, tx. with Thiabendazole ]

Q. Oh, please note that ALL OF THE CASES YOU WILL SEE ON THE USMLE WILL LIKELY HAVE A HISTORY OF TRAVEL!

That said, you have a male pt, 30, with epilepsy coming in after eating "raw pork". What is the helminth and the treatment? [

A. the bug is a tapeworm--Taenia solium and you give Praziquantel and Niclosamide and a steroid to relieve CNS pressure because this bug swims everywhere, even in the CNS! (Pic given. slide)

THE CASE WILL give travel to Southeast Asia or maybe Africa. ]

65

Q. A traveler comes from Africa (could also be a West Alaskan Indian), and had told you he ate coyote and dog poop as a college dare! He is ASYMPTOMATIC but you see cysts in his lungs on X-ray. What's the bug and TX? [

A. Give him Albenza which is trade name for Albendazole which works by depleting ATP, and the bug if asked is Echinococcus. For this and the other tapeworm, Taenia, the guy could be scratching his rear end a lot so wash your hands! ]

Q. A pt of yours came back from Brazil and has dysuria and nausea. Plus he told you he ate a bunch of snails at a local exotic restaurant. What's the bug and tx? [

A. He has the famous Schistosoma Haematobium. In US it is rare because they don't usually eat a lot of snails! But know this fluke has many subtypes and can clinically present LIKE ANTHING! The NBME will have to be very specific. One key is it results in granulomas! Treat with Biltricide which has generic name Praziquantel. ]

Q. A pt returns to your clinic fr. Latin America with signs of Asthma. But a stool sample shows a round curved worm (slide is given). YOUR ATTENDING TELLS YOU THIS IS THE MOST COMMON HELMINTH INFECTION IN THE WORLD! You are looking at what and will treat with what? AND also seen is what is MOA of the drugs? BONUS, you must know. [

A. YOU are looking at ASCARIASIS. So common. Treat with

Mebendazole (WHICH WORKS BY BLOCKING GLUCOSE UPTAKE). AND pick Pyrantel pamoate (WHICH IS A NEUROMUSCULAR BLOCKING AGENT WHICH PARALYZES THE ROUNDWORM). I THINK Kaplan AND Error! Hyperlink reference not valid. mentions these bugs but not ALL THE NECESSARY and tested material is given.

This is FECAL ORAL SPREAD ]

Q. Don't you dare confuse this with Ascariasis. This nematode is quite prevalent in the US. That will be the give away and so will the fact that your peds patient is scratching his behind. Give me bug and drug [ A. Watch out, this one I am told is confused with Ascariasis, but it is Enterobius vermicularis and the case seen is a kid with an itchy "butt".

Treat with Pyrantel pamoate. ]

Q. This is a BIGGIE in the US, so you don't need a history of travel: HERE goes: [

A woman patient comes to you after sampling raw spiced pork sausage links (classic case). She has myalgias and PERIORBITAL EDEMA. What's the bug and drug and MOA of drug? [

A. This helminth is the ubiquitous Trichinella. Very common the US.

FOR ALL OF THE HELMINTHS QUESTIONS, THE NBME USUALLY GIVES A EM OR HISTO SLIDE BECAUSE MANY OF THEM PRESENT WITH SIMILAR VAGUE SYMPTOMS LIKE DIARRHEA, MYALGIA, ETC. SO WATCH CAREFULLY FOR THEIR CLUES WHICH THEY HAVE TO PROVIDE.

Treat Trichinella with Thiabendazole!

Again, Trichella is assoc. with pigs if all else fails. ]

Q. This helminth is rather distinct so you likely won't have trouble!

Hey, you get a patient who came from a trip photographing wild animals in AFRICA (let's say Ethiopia). He comes to your clinic and you see hypopigmented (leopard spot like) lesions on his legs. He photographed from a riverbank (HINT). Give me bug and drug and MOA of drug? [

A. HERE we are:

This is "river blindness" or *Onchocerca volvulus*. BUT THE MOST COMMON PRESENTATION IS NOT BLINDNESS WHICH IT MAY EVENTUALLY CAUSE, BUT SKIN LESIONS!

Transmission is by black flies, along riverbeds, mostly all in Africa. Treat with IVERMECTIN, which works and binds selectively with glutamate-gated chloride-ion channels in invertebrate nerve and muscle cells. ]

Q. Here is a MUST KNOW:

A post college grad comes to you who came back from the PEACE CORPS. She volunteered her time so well, but this is a crisis. She is thin and athletic, and pretty but sadly one of her legs looks swollen like an ELEPHANT'S. What's the bug and drug you give? [

A. This is too bad, she was trying to do good...This is a classic NBME example and very common case of Bancroftian Filariasis or *Wuchereria*

68

bancrofti where a person is bit by a mosquito and has lymph node swelling everywhere. Common is a foot and/or leg elephantiasis. Treat FAST with Ivermectin or Diethylcarbamazine or she will lose her precious leg ]

Q. While we are on the subject of these parasites, here Nematodes, we spoke of a drug often used called Mebendazole. What is the MOA? [

A. Mebendazole is often used for treatment of eosinophilic enteritis; inhibits microtubule polymerization by binding to cytoplasmic b-tubulin; by affecting parasite's intestinal cells, prevents use of nutrients and essentially starves parasite to death! Sorry parasites...esp. if you are Buddhist, I guess even a parasite would be sacred! ]

Q. We are slowly winding down the NBME's list of parasites...

**BUT HERE IS A BIGGIE THAN AFFECTS UP TO ONE BILLION PEOPLE!**

You see one of your dear patient who came back from Puerto Rico (could be other places too). Now, he complained that a month ago he started itching, THEN coughing, THEN having diarrhea! Terrible! He is begging you to diagnose him because he is starting to look anemic!!

What is the bug and drug????????? Oh, also what does his blood smear show? [

A. You are staring at Anclostoma or HOOKworm disease which is SOOOOO prevalence around the world. You should look for travel history. Another related hookworm is Necator Americans.

When the bug hatches in the intestine, you get IRON DEF. ANEMIA, so

69

blood smear will show microcytic RBCs.

You treat with Albendazole or Mebendazole. ]

Q. This is a medumeer, but you have to know this too:

In your peds clinic, a kid comes in with vision problems and his mom said he had gotten a couple of new puppies. He also has wheezing urticaria and he lives in Southeast US. What is the bug and drug?

This is kinda hard because the differential is HUGE, but the association of:

puppies=southeast US=eye stuff gives it away easy. OK, so go ahead!  
[

A This is classic for Toxocariasis. You treat with a drug called Diethylcarbamazine but Thiabendazole can be used too. Puppy poop has this. You cannot miss this and accidentally treat with antibiotics thinking you have Pasturella (bacteria).

So how will you KNOW? Well, the NBME will give you a picture and labs. Remember eosinophilia? It can be as high as 80% with high IgM!!!! Oh, I should make that my next CONCEPT!

We have been going over the parasite bugs the NBME WILL test you on. And they frequently have things that will distinguish them from bacteria.

- 1) You may see a clinical history with stages (first intestines, then lungs, etc. because these guys lay down larvae)
- 2) You MUST look for the clue for labs and sometimes my students say they completely skip the lab section because they are in a hurry. ONE TWO MILLIMETER SPACE has the info HIGH EOSINOPHILIA! If you miss this, you may treat your patient with antibiotics on your test and get the question wrong.
- 3) Also, a lot of these bugs are not endemic to the US. So look for a history of travel.
- 4) There are only a few drugs here, so please don't forget them ]

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Q. Here is one that has been reported POPing up, so you better know it because it was in a newspaper and...

A Japanese family just came to the US 3 months ago and then went straight to your clinic. One of the kids has serious pulmonary signs and was treated for Tuberculosis. HE IS NOT BETTER. Worried parents gave you a history that he was treated by his older grandma in Japan with raw crayfish for health. You are glad they came to you because you know you are not looking at TB but rather....? And you will treat with ???

A finally the drug was what MOA???? [

A. This is popular with NBME because doctors mistake this deadly PARASITE with other things like TB or coccidomycosis and then a BIG lawsuit occurs.

So here you have a big clue about the Japanese ethnicity and the ingestion of crayfish and the lung findings.

This is pathognomic for....Paragonimiasis.

Please treat with Praziquantel. You must know...

Praziquantel again that it inhibits microtubule polymerization by binding to cytoplasmic  $\beta$ -tubulin; by affecting parasite's intestinal cells, prevents use of nutrients and essentially starves parasite to death. I think I mentioned this before, but I am repeating it because it is very important.

thought as I finish up the parasites that you really try to LUMP them somehow. I think of these because they work for me, but you should use some mnemonic because they are kinda hard to distinguish.

Taenia> Sounds like Tan-in-sol (sun) while Praying (praziquantel)

[These are weird mnemonics but I think you need some and personalize them like since I like to pray a lot, I can think of Tanning and Praying so I associate Taenia with Praziquantel for the drug treatment]

Strongyloides> "strong thighs" (Thighs sounds like) Thiabendazole

Onchocerca > "On cocaine via IV" (IV for Ivermectin)

Corny, but the parasites need this because their names are weirder.

Again, try not to confuse the parasites and bacteria. Look for Travel, look at labs, and look for symptoms that wax and wane over a month as the parasite goes through larvae stages ]

Let's move on,

I'm quizzing you from before...

Remember my original case of the 2 year old with Chronic Granulomatous Disease which we discovered is REALLY BAD, what is the name of the enzyme that was lacking? Do you remember? Were you paying attention? If not, that is OK, I am not upset at all, but you should keep reviewing my HY posts! [

A. Answer is NADPH OXIDASE

Our phagocyte oxidase system is an NADPH oxidase enzyme complex consisting of 4 component proteins. Membrane-bound gp91 and p22 make up the b and a subunits of the heterodimer cytochrome b558 portion of phox gene. But for us, we need to only remember NADPH OXIDASE, not distractors like NADH OXIDASE or NAD<sup>+</sup>OXIDASE or NADPH REDUCTASE! ]

IMMUNO

Q. The NBME wants you to know a simple point about VDJ recombination. It is a very basic concept which explains antibody diversity. But if I say it exactly I would be repeating NBME material b/c it is so specific so instead I'll ask you to read on it for just two seconds. Of course, if I can think of a way to present it indirectly which I am always doing then I will. But HERE, let me ask you guys, at the very least, which chain, the H (Heavy) or L (Light) carries the 3 gene

73

segments? And in CLASS SWITCHING, which antibody, IgM, IgG, IgA, IgD, or IgE is most "primitive"?

BIG CONCEPT, and some tests had a disproportionate amount of IMMUNO.

[

A. While you read up on VDJ, know that the Heavy chain has the VDJ and there is DNA rearrangement. Know the L and H chains are made SEPARATELY in the CYTOPLASM by means of DISULFIDE BONDS!!! The LAST step is the addition of the CARBOHYDRATE moiety. (Look and remember my capital letters...).

Second, at first, all B lymphocytes carry IgM specific then after undergo class switching to the others (If you were lost here, YOU REALLY NEED TO KNOW IMMUNO AND REVIEW) ]

Q. OK, here we go, a patient presents with dyspnea, endless differential, but here are the secondaries for ARDS:

1) Pretend you already diagnosed ARDS, a deadly illness, what cell is responsible for the distress?

2) OK, they NBME wants you to understand they will ask you cases (so what are the main causes?)

3) We know there are a lot of causes of Pulmonary Edema, but how can you differentiate ARDS edema and Cardiogenic edema?

ARDS carries a 50% death rate. Know it or Die! [

A. 1) Neutrophils

2) Ischemic shock/Endotoxic shock/DIC; breathing really hot air; acute pancreatitis (weird, eh?), drug use

3) It is called Pulmonary Capillary Wedge Pressure test (LV) LOW in ARDS, HIGH in CARDIOGENIC! ]

Q. THIS IS A GREAT CONCEPT:

OK, let's dabble in immune just for a change of pace, for just a second, we will revisit later. We need to know the following..

Whew! I am getting tired again, I need a break so I will lump a couple of KEY factoids:

1) Could you pick out the right ratio of T to B cells?

2) YOU know the T cells pass through thymus for thymic education (review if what I just said is foreign), do the B cells pass thru thymus? If not, where (amongst a series of choices of course)?

3) Which IL type boosts up T helper cells? [

A 1) 3:1

2) B cells don't pass thru thymus but the precursors mature in GALT and Peyer's patches.

3) IL-2

ALL OF THOSE ARE MUST MUST KNOWS, THE CONCEPT ARE IN THE

BRAIN OF THE NBME, BUT I PICKED MY OWN WAYS TO MAKE SURE YOU UNDERSTAND!!! KNOW THAT NBME WILL ASK THE ABOVE CONCEPTS IN WEIRD WAYS, SO AFTER THE FIRST READING OF THE QUESTION, YOU WILL BE LIKE "HUH?" THEN FOR EXAMPLE THE ABOVE THREE CONCEPTS WILL COME TO YOU AND THEN YOU WILL SAY "OH, I KNOW THIS!" ]

Q. OK, after this I need a few minutes break....

OK, remember that to really learn you need to compare and contrast so that is why I think I will "LUMP" my HY by subjects if I can at times. To know what is BLACK, you need to see WHITE, etc.

SOO>>>...

We know IL-1 and TNF-alpha makes your temperature go up, so which IL revs up IgA? [

A. IL-5 ]

Q. All, the NBME likes to ask things in weird ways:

We just covered helminths. Which IL is most involved? [

A. BIGGIE POINT: SAME ANSWER AS BEFORE IL-5. That is how NBME tricks you. You may "memorize" what I just asked, IgA is stimulated by IL-5, but then when I bring up the concept that IL-5 revs up both IgA (intestinal mucosa) and Eosinophils, your brain may hiccup! See, are you starting to understand???? ]

Q. IMMUNO:

Which mediator is responsible for endotoxin septic shock and makes you have cachexia (like in cancer)? And then, what is the MECH? [

HARD HARD, BUT MAJOR POINTS.

A.

TNF alpha,

1) secreted by MACROPHAGES

2) It causes cachexia by inhibiting lipoprotein lipase in adipose tissue.

ALSO, FOR ICING ON THE CAKE, KNOW TNF-A also revs up IL-2 and B-cells. ]

Q. Here's one more at least:

A patient of yours is predisposed to TYPE I hypersensitivity. Which IL is mostly responsible. This is a great great question.... look below after guessing... [

A. Surprise, I bet you guessed IL-1 or TNF-a BUT NNOOOOO!

The answer is IL-4 IL-4 revs up IgE, WHICH THEN is responsible for anaphylactic shock.

THIS IS AN ULTIMATE CONCEPT. MANY STUDENTS JUST LINK IL-4 TO IgE, which is fine because some versions of the test will be that straightforward. BUT SOME OF THE TEST TAKERS WILL BE ASKED JUST LIKE I JUST DID, INDIRECTLY AND WITH A SECONDARY. It is not a HARD question, but you can GET EASILY DISTRACTED!

77

DO YOU GUYS AGREE????? YOU HAVE TO PONDER AND REALLY THINK!

]

Q. Some of you will be asked:

Which IL revs up stem cells? [

A. WOW, the answer is IL-3

But some of my students got it wrong because they read Error! Hyperlink reference not valid. and it said IL-3 = Bone marrow.

So they Blanked! They KNEW the answer, but they blanked because they did not stop to recall that stem cells are in the bone marrow.

See, see how easy it is to get tricked? Please let me know if you agree.

]

Q. We MUST BE LUMPERS, (lump info together), it is more efficient, believe me it is educational theory...

So, which IL is part of the acute phase other than IL-1?

Also, which IL does the same as GM-CSF? [

A. IL-6

then IL-3 is like GM-CSF!!!! ]

Q. You KNOW MHC I = T-cells  
AND MHC II = B-cells (these are loose associations), but tell me,

Mature MONOCYTES secrete which two cytokines?/b [

A. Mature monocytes are macrophages and they are the ones that secrete IL-1 and TNF-alpha ]

Q. Great question:

Give a place where macrophages are fixed in tissues and name a mediator that activates them to move!!!! [

SUPER DUPER POINT(s)!

A. Kupffer cells of the LIVER and C5a!!!!

YOU MAY THINK I AM BEING TRIVIAL, BUT HINT HINT, I AM NOT!!!  
KNOW THESE!!!!!!!!!!!! DON'T FORGET! ]

Q. Differentiate NK T-cells with cytotoxic T-cells!!! It is things like this which keep students from passing! [

A. NK, or natural killer cells specialize in killing virus infected cells and cancer cells but unlike cytotoxic T cells, THEY ARE ACTIVE WITHOUT PRIOR EXPOSURE TO THE VIRUS, ARE NOT REVVVEEED UP BY CONTACT, AND ARE NOT SPECIFIC!

AND, THEY DO NOT HAVE TO PASS THRU THE THYMUS TO MATURE.

79

(You DO know the cytotoxic T-cells have a receptor, NK's don't!)

(NK's don't need MHC to act)

Since NKs activated by IL-2 are being used in cancer research, is there any wonder that what I JUST WROTE WILL BE ON YOUR TEST?! ]

Q. IT is the WONDER WOMAN of concepts:

Which ILs rev up growth and maturity of B-lymphocytes? [

A. The answer is IL-2,4,5!!!

Say it again, 2,4,5

Again, 2,4,5

You "may" be shown a pic.

I KNOW THIS STUFF IS HARD AND BORING AND SO ROTE MEMORY, BUT IT IS HY, YOU MAY NOT THINK IT IS, BUT IF I CAN HELP EVEN ONE OF YOU GET THEM ALL STRAIGHT, WE WILL DANCE TOGETHER IN HEAVEN. ]

Q. This is the KENTUCKY FRIED CHICKEN 20 PACK concept:

You all know CD-8 binds to MHC-1, but if I give you 5 mult choices, which IL revs it UP!!!! [

A. IT is IL-2 !!!!!!!!!!!!!!!!!!!!!!!!!!!!!!! Which also stimulates itself (Kinky, eh)

NBME LOVES THESE, DRAW A PICTURE UNTIL YOU KNOW IT COLD, IT

IS VERY CONFUSING. ]

Q. LOOK, you all know all T cells have CD3 (That factoid alone can help eliminate wrong choices like the leukemia stuff), but, what does CD3 do?

Is it using the cAMP pathway? [

A. This,,,,,my friends, is the BATMAN of facts:

CD3 molecules transmit into that the antigen receptor is OCCUPIED!

This works NOT by cAMP but by the IP3 Ca pathway.

(Music PLaying..) Instead of hearing "This is CNN", you are hearing "This is the NBME"

WE ARE IN THE NBME MATRIX, where's Keanu?

RE: HY Concept 182,

KNOW COLD that B Cells do not have CD3!!!! AND

B-cells have IgM on the surface BUT T-cells DO NOT!!!

Repeat this over over over over over over over again! ]

Q. This is the Green Lantern of concepts:

Which 3 cytokines bring neutrophils to the scene (pretend I show you a histo slide pointing to a neutrophil and THEN ask the same question)

81

Secondaries, secondaries... [

A. They are

IL-1, IL-6 and TNF-alpha = acute phase response

Are We Getting Anywhere Yet? ]

Q. FRIENDS, I told you IL-3 revs up bone marrow, now tell me:

What is different about the T-cells that make IL-3 (vs. others)?

Now tell me which mediator is used in cancer chemotherapy to rev up some neutrophils to stave off infection? [

A. IL-3, unlike the others are ACTIVATED first

AND WHAT A CONNECTION:

IL-3 IS SIMILAR TO GM-CSF (colony stim. factor)

KEY: IT IS GM-CSF THAT IS USED IN CHEMOTHERAPY ]

Q. Oh boy, now we get to complement!!!

This can get really really confusing! If I merely post my HYers, you will be lost unless you quickly review an IMMUNO book and look at the COMPLEMENT CASCADE. You WILL be asked which complement factor does what, NBME is very specific! There are literally 100 questions possible and more just on the diagram of the complement cascade!

82

So, I will ask only one or two questions here:

HOPEfully you know for example that C3b opsonizes bacteria, but which factor (s) neutralize viruses? [

A. C1, 2, 3, and 4 neutralizes viruses in the CLASSIC pathway, and complement:

- 1) kills GRAM-NEGATIVE BACTERIA
- 2) IgM and IgG activate complement in the classic pathway,
- 3) But, Endotoxin and nonspecifics work in the alternative pathway!!!

(THE NBME can ask SO many questions on just the words above, that is why this test is concept based. They could give a list of bugs and ask which one does C3a work on and you are scratching your head, but then you notice that all the bugs are gram positive except ONE! And then you will pick the Gram NEGATIVE bug!) (You may be distracted for 10 minutes trying to recall what C3a does (anaphylaxis), but YOU WASTED YOUR TIME!

Also, of course, I could ask you what OTHER complement works like C3a? Then you have to know it is C5a....

And so on , and so on and so on. Do you see how EVERYTHING IS INTERCONNECTED AND WHY SIMPLE RECALLS DON'T WORK? If you take the time, you can see into the NBME's mind and KNOW it all. ]

Q. So after reviewing, which complements are part of the membrane attack complex (MAC)?

And, which complement do both pathways meet at?

(two questions of candy bars [

A. C5b thru C9 = MAC

And both classic and alternative pathways meet at C5.

Tattoo the above facts into your brain! ]

Q. CANNOT BELIEVE IT, I ALMOST FAINTED BECAUSE I ALMOST FORGOT TO TELL YOU THAT THE COMPLEMENT SYSTEM MUST BE REGULATED OR..

The system can overreact and destroy our good cells. So..I told you C1 is an esterase right (no, I didn't, and there is a another possible question!). OK, what factor blocks C1 and what happens if you lack C1?

Next, give me another case: Human cells have DAF or (decay accelerating factor) to protect themselves. What factor does DAF work on?

What diseases arise if the above controls are LOST? [

A. Your body has C1 inhibitor (rather unoriginal name) to block C1.

Your DAF blocks C3b thus protecting your cells.

If C1 inhibitor and/or DAF is gone, your capillaries will weak, you will get PNH (hemoglobin in your urine at night) OUCH! ]

Q. SPEAKING OF IMMUNO, YOU WILL SEE... ..

Interferons, because they are DRUG and part your body's defense..

They are GLYCOPROTEINS (Everything I sort of BOLD is an unforgettable word/point), and they protect healthy cells and virus replication. KNOW there are alpha, beta, and gamma interferons:

alpha (fr. WBCs) interferons and beta (fr. fibroblasts) are triggered by viruses and target viral mRNA.

1) NOW, GAMMA interferon are the third interferon, they are produced by?

2) They active what process?

3) Gammas rev up what cells? [

A. 1) Gammas are made by activated CD4 and CD8 T-cells.

2) THEY rev up PHAGOCYTOSIS.

3) This by those NK, macrophages, neutrophils and revs up MHC I and II antigen presentation, which is like a plate of food that attracts the phagocytes. Finally, Gammas revs up B-cell antibody production.

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Q. ok, BIG POINT:

You have to understand the basics of activation. IF say an antigen presenting cell (Dendrocyte, B-cell, Macrophage) [T-CELL ARE NOT ANTIGEN PRESENTING!!!!], binds an antigen (virus), then CROSSLINKING occurs and the cell gobbles up antigen and then PRESENTS IT ON THE SURFACE. A lot of you know that, but THEN you

must follow the storyline. And lovely young helper T cell comes along and attaches or holds hands with the antigen presenting cell. THEN, the T-helper cell "blushes red" and is so happy she throws out IL-2, IL-4, and IL-5 that stimulate both B-cells and T-cells (IL-2 here). Some of the activated B-cells from what kind of cells in the secondary response and what is the most common surface Ig? [

A. A few activated B-cells turn INTO MEMORY CELLS (BEFORE, THEY WERE PLASMA CELLS), and they usually have IgG on top of them for rapid response to reexposure.

IMMUNO IS REALLY TOUGH SO I HOPE YOU GUYS DON'T GET TOO MANY QUESTIONS, BUT THE GOOD THING IS THAT THE NBME IMMUNO QUESTIONS OFTEN RANGE FROM SUPER BASIC TO SUPER DUPER HARD.

GET THE EASY ONES RIGHT! ]

Q. cannot break the copyright rules, but there was a question where the concept I can describe so you won't miss it.

IT is very very basic. They, many of you will get variations of the same concept where you are given a pic. of that infamous Y shaped Antibody and there are like a thousand questions about same concept. Like, let me make up something original but applicable:

- 1) Is the Constant Light Chain region part of Fab fragment or Fc fragment?
- 2) Is the CARBOXY terminal part of the constant or variable region? (There are ways with arrows to address this, so know this)
- 3) What kind of bonds KEY PT, holds the chains together? [

A. 1) Fab fragment

2) Heavy chain

3) Disulfide bonds, know which drugs can cleave these]

GET the concepts

Q. 1) Give that famous Y antibody with arrows, where does complement bind (Fc or Fab portion?)?

2) POINT to where CMV virus attaches.

3) Where can I find sugar side chains? [

A. 1) Fc portion

2) Both L and H hypervariable regions

3) Fc fragment ]

Q. KNOW that LIGHT chains only lie in the AMINO TERMINAL and are part of only the Fab fragment!!!!

Q. SUPER DUPERS:

MOst know that babies have IgG from Mom until 6 months of age (a key pt like ..uh on a graph), can the baby defend itself against syphilis at one month? [

A.  
YES, the fetus can make IgM. ]

Q. 1) Whoa! you see an EM of an Ig that is a dimer. Where in the body is it found and MOA? Does this fix complement?

2) Whoa! you see an EM of an Ig pentamer! What's so special here?

3) The only Ig to cross the placenta, this dude is most dominant in 2nd response about is what percent of total Ig?

4) You see an Ig in a baby's cord blood that the IMMUNOLOGIST tells you is rather unknown what it does? what is it?

5) You see an EM of an Ig that binds a basophil on a smear! Does this one fix complement? What else is special here? [

A. 1) IgA (also can be monomer). See in saliva, tears, gut, vagina, etc.

2) IgM is the PRIMARY response, most efficient in agglutination

3) IgG of course - 75% of all

4) IgD

5) IgE, anaphylactic allergies DOES NOT FIX COMPLEMENT.

[for example you may be asked a patient has a hookworm infection, which Ig is reved up? = IgE, right, remember?]

Q. OHHHH! Superkey!

T/F, Delayed hypersensitivity is a function of antibodies, right, huh? [  
A. NOOOOO.

Type IV Delayed is CD4 T-cells! Common mistake! ]

Q. Great question:

A patient of yours tries a new cosmetic cream and then presents in a couple of days with eczema. Which HYPERSENSITIVITY (I, II, III, IV) is this?

KEY POINT, I won't bug you with all the possibilities, but you must MASTER ALL THE FOUR HYPERSENSITIVITIES AND THE POSSIBLE OFFENDING AGENTS, THEY WILL BE ASKED! [

A. This is not not not Type I (common mistake), but Type IV. Often is you see stuff like a case of neomycin or soaps, and then a reaction a day after after reapplication, you are looking at TYPE IV. Review all of them...

like Glomerulonephritis is TYPE III (NOT TYPE II)

like Goodpasture's is TYPE II (NOT TYPE III)

(see look above, some student just think kidney stuff-Type III, and they get stuff wrong!)

like the complement system is activated in TYPE III

like Coombs Test is associated with TYPE II ]

Q. I heard of 500 questions/ways to address the concept that:

You know T-cells have CD3,4,and 8 on their surface. Which CD is a suppressor function? [

A. YEAH, CD8 has both cytotoxic and suppressor functions they suppress B cells and cellular immunity. ]

Q. Quiz to know if you are reviewing wisely:

1) What cells are involved in AUTOIMMUNITY?

2) Graft rejection? [

A. 1) B cells

2) T cells

[I CONFESS I CANNOT COVER ALL OF IMMUNO, IT IS SO CONFUSING AND ENDLESS, BUT I JUST PRESENTED SOME OF THE HIGHEST YIELDING STUFF ]

Q. There exists out there a diagram of the difference between:

TH1 and TH2 cells. YOU HAVE TO KNOW THE DIFFERENCES!

1) Which ILs are made by what?

2) IL-12 induces TH1 or TH2

(you have to read these stuff also on your own) [

A. Th1 revs up CD8 (T-cells) and macrophages (APCs)

Th2 revs up B-cells via IL-4 and IL-5

Gosh these are ultra high yield but so much I think I need to SCREEEAAMMM!]

TOMMYK POSTS (201-231)

Q. SUPER HYers that have been rounded up in the Wild West of Usual Suspects in IMMUNO Questions in the MIND OF THE MATRIX NBME!

1) A pt comes in and you see anti-centromere antibodies? Dx?

A. CREST syndrome

Q. most know Anti ds DNA and anti ANA is Systemic LUPUS, but give me the one if Drugs induce lupus? [

A. anti-histone!!

Think HIS-STONE of COCAINE (drug), so assoc. histone with drugs ]

Q. Pt comes in and has skin lesions that are peeling off, ouch!...pathology lab reports which autoantibody [

A. likely pemphigus and anti-epithelial antibody ]

Q. As I am doing, there are a WHOLE SLEW of autoantibodies that you must know that I cannot cover all, know them cold because they are easy points if they are highly specific.

Quickie, can you reverse chronic kidney rejection with cyclosporin A? [

A. NOOOOOOOO,

but you can suppress ACUTE REJECTION! ]

Q. BIGGIE JUICE?

A transplant patient comes to you and cries because after a year her kidney transplant makes her eyes yellow and her tummy is FAT (hepatosplenomegaly). What is the MOA? Think. [

A. This one was a little tricky,

NOT so much chronic rejection symptoms, but think

Graft vs. host disease!! All the organs are systemically knocked out! ]

Q. You see a slide with large cells and hyaline bodies in the last female kidney transplant patient. What is the virus (HINT) and the Dx? [

A. This is good HYer. She is immunocompromised from cyclosporine, so she is at risk for CMV, which you see. Give ganciclovir (Not acyclovir), if she is resistant still, give foscarnet.

]

Q. This connects with my previous concept:

KEY!

Why did you give her Ganciclovir and not Acyclovir? And if she was resistant, why did Foscarnet work???? [

A. ganciclovir IS phosphorylated like acyclovir, but it LOVES CMV DNA polymerase (MOA). Foscarnet worked because it did not need viral kinase activation!!!! (resistance issue)

WOW! ]

Q. WE JUST TALKED ABOUT acyclovir, gancyclovi, foscarnet.

Which body organ is at risk of toxicity? [

A. all are nephrotoxic and ganciclovir can cause pancytopenia! ]

Q

We just mentioned CMV right?

Your door opens. The patient reports decreased visual acuity, floaters, and loss of visual fields on one side. Ophthalmologic examination shows yellow-white areas with perivascular exudates. Hemorrhage is present and is often referred to as having a "cottage cheese and ketchup" appearance. Lesions may appear at the periphery of the fundus, but they progress centrally.

OKOKOK, this is CMV, I need you to know CMV retinitis is common in HIV, but tell me:

The VIRAL FAMILY, and DNA Structure/Envelope [

A. CMV is very tested. (As an aside, it is horribly affecting to unborn babies), IT along with VAV and EBV and HHV are all HERPES viruses with DS (Double strand), linear envelope

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Q. But wait there is MORE,

our poor CMV patient has HIV, right? Concept is what is the structure of HIV????????? be specific. [

A. This RNA virus has an envelope, SS+, square, and is one of the only two RNA viruses to replicate in the NUCLEUS! ]

Q. A child comes in with pink eye and half his kindergarten has symptoms of this common virus? Give me structure? [

A . Adenovirus is DS linear without an envelope! You have to know the details because one of the answer choices will have DS linear with envelope. Everyone limits to two choices. Don't be trapped!

]

Q. NOW, you see a mom with a child coming in with a rash on his cheeks and is tired a lot. What virus is this for his classic combo? And give structure!!!!!!!!!! [

A. Parvovirus B19, 5th disease, shown a picture, no envelope, SS linear

(This is the only DNA virus that is SS, YOU HAVE TO START LUMPING IN EVERY WAY YOU CAN UNLESS YOU ARE GOD, AND ONLY GOD DOES NOT HAVE TO LUMP)

94

Another LUMP,

Hi (Hepatitis/Herpes) Pox (Poxvirus) Lady, holding an ENVELOPE with a Valentine's Day card!

MNEMONIC for the 3 DNA viruses with an envelope, the others DON'T have an envelope.

OH! INCIDENTALLY, TODAY IS VALENTINE'S DAY. SO HAPPY VALENTINE'S DAY! ]

Q. Whew, I am getting tired, but>>>

A child comes in with his face looking like chickenpox but serology tells you it is Measles. Also his physical reports a grayish spot on the inside of his mouth before the measles started (Koplick spots). Give me the structure? [

A. This is a NEGATIVE sense, SS, linear, NONSEGMENTED.

UGLY, UGLY. This structure stuff IS ALL OVER THE NBME's MIND, but it is so hard to master. Click on my posts over and over while covering the bottom part with the answer until you make NO mistakes...

I am devoting a lot of effort, so DON'T LET ME DOWN, LET'S WIN! ]

Q. THEY may give an EM with the previously mentioned MEASLES VIRUS, what does the capsid look like and what are the 3 other viruses in this family? [

A. The capsule is a HELIX, and RSV, Croup virus, and Mumps are all part of this Paramyxovirus family. ]

MAN, this is a PAIN! Right?

Q. HERE IS A CLASSIC, LIKE HAPPY DAYS AND THE FONZ! OR Laverne and Shirley...

You see a female young sexually active patient with genital warts you biopsy to be HPV. (SO MANY SECONDARIES, like cervical cancer/cone biopsy needed/CIN grading/colposcopy) EVERYTHING IS CONCEPTS!

Sorry but to the case...the HPV is what structure and family? [ A.

This is a Papovavirus, with NO envelope, DS and circle shaped! Another secondary is back to your HIV patient, he can get another virus from his HIV that slams his brain: JC virus...just mentioning...

Are you guys getting these? These are so boring and rote memory.... ]

Q.  
Oh dear, you will see a million of these:

A kid comes in with the common cold and serology says it is not adenovirus. What is the structure?

Q. OH BOY,

This ain't OLD YELLER, but a raccoon that bit one of your peds kids and his serology is Rabies!! Quickly, structure! But first you gotta be quick and give the kid Imogam/Human Rabies Immunoglobulin. [ A. this neg stranded virus is SS and HELICAL ]

Q. Emergency!  
Another peds patients came from Africa with serology of EBOLA!  
Structure! Please [

A. Ebola is neg. sense, enveloped, linear, helical (JUST LIKE RABIES)! Your poor patient must be isolated because this virus cause vascular hemorrhages!!!! And NO DRUG (proven) at least is available! AHHH! ]

Q.  
BIG ONE!

A case of a peds kid comes with a 4 day rash over his trunk (you need serology so I tell you it is Rubella). What is this Togavirus's structure?

[

A. Here is a positive sense, SS, linear, square virus, with an envelope. You will get a couple of exactly these concept questions which will make you have diarrhea because it is so hard. Think of mnemonics.... ]

Q. YOU HAVE TO KNOW THIS ONE, because it is EVERYWHERE!

Case: You have a peds kid with serious diarrhea from a virus. What is this everywhere virus and the structure???????

A. THIS IS ROTAVIRUS, a mainstay in peds offices.

IT IS THE ONLY RNA VIRUS THAT IS DOUBLE STRANDED!

And it is linear, square, with an envelope.

THIS STUFF IS REALLY HARD, I THINK I AM GETTING DIARRHEA MYSELF!!!!!! KEEP CLICKING MY POSTS TO QUIZ YOURSELF, this part I think is the hardest because the answer choices will be so hairline similar. ]

Q. CLASSIC GRANDDADDY QUESTION !

Case: A couple of your newlywed patients go on a cruise ship. Instead of a good time, they come back to your clinic with the worst honeymoon ever!! All everyone on the ship did was diarrhea!!!!!!!!!! (You are walking up a ladder and you are hearing something spatter..diarrhea..uh..uh...you are walking down the hall and you are hearing something fall diarrhea...)

What is this classic bug and the structure? [

A. This is the FAMOUS NORWALK Virus. IT is SS positive sense linear and square with NO envelope.

I am hoping that for these last series of posts about viral structure I see that there are thousands of views because that is what it WILL take to master them and pass them.

IT IS SO BORING RIGHT? AND SO HARD. I mean, gosh!

Oh, a quick personal mnemonic... PCR we know stands for that DNA amp test. And so remember PCR stands for PICORNA, Calci, Reo viruses. If you recall the families, then PCR RNA viruses are the only ones without an envelope.

YOU HAVE TO REPEAT THESE A THOUSAND TIMES, there is no other way. All the people I spoke to said this was the hardest thing on their USMLE because they could get the bug right, but they were like pos or neg sense, whatever? ]

Q. HERE IS A MEGA HY and a lumper:

Two patients of yours walk in with antibody specific for Hepatitis A and E

Another comes in with a tattoo and she has Hep C

A third jumps in your office crying bc she has Hep B

Give me the structures (NBME WILL ASK YOU THIS) [

A. Good, we can LUMP Hepatitis A and E with SS pos. sense, linear, square and no envelope. RNA

However, Hep C is also SS pos. sense, linear, square, but ENVELOPED!  
RNA

NOW Hep B is a DNA virus curved on EM WITH an ENVELOPE!

You feel you want to avoid this, but the secondaries will address these.. ]

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Q.

Case: A rocker teen comes in with serology positive for COXSACKIE B,  
AN NBME FAVORITE.

Two questions:

What disease and sorry sorry to ask, but give me structure!!!

A. This bug is part of Picornaviruses and is like Hep A and E in that it is

!) positive sense, RNA, SS, and square.

2) The disease is MYOCARDITIS [

I think this is all so hard you need a mnemonic so let me give you all one and you make one up yourself or you are dead b/c it is so much mumbo jumbo: RNA viruses first:

For the POS. SENSE, I think of the viruses that are not SO BAD because they are:

Rhinovirus, Coxsackie, Hepatitis A, E, C, Rubella (non-congenital one), Coronavirus (common cold), and HIV....(HIV I think is now not SO BAD because of the new drugs)

(The NEG SENSE are all the other RNA viruses)...(for example Rabies and Ebola are neg. sense because it is so negative/bad to get them)

For getting straight the strands, know ALL OF THE RNA viruses are SS except for Reo/Rotavirus which are DS.

For the Capsule, aside from Corona (common cold) which is not THAT DEADLY, THE DEADLY VIRUSES ARE HELIX shaped (e.g. INfluenza on an older man, untreated mumps, rabies, ebola, LCV, Hantavirus (hemorrhagic fever) The others are all square...

NOW, the DNA viruses you identify because they are HAPPY!

(H) Hepatitis B

(A) Adenovirus  
(P) Poxvirus  
(P) Papovavirus  
(P) Parvovirus B19  
Y

All the DNA viruses are DS except Parvo  
You send an ENVELOPE with an p OX to HP (Hewlet Packard Co) [The p  
OX stands for poxvirus and the HP stands for Hepatitis B and Herpes]

These mnemonics work for me, but you NEED some otherwise it is  
hopeless.... Try to be creative!

I think this is all so hard you need a mnemonic so let me give you all  
one and you make one up yourself or you are dead b/c it is so much  
mumbo jumbo: RNA viruses first:

For the POS. SENSE, I think of the viruses that are not SO BAD  
because they are:  
Rhinovirus, Coxackie, Hepatitis A, E, C, Rubella (non-congenital one),  
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- (H) Hepatitis B
- (A) Adenovirus
- (P) Poxvirus
- (P) Papovavirus
- (P) Parvovirus B19
- Y

All the DNA viruses are DS except Parvo  
You send an ENVELOPE with an p OX to HP (Hewlet Packard Co) [The p OX stands for poxvirus and the HP stands for Hepatitis B and Herpes]

These mnemonics work for me, but you NEED some otherwise it is hopeless.... Try to be creative! ]

Q. THE WAY NBME'S VERSIONS OF THE TEST ARE THAT SOMEONE IN CHINA WILL BE ASK TOXOPLASMA, SOMEONE IN USA WILL BE ASKED TRICHOMONAS, SOMEONE IN INDIA WILL BE ASKED PLASMODIUM, ETC. SO THE CONCEPT IS THAT TO ANSWER CONCEPT #47 (E.G.), YOU HAVE TO MASTER SAY 50 FACTS TO GET THAT ONE QUESTION RIGHT BECAUSE YOU DON'T KNOW WHICH VERSION YOU WILL GET... SO BACK TO THE CONCEPTS:

You KNOW Trichomonas is SO COMMON in clinics and you know the

drug? [

A. metronidazole ]

Q. You have a patient with a history of travel to Mexico. Did I ask this?  
I dunno..

He has bloody diarrhea, that should be enough you know the bug to treat him with what drug? AND, the next day he comes back after a cocktail party and said he threw up and had a red rash on his face? What happened? VERY VERY TESTED AND KEY POINT THAT I THINK IT IS GOOD THAT THE NBME TESTS THIS. [

A. This is classic dysentery from *Entamoeba histolytica*/amebiasis. He may present with abd pain like pancreatitis.

The IMPORTANT drug reaction with METRONIDAZOLE (which is trade name Flagyl) is a Disulfiram like reaction with alcohol!!!!!!! ]

Q. This WILL BE ON YOUR TEST (well if not YOU then the GUY NEXT TO YOU):

A patient with travel to India (a great country with an exploding GDP), comes back with anemia and he tells you he was bitten by a mosquito. What bug and drug? Be specific!!!! [

A.

*Plasmodium malariae*, give him Chloroquine and or Quinine.

KNOW a side effect of Chloroquine is visual changes and tell him not to take with Mg antacids because it delays absorption and people with *Plasmodium* may take an antacid due to tummy ache ]

Q. NOW, another traveler, this time from Africa, comes with bitten by

103

a mosquito and you see the typical malarial signs:

FEVER, VOMITING, MILD SEIZURES, ANEMIA, ENLARGED  
SPLEEN/LIVER

Which Plasmodium does he have most likely? [

A. NBME wants you to know that Plasmodium falciparum is more common in Africa while in the previous case Plasmodium Malariae is more common in India. There is a prodrome and time lag since:

The bite of an infected mosquito introduces asexual forms of the parasite, called sporozoites, into the bloodstream. Sporozoites enter the hepatocytes and form schizonts, which are also asexual forms. Schizonts undergo a process of maturation and multiplication known as preerythrocytic or hepatic schizogony. In Plasmodium vivax and Plasmodium ovale infection, some sporozoites convert to dormant forms called hypnozoites, which can cause disease after months or years. Very important to know the above mouthful of words I wrote!

OH, you treat Falciparum and Malariae the same way, Chloroquine ]

Q. We MUST COMPARE AND CONTRAST FOR THE INFO TO STICK...so

You get a traveler from India with that mosquito bite and the aforementioned MALARIAL symptoms, the travel and symptoms will NAIL the diagnosis for you. But this time she complains the malarial symptoms recur and relapse over the past two years. What two bugs do this and what drug must you add to the regimen and WHY? [

A. The forms Plasmodium Vivax and Ovale are cyclical and have dormant stages called hypnozoites in the liver. So, you must ADD

PRIMAQUINE to the regimen.

KNOW THE MOSQUITO'S NAME is Anopheles. Even mosquitos like the sound of their own NAME! ]

Q. You are shown a slide of the horrible Pneumocystis carinii in an HIV patient.

- 1) What is the lung X-ray classic finding?
- 2) Method of infection
- 3) Drug of choice?
- 4) When should prophylaxis have BEEN STARTED? KEY POINT, give T-cell count (hint ) [

A. 1) Perihilar interstitial infiltrates

2) Inhalation of cysts

3) Trimethoprim-sulfamethoxazole (Bactrim, Septra, Co-trimoxazole)

4) Probably CD4 count less than 200 and not on PCP prophylaxis.]

Tommyk posts 232-300

Q. We were on the topic of ... hmm... let me first tell us that the NBME needs you to understand the RECEPTOR AND 2nd MESSENGERS.

HERE is ONE that NBME loves:

The Ryanodine receptor

What are they and what ion triggers them

A. The Ryanodine receptors acts as sentinels for Ca in the sarcoplasmic reticulum, so remember the receptor type is an Ca channel.

105

Q. NOW, you KNOW the NBME begs you to study LUNG TISSUE.

So, if I present a clinical case and a histo slide of the LUNG with arrows of course,

can you point exactly to a

- 1) Endothelial cell
- 2) Type I pneumocytes
- 3) Type II pneumocytes
- 4) Clara cells?
- 5) Dust Cells? (What are Dust Cells by the way?)

A. Sorry, but you have to grab your histo atlas, but do so NOW!

KNOW Dust Cells DC are macrophages

Q. Quick review:

HERE IS AN EMPEROR OF NBME CONCEPTS; YOU GOTTA LOVE IT!

I present a case of a patient named Mr. Wiggles who comes to you after received Isoniazid tx for TB. He is acting goofy, has diarrhea, and his skin is inflamed.

- 1) The secondary/tertiary is What is the function of compound missing?
- 2) What is the compound missing?
- 3) What AA does this come from?
- 4) What dx does he have?

THIS IS 100% NBME'S THOUGHT PROCESS...

A. 1) Redox Rxs (recall NAD, NADH)

- 2) Niacin (vit B3)
- 3) Tryptophan
- 4) Pellagra

There are like 20 questions from the above concept. Think hard, and USE THE FORCE, LUKE..or LEA if you female.

Q. While on vitamins,

LOOK, it is common knowledge that Vit A def causes eye problems, and excess causes hair loss, and muscle pain, AND you have to be careful to give RETIN-A to your pregnant patients (This will be in NBME's mind), but

LOOK NOW AT Vit B1 (thiamine). You will face this from alcoholics:

- 1) What heart disease is he going to get?
- 2) What rxn is this a cofactor for (give 2)?
- 3) 2 main def. diseases please?

A. Again, at least 20 questions from this ONE concept:

- 1) dILATED Cardiomyopathy
- 2) This is a cofactor of OXIDATIVE DECARBOXYLATION of pyruvate and it is a cofactor for TRANSKETOLASE in the HMP SHUNT.
- 3) of course.... Wernick-Korsakoff syndrome and Beriberi

(I ENCOUNTERED THIS A HUNDRED TIMES IN MY MEDICINE ROTATION, OBVIOUS THE NBME WANTS ME TO MAKE SURE YOU KNOW IT).

of course, give the alcoholic thiamine before sending him to AA.

107

## Therapy

Q. OHMIGOSH!

A patient of yours named WilliWonka comes in with cracked lips and difficulty seeing, itchy rash, and the corners of his mouth are dry. What vitamin? What Reaction? What product?

A.

This is tricky because it looks like Vit A def.

But Vit A, for for muscle and hair stuff...

NOW, we are talking Riboflavin (B2)

2) Oxidation and Reduction

3) FAD

Q. In case they ask, which they WILL, they will ask in a way like which vit is toxic if overdosed and you will see a bunch of vitamins and you have to pick the FAT SOLUBLE one. Soo.

On your college campus, unfortunately, the girls from sorority DEKA are FAT. (Vit D, E, K, A)

ALL THE REST ARE WATER SOLUBLE.

BUT THE NBME TOLD ME YOU SHOULD KNOW THAT:

1) What disease can result in DEKA deficiency?

A. Cystic Fibrosis, and Celiac Sprue are two examples.

KNOW ONE THING I FORGOT ABOUT VIT B12 which is NOT a member



A.. 1) ALL reactions involving CARBOXYLATIONS (CAREFUL HINT HINT, I SAID CARBOXYLATIONS NOT DE-CARBOXYLATIONS, A COMMON MISTAKE) are wiped out. Look them up.... (e.g. methylmalonyl CoA, Oxaloacetate)

2) Lovely Biotin is missing.

ESSENTIAL POINT:

ON PREVIOUS CONCEPT, KNOW BIOTIN DEF. IS OFTEN ASKED BUT NOT THAT COMMON. IT IS ALSO SEEN IN PATIENTS WITH

TPN (TOTAL PARENTAL NUTRITION)  
AND LONG TERM ADMINISTRATION OF ANTIBIOTICS SINCE  
INTESTINAL FLORA MAKE BIOTIN AS WELL AS YOUR EATING IT IN  
FOOD.

REMEMBER, CONCEPTS CONCEPTS, THIS QUESTION ARE NOT  
SHORTCUTS. I HEARD A RUMOR THAT NBME HAS MILLIONS OF  
QUESTIONS!!!!!! COULD THAT BE TRUE

Q. dunno why, but ALL MY STUDENTS SAY THAT THEY MIX UP FOLIC  
ACID AND VIT B12, THINKING THEY ARE THE SAME!

For folic acid (1000 questions here like you have to give to pregnant  
females, def. causes neural tube defects, most common vit def in US,  
etc. etc. etc. smear is macrocytic anemia, etc etc.)

Here, what two MOA is FOLIC aCid crucial in?

A..

Folic acid is crucial IN:

ONE-CARBON TRANSFER REACTIONS!!!!  
THEY are needed for METHYLATION REACTIONS!!!!

KNOW THAT FOLIC ACID IS NOT STORED IN THE BODY LONG SO EAT  
YOUR SPINACH LIKE POPEYE! While Bit 12 is stored for YEARS!

KNOW THAT PABA = FOLIC ACID PRE IN BACTERIA

KNOW DAPSONE AND SULFAMIDES RELATED TO FOLIC ACID

KNOW FOLIC ACID IS CRUCIAL FOR DNA AND RNA SYN VIA THF.

THIS CONCEPT HAS 122 POSSIBLE QUESTIONS (i MADE UP THE  
NUMBER 122, BUT IT IS PROBABLY ACCURATE)

Q.. QUICK OFF THE SUBJECT:

valuemd.com

What is the resistance mech of ACYCLOVIR?

A.. Resistant is the mutation of viral thymidine kinase. Think of the  
mechanism. LIKE tell me what is the resistance of a quinolone. YOU  
CAN FIGURE THE RESISTENCE questions IF you know the  
mechanism !!!!!

YOU CAN DO IT!

Q. Let's finish NBME's wanting you to understand treatment of  
protozoa.

Again I feel stupid now, but the NBME wants you to know Chagas

111

disease. It is so important because the infection rate in South America is SO prevalent. I feel stupid saying this but YEAH it IS NBME "content" but so is what I talked about before like rhinovirus and myocardial infarction. I again just heard someone tell me, "Why are you helping them, YOU did not get the same help..." I am almost crying because this is against what I believe is the spirit of humanity and of love and education. Whew...you WILL be given a case of a man who traveled to someplace like Brazil and he has malaise, arrhythmias, and mentions he was bitten by a fly. You see these spotty flagellates under a peripheral smear and suspect Trypanosoma cruzi. What drug will you pick up?

A. The two drugs of choices are:

Benznidazole and Nifurtimox

Q. OK in your peds clinic a patient 17, named MickyMouse walks in with his mom. She says they came back from abroad Soviet Union and the kid has very smelly diarrhea that won't stop. His stomach is distended and you take a stool sample. YOU SEE UNDER THE MICROSCOPE, cysts. Your attending comes in and hints that this is the MOST common pathogen/parasite to hit children. You give him the right medicine and know he is going to a wedding where beer is the drink of choice. What is the drug and the side effect with beer?

A. YOU given him Flagyl (Metronidazole) and you warn him about a disulfiram like reaction.

OK OK another parasite:

A friend of yours named Willy Wonka just arrived from a meeting in West Africa where he was bitten by a fly. He has a mild fever and

112

lymphadenopathy and a chancre on the bite spot.

Need a hint:

The fly is a Tsetse....

What is the disease and the med?

Q.. Ah, another of your patients is only 2 yrs old with HIV positive. He lives in San Diego in a place where his mom brings him to a day care. After removal from the day care, the child has voluminous diarrhea, up to 15 liters a day, and you see cyst in the water sample. What is the bug and drug?

A. This one is key because it is so common in the US.

IT is Giardiasis. The ONLY treatment here is Bismuth and "Kaopectate".

Don't pick Metro as the drug. You will be wrong!

tommyc

all my books say metro is drug of choice against giardia

i dont know the drug you mentioned

can you describe its mao,please?

Sorry, but I made my first REAL BOO BOO error. Yes give Metro for Giardia.....I was thinking of Cryptosporidium.

Giardia and Cryptosporidium can present so similar on your test so the USMLE has to provide a PICTURE of Giardia Trophozoites OR

For Cryptosporidiosis they have to give an ACID fast slide with cysts.

113

IF you quickly look at BOTH ON a Google search with a visual, you will never mistake them. Thanks..

One of the keys for my passing the test that helped was that I RIPPED through the tutorial and saved 15 minutes.

NOW, you cannot USE MORE THAN an hour for each block, BUT, you can ACCUMULATE break and lunch time which is 45 minutes. THUS, if you can pace yourself fast, you can earn more points I think.

Because a lot of the info is FAST recall, I did NOT take lunch and RIPPED PAST THE TUTORIAL. So I took these longer breaks where I SUPER CRAMMED THE HY STUFF, like pharmacology side effects.

That helped a lot because some of my friends did not do this and they only had enough time for quickie bathroom break and spent lunch talking it with friends.

But since I used it for CRAMMING, I COULD PROMISE YOU that it worked because some of the facts WERE IN MY short term 5 minute memory. Then I would run back into the testing room and go through the 50 questions really fast for what I could remember quickly off the top of my head so to speak, then I went back to the "THINKING" problems.

It really worked for me. As I said before, think of what works for YOU. The day before the test, I SLEPT AT 8:00 pm (I ADMIT I TOOK SOME OVER THE COUNTER SLEEPING PILLS AND QUIET MUSIC), disconnected the phone and awoke at 2:00 am. Then I studied like mad because that worked for me since so much of the test is quick recall and your memory fades fast.

BUT THIS IS WHAT WORKED FOR ME. it may be different for you. Again, you have to eat a BIG breakfast because my strategy is to skip lunch. Also, I had a friend drive me to the testing center so I was studying like mad all the way til the second my computer turned on. I promise that it helped me in my case.

Plus, when I signed out and in, I signed out my signature REALLY MESSY AND FAST to save seconds...some of my friends took like a MINUTE to sign out.

I maximized everything.....you should too.

And as I mentioned bring TYLENOL or ASPIRIN because it saved me after the 4th block!!!!!!!

And bring a sweater just in case!!! And hard Candy in your pocket.

There is a study that says that caffeine helps your brain...but if you take cafeeine pills you may have to urinate and you can't leave within a block.

ONE OF MY STUDENTS KNEW THIS AND CONFESSED TO ME THAT HE ACTUALLY WORE A DIAPER, A DIAPER!!! And he urinated in it so he could save breaktime for max. cramming.. I am not sure if you want to go that far, but this test is a LIFE event, so think of everything to gain advantage!

A few of my students, actually just a couple, got in trouble...

here is why.

Some centers are run like a military zone thru company Prometric. ONE guy put his hands in his pants. That is all, and his test was "FLAGGED" and his score delayed. Another took off her SWEATER during a block and HER TEST WAS FLAGGED! Both cases were dropped, but it delayed your score. SO, just be careful my brothers and sisters. LOVE, tommyk

ONCE AGAIN, on the PARASITES, the NBME WANTS ME TO TELL YOU THAT MOST OF THE QUESTION HERE ARE TREATMENT, the BACTERIAL QUESTIONS ARE A LITTLE HARDER BECAUSE THEY ADDRESS RESISTANCE ETC. BUT YOU NEED MNEMONICS: SO LET ME OFFER A COUPLE AS AN EXAMPLE AND THEN YOU CAN MAKE YOUR OWN, BUT YOU HAVE TO MAKE YOUR OWN!

When we think of the PROTOZOANS,

For Trypanosomiasis (African Sleeping) I think of the evil Apartheid of Africa (Another great EXPLODING GDP nation) and a boy who "TRIPPED on a White Soldier's PAN, and said "Sorry Sir") [Thus you equate Trip-PAN-osma with "Sir"amin]

For Pneumocystis carinii, I know the abbreviation is PCP, and the tx. is TMP so I think of the idea of acronyms and say PCP-TMP, PCP-TMP PCP-TMP and then it sticks.

For Plasmodiums (Malaria which means "bad" "air") I think that Ovale and Vivax are the 2nd half of the English alphabet and so is the tx PRIMAQUINE b/c it starts with a P. Malariae and Falciparum are the first half of the alphabet (M and F) and the tx is Chloroquine, which has a C which is also the first half of the alphabet.

For Trichomonas, I had my OB/GYN rotation already and we used it and the so famous acronym is GET on the metrobus, so G-Giardia, E-Entamoeba, T-Trichomonas.

For Toxoplasma, I think of TOXic SULFUR gas, so I equate Toxoplasma with Sulfa drugs.

Finally, with Chagas Disease (T. Cruzi), I recall reading about the genius of Dr. Chagas and how he isolated the protozoa, it is a great story and I think he must have been so NICE and smart, so I equate Chagas with Nifurtimox. (The NI is NICE and the NI is Nifurtimox)

This is cheezy, and I think it is a good example because you need to make some up or you will forget!!!

MY PERSONAL USMLE SCORE would have been so much higher if I was better prepared. I did not know what to expect so I sort of lost track of time and had to bubble in answers at random because:

YOU HAVE TO PERSONALLY KEEP TRACK OF YOUR TIME AND BREAKS

THEY GIVE YOU A PAD TO WRITE ON, USE IT! AND WEAR THE EAR PLUGS THEY GIVE YOU.

THE ADVICE I GAVE BEFORE ABOUT THE CRAMMING IN BETWEEN SAVED ME AND GAVE ME A DECENT SCORE BUT NOT THE SCORE I COULD HAVE EARNED.

THAT IS WHY I AM DOING WHAT I AM DOING NOW.

Before, as I said, when the NBME asks about the parasite HELMINTHS, you will mostly be asked the tx. Also, you usually will be given TWO BIG HINTS like the EOSINOPHILIA and a slide of the bug. The HELMINTHS are notably distinctive b/c like the hookworm looks like it IS HOOKING its fangs of teeth into Small intestine tissue.

So here we go.

For Onchocerca (river blindness) I think of a person ON an IV in the hospital. So the IV is IVermectin! ON...IVermectin!

For Filariasis and Toxocariasis, I see the FIL and the TOXO, and CAR and group them into automobile themes (FIL is fil er up in a gas station). Then I think that cars are a leading source of death in accidents, and DIE-thylcarbamazine is the tx.

For Trichinella and Strongyloides, I think of "Tri-ing to get Strong Thighs" when you exercise. And both need Thi-abendazole (Thiabendazole).

For the roundworms, HOOKworm, Enterobius and Ascarius...well I think of a homosexual theme.... listen... Ascaris sounds like scary and Enter-obius sounds like enter. So I think it is A-SCARY that a guy would ENTER someone meBEND (ing) azole [Mebendazole]. So then I never forget the image of Ascariasis and Enterobius treated with MEbendazole. And the hookworm is easy. A HOOK is BENT, so you treat Hookworm with meBENDazole.

For the tapeworms, I have the PRAYER THEME:

I knew a guy name TAE who PRAYED to the SUN (All the tapeworms

118

need Praziquantel)

Paragonimus= Paragon means "model" I think of a model citizen

PRAYing

Echinococcus= has words Eck!!! it is AL! (Albendazole)

Schistosoma= sounds like "S-H-I-and another letter that completes a bad word", then I then I have to PRAY to get forgiveness. Thus

Praziquantel again (Praziquantel sounds like prayer)

The above is very weird but YOU HAVE TO HAVE SOMETHING or you will forget. I am just sharing my weird stuff to give an example. YOU must make some yourself

Q.. OK, still LUMPING ALONG..

You see three patients:

Patient A has cystic fibrosis and another Patient B after bone marrow transplant. Patient C has HIV. The bug I am referring to has very NONSPECIFIC findings so the question on your test has to give a slide of the organism. The keys are:

Here, you see 45 degree branching hyphae.

The three above cases are classic cases that are so common the NBME cannot give an atypical example.

Give me the bug and drug!

A. A three cases are Aspergillosis. Classic cases....

The facts of HIV, cystic fibrosis, marrow transplant are good, but the ultimate key is 45 degree branching hyphae!

Q. OK, for these groups of FUNGUS, you know most of us with good immune systems will not see this but...

Case. You have a patient with HIV and presents with nonspecific findings like fever, etc. but he complains of some mild chest spasms and a stiff neck. Under the scope, you see little bugs that have a capsule around them swimming with a stain of India ink.... this is KEY for what yeast infection?

YES, you know it to be Cryptococcus and he has pulmonary and meningitis. You equate this with pigeon poop, but almost 80% of HIV cases are correlated with Cryptococcus. The KEY is the slide with the bugs swimming in halos, and the India INK stain, which is mostly used.

MY goodness, I must be getting tired:

For this bug too you treat with Amphotericin B!

Q. OK, let's keep going with the immune system dysfunctional diseases:

AGAIN, often the NBME will LIKE HIV and Diabetes cases with the immunodepressed. During my medicine rounds, these were everywhere, and it is logical that NBME wants you to know them.

Case: HIV male, 27, presents oddly like Guillain Barre. He says he has lower back pain and urinary incontinence and lower limb weakness that is progressing. Hmmm.. you are thinking a huge list including prostate issues, BUT the NBME must give you some more: So, his CBC comes back with a bunch of clover leaf shaped lymphocytes. (A lot of this is NOT in Error! [Hyperlink reference not valid](#). but I know the

120

NBME wants you to know them). OK, what is the bug and drug? (HARD ONE)

A. This is another common opportunistic infection of

HTLV-1 !!!! or Human T-Cell Lymphotropic Virus

This is seen with HIV positive patients! There is no drug for this!!!  
Maybe some steroids...

This disease was already covered so I won't repeat it, BUT,

WHILE we are on the subject of immunocompromised people,

KNOW that they are susceptible to all kinds of LYMPHOMAS, e.g. if they have non Hodgkins, you will get a peripheral smear instead of like a slide with a yeast or fungus.

Q. NEXT:

You see into your clinic two people:

Bob has had a bone marrow transplant  
Bill has HIV.

Both have very distinguishable purpuric skin lesions all over his trunk and a raised lesion on the inside of their mouths. They have the constitutional symptoms of fever, weight loss, weakness, diarrhea, flaky skin. Bill, but not Bob is homosexual fr. history. What is this defining lesion and treatment?

A. this is pathonmonic for Kaposi's Sarcoma.

121

It IS the AIDs defining lesion.

- 1) You will see the skin stuff, and be asked the virus is HHV-8, (a herpesvirus)
- 2) I put the other guy in the example because a small percent of cases follow bone marrow transplantation. Watch for it.
- 3) Treat with Paclitaxel and Doxorubicin!

Q.. An immunocompromised person on your test, either HIV or bone marrow transplants, will present similar so you must be a clever detective:

An HIV positive woman named Jill comes in with a NON-productive cough, fever, dyspnea. Her CD4 count is under 200 as is with all these cases. So, the NBME has to give you some clues. For instance, this cases has no skin lesions so you can rule out Kaposi's, but, labs come back with a silver stain with yeast like circles that look like CRUSHED PING PONG cojones (this is fungus, and it is black). What does she have?

A. This is classic as PCP or Pneumocystis carinii is found in 75% of those without HAART treatment. PCP is very very high on your differential with HIV patients.

**YOU MUST TREAT AND PROPHLYAX with TMP-SMX!!!!!!!!!!**

**VERY QUICKLY, NOTE** that with all of these immunosuppressed people they present in a similar way with lung stuff, fever, diarrhea, etc. So

122

the NBME has to give you a picture...

SO PLEASE GO TO WEBPATH OR ANOTHER SOURCE and quickly GLANCE at the organism. Some of them are, rather most, are distinctive.

OK?

Oh, usu. their T-cell count is under 200

Q. Another HIV patient comes in with white plaques on his mouth and she has some mild genital lesions. This organism can hit any organ but you see a slide of pseudohyphae on a KOH stain; yeast like stuff too. Again, bug and drug?

A. HERE is the famous CANDIDA ALBICANS!

The pseudohyphae in KOH gives it away plus the genital involvement. Treat with NYSTATIN!

Q. The NBME say you must recognize this disease which is often mistaken for Kaposi's Sarcoma. Remember the presentation. But this time, the NBME tells you the patient is homeless and has cats living with him.

Again, dx and tx?

A. You must catch this subtle difference b/c the drug is different..

Here you have those CATS and you treat with Erythromycin.

I forgot to mention this HUGE HIV disease which is called:

Bacillary Angiomatosis

Q. Here, you have 4 patients come in from different locations but all have similar symptoms that are SYSTEMIC:

They all have fever, chills, SOB, fatigue, skin stuff. All let's say are immunocompromised (but not as much as those with HIV). So...NBME must give clues because otherwise you are helpless. NOW, before we go further, you must know you have to rule out cancer and TB or LAWSUIT time...

Mr. One lives in the Great Lakes area

Mr. Two lives in Arizona

Mr. Three lives in Ohio

Mr. Four lives in rural Brazil

All the slides show dimorphic fungus. Bugs and Drugs?

A.. OK, I chose the non typical places:

- 1) Blastomycosis, Great Lakes can also be Mississippi R eastern US
- 2) Coccidioidomycosis, Can also see in California, SW USA, N. Mexico
- 3) Histoplasmosis, Mississippi and Ohio River valleys
- 4) Paracoccidioidomycosis Brazil and Latin America, rare in US

SO, listen up, the presentations are similar and even the slides all look similar like dimorphic fungi should, but the good thing is that the lines are deep due to location of patient's travel.

Know you may see a lot of cases with bat, pigeon, bird poop. Know

124

Histo is by far the most common, and YOU CAN TREAT ALL WITH AMPHOTERICIN B!

Q.. Now the NBME will definitely want you to master BACTERIA and the difference between gram pos and neg,/exo vs endotoxin.

NOW I can cover all the Gram positive vs Gram neg bugs and the classification, but this is BEST DONE BY DIAGRAM, unlike if I ask you a drug and the MOA. Thus, I must ask you to review the above subjects because they are easy points.

I WILL ASK ONE QUESTION THOUGH, which, exo or endo toxin activates the coagulation cascade??????

A. via the Hageman factor, Lipid molecules in endotoxin activate the cascade to DIC!!!!

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Q. OK, here are some directs:

What bug is Bacitracin sensitive, B hemolytic and has streptolysin O and an erythrogenic toxin? GOOD POINT

A. This is Strep pyogenes

Q. Now where were we?

Just for a breather, let's move to pharm for a little while.

Tell me, NBME wants you to be educated about MOA and esp. side effects of drugs...

We cannot cover everything, but let's have a go:

[First, please know a few basic basic equations on calculating maintenance dose and loading dose and Vd and Clearance and half life, they are VERY basic]

BUT FIRST, TELL ME THE DIFF BETWEEN PHASE I AND II METABOLISM?

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"All USMLE cases are original and are expressly not from questions seen, recalled, paraphrased from the real USMLE, the material is for the purpose of the education of future physicians and the safety of their patients."

A. Phase I has redox reactions with cyt. 450 and Phase II inactivates the drug via either sulfation, glucuronidation, conjugation, or acetylation.

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"All USMLE cases are original and are expressly not from questions seen, recalled, paraphrased from the real USMLE, the material is for the purpose of the education of future physicians and the safety of their patients."

Q. Case: pt comes with malaria. You prescribe primaquine. But he tells you he take a H2 blocker starting with the letter "C" (Hint)

Tell me:

- 1) Drug
- 2) What is danger here?
- 3) The other drugs NBME wants you be aware of that have a similar effect.

GIANT CUPCAKE QUESTION

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"All USMLE cases are original and are expressly not from questions seen, recalled, paraphrased from the real USMLE, the material is

126

for the purpose of the education of future physicians and the safety of their patients."

- A. 1) Cimetidine block the metabolism of drug in the liver by depressing the P450 system so PRIMAQUINE will be in body longer, more toxic.  
2) same as above  
3) Think mnemonic "SICKe"

Sulfa drugs  
I soniazid  
C imetidine  
K etoconazole  
e erythromycin

---

"All USMLE cases are original and are expressly not from questions seen, recalled, paraphrased from the real USMLE, the material is for the purpose of the education of future physicians and the safety of their patients."

Q. CLASSIC like an Indian Motorcycle:

Female pt on oral contraceptives, but she gets pregnant. Which drugs could have caused this involving P450 system in liver?

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A.. Drugs that rev up the P450 system result in the drug metabolized too quickly:

127

THIS YOU HAVE TO KNOW:

Think of a "GReasy (Griseofulvin) RIF-raf (An USA slang term for homeless, Rifampin), jumps into a CAR(bamazapine), with a Queen (Quinidine) injecting PHenobarbital and PHenytoin." Imagine this case and say it 20 times...

Those are the drugs I need you to know.

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Q. A young lady comes in asking for oral contraception with history of stasis. What are you worried about?

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A. Thrombosis

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"All USMLE cases are original and are expressly not from questions seen, recalled, paraphrased from the real USMLE, the material is for the purpose of the education of future physicians and the safety of their patients."

Q. African American male comes in with G6PD deficiency (HUGE

128

CONCEPT).

What drugs lyse his RBCs?

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"All USMLE cases are original and are expressly not from questions seen, recalled, paraphrased from the real USMLE, the material is for the purpose of the education of future physicians and the safety of their patients."

A.. Think of him spinning and dancing..

SPINN

S ulfa drugs  
P rimiquine  
I soniazid  
N SAIDs  
N itrofurantoin

---

"All USMLE cases are original and are expressly not from questions seen, recalled, paraphrased from the real USMLE, the material is for the purpose of the education of future physicians and the safety of their patients."

Q. Pt of yours on a med comes in with a breakout red rash. What drugs caused this?

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A.. Think only of the drug vancomycin, which MOST KNOW CAUSES RED MAN SYNDROME!

VANComycin, LOOK AT THE FIRST FOUR LETTERS VANC, THEN

Vancomycin

Adenosine

Niacin

Calcium channel blockers

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"All USMLE cases are original and are expressly not from questions seen, recalled, paraphrased from the real USMLE, the material is for the purpose of the education of future physicians and the safety of their patients."

Q.. PRE menopausal woman with Hot flashes. What drug likely did this, IF she had a family history of breast cancer.

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A. Tamoxifen

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"All USMLE cases are original and are expressly not from questions seen, recalled, paraphrased from the real USMLE, the material is for the purpose of the education of future physicians and the safety of their patients."

Q. Granulocytopenia is seen in a pt whom you gave what meds? Most common ones?

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A. Think the word granuloCytosis. Say it aloud with the C, C, C. Think the letter C three times.

Then,

Clozapine  
Carbamazepine  
Colchicine

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"All USMLE cases are original and are expressly not from questions seen, recalled, paraphrased from the real USMLE, the material is for the purpose of the education of future physicians and the safety of their patients."

NBME requires all doctors to know what drugs cause SLE?

Think of a girl with nice HIPPs (SLE is usu. females), so,

Hydralazine  
Isoniazid  
Procainamide  
Phenytoin

GOOD WORK!

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"All USMLE cases are original and are expressly not from questions seen, recalled, paraphrased from the real USMLE, the material is for the purpose of the education of future physicians and the safety of their patients."

Q. An alcoholic pt of yours comes in with hepatic necrosis. What drugs are commonly seen doing this?

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A. Imagine your alcoholic pt and ask him..

"did you HAVE a drink?" since alcoholics have liver probs. this relation is strong. Look at letters HAVE, spec. HAV in have...

Halothane  
Acetaminophen  
Valproic acid

Keep Keep remembering by repetition, you have to remember the MNEMONIC FIRST!

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"All USMLE cases are original and are expressly not from questions seen, recalled, paraphrased from the real USMLE, the material is for the purpose of the education of future physicians and the safety of their patients."

Q. A pt of yours has a UTI. You gave him a drug and his skin is

132

peeling, even the soles and palms. What drug(s) caused this?

You should think..."I must think a SEC!" Look at letters SEC...

S ulfa drugs  
E thosuximide  
C arbamazepine

Great. Now YOU MUST REPEAT THIS OVER AND OVER! IMAGINE THE MNEMONIC FIRST!! This is how your mind works.

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"All USMLE cases are original and are expressly not from questions seen, recalled, paraphrased from the real USMLE, the material is for the purpose of the education of future physicians and the safety of their patients."

Q. I agree with the NBME's heavy duty coverage of side effects. BEFORE you even treat a patient, you should know what it could do to them if things go wrong! So, in support of the NBME..

Think, over and over, "I SAT in the sun and my eyes hurt from the glare."

Look at the letters SAT...

S ulfa drugs  
A miodarone  
T etracyclines

---

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their patients."

A. sorry the mnemonic with the SAT in the sun causes:

### PHOTOSENSITIVITY!

---

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Q. After some meds, three patients of your cannot breath well...and their lungs are fibrotic on exam. What are the three drugs?

---

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A. Lung Fibrosis

OK, one is Bleomycin (I remember this one because Bleo- sounds like Blow, and you use your lungs to blow, hence the lung association)

NOW, think I exercise regularly and have strong LUNGS and ABs (short for abdominal muscles). AB...

A. miodarone

B. usulfan (a drug for chemo for CML)

---

"All USMLE cases are original and are expressly not from questions

seen, recalled, paraphrased from the real USMLE, the material is for the purpose of the education of future physicians and the safety of their patients."

Q. Pt of yours comes in with aplastic anemia, aa. What drugs are common for this?

---

"All USMLE cases are original and are expressly not from questions seen, recalled, paraphrased from the real USMLE, the material is for the purpose of the education of future physicians and the safety of their patients."

A.. Think Aplastic anemia has the letters abbreviation, AA for Alcoholics Anonymous. Then associate an Alcoholic CAB driver who hits you and your blood gushes out!! Yuck! But... look at the word CAB...

Chloramphenicol  
Aspirin  
Benzene

valuemd.com

See how easy it can be? But you must keep on repeating...again, again...CAB...CAB, then think of drugs, then think of CAB, then think of Drugs, IT WILL STICK...

---

"All USMLE cases are original and are expressly not from questions seen, recalled, paraphrased from the real USMLE, the material is for the purpose of the education of future physicians and the safety of their patients."

Q.. You gave your patient an antibiotic and she comes back with neuromuscular damage. What drug caused this?

---

"All USMLE cases are original and are expressly not from questions seen, recalled, paraphrased from the real USMLE, the material is for the purpose of the education of future physicians and the safety of their patients."

A. Know that saying with the Parrot who keeps repeating "Poly wanna cracker?" You know it... it is so silly and neurotic for the bird to repeat it.

So....

Poly = NEURO tic

(Poly stands for Polymyxin)

---

"All USMLE cases are original and are expressly not from questions seen, recalled, paraphrased from the real USMLE, the material is for the purpose of the education of future physicians and the safety of their patients."

Q.. You gave a patient of your a med for arrhythmias but she presents with Ventricular Tachycardias! (i.e. torsade de pointes meaning "twisting of the points" in Latin)? What drug has SE?

---

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A. Think of beautiful QUeen that is SO gorgeous she makes your heart thump (like ventricular tachycardia)! See the capital letters QU and SO in the expression?...

136

Qu inidine  
SO talol

Easily associated with ventricular tachycardia now!!!

---

"All USMLE cases are original and are expressly not from questions seen, recalled, paraphrased from the real USMLE, the material is for the purpose of the education of future physicians and the safety of their patients."

Q. You have a cancer patient on drugs you gave that presents with Ototoxicity and Nephrotoxicity. What 3 drugs w/ SE?

---

"All USMLE cases are original and are expressly not from questions seen, recalled, paraphrased from the real USMLE, the material is for the purpose of the education of future physicians and the safety of their patients."

A. Here is a VERY good mnemonic...

Think of a wild CAIF (CALF, you know those young horses) that kicks you in the kidney (Nephrotoxicity) and then your head (Ototoxicity).

Recall Ototoxicity is damage to CN VIII so you are dizzy which makes perfect sense!

Again,

Cisplatin  
Aminoglycosides  
I (nothing here it is a lower case letter)  
F uroseamide

MAKE A PICTURE IN YOUR MIND, THEN REPEAT THE MNEMONIC THEN SAY THE DRUG. IT DOES WORK!

---

"All USMLE cases are original and are expressly not from questions seen, recalled, paraphrased from the real USMLE, the material is for the purpose of the education of future physicians and the safety of their patients."

Q. Case: A peds patient of yours comes in from an antibiotic that you gave him that is giving him joint pain in his tendons. What drug did you mistakenly give him?

---

"All USMLE cases are original and are expressly not from questions seen, recalled, paraphrased from the real USMLE, the material is for the purpose of the education of future physicians and the safety of their patients."

A.. There is inflammation of his tendons due to Fluoroquinolones.

Think "Fluoroquinolones sounds like Floor-oquinolones" See the word Floor. It is hard material. And so if a kid falls down on the Floor, he will bust his tendons.

Floor=Tendons

---

Q. This is a must know:

An OB/GYN pt of yours has Trichomonas which you treated with Metronidazole. Tonight she is going to a cocktail party. What do you warn her about? (THIS IS REALLY ONE OF THE MORE TESTED ONES)

---

A. Your patient will have a flushing of the face, nausea, and vomiting called Disulfiram reaction.

So this is a great mnemonic, think "Female in PMS (premenstrual syndrome) looks really sick and nauseous, and is vomiting (Disulfiram reaction).

Procarbazine (a cancer drug)  
Metronidazole  
Sulfa drugs

(The last two are favorites of NBME)

Q. The boards and hospitals are in LOVE with this one:

A male patient of yours with gastric ulcers on cimetidine complains of big breasts. Whoa! That ain't good...what other drugs cause gynecomastia?

A. Think...of a guy with big breasts. Isn't that SICK?

Look at letters SICK..

Spirolactone  
Inebriated (This word means drunk with alcohol)  
Cimetidine  
Ketoconazole

So, Spirolactone, Inebriated w/Alcohol, Cimetidine, Ketoconazole cause SICK big breasts on a male.

You likely know this, should I give my memory mnemonic?

Penicillin causes anaphylaxis and INH causes hepatitis....I saw these both in my medicine rotation so it is second nature to me.....

I just mentioned them to you b/c these are heavily tested.

Q. This is also a HUGE SE, so you must know cold:

A bipolar patient of yours is on a med and complains of excessive urination. What drug?

A. This is Lithium. It causes Diabetes Insipidus.

Think this..."IF you Lie, then you should Die-(abetes)"

Li=Di

Q. OK, just in case, did you know the famous one:

ACE inhibitors CAUSE coughing (from bradykinin).

But, let me ask, a patient of yours is depressed and you medicated him. But he comes back complaining of the inability to read near vision and a fast heart rate. What drug did you give

A. he is on a tricyclic antidepressant like imipramine which has anti muscaric side effects, thus mimicking atropine:

This one I remember differently. Let me explain....Tricyclics cause your eyes to be dry (hallmark sign) so you are not crying. Since you are not crying, you must be on a drug which keeps you from getting depressed (hence, antidepressant drug).

Tricyclics=antidepression.

Q. This concept is actually a suggestion:

YOU MUST NOT UNDERESTIMATED DRUG SIDE EFFECTS, they are a MUST KNOW!

SO, YOU ALSO MUST KNOW THEM BOTH WAYS, IN OTHER WORDS, THEY CAN PRESENT A PATIENT WITH A COUGH AND CHF AND THEN YOU HAVE TO PICK ACE INHIB. OR THEY COULD TELL YOU A CLASS OF DRUGS LIKE THE TRICYCLICS AND THEN ASK YOU WHAT SIDE EFFECTS OR DRUG REACTIONS OR EVEN MAKE YOU POINT TO A DIAGRAM WHERE THE DRUG DOES THE ACTION. THAT IS LIKE 3 OR 4 STEP THINKING, BUT THE NBME NEEDS YOU TO DO THAT!!!

---

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---

A.

The steroids makes her susceptible to osteoporosis and heparin can cause the same effect.

---

Q.  
I SAVED THE BEST FOR LAST:

A mother comes to your clinic in emergency because her baby looks pale and the baby's heartbeat is slow and barely audible. The baby was given an antibiotic by an inexperienced med student!

Drug and name of condition please?

A.. The lack of glucoronyl transferase activity in the newborn will delay the metabolism of the chloramphenicol to the inactive form. The result will be an elevated chloramphenicol level which can cause cardiovascular collapse and death.

Q.. A pt of your overdoses on HEPARIN. What do you do?

A.. protamine sulfate

Q. A hypertensive patient of yours left out the pills and her young child ate a bunch of them, her heart is very slow on exam. What do you give?

---

A. For B Blockers, you give Glucagon.

Q. A patient of yours swallowed a bottle of weed killer. What do you give him?

---

A.. For organophosphate poisoning, you give either pralidoxime or Atropine!

---

Q. A depressed patient of yours tries to kill herself by swallowing a bottle of imipramine. What do you give her?

A. you give sodium bicarbonate.

Q. An infant swallowed a bottle of Fe pills. What do you do?

A. you give her Deferoxamine!

Q. A peds patient of yours comes in having eaten lead paint chips in his old house. What 2 drugs must you give?

Bonus? What is the difference between the 2 drugs?

A. You could do a gastric lavage, but if lead levels are high, give both dimercaprol and Calcium EDTA.

The difference between the two is that Dimercaprol (BAL) crosses the Blood Brain Barrier, and CaEDTA does not!!!

Q. A peds patient of yours presents with choleralike symptoms with diarrhea massively and there is garlic smell on his breath. What toxin did he eat and what do you do?

A. this is a perfect presentation of Arsenic poisoning from a child eating rodenticide. Quickly, give a chelator like Dimercaprol/BAL. And put him on liquid support due to the diarrhea.

Q. A 37 yo male patient of yours went on a fishing trip in the Far East and ate a LOT of shark fin soup. He comes to you with headache, memory loss, ataxia, vision troubles, memory loss, he is in BAD shape. What happened and what do you do?

A. Whoa!

This is a classic case of methyl mercury poisoning. People think of

thermometers but most cases involve the consumption of fish in polluted waters. Chelate FAST with Dimercaprol...

Q.. Hey I must ask you guys the mechanism of action of Dimercaprol!!!!

A.. This is KEY:

In the last case, mercury binds to the body's ubiquitous sulfhydryl groups. Thes BAL are thought to compete with sulfhydryl groups in binding methyl mercury by using its thiol groups.

Watch out, because the newest agent is 2,3-dimercaptosuccinic acid (DMSA) which is proven to be superior to BAL. NOT IN THE textbooks like [Kaplan](#) and [FA](#), but may be in NBME's mind.

Q.. Bizarre!

A patient walks into your clinic all giddy and acting hysterical, has SOB, is dizzy and his job is working at a plastic manufacturing plant. What did enter his body (HINT: you see this in 007 James Bond movies)? And what do you give?

A. He has cyanide poisoning, as in the plastics industry it is part of the solvent. He inhaled the fumes. You must give Sodium Nitrite., FAST!

- HY 300- 320

- HY 300: Bizarre!

A patient walks into your clinic all giddy and acting hysterical, has SOB, is dizzy and his job is working at a plastic manufacturing plant. What did enter his body (HINT: you see this in 007 James Bond

movies)? And what do you give?

..... He has cyanide poisoning, as in the plastics industry it is part of the solvent. He inhaled the fumes. You must give Sodium Nitrite., FAST!

- HY 301: NOW, we know that with heavy metal poisoning we chelate with Dimercaprol, that includes silver, copper, and gold if anyone actually going to eat such precious metals. BUT, what is the main cause of death from heavy metal poisoning overall?..... it is encephalopathy of the brain.

- HY 302: I love this case because it involves chemistry and you MUST be aware of it. Two scenarios:

Patient A is getting a large lidocaine dose for LP:

Patient B is working with aniline dyes in a factory:

Both present to your clinic the same way, with tachycardia, and symptoms of CYANOSIS like lip/skin discoloration (hint).

1) What is the disease?

2) Mech of disease

3) Treatment

4) Mech of Action of tx?.....A:

1)Both of these cases are of methemoglobinemia!

2) Any oxidizing agent converts Fe +2 to Fe +3.

3) As such the hemoglobin cannot bind the oxygen in this ferrous form.

4) Give methylene blue and LATER bicarb and hydration for the acidosis. Long term tx for chronic cases is Vit C! The methylene blue acts as a cofactor in the NADPH-dependent metHb reductase system and reduces the iron to ferric form which can bind the oxygen.

How's that for combining the sciences!

- HY 303: I am almost out of gas. But here goes...

Everyone knows you give acteylcysteine for Tylenol overdose, but

what if your patient overdoses on Streptokinase that your inexperienced intern gave him? What do you do?..... Cool.  
You treat with aminocaproic acid

- HY 304: Oh no!

A peds patient of yours comes in hyperactive, breathing heavily and his mom said he ate a bunch of baby aspirin!! What do you do???..... someone told me while I was teaching that [FA](#) is different, but I stick to my guns:  
You perform gastric lavage and give activated charcoal., also: just wanted to add that you also alkalinize the urine with some drug like acetazolamide.

- HY 305: A med student colleague of yours is studying for USMLE Step 1 and has not sleep for a week. He is dosed on Ritalin, amphetamines. You bring him to the doctor and your friend is hysterical and sweating, and his eyes are dilated. What did the doctor do? .....Again, [FA](#) and [Kaplan](#) say different,

but my experience in clinics say:

You administer activated charcoal, give him a benzodiazepam, and MAYBE haloperidol if he is uncontrollable.

- HY 306: Great concept!

A male 40 yo patient of yours is brought to the clinic after a suicide attempt by ingesting a bottle of Benadryl (diphenhydramine). He presents with delirium, hot skin without sweat, he cannot pee and the ECG has arrhythmias.

- 1) What mech is going on?
- 2) What drug do you administer stat for his general symptoms?

3) What drugs do you give for his restlessness and cardiac symptoms?  
.....The diphenhydramine is giving him severe anticholinergic symptoms.

- 1) Give Physostigmine stat (an acetylcholinesterase inhibitor)
- 2) After his Ach goes back up, give...
- 3) Benzos for his restlessness and Sodium bicarbonate for his arrhythmias. Bingo!

- HY 307: You have patient with non Hodgkin's lymphoma. He is on high dose methotrexate therapy and the MOPP regimen. What is the MOA of methotrexate, again (I asked this) and what do I need to give him due to high dose methotrexate? .....Methotrexate is a folate antagonist at dihydrofolate reductase. You need to give the patient Leucovorin calcium which is a derivative of folic acid but does not need dihydrofolate reductase. This is called leucovorin rescue.... Also, give the patient L-asparaginase (produced naturally by E-coli) which catalyzes L asparagine to aspartic acid!

- HY 308: Now where was I?  
Hmmm...LET's roll through more drugs...  
What is the SE, Use, and MOA of acetazolamide?.....SE:  
Urolithiasis, Ca most likely.  
Use: use if your pt. is alkalotic.  
MOA: Blocks enzyme carbonic anhydrase! So bicarb spills out into the toilet!

- HY 309: You can also use acetazolamide for Glaucoma because aqueous humor production is decreased!

- HY 310: Hmm.. let's NOT lump all the drugs because then it will be

TOO easy to answer the questions! So, let us drill away randomly!  
We know Buspirone is an anxiolytic, which receptor does it act on? Be specific!..... 5 HT 1A receptor!

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- HY 311: You may know Clonidine (as well as methyldopa) is an alpha 2 agonist to control HTN, but if I give you a diagram, Are the receptors PREsynaptic or POSTsynaptic predominantly? Second, True or False, is there ANY time clonidine causes HTN?..... Clonidine's receptors are mostly PREsynaptic.

AND, the NBME WILL ASK YOU THAT if given IV push, you will get momentary HTN from a few postsynaptic alpha 2 receptors on vascular smooth muscle! The NBME likes you to answer in graph form!!!!!!

- HY 312: HEY,

Simply, you need to be able to eliminate wrong answer choices! So..

1) WHAT is the SEs of chlorpromazine?

2) Receptor?

3) Major usage?..... This is an antipsychotic. Its main SEs are from blockage of cholinergic, adrenergic, histaminergic, and dopaminergic receptors! all of them!  
So,

you see the dry eyes, mouth, constipation, can't pee; The adrenergic block will make you hypotensive when standing suddenly, and you get impotence. The Histamine effect could make you tired and stop any rashes from forming. And the D2, again it is the D2 blockade that will give the extrapyramidal dysfunction: Parkinsonism mostly. After a few

months you will get the rigidity, fever and then Tardive dyskinesia!

- HY 313: Case: you are treating a woman with Parkinson's disease. OK, OK it is an ergot alkaloid called Bromocriptine.

1) SEs?

2) Mech of Action?

3) What other diseases does it treat?..... 1) Watchout for first dose cardio failure!

2) This is a D2 agonist and D1 antagonist!

3) Also used to treat hyperprolactinemia, amenorrhea, galactorrhea.

- HY 314: The readers of this board seem to convey they understand that B1 blockers are used for HTH, Angina, Arrhythmia. But what is an important SE in diabetes patients?..... In diabetes, you can mask the tachycardia from B1 blockage due to hypoglycemia and you can get impotent and reflux!

- HY 315: Quicky:  
Other than OXYTOCIN, what Ergot alkaloid is used to contract the uterus in pregnancy?..... well, I better say AFTER the delivery b/c if you use it during pregnancy, your purpose is to abort the fetus.  
Here, the answer is ergonovine!

- HY 316: As a group, we already discussed Chloroquine! But tell me the imp. SEs!..... Beware of G6PD deficiency hemolysis, and warn patients about vertigo and itchy skin.

- HY 317: OK, think of cholestyramine...  
Give me 1) SEs 2) MOA 3) medical use.....Answer:  
1) This anti lipid can cause constipation and in higher doses produce fatty stools, and fat soluble vitamin deficiency!  
2) This works by binding bile acids and stops absorption of cholesterol in the gut. They diverts hepatic cholesterol to make more bile acids, upregulates LDL receptors and thus lowers plasma LDL  
3) it is an antilipid
  
- HY 318: Which is the only adrenergic receptor to work by the PIP Ca cascade?..... alpha 1, the others work via cAMP!
  
- HY 319: The anti Parkinson drug Levodopa is used with Carbidopa. Why?  
And what category of rxn is levodopa to dopamine?.....  
carbidopa prevents peripheral utilization by blocking the enzyme dopa decarboxylase (which is answer #2)
  
- HY 320: I am sure that the NBME wants you to know about INSULIN...  
1) MOA in Adipose:  
2) MOA in Muscle:  
3) MOA in Liver:  
4) What ion is eliminated when given with glucose as tx?  
5) Do you know ALL the enzymes affected by INSULIN?.....

answer: 1) Activates Lipoprotein lipase pulls glucose inside  
2) In muscle it stimulates glycogen synthesis, and K and glucose uptake  
3) In liver it makes glycogen by...(tyrosine kinase activity) and works

on all the irreversible steps of glycolysis and glycogen synthesis.  
4) It is used with glucose to get rid of K!

321. ARE we all on DRUGS? Yeah!

OK, NBME wants you to know diabetes drugs COLD like

GLYBURIDE!

you know it is a sulfonylurea that simulates insulin release from B cells. But what ELSE does it do at what channel?

322. am i getting sloppy, the answer to HY Concept 321 is glyburide acts on K channels that are ATP sensitive.. watch out it can cause hypoglycemia.

NOW, with Isoniazid, you give what VITAMIN to min. toxicity?? HY ultimate!

.....Vit B6

323. HUGE HUGE HUGE!

What is PHENOTOLAMINE? EXACT, please?

WHAT DRUG IS IT RELATED TO THAT SOUNDS SIMILAR BUT HAS IRREVERSIBLE EFFECTS?

.....

IT is a NONselective alpha blocker! NONSELECTIVE....

KNOW that PHENOXYBENZAMINE is close but IRREVERSIBLE! Both are used for pheochromocytoma but cause prominent orthostatic

hyPOtension.

324. Tetracyclines...

YOU KNOW the MOA exactly?????

SEs?

What common drink impairs its absorption?

.....

hey, these binds to 30S subunit and blocks aminoacyl t RNA. Now, KNOW the drug uses an energy dependent active transport pump.

SE include fatty liver and brown teeth in kids.

And, milk and antacids block absorption!

325. We KNOW now that primaquine, you avoid in women with connective tissue disorders. BUT, in pts. with G6PD def., what other than hemolysis can it cause?

.....

Dont' give to pregnant women and in G6PD def. we get methemoglobinemia? REmember?

327. QUICK QUIZ, to learn effectively, you must compare and contrast!!!!

So, FAST, without hesitation, KNOW and tell me:

1) TWO alpha adrenergics AGONISTS that work only on alpha 1 receptor (hint, letters M then P)

2) ONE alpha adrenergic BLOCKER selective for alpha 1 only!

- 3) Does dopamine work on alpha 2 agonism?
- 4) Quickly, yell out a NON selective B adrenergic AGONIST!
- 5) SUPER FAST, scream out a NON selective B adrenergic BLOCKER (dangerous for asthma patients, see the connection?)
- 6) SUPER FAST, wail out a B1 selective blocker starting with letter M!

KNOW THESE UNTIL YOU CAN DO THEM IN YOUR SLEEP!

.....

- 1) methoxamine, phenylephrine
- 2) prazosin
- 3) NO!
- 4) Isoproterenol
- 5) Propanolol
- 6) Metoprolol

328. Ah, got you!

HERE IS A STRAY CAT QUESTION! BUT exactly the kind of question NBME WILL ASK...

You have to address anemias.....iron deficiency is most common, but two BIGGIES TO KNOW IF YOU KNOW CONCEPTS:

- 1) Do you give drug Ferrous Sulfate or Ferric Sulfate?

2) and, DO you give the meds to thalassemia or sideroblastic anemia pts?

.....  
1) MUST give in the form Ferrous Sulfate

2) NO, you don't give iron to these patients with anemia!! BIG LAWSUIT ISSUE AND A USMLE STEP 1 mustknow.

329. WE addressed methotrexate and its function on DHFR, but again, if your patient shows signs of toxicity, what drug can you give, and the name of this process?

.....  
Called Leucovorin rescue, give a form of folinic acid.

330. THIS MUCH LOVED DRUG IS used in leukemias, chorionic carcinomas, ectopics, rheumatoid arthritis, etc.

KNOW THESE, and does this drug cross the CSF barrier?

.....  
no, not very well!

331. We reviewed that Amrinone is an inotrope. What enzyme does it work on?

.....  
This CHF drug blocks phosphodiesterase and pumps up cAMP, just like your coffee!!!!

332. I am thinking of a drug used for Parkinson's...

1) What MOA?

2) What OTHER common drug starting with letters AT... is similar in that the MOA raises pulse?

3) This alkaloid can result in what psychological effects?

.....

Benzotropine, works like ATROPINE, but can readily move into the CNS.

2) It blocks the disinhibited cholinergic neurons and THUS LOWERS ACh levels. (This is due to the fact that in Parkinson's, you lose the dopaminergic neurons that block the ACh in the SUBSTANTIA NIGRA)

3) Watch out for SE like atropines AND DELIRIUM!!!!!!!!!!!! This is an antimuscarinic!

333. We already reviewed fluoxetine, an antidepressant.

Tell me main commonest side effects???????

.....

MUST KNOW,

this drug is used by just about half the USA....and the world... (exaggeration) but...

it causes antimuscarinic side effects....and sometimes unwanted erections!!!

334. We reviewed Chloramphenicol and it binds to 50 S,

but the NBME will likely go a step FURTHER.....

so, give me quickly the ENZYME IT WORKS ON.

two, give me the most feared SE in adults, then

in children...

.....  
REMEMBER the concept of all concepts that the NBME wants you to  
KNOW SO MUCH about a relatively small universe of things....

HERE, we have chloramphenicol hits PEPTIDYL TRANSFERASE.

MOST WORRISOME is aplastic anemia in adults and the infamous Grey  
baby syndrome in babies....

Are you remembering these?????

335. HUGE HUGE HUGE concept.

Your patient is a PREGNANT LADY with SEIZURES! Remember  
pregnancy is SO KEY ON USMLE. So, what is DOC?

.....  
would you believe, phenobarbital?

336. BIG CUPCAKE question:

What compound does strychnine work on?

What is the antidote of choice?

REVIEWING!!!!

.....  
This blocks glycine receptors leading to seizures.

YOU can give diazepam, among other choices....

337. This question tests your conceptual knowledge of cancer drugs...

Give me the exact MOA of 6-Mercaptopurine?

Used for ALL and Hodgkin's and IBD, what enzyme gets rid of it?

.....  
This cancer drug is acted on by HGPRT, an enzyme in the purine salvage pathway. It is phosphorylated to TIMP which blocks IMP to AMP. Then, PRPP is blocked from making ribos-5-phosphate which regulates purine synthesis, ending all in the result of lower DNA and RNA

IT IS METABOLIZED BY XANTHINE OXIDASE VIA METHYLATION AND OXIDATION.

SEE....ALL THE SAME ENZYMES KEEP COMING BACK IN HYers!

338. Big one!

YOU KNOW Amantidine is used in Parkinson's and Influenza! But give me MOA? Can you point on a diagram which step of viral rep it works on?

.....  
hey, know it works on INfluenza A via blocking assembly and uncoating.

339. YOU HAVE TO MEMORIZE AND KNOW ALL THE CEPHALOSPORINS, WHICH ARE FIRST SECOND AND THIRD GEN, UNTIL YOU CAN DO IT FROM SCRATCH!

But, let me ask, if you know, then I can be happy you know the concepts:

What is the main notable difference between 1st and 3rd generation

cephs?

.....

Other than affinity for gram negs, 3rd generationers can cross the BLOOD BRAIN BARRIER!!

340. We previously reviewed MAO inhibitors, but I was surprised that so many of my students could not recall a single one!!!!!! Name at least three..

Second, can you pt to where it will work on a diagram?

.....

Here's three of them: Selegiline, Tranylcypromine, Phenelzine

Remember key things like watching out for tyramine containing foods? Know that MAO-A IS serotonin's affinity and MAO-B is involved in anti-Parkinson's!!

341. Heres one that is missed a lot:

NITROFURANTOIN is anti UTI med. What grp of people is this contraindicated in ?

.....

G6PD def. patients!!!

342. NOTE: NOt a question but KNOW that

when you give ISOPROTERENOL for bradycardia, ask pt if she is hyperthyroid or diabetic!

343. We previously discussed ATROPINE,

an antimuscarinic,

TELL ME THE THREE RECEPTOR SUBTYPES AND 2ND MESSENGERS

TELL ME ALSO WHEN IS IT USED MOST OFTEN?

WHAT IS THE NAME OF THE DRUG THAT IS RELATED FAMILY TO TREAT MOTION SICKNESS?

.....

M1 is in the CNS and works via IP3 and Ca

M2 is in heart and works via K and cAMP

M3 is in smooth muscle and works via IP3 and Ca

NBME LOVES THE ABOVE INFO, AND YOU MUST KNOW THE PICS,

You often see atropine for organophosphate poisoning. And Scopolamine is used for motion sickness.

344. We covered EDROPHONIUM. What is this MOA? What dx does it work on?

What is the related drug for LONG TERM USES OF SAID dx?

.....

This is an Acetylcholinesterase inhibitor which pumps up ACh at NMJs. you use this to diagnose myasthenia gravis!!!!

Pyridostigmine is used for chronic myasthenia gravis!

345. BIGGIE PT:

We spoke of Sulfa drugs, so many of my patients were allergic to sulfa drugs....

Thus, tell me the enzyme that sulfonamides block?

YOUR ATTENDING WILL SMACK YOU SILLY IF YOU GIVE TO WHAT 4  
HUGE GROUP OF HUMANS!!!!?????!!!!!!?????

.....  
This PABA analog, part of TMP-SMX, blocks dihydropteroate  
synthetase.  
DO NOT... give to

PREGNANT WOMEN  
PTS, w/ history of STEVENS JOHNSON SYN  
G6PD def patients  
PTS with a history of renal stones

IF THE USMLE DOES NOT ASK YOU, YOU WILL BE ASKED DURING  
YOUR MEDICINE ROTATION!!!

346. Quicky:

Yohimbine is often presented in as NBME case everywhere...what's the  
MOA?

.....  
THIS DRUG IS AN ALPHA 2 BLOCKER.

SOME THOUGHT IT WAS AN ALPHA 2 AGONIST.....

remember, DON'T MIX ANTAGONISTS WITH AGONISTS, IT IS AN  
EASY COMMON ERROR...

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347. A GIANT:

YOU WILL SEE Glaucoma.

Give me the B-Blocker that is the DOC? What is its MOA?

But what OTHER adrenergic drug can be used and ITS MOA?

BIG SENSITIVE Point!

.....  
Timolol is the DOC via lowering both production and secretion of aqueous humor.

NOW, an alpha adrenergic AGONIST like epinephrine or BRIMONIDINE can be used which works similarly but also improves drainage from the canal of Schlemm!!!!

Surgery is iridotomy....

348. JUST KNOW THIS CONCEPT ASKED A TRILLION WAYS:

Epinephrine is a pure adrenergic agonist: both alpha 1,2 beta 1,2

But NOREPInephrine has NO Beta 2 activity!!!!!!!!!!!!!!!

AND Phenylephrine is a true alpha activity only drug.

AND Terbutaline is a a true BETA activity only drug. (like albuterol)

349. To save myself time, I am combining...but you must must must KNOW

the GLAUCOMA, because in clinics, it can lead to sudden blindness without symptoms at times. YOU could be sued for one million dollars easy if you miss this...

KNOW...

As we discussed there is open angle and closed angle glaucoma...

CLOSED ANGLE is when the canal of Schlemm is blocked and is an emergency...

OPEN angle is most common...

We talked about the adrenergic meds, but also KNOW that

Prostaglandins like latanoprost are used...

an alpha 2 agonist like Brimonidine can be used...

A carbonic anhydrase inhibitor can be used via MOA of lowering bicarbonate production and sodium transport and thus aqueous humor formation...

and finally, a cholinomimetic like pilocarpine can be used, but a friend of mine who is in ophtho says this is going out of style...(the miotics)

350. EVERYONE I ever spoke to said CNS pharm is vital and we just discussed GLAUCOMA. NOW, let's quickly go over BPH, benign prostatic hypertrophy...

YOU, Dr. ValueMD can use terazosin or prazosin for BPH (the only diff is that prazosin is shorter acting). (Students think of alpha blockers only for HTN, but recall that alpha receptors are everywhere, like, in the bladder?)

[You could use Phenoxybenzamine, but that is a bit too much, eh]

Second line is FINASTERIDE, which you recall is a 5 alpha reductase inhibitor, MOA please? You remember?

.....

Everywhere I went, I saw BPH (like CHF, Diabetes, etc.).

Finasteride hits 5 alpha reductase and lowers testosterone production which decrease prostate size.

I did not ask, but for all cases of BPH, YOU HAVE TO DO A RECTAL EXAM! You must rule out malignancy.

351. REally quick:

IF you see pentamidine on your test/clinics, what BUG are you treating?

.....

PCP or African sleeping sickness...

352. HERE IS a good question that tests your ability to understand a KEY!

We spoke of methoxamine as an alpha agonist 1. IS IT inactivated by a MAO?

.....

This drug...which works via PIP and DAG and IP3 and Ca. all important...used to treat Atrial Tachycardia...is NOT a derivative of catecholamines!!!

353. HEY, this a a sweetie!

KEY KEY KEY KEY....What drug did I mention BEFORE which is used ABOVE THE DIAPHRAGM for anaerobes which has the SAME MOA as ERYTHROMYCIN???? THE NBME IS ALL SECONDARIES, (thus their 350

question STEP 1 is LIKE a 10,000 question test!!!!!!!)

And what is the mech of RESistance of this drug?

IF YOU KNOW THIS, YOU ARE DOING VERY VERY WELL!!!

.....  
We are talking about CLINDAMYCIN! via the 50 S subunit!

And the mech of resistance, do not forget the NBME loves these resistance questions because they are BIG points in rotations and in university research in USA...

Bacteria methylate the 50S binding site!!!! and stop the drug permeabilty through membranes!!!!

354. Quickly, we discussed this..

What is the MOA of Gemfibrozil? IOW, what enzyme does it work on? For what disease?

.....  
This revs up lipoprotein lipase and breaks down VLDL!!!

355. ORLISTAT IS NOW SEEN ALL OVER ALL USMLE STEPS! HOW IS THIS ANTILIPID DIFFERENT FROM cholestyramine?

.....  
This statin is a HMG CoA red inhib. and cholestyramine is a bile acid binder that revs up bile acids and thus lowers blood LDL.

Be ready to answer the most common side effect of ALL statins!!!

THE ANSWER IS MUSCLE PAIN!! IF USMLE STEP 1 does not ask you, you WILL BE ASKED DURING YOUR MEDICINE ROTATION AS OFTEN

AS THEY ASK YOU YOUR NAME!

356. NOW, another anti lipid is PROBUCOL, what neg. thing does it do to one of the cholesterol types..? def a must know!

.....  
This antilipid pushes DOWN HDL which is BAD!

357. YOU will have to know ALL the antacids! THEY ARE EVERYWHERE!

Tell me the three main types, what specific advantage each one has, the SEs of course, and what PATIENT DO YOU DEF. NOT GIVE THEM TO????

HUGE GIANT HUMONGOUS CONCEPT! Think first before answering....this one esp.

4 sep. questions here!!!

.....  
Calcium (the famous brand is TUMS) also lowers K (remember the previous concept somewhere where I told you you give insulin and Calcium salts to HYPERKALEMICS!! ALL CONCEPTS TIE TOGEHER!)

Aluminum antacids bind phosphate and lower phosphate levels!!!!  
(Think Al-phos...sounds like al..batross the bird..worked for me)

Magnesium antacids (famous brand Milk of Magnesia) is used for constipation too!!

NOW SIDE EFFECTS! (SEs):

Calcium: OVERDOSE can rev up acid secretion!

Aluminum: SE is constipation!

Magnesium: SE is DIARRHEA!

ALL THREE CANNOT BE GIVEN TO RENALLY COMPROMISED PATIENTS...IF YOU FORGET THIS, YOUR ATTENDING WILL LOSE HER JOB AND YOU WILL BE DEPORTED BACK TO YOUR HOME COUNTRY!

358. Now, I am thiining of a famous antifungal we previously discussed... to treat HIstoplasmosis, Candida, and Cryptococcus!

WHAT FAMOUS DRUG IS THIS (starts with letter A and rhymes with lamp)?

What is MAJOR SE you watch for?

What organ does it have trouble reaching, thus, what drug do you co administer????????????????????????????????

.....  
this is AMPHOTERICIN B!

IT SELECTIIVELY BINDS ERGOSTEROL, which is a fungal type memb., and you watch for liver and kidney pts, and it can cause FEVER and CHILLS!

It has some trouble entering the brain, so give with flucytosine for HIV Cryptococcus Meningitis!!!

359. This POPULAR DRUG is given OFTEN to patients with SEPTIC SHOCK (my own grandfather died recently of this (starts with letter "N"))

NOW, what patient condition is contraindicated???

.....  
NOREPINEPHRINE, you avoid in hypovolemic patients!

360. Patient of yours named Ben comes in with BP 250/110 !!!

What drug do you grab for IV drip!!!?

What enzyme does this drug act on?

What dangerous SE you watch out for?

.....  
This is DOC for HYPERTENSIVE CRISIS!

It drops PREload and AFTERload via vasodilation via GUANYLATE CYCLASE!

WATCH OUT FOR CYANIDE TOXICITY!

361. WE MUST KNOW THAT:

Ibuprofen is used for HEADACHES and to close a PDA in a baby!!!

WHICH EXACT two prostaglandins are blocked downstream here that kept the patent ductus open?

.....  
YOU HAVE TO KNOW:

This NSAID indirectly via COase inhibition BLOCKS PGE2 and PGI2

362. THIS WILL ANSWER 1000 QUESTIONS:

BE READY, IN A GIANT PICTURE OF A CELL WITH ARROWS EVERY PLACE:

WHERE EACH MAJOR ANTI BACTERIAL ACTS...

FOR E.G., WHERE WITH AN ARROW DOES POLYMYXIN WORK?

.....

POLYMYXIN acts with the arrow pointed at the cell wall... KNOW EVERYTHING HERE>>>

Sulfas block at step right before DHF on dihydropteroate synthetase....

Clinda and Erythro block translocase at 50 S.....

Vanco blocks the polymerization step so single peptidoglycans cannot grow..

ON THIS YOU MUST SEE ON A DIAGRAM!!!

363. Someone asked about Q-Fever...

IT is called Q because its first discovery was a query or mystery...

It is from bug *Coxiella burnetti*..

You catch it from ticks in Montana but the bug is everywhere.

Give DOC Doxycycline..

364. Here is an answer to a Value MD brother/sister but PLEASE, if I do not get back to your question, I gave my private email on Yahoo! to a few people and I am WAY behind. I will try to catch up but If i don't, then I am so sorry.

SOMEONE asked me about PACLITAXEL, which is TAXOL... it is used

for all types of cancers in clinic and blocks microtubule formation.

It causes BAD leukopenia and can be cardiotoxic!

365. Someone asked about MOA of flucortisone and what it is?

Answer is that it is used for ORTHOSTATIC HYPOTENSION. It works by sucking back the Na and driving out K. The RENIN is reved up, restoring BP.

366. Some people said their test is this week and wanted me to keep em pumped up so I will try my best..pray please for physical strength so I can push forward. YOUR prayers are working because last hour I felt so tired but someone's prayer LIT me up and I felt strong again!

Q) Someone asked me about how Praziquantel works since we talked about it a lot with parasites... again the MOA...

Praziquantel (Biltricide) -- Parasite cyst murderer agent that destroys approximately 75% of cysts with a single course. Increases cell membrane permeability in worms, resulting in loss of intracellular calcium, massive contractions, and paralysis. Causes vacuolization and disintegration of the schistosome tegument, followed by attachment of phagocytes to the parasite and then the LAST RITES of the parasite, sorry parasite... bitter tabs though.

367. If I can have time, I will try to correspond with each of you that asked about personal study schedules. There are SO MANY SAMPLE schedules out there, but you have to tailor your own. However, you have to make it so the micro and anatomy are last because they are

169

the most easily forgotten. Physio should be first. You need breaks of course during the day, BUT short ones. I told all my SERIOUS students they need to study at least 10-14 hours per day for a 3 month period. They must have a scientific method to ASSESS their progress to know if they are being EFFICIENT. One student of mine studied one year and still she failed...the study plan was not efficient. Everyone is different though. When I have more time, I will try to answer each one of you.....

Q) ON MY PREVIOUS POST, I got a WINDOWS MESSAGE to clarify the name of a disease that causes orthostatic hypertension...

A) There are a lot of them, BUT the common category is AUTONOMIC FAILURE SYNDROMES like SHY DRAGER synd.

368. Someone asked about URINARY INCONTINENCE, definitely a HY subject..

YOU will see this all over the place during OB/GYN..

Q) What is MOA of Oxybutynin? What enzyme does it act on?

A) Oxybutynin (Ditropan) -- Useful for urinary incont. Inhibits action of ACh on smooth muscle and has direct antispasmodic effect on smooth muscle which in turn causes increase in bladder capacity and decrease in contractions.

170

369. Q) Was, where does Beclomethasone act?

A) It first binds to receptor in cytosol and is carried to nucleus to a ZINC FINGER DNA BINDING PROTEIN. TRANSACTIVATING STEROID RESPONSE GENES

GOOD QUESTION AND HUGE HYer!

370. Q) What is the function of Probenecid? What dx and MOA? SEs?

KNOW THIS ONE!

A) This blocks reabsorption of uric acid and enhances excretion. DON'T USE IN ACUTE gout but only for chronic gout. ...

This works on the PCT in kidney....

SEs are HY... can cause uric acid stoneS!!!

371. Q) Someone asked about Chorionic villi sampling. Def. HYer too...during week two, extensions of the cytotrophoblast cells called chorionic villi formed and projected into the syncytiotrophblast cell mass. During week 3, these villi enlarge and blood vessels grow into them, forming highly vascularized structures, completely surrounding the chorion. This intricate network of embryonic vessels is now close to

171

the syncytiotrophblast lacunae which are filled with maternal blood. This forms the placenta!

CHORIONIC VILLI TESTING cannot detect neural tube defects like the alpha feto protein test done later AT WEEK 16 AROUND....

YOU CAN DO THIS TEST EARLIER THAN AMNIOCENTESIS!

372. Q) Still on drugs...

We know Bleomycin blows out LUNGS (bad SE), what phase of cell cycle does it act in? Binds to what ion?

A) G2 phase, binds to Fe in oxidase and "cuts" DNA.

373. Here is YOUR answer:

Case: The famous drug Robitussin PM has a cough suppression agent called DEXTROMETHORPHAN. What is the MOA? What receptor?????

A) Dextromethorphan has shown agonist activity at the serotonergic transmission, inhibiting the reuptake of serotonin at synapses and causing potential serotonin syndrome, especially when used concomitantly with monoamine oxidase inhibitors (MAOIs). In addition, dextromethorphan and its primary metabolite, dextrorphan,

172

demonstrate anticonvulsant activity by antagonizing the action of glutamate, wow, an super HYer.

374. Q) What BAD side effect is involved with STATINS if given with Gemfibrozil?

A) Rhabdomyolysis!! Watch for it!

375. Q) Biggie question: What drug used for Candida topically works by the same MOA as Amp B?

A) Classic question: answer is Nystatin has same MOA....

376. HARD QUESTION but reviews your fungals...

q) For Cryptococcus meningitis, you used AMP B...what two other drugs starting with letters, FLU... completes the treatment...MOA too please??

A) Use Fluconazole and Flucytosine. All the -azoles work against ergosterol, but Flucytosine is an antimetabolite!!!

377. Q) I know you know that H2 blockers, antacids, and OMEPRAZOLE are good for GI diseases..

BUT, what drug is also used for peptic ulcer disease that CANNOT BE COADMINISTERED WITH H2 blockers????

A) SUCRALFATE: this protects peptic ulcer tissue and YOU CANNOT GIVE WITH ANTACIDS BECAUSE SUCRALFATE NEEDS ACIDIC ENVIRON.

378. I saw a drug called CYTOTEC for stomach ulcers given out all the time.. This is generic MISOPROSTOL.

Q) What group of people is there you CANNOT give to? Also give me MOA?

A) This prostaglandin E1 STIMULATES mucus production and..

YOU CANNOT GIVE TO PREGNANT WOMEN BECAUSE THEY MAY ABORT FR. CONTRACTIONS TO UTERUS!

379. Q) What two opioid agents are commonly used as an antidiarrheal? One of these "sounds" like Dextromethorphan, an anticough. MOA?

A) Diphenoxylate and Loperamide are OPIOIDS for diarrhea control. But stop GI contractions from mu and delta receptor activity!

380. You first learned in college that Vasopressin is ADH acting on V2 receptors in the COLLECTING DUCT!

Q) But what is MOA on V1 receptors and what is the dx it treats?

Q) Also, on V2 receptors, it also releases what two coag factors????

A1) On V1, it vasoconstricts the splanchnic bed. So, use for GI bleeds.

A2) VWF and Factor VIII !!! KNOW THAT!

381. Give me exact MOA of Daunorubicin and Doxorubicin? SE?

A) This acts on DNA and cuts it hence blocking DNA AND RNA syn. It works by intercalating and is cell cycle nonspecific!

SE is Cardio damage. "don't let Dawn break your heart"

382. What is MOA of Etoposide???

a) ANti-cancer...Works in S phase and binds DNA topoisomerase II, thus breaking and stopping DNA and RNA production!

383. HEY, I am thinking of antiarrhythmic that works via blocking Na channels for Class IC and raises depolarization threshold in PHASE 4, (Be able to identify changes in graphs). It is ONLY USED IN SERIOUS cases of V-TACH for people with a decent cardio function.

Who am I? (Starts with letter F)

Answer: Flecainide

384. In a famous movie, a doctor used Dopamine to aid a patient with

175

poor renal perfusion via the...

D1 receptor.

Tell me what other receptors are revved up if I increase the DOSE of DOPAMINE? very important...

Answer: First, the D1 is activated. Then the B1 is activated, then if keep increasing, the alpha-1 is activated!! Thus, cardio function and vasoconstriction occur...at higher doses! Can you graph it?

385. WE ALL KNOW WARFARIN IS CRUCIAL HY...

1) WHAT IS MOA, GIVE ME EVEN THE ENZYME!!!

2) WHAT KIND OF RXN (HYDROXY OR CARBOXY) DOES IT DO?

3) GIVE TO PREG WOMEN?

A1) THIS VIT K FACTOR blocks epoxide reductase! The famous Vit K is a cofactor in CARBOXYLATION REACTIONS!

A2) CARBOXY

A3) NO!!

386. What is MOA of Dipyridamole?

A) This ANTIplatelet works by revving up cAMP and blocking platelet adenosine uptake...it also decreases vascular tone so OPENS UP blood vessels?

387. Give me MOA and tx involved with Azathioprine!

A) This blocks purine conversions via IMP dehydrogenase block...is a purine analog used for KIDNEY TRANSPLANTATION!!!!!!!!!!!!!!!!!!!!!! Via immunosuppression!

388. Yeah, you know HYDRALAZINE...but tell me the second messengers???

Ans: This vasodilator can cause LUPUS but activates Guanylate cyclase...reving cGMP...causing hyperpolarization!!!!

WOW!

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389. I am thinking of a class IV antiarrhythmic like Diltiazem. What channels, exactly,...do they both work on?

A) L type Calcium Channels. Stress the "L". This slows conduction at the AV node.....oh, the drug is VERAPAMIL....

390. Tamoxifen, a breast cancer ANTIESTROGEN, revs up which hypothalamic hormone?

ans: GnRH

391. Which cell cycle pt does Vincristine act on? What dx? What bad SE? What protein does it bind? What is mech of resistance?

A) METAPHASE; for leukemias/lymphomas; peripheral neuropathy along with the others like hair loss,etc. ; it binds a protein called TUBULIN. The mech of resistance is tubulin gene amplification!!!

Are you getting all of this????

392. Case: A patient of yours is on antiretrovirals....AZT is not working,

YOU SHOULD ADD ON ddC or ddl.

Q) What is organ affected in SEs of ddC or ddl?

A) Pancreas...and you may face neuropathies...

393. Case: You have a pt on cancer chemo. She complains of nausea and you give... name at least two antiemetics for cancer rxns... and what receptors do they act on?

Answer: Tricky, I am not thinking of the opioid drugs. The ones usually used are the anti dopaminergic ones...that block the reticular formation in the medulla brain stem slide. Two eg's are Metoclopramide and Droperidol!!!!

394. OTHER than nitroprusside for HTN emergencies, you can use DIAZOXIDE, its MOA is thru ATP and K channel activation...which as you know, when K channel open there is hyperpolarization...etc. relaxation of vessels...vessels open up....etc. What is this drug's SE?

Ans: Hyperglycemia in Diabetics via B cell inhibition in pancreas!

395. What is the dx and MOA related to cytarabine?

178

ans) This ANTIMETABOLITE acts during the S phase and incorporates ArabineC into DNA. RNA growth continues and leads to IMBALANCE, and stops growth mostly used for cases of AML.

396. KNOW ALL ABOUT digitalis...it WILL BE ASKED. KNOW MOA...? What patient condition is contraindicated?

A) Binds Na/K/ATPase pump...Ca revs up...increased contractility...AV nodal velocity goes down...

DON'T GIVE TO HYPOKALEMICS! AND THOSE ON CLASS I ANTIARRHYTHMICS...

397. SO KEY:

What is MOA of 5-FU?

Answer: 5-Fluorouracil works in S phase and is converted to 5-FdUMP. This now blocks thymideylate syn, blocking DNA syn, so there is loss of balance as RNA and protein go up....thymine is LOST.

398. What receptor does IPRATROPIUM work on? What dx?

A) This asthma drug is SO common and blocks ONLY the M3 muscarinic receptors in the lungs (b/c it is inhaled).

399. Everyone needs to know birth control...etc.

What is MOA of progesterones for birth control and what three conditions is it commonly used for?

answer: In the nucleus, it binds zinc finger binding protein and lowers GnRH, and the LH and FSH surge.

You give to DUB pts with too much estrogen secretion, endometriosis, and fibrocystic change, along with the birth control reasons!

400. For the USMLE STEP 1, you must go beyond things like, "streptokinase is used for tx of thrombosis." SO, what is the exact, and I mean exact, MOA of streptokinase??? Can you point to where it acts if I show you a coagulation cascade diagram? These are original questions but are EXACTLY the LEVEL you need to PASS the MONSTER EXAM..

Ans: This streptokinase binds plasminogen, activating its active site, thus revving up plasmin which then busts up clots and factors V and factors VIII.

WHEW!!

401

I am so happy Step 1 came in. As I mentioned, I am tutoring a student who happened to live in my hometown since I gave out my email to a few people who told me they LIVE IN MY US CITY! So I am tutoring him because he is panicking. I am NOT CHARGING ANY money so do you guys worry. Please say a quick prayer that he passes!

Q) What is the Shilling test used for?

A) So KEY!. We use it to identify pernicious anemia by giving a pt. vit

180

B12 and seeing if intrinsic factor is present!

---

402

This was a concept that someone e-mailed me that I think I incorrectly responded to:

Q) If you inhibit aldosterone release thru penicillamine,

a) then you will decrease RENIN levels. (choices she gave included Ang 1, Ang ii, etc.)

---

403

The NBME will ask a lot of questions about serum electrolytes and DIURETICS. This is come up over and over and over again!

LISTEN, for FHA (Acronym Federal Housing Authority). (which stands for Furosemide, HCTZ, and Acetazolamide), YOU LOSE K (cash) FROM THE SERUM!!!!!!!!!!!! (Hypokalemia results)  
(Think of mnemonic, if the FHA comes, you likely have no Kash!)

LISTEN, if your patient wants a H (HI)- Fidelity (F) stereo, he has to BUY IT UP (B=Bi-carbonate) (Hydrochlorothiazide and Furosemide stands for the H and F), then you must BUY IT UP! ("Buy" sounds like Bicarbonate, and BUY IT UP means the "Buy"carbonate HCO<sub>3</sub> levels in your blood will increase!) (LINK:metabolic alkalosis)

LISTEN, all Diuretics lower Magnesium, so THINK that if you sit down to pee (diuretic), you will also have a big MASSIVE GLOB of poop. The M in Massive and the G in Glob of poop stands for Mg coming out. (hypomagnesium)

181

LISTEN, ALL Diuretics raise uric acid in serum, lowering it in the renal tubules. This mnemonic is easy...DIURETIC SOUNDS LIKE URIC ACID both have "UR". Say it fast. then you will connect! And Diu"r"etic has "r" for raise in serum. (This is of clinical importance as Thiazide Diuretics are used to treat uric acid stones because while serum levels stay high, renal tubule levels remain low!)

LISTEN, the acronym ASA stands for aspirin, and aspirin Overdose is acidic. So the A (Amiloride), the S (Spironolactone), and A (Acetazolamide) . An aspirin Overdose gives you metabolic acidosis!!!!!!

Also KNOW the connection that the K sparing diuretics STAYS in serum. (S=spironolactone, T-triamterene, A-amiloride) (Usually, I found it helpful to recall that H and K stay together usually...) HCO<sub>3</sub> leaves in the urine...

LISTEN, for Calcium ions it is tougher. But this works for me. I think of a S-Ca-fFold falling down a building. (You know, for painters). So, Calcium administration makes S (Spironolactone) and F (Furosemide) "fall" out of your urine! See the Ca in word S-Ca-Ffold!!!! (This is clinically important because LASIX or furosemide is given to CHF patients every second which predisposes them to Ca stones in kidney!

REPEAT THIS HIGH YIELD CONCEPTS UNTIL IT STICKS!!!!!! BEcause you will likely be asked this on your test because of the relation to renal stones....Ca for e.g. causes stones. Hypokalemia, Hyperacidosis...And this is SO IMPORTANT, you must repeat my mnemonics over and over.

They worked for a lot of people! And I spent a long time making up these memory mnemonics. Don't let them go to waste! Someone told me my mnemonics saved them! Thanks but please they are for everyone and they are useless unless you repeat them.

Please e-mail me if you like the mnemonics. If they are bothersome, I will stop submitting them!!!!

---

404

I am getting so much mail and I love them but my ValueMD mailbox is not letting me reply.. I just want to say to some of you that I would LOVE to visit your HOMECOUNTRY if I go overseas! And I am doing fine thanks to your prayers! And please pray that the student next to me that I am tutoring for STEP 1 (hence my absence today) will pass this Saturday! He is crying and desperate. I am serious...

OK, while he is doing one of my exercise drills...

Q) Important point: NBME wants you to know the difference between procaine and meprivacaine. What is it?????

A) PROCAINE and LIDOCAINE is an ESTER based local anesthetic like the Novocain your dentist uses!

AND, MEPIVACINE sounds so similar but it is an AMIDE based local anesthetics.

This is so important because the esters are shorter in action!!!!

---

405

Step 1 is right, the NBME WILL WANT YOU TO understand ALL second messengers because all their questions are secondaries, tertiaries, tetraaries (spelling is wrong), etc. I cannot stress enough this POST...

SO, LISTEN VERY CAREFULLY:

For the second messengers, you must have ORDER to remember

183

subtypes and the sub-subtypes which the NBME will definitely ask you:

(1 subtype) Repeat this story, You, say you are a male, takes out on a date a beautiful girl with an "A1" great BIDI ("Body" which is stimulating for you) (Receptor A1, B1, and D1 are stim in B1,D1.). Or you can think acronym one 1 BAD date with a lovely lady. But recall stimulatory.

(2 subtype) Then you both are so hungry, hunger stimulation, so you both order H 2 Hamburgers, so receptor H2 is stimulatory! BUT, all have exceptions! You open your handheld Palm Pilot to write her phone number down and note it is powered by an AMD processor (a company that INHIBITS the dominance of Intel Corp) (The Acronym AMD is A2, M2 (found in the heart), D2 which are all INHIBITORY). OR...think you are 2 M.A.D. because you forgot your best necktie.

(3 subtype) Now, you take her to see the movie "Matrix 3" (M3 receptor) and you hold her hand during the movies and because you are nervous, your hand sweat glands are stimulated! M3 receptors in sweat glands are stimulatory when activated! (MOISTEE..sounds like M3)

NOW THAT WE KNOW WHICH ARE STIMULATORY AND INHIBITORY:

REMEMBER: 1) All the subtype 1s (like alpha 1, M1, V1, H1, etc.) are STIMULATORY and work via the Phospholipase C, PIP, IP3, DAG, Ca, Protein Kinase C. Try to link the word ONE-C or say C-ONE. EXCEPT for acronym BIDI or sounds like BODY. The B1 and D1 (Beta one and Dopamine 1) are stimulate by ATP, cAMP, Protein Kinase A! (Think all these messengers have the letter "A" in it for the expression "A" great "BIDI" (body). Recall the letter "A" stands for Protein Kinase A.

2) NOW, LUMP the subtype 2s: (like alpha 2, muscarinic 2, dopamine 2). Activation, here result in INHIBITORY via adenylyclase, PotassiumK, cAMP downreg. and Protein kinase A downreg.. EXCEPTION...here is H2 in the stomach which is stimulatory but STILL involves Protein Kinase A. Think of eating 2 hamburgers because your hunger is so stimulated! But, do not confuse that most of the 2s are MAD (M2, A2, D2)!

3) NOW, LUMP the subtype 3s: (which there is only one you need to worry about which is M3. These attached messengers are PIP, IP3, DAG, Ca, and Protein Kinase C. Think you did "see" or "C" the movie, right?

BIG HINT: For the 1st and 3rd messengers, think "generally" the "1" and "3" subtypes like alpha 1 are connected to PIP, IP3, Ca or Protein Kinase C. "13C"

For the 2nd messenger, which think is in the MIDDLE of the pack, it is adenylyclase, ATP, cAMP and Protein kinase A (which ALL have the letter "A" in them. "2A"

It sounds hard, but if you say them aloud a few times, you WILL remember. ONE of my students said my original mnemonic was worth its weight in gold. I don't know about that, but THANKS TO HIM. Don't worry, I will ALWAYS stay HUMBLE!!!! love, tommyk

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Oh, just so I know, are these mnemonics helping? Please let me know because if they are not helping, then it is a waste of time for you to read them!

---

406

Case: Another movie with a cousin of Brandon Lee in the movie "The

185

Crow". He is shot by a fake bullet, but is bleeding heavily. What drug that is a LYSINE analog do you grab to try to save his life???????? (starts with letter A)? MOA OF COURSE?

Answer: Aminocaproic Acid which thrombolyses clots to stop the bleeding. It binds and inactivates PLASMIN from binding FIBRIN!

407

Case: Two patients of your walks in. Mr. Brown cannot pee. Mr. White has myasthenia gravis. What drug (starting with letters NEO do you use and its MOA?

(I really did like the movie Matrix as you can tell)

Answer: You grab Neostigmine!!! An inhibitor of enzyme cholinesterase! (This of course, boosts ACh is the system!)

---

408

Q) What don't aminoglycosides work on anaerobes?

A) Because aminoglycosides need O<sub>2</sub> dep. transport and anaerobes don't have these.

**BIG KEY POINT OF RESISTENCE!**

---

409

Q) For all young women with HTN, you learn in clinic you always ask if they are on birth control....big point....

NOW, what contrib. does estrogen play to stop ovulation?

A) In the hypothalamus, the estrogen receptors are overwhelmed, so FSH drops!, thus, helping to stop ovulation!

186

REMEMBER, DON'T GIVE ESTROGEN TO HTN PATIENTS OR YOU WILL BE SUED!

---

410

IMPORTANT:

What is MOA of Clomiphene citrate?

A) This infertility drug works b/c as a WEAK antiestrogen, it weakly counteracts GnRH at the hypothalamus, so FSH and LH rev up and ovulation occurs. Key is it is a WEAK estrogen!

---

411

You know that classic pressure curve diagram and the BIG diagram with EVERYTHING like EKG, Ventricular Volume, Heart Sounds, etc. etc.

I guarantee everyone will face this on their test. But more important, realize that some mentor told me that at least 10,000 questions can be asked because it is SO diverse. The concept is not that hard, but see if you can draw them from scratch (where the S1 is, where isovolumetric contraction is, where atrial pressure is lowest, etc. IT IS ENDLESS)

---

412

Remember, this is a NBME favorite:

Odds ratio is quickly calculated as  $OR = ad/bc$

AND Case Control studies = Odds ratio

AND Cohort Studies = Relative risk

(Think: "This Case is ODD to make the relation that Case control is Odds Ratio" AND think that a "Cohort" is a grp of people starting together and people's personalities are all RELATIVE."

---

quickly though,

give me the difference and point to a histo slide of:

a) oligodendroglia

b) Schwann cell

ANSWER: Both Myelinate, but Oligos =CNS axons and Schwann=PNS axons.

BIG POINT that is often asked in relation to tumors....

Think of Schwann or Swans flying off...to the periphery....

---

413

For the embryo arch problems (one of my students got a whole bunch of them, see you can never tell)...

Just remember that Branchial arch 1= Ms (Masseter, mangle, etc.) and has nerve CN V3 "IV3 rhymes"

Just recall that Branchial arch 2 has a lot of Ss in it (Stapedius, Stapes, etc.) and has nerve CN VII "The Roman numeral VII has "two" II it it"

Just recall that Branchial arch 3 has pharyngeal stuff and CN IX "3P9" rhymes.

188

Just recall that Branchial arch 4 and 6 have an "levator in the larynx" (levator veli palatini and larynx muscles) and CN X. "Think of the expression, For Sex" (4=sex and "s" is first letter of sex) {But I personally do not advocate sex before marriage, I just felt I had to put that point in}

"Another hint for the order are the odd numbers til ten"= CN V, III, VII, IX, X

for arches 1 to 4/6. See CN five has the 3 branch. Just go in order....

---

414

For the infamous Pharyngeal pouches,

just think... you know there are 4 pouches:

1=M iddle ear

2=P alatine tonsil

3=T hymus

4=S uperior parathyroids

"Think the acronym MPTS or Many People Throw Stones, then work your way down the head anatomy from ear down to parathyroids"

(It gets more complicated of course, but this should help a lot")

---

415

Good one:

BIGGIE:

Case: Pt with infertility and Urinary tract problems. There was an

189

incomplete fusion of the paramesonephric ducts. What is the dx?

Answer: Bicornuate UTERUS

---

416

To recall the all important lower injuries,

"Think of actor Johnny DEP falling into a PIT" (I know, Dep is spelling w/ 2ps, but still" It is easy to remember cause ALL of girlfriends think he is best looking guy around.

DEP= Dorsiflexion and Eversion is Peroneal (Common Peroneal)  
Then, for the essential levels which they will ask, "Think, Johnny Dep is  
So good 2 Look 4" hence L4-S2 injury. (See, the S in So and then the  
2, then the L in Look then the number 4)

PIT= Plantarflexes, Tibial nerve Inverts. Think, "If I L ook 4 hiim in the  
PIT and find him, I will be So 3-illed" (in other words, If I look for him  
in the pit and find him, I will be so thrilled) (This completes the  
association with L4-S3 nerve roots)

---

417

Bold CASE:

A patient of yours named David comes in and cannot move his hips  
and there is no knee reflex. What cords are damaged?????

Ans: L2-L4 "Think, you have to link stuff to save memory brain space

190

so think this: David is in PEDiful (pitiful) shape. Since we linked PED with Lr and L2 already, the association produces the answer L2-L4 are lesioned.

---

418

YOU MUST KNOW BASIC ANATOMY like the mastication.

So you must associate V3 with the Masseter muscles which is easily because the mastication muscles all start with letter "M".

HERE, I think always of eating since we are dealing with mastication....so I say MMM...I'm Very 3-illled to eat (MMM are the masseter muscles and Very is V and 3 for thrilled) See? If you think it, it will work!!!!

---

419

YOU HAVE TO KNOW THE HYPOTHALAMUS COLD!!! Everyone has a version because the hypothalamus is SO VITAL! HERE goes...

This is from my neuro teacher and myself!

You will be asked to differentiate the anterior and posterior hypothalamus:

- 1) "So think A for anterior hypothalamus is A for autonomic regulation"
- 2) "If you get spanked on your POSTERIOR, you will get SYMPATHY" [posterior is sympathetic]
- 3) The SEPTATE nucleus is SEX urges. "They both only start with "S" "
- 4) The suprachiasmatic nucleus controls the Circadian rhythms. "For this, I think of SUPERMAN (suprachiasmatic) CIRCLING (Circadian) the globe!
- 5) The ventromedial nucleus controls appetite. So, this one is easy..I

191

think "I am VeryMuch Hungry" V-Ventro, M-Medial

6) You know Oxytocin and ADH comes from Neurohypophysis from college biology, so no student ever asked me for a mnemonic but you can remember the name NOAh for association.

7) The Supraoptic nucleus controls thirst. So I think that Supra Optic sounds like Super Openorange juice, which makes me thirsty.

---

420

YOU HAVE TO PICK IN A DIAGRAM THE LESION IN THE BASAL GANGLIA

1) OF HUNTINGTON'S

2) Parkinson's

3) Hemiballismus

4) Wilson's

A1) If you HUNT, you must shoot STRIat (straight) = Striatum

A2) Lesion in compacta nigra (I Parked a Compact Car)

A3) Subthalamas

A4) Wilson's = He Wil (Wilson's) go STRaight (Striatum) around the Globe (Globus Pallidus)

---

421.

Dear Family,

Again, the below is not copyrighted and is original, but it will seem like an actual USMLE step 1 case you will see because it resembles the format of what they feel is important for you to know. Please study it because you will face the same format and "feel" again and again in similar concepts:

Case: A pediatric patient of yours named Valentine comes in with vague presentations involving sweating, poor feeding, respiratory issues, malaise, tantrums, trembling, confusion at times. History shows the following: Valentine has on your physical hepatomegaly, hyperlipidemia, and growth retardation, and his sugars are low. (NOW, NBME USMLE STEP 1 cases can be VERY VERY LONG...do you know what this child has? If not, I need to give more information....) More labs come back and you note that there is glycogen filling up in the body's cells. Obtain a lipid profile. Modest elevations in very low-density lipoprotein cholesterol and triglyceride levels sometimes occur. Evaluate blood and urine for ketones, especially after a brief fast. Fasting ketosis is prominent. (NOW, I believe here a well-prepared student should tell me the diagnosis...but remember, the USMLE is about secondaries to the disease, so here is the diagnosis and the relevant secondaries that are within the NBME sphere of focus)

Answers I am looking for:

- 1) The disease is of course Cori's disease, a glycogen storage disease. (Incidentally, the husband and wife team won the Nobel Prize for their work)
- 2) If I asked what TYPE it is, you should tell me TYPE III. Don't confuse it with TYPE I or the others. There ARE clinical differences....
- 3) If I asked if gluconeogenesis is impaired, you should tell me that it is NOT IMPAIRED...a very common student mistake)

193

- 4) If I asked you the MOA, you should tell me that the debranching enzyme is deficient. REPEAT, DEBRANCHING ENZYME DEF.
- 5) If I asked you the enzyme itself which is a favorite on the NBME, you should tell me it is alpha 1,6 glucosidase (NOT beta 1,6 glucosidase, NOT alpha 1,4 glucosidase, NOT gamma 1,6 glucosidase, etc. which can be all in the answer choices)

Again the above is 100% made up by me and is property of ValueMD and even though it LOOKS like an actual NBME case, it is an original presentation and not a recall. But, I would not be surprised if it exists somewhere in the vaults of the NBME's sphere of focus. This is just what I feel is a VERY VERY illustrative example of a "model" NBME USMLE Step 1 question. I feel I need to say this so that you all do not think I am violating copyright infringement, but rather educating in my own legal way.

Because the NBME also stresses PICTURES and TREATMENT and or DRUG, you should also know what the patient will present as and how you will treat them. THEN you may be asked what are the SIDE EFFECTS of the treatment and the long term consequences.

IT looks impossible, but it is just like remembering your aunt's birthday...except of course you have like one million aunts. YOU can do it, my professional memory studies show most everyone can, but AT DIFFERENT SPEEDS. And you MUST HAVE THE RIGHT CONDITIONS AS I EXPLAINED BEFORE (NO 2 hour study days with the TV on, etc.)

I broke my own rule of putting my concepts out of order by clicking on them via replies, but I HAD to add that this case is almost exactly the format and content of what NBME wants you to master.

I am writing this because someone asked me if this was too much detail because it moves past [FA](#). While I agree [FA](#) is excellent, you must go beyond it...

Love, Tommy

422.

This is not a case like the previous one but I think it is just as important to say:

- 1) You must understand how "to study" such vast material.
- 2) This is unlike recall only a pretty girl's telephone number. You must learn the material in LUMPS, so that is why my HYers are lumped. Again, to know what is purple, you must know what looks close..so you must know what black, blue, and deep green look like...
- 3) Repeat the information in GROUP in pre defined intervals.
- 4) KNOW the NBME wants you to get the COMPLETE picture. Anything goes.

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423.

A reader just emailed me something that I MUST INCLUDE:

- 1) KNOW that the typical words presentations are being eliminated. For instance, for the dx POLYARTERITIS NODOSA, know that the words "cotton wool" spots will NOT be given, but the words will be described in detail in other less obvious or unique words.

2) Thus, buzzwords are becoming LESS AND LESS important. Ten years ago, when the USMLE STEP1 was a two day 700-750 question test, there were a lot of ONE LINERS, so BUZZWORDS WERE in vogue and used a lot. NOW, times have changed.

3) NOW, understand that VARIETY is flowing into the USMLE STEP 1. That means more diagrams, MRIs, pictures, photographs, CTs, flowcharts, biochemical pathways, and variable answer choices (not just A-E). My sources tell me the test is starting to "become alive" in a way.

424.

Sorry, this is not a case question but know this...

And I do not think the NBME will mind me telling you this...

USMLE STEP 2 materials are appearing in STEP 1!!!!!!!!!!!!!! Many many have told me that they had questions asking what is the best NEXT STEP in management of the patient. They could be theoretical questions...but really who knows

425.

I just received another notice..seriously....from a student who took the STEP 1 and told me to rely this info to you.

Thus, I am making it a "concept" because it is so important...

The student came in the testing room and she was seated right next to the door. During one of the sections, there was talking outside by someone in the waiting area. Since she was SO SO nervous, it really hurt her, although she could not tell me if it messed up her questions, but she did think it may be slowed her down so she had to guess on the last question.

Q) So, what can you do about this?

A) Make sure they offer and make sure you wear your earplugs. I have heard of some students who are seated by the door that asked to be moved away and were granted their wish, but I am not sure about this because I have no proof. Don't underestimate this concept because if you are nervous, HAVE no earplugs, are seated next to the door and freaked out, that could be VERY BAD.

426.

Someone just keeps telling me test center advice...(I am going through my emails too).

BUT I AM INCLUDING THIS ONE BECAUSE I FEEL IT NEEDS TO BE A CONCEPT...

Bring two government IDs which your exact spelling on it. One of the students was PULLED OVER BY THE POLICE on the way to her testing center (maybe she did not know the way and made an illegal u turn or maybe she was speeding). But...listen...if you get caught by the police in your vehicle for speeding or something...in MANY of the USA states they WILL TAKE YOUR LICENSE ON THE SPOT, it is policy...the person who told me this lives in USA Chicago ILLINOIS. BE careful, but bring TWO government picture IDs. NBME centers will not let you take the test if you don't have pic ID! Then you will wail out in agony!!!!!!!!!!!!!!

427.

Quickie case because people often get this confused!

LISTEN...the marker for Wegener's granulomatosis is C-ANCA. The marker for Polyarteritis nodosa is P-ANCA.

This IS a crucial fact even though it seems so small!!!!

428.

I don't think this is copyright infringement b/c I am describing what the NBME is asking you to understand, but here it is:

1) KNOW at least the very very simple basics of general chemistry and physics. Two of my students got these questions.

I cannot repeat the exact ones so I won't break copyright, but it had to do the delta G Energy stuff and enzymes for General Chemistry; and the PHYSICS questions had to do with LUNGS, gases kinetics; and another PHYSICS question had to do with flow equations and BLOOD VESSELS(remember the liquids and solids equations?)

429.

While we are on the subject, just for completion, I MUST SAY THIS:

ONE OF MY STUDENT TOLD ME HIS COMPUTER MALFUNCTIONED DURING HIS TEST! during his personal test day!!!

He said that he came out and the proctor told him that this never happened before on his watch, and then told him to call a place called CANDIDATE CARE and gave him a card.

I have NO IDEA what the computer problem was, or even if the student's problem was legitimate but I left the need to tell you what to do if such a crazy thing happens.

I was told that he was informed that he may get another testing day later, of course though he will not be charged as such.

430.

198

I learned this is VERY CRUCIAL:

Case: You open your door and in comes a guy like Beavis who asks you, a pathologist, to identify a bug on a slide from a patient with respiratory symptoms:

YOU see fungi appearing organisms. They have long branching filaments/rodlike structures under microscope. You are thinking MUCOR, but Gram stain produces gram positive rods.

Q) What bug is it? But the NBME will likely want you to tell the difference between it and another related morphological bug that is on the skin.

A) You are looking at NOCARDIA, an acid fast aerobe you can catch walking barefooted in soil. You can get respiratory symptoms.

A2) Now the secondary is the bug ACTINOMYCES, a VERY common bug growing on your skin. It can infect the sinuses and is a gram positive anaerobe forming sulfa granules.

WOW, see how interesting this is?

431.

ALL NBME questions are noted by their test takers as being so VARIED...that is because of the sheer number of questions that they have and the number of people writing the questions.

SO, some of the questions will be very direct and SHORT.

AND others will be so LONG that you have to SCROLL down to read the entire QUESTION! Some questions I was told seconds to answer, some took almost 6-7 minutes due to 20 different lab results they had to study!

That said, here is one:

ORIGINAL VALUE-MD CASE: A 16 year old patient named Thelma comes into your clinic with burns all over her body (1st and 2nd degree). After discussing with the family to rule out abuse (which you are required by law to do), you must start with what?

Ans1) IV fluids

Q2) Then, you note that her tetanus booster is two years ago, so what do you do?

A2) You do NOT have to give a booster, the time interval is 5 years...

Q3) What two main bugs if asked are you worried about initially?

A3) Pseudomonas of course and also Staphylococcus Aureus. (You must smell the wound site, if it smells "fruity" like grapes, Psudomonas is more likely).

Q4) So, the NBME and attending asks you what meds will you grab?

A4) You must grab 3 types: Morphine, NSAIDS, and an Antibiotic

Q5) Grab MORPHINE SULFATE FOR THE PAIN. But what do you be aware of before administering it?

A5) Ask her if she is taking MAOs for depression. Also, ask her about hypersensitivity and if she is pregnant because the respiratory

200

DEPRESSION may hurt the baby.

Q6) After NSAIDS (no questions here) what two antibiotics are recommended typically today in the USA in this case (AND YOU MAY HAVE OTHER CHOICES ON THE TEST AND IN LIFE--this is ONLY A GUIDE BUT A USEFUL ONE)?

A6) Grab Silver Sulfadiazine and Neomycin. They should be good coverage. BUT, they are NOT the ONLY choices...so if on your test or in life you don't HAVE those choices, just pick the ones with Staph and anaerobic coverage!

See ya!

432.

This post has both answers to reader questions and a case:

Q) Why is [FA](#) not good enough for everything? And why is the students' mental processes in error? And what can be done?

A) Consider a classic case and question I posed to a group of my students: I asked, "IF YOU ARE ASKED ABOUT LESCH NYHAN DISEASE WHICH IS A NBME FAVORITE, TELL ME ALL YOU KNOW?" I presented a case with a boy in his teens with A HISTORY OF OCCASIONALLY FLANK PAIN!

Their response was the EXACT WORDING, "Self mutilation, Nail biting, Retardation." That was it. Then after waiting, one of them said, "HGPRT".

OK, but the NBME knows most med students are the best of the best and will know these 3 bits of info so you will likely NEVER see them. I remember one student told me that NBME presented the disease with

201

a presentation that was close but not in those EXACT WORDS, (like nail biting). So, my QUESTION TO YOU is the following:

Q2) What is causing the flank pain?

A2) Kidney stones from excessive URIC ACID (that can be another question!)

Q3) You find out that he is on a thiazide diuretic medication for these kidney stones and a uricosuric called probenecid because his last doctor thought/heard that thiazide diuretic treat kidney stones and since uric acid is the problem, he gave him PROBENECID? Do you agree with his last doctor (hint: he was an inexperienced sub intern)?

A3) NO, he was wrong, the thiazides are contraindicated from Lesch Nyhan pts. and the uricosuric will only make stone formation WORSE.

Q4) Therefore, what do you change his meds to? And MOA of new drug please?

A4) He needs Allopurinol.

Q5) What ENZYME does allopurinol inhibit?

A5) It inhibits XANTHINE OXIDASE.

Q6) The NBME and USMLE give all the above in a case and then ask if

202

anything else you should give him for prophylaxis that is NOT a prescribed drug? (VERY IMPORTANT)

A6) You MUST give him hydration.

Q7) If I gave you a picture of the brain on MRI with arrows, which structure is affected in this dx?

A7) Pick the arrow pointing to the basal ganglia.

Q The NBME and Attendings LOVE to asks this in mult choice form: What is the genetics behind Lesch Nyhan?

A X linked recessive, so you mostly always see it in MALES. (although theoretically possibly presented in females which it has been reported, the NBME will not ask a bizarre EXCEPTION)

PLEASE PLEASE, use this and others as a MODEL for what you NEED TO KNOW. The one step questions like "What is the capital of New Jersey, USA?" Answer: "Trenton". They are GONE! (naturally, you won't be asked USA geography...but you need to get the concepts and THEORY)

Please keep asking me about BUZZWORDS. There are being slowly ELMINATED. IT does not mean you should forget all of them, because they may present the buzzword in OTHER COMMON words. But know this fact while you study!!

Did that answer your questions? (I am addressing my email question readers)

203

433.

Q) Key concept: An accident victim comes to your clinic named Louise. She has a hemoglobin level of 9. Your attending asks you if you will IMMEDIATELY transfuse. She is alert and oriented times 3.

A) NO, you transfuse usually in clinics (and boards) if the patient is showing clinical signs. Even if her Hemoglobin is low. BUT, that said, if her hemoglobin was under 7 (remember that number), then pick transfusion. I know I would....what do you guys think?

434.

Q) Case: This is a tough tough subject but a HY one: You have an older patient named Robert who comes to you with mild depression and dementia. (BE CAREFUL, DISTRACTORS ARE ALZHEIMER'S, etc.). But I tell you that during PE, I touched his facial nerve and it twitched. And his PE reveals some muscle spasms (tetany). HE also presents with mild KIDNEY disease....

So if I ask what mineral(mineral, specifically) is deficient which is specifically related to his tetany and presentation, which one will you PICK? What dx? (HINT: this is not dementia)

A) Calcium is deficient. Think of the link with the kidney and its regulation with Vit D which is needed for Ca. I saw this exact CASE during one of my on call nights!

Q2) What typical sign is found on ECG which confirms your suspicion?

A2) The QT interval is lengthened. This is CLASSIC..

(Again, CONCENTRATE ON THE FORMAT, of the above case and

204

secondaries. These are NOT from [Kaplan](#) or NMS or big publishing house. They are from solely my experience as a teacher which I FIRMLY believe are better suited for you for STEP 1 and the clinics, because they do not go TOO light or TOO deep into the material...like the story of Goldilocks and the BEARS, the soup is just right. NOW STUDY STUDY STUDY STUDY, until you collapse!! Do it NOT for yourself, but for your future PATIENTS WHO NEED YOU!

435.

BIG POINT:

Case: A female pt of yours named Wilma comes in with vaginal bleeding with red lumps of cherries that are coming from her vagina. She believes she is pregnant from high HCG. BUT...I know you are NOT going to choose "abortion" as a choice because I am telling you that there are weird size and date assesment problems in history... BUT, if you need more...the NBME and attendings will tell you that there is a BUZZWORD...a snow storm pattern on ultrasound and no fetus.

Q) NOW, you should tell me the dx, (IF you guessed it before the buzzword then you are doing great!)

Q2) Tell me the karyotype IF the mother's chromosomes contributed. HARD, but definitely NBME wants you to know.

Q3) What condition does she have PRIOR to her third trimester involving her BP?

Q4) Treatment Rx?

Q5) What dx can happen if you don't treat?

205

Q6) What enzyme does the drug I asked you for (which starts with the letter "M") act on?

A1) Hydatiform MOLE if only the father's genes came on board. It is called an incomplete mole if the mother contributes her genes and you will see fetal parts...

A2) 69 XXY, be careful, I asked you about the mom so this question addressed an INcomplete mole, not a hydatiform mole which is 46 XX...which only involved the father..

A3) Her BP is very high which is called "pre eclampsia". Which YOU MUST address promptly. If she is of right gestation, you must deliver... (This is a concept and question by ITSELF!!!!)

A4) Give methotrexate and monitor HCG after delivery until it goes to zero.

A5) Choriocarcinoma or INVASIVE MOLE!

A6) Methotrexate acts as you recall on my previous posts acts in the SYNTHESIS PHASE of the cell cycle and block DHFR or dihydrofolate reductase.

AGAIN, TO ADDRESS A READER QUESTION, PREVIOUS TEACHERS OR EXAMINEES ARE THE VERY BEST SOURCE OF QUESTIONS OF CONCEPTS FOR THE USMLE STEP 1. It takes a lot more work (I think I spent about 600-700 hours already), but YOU EXPERIENCED ONES ARE IN GREAT POSITION TO WRITE THE BEST POSSIBLE QUESTIONS SINCE YOU KNOW WHAT THE NBME NEEDS YOU TO MASTER, PLUS YOU HAVE CLINIC EXPERIENCE FROM ROTATIONS, and STEP 1

ADDRESSES A LOT OF 3rd YEAR CLINIC STUFF! JUST DON'T VIOLATE COPYRIGHT AND REPEAT EXACT QUESTIONS...think of the concepts and make up your OWN UNIQUE QUESTIONS, then the NBME will be HAPPY with our attempts!

436.

Copyrighted Original ValueMD Case:

Case: A patient walks into your clinic named Bruce and is a farmer's helper living in Indiana. He is asymptomatic but has an radiograph with a coin lesion (1 cm sized) that is calcified on a upper lung lobe. The lesion has not grown in 18 months (from his chart), and he has no PE symptoms otherwise. He is otherwise obeying HEALTHY habits (no drug use, smokies)

Q) What is the dx?

A) Because he lives in the Midwest USA, he likely has a benign granuloma from histoplasmosis since he also works on a farm. Since the lesion has not grown in 18 months, it is mostly likely NOT LUNG CANCER WHICH MUST BE RULED OUT AND YOUR ATTENDING WILL KNOCK YOU SILLY IF YOU MISS THIS AND HE the patient...DIES. The attending will lose his house, his car, and his friends.

Again, the clues that the lesion is only one cm. Second he has good health habits. Third, the lesion has not growth in 18 months and he has no other symptoms which pushes your thinking into a benign HISTO Ca lesion..

437.

Here we go again, today is Sunday, and church and prayer day. Now that I can take a quick break away from praying, here is a question:'

207

ValueMD copyright case: You are on a plane bound for Los Angeles to do a lung transplant. Sitting in the middle seat, you have two passengers sitting next to you. The man on the left Bob, excited he is sitting next to a budding doctor, asked you a couple of questions:

Q) "I just took these drug called Edrophonium because my IM doc wanted to see if I had a disease...I cannot recall the name, what is it?"

A1) This short lasting drug is used for diagnosis of myasthenia gravis.

Q2) Then Bob asks, "I ran out of meds and my friend gave me a drug called Bethanechol." He said it should work the exact same for my dx MOA. Is that true?

A2) NO! Bethanechol is ALSO a cholinomimetic, but HAS a different MOA. It is used often in OB patients for urinary retention, and it is a direct muscarinic agonist. His drug, Edrophonium is a CHOLINESTERASE INHIBITOR, and thus works indirectly by keeping ACh in the junction longer...

Q3) He pulls out a drug pharmacy box with a drug called Neostigmine, which his IM doc gave him. He then asked you the MOA exactly?

A3) This, like Edrophium, is a cholinesterase inhibitor. But it lasts longer so it is used for myasthenia gravis chronically. Its MOA is that it CARBAMYLATES cholinesterase at the NMJ, and causes the cholinesterase to stay inactive to HYDRATION RXNS.

Q4) Then Bob asks you what would happen to his AV node in his heart

208

in case he took the entire bottle by mistake?

A4) This class of drugs will have THE PARASYMPATHETIC EFFECTS at high doses. Remember the M2 receptors in the heart? (THEY WORK VIA a 7 MEMBRANE G COUPLED RECEPTOR that is INHIBITOR in this case. Second messengers are ADENYLATE CYCLASE WHICH HYPERPOLARIZE WITH POTASSIUM, then lowering cAMP, then PROTEIN KINASE A is lowered....DO YOU RECALL ALL THIS? Can you label all the protein enzymes in a blanked out flowchart? THIS IS A VERY VERY VERY IMPORTANT concept you cannot forget. Do you recall my mnemonic with the chip company AMD inhibiting dominant chipmaker Intel so letters A, M, D for the 2nd subtype of receptors as AMD is second to Intel are all working via the same MOA!) So, the answer is that the velocity through the AV node will be reduced!!!

Q5) Next, Bill sitting to your right says that he was a FORMER PSYCHOTIC who overdosed with a D2 blocker drug and the ER have him PHYSOSTIGMINE. He asks you why couldn't he use Bob's drug NEOSTIGMINE?

A6) CRITICAL PT I mentioned long ago....PHYSOSTIGMINE can cross the all important Blood Brain Barrier and so is used for antimuscarinic cases of D2 antipsychotic drugs in the CNS...NEOSTIGMINE CANNOT CROSS.

Q7) Then, he asks you how will he know if he took too much PHYSOSTIGMINE?

A7) Remember, lump stuff together. This is a cholinomimetic and will cause the associated symptoms which you MUST KNOW ALL OF THEM like miosis, it will make people feel like urinating, etc.

Q) Why then is Physostigmine more dangerous than Neostigmine?  
Think about it first...

A Because it can cross that Blood Brain Barrier, an overdose will lead to respiratory depression and cardiac depression.

IF YOU REALLY UNDERSTOOD THE ABOVE CASE, YOU ARE DOING WELL!! Really focus on the words that I capitalize and know that you need to know EVERYTHING in detail. REREAD the above text over and over. I spent so much effort to give a NBME-philic case you can model your thinking around...

Tommy

438.

You know all about ATYPICAL PNEUMONIA from MYCOPLASMA pneumoniae right? But tell me three things quick!

1) Is the cough productive?

A1) NO

Q2) Are the antibody titers WARM OR COLD?

A2) They are positive COLD antibody titers.

Q3) Is the treatment Penicillin G or Penicillin V or NEITHER?

A4) This one needs a protein blockers like Erythromycin.

210

439.

You all know the most common primary bone tumor the NBME will ask is MULTIPLE MYELOMA.

Q) What is the 2nd most common primary bone tumor?

A) Osteogenic Sarcoma...do you know the age, and tx, and side effects??

440.

YOU all know that Glioblastoma Multiforme is an NBME favorite and is the most common primary brain tumor in ADULTS.

Q) But is this the same in children?

A) NO! The most common primary brain tumor in kids is medulloblastoma. Could you point it out in an MRI? Do you know the Rx?

441.

Q) You know how to spot a clinical case of the CREST syndrome in a women right? First, think about it...Very important...when the labs come back, which autoantibody are you looking for?

A) The anti-centromere antibody.

211

442.

Q) We review a child with Celiac Disease and you KNOW who diet he must follow. Right now, as a review, tell me what lab antibody type are you looking for to confirm the diagnosis?

a) Antigliadin antibody

443.

Q) LUPUS in a women can be so devastating...

So tell me the two antibodies for SLE and THEN tell me the antibodies for SLE that was drug induced, and THEN tell me the drug which could have caused this crisis!

A) Naturally, you are looking for anti double stranded DNA and ANA antibodies (single stranded DNA antibodies are a common error) Also, you KNOW that my mnemonic is "Women have nice HIPPS." So...

H ydralazine

I zoniazid

P Phenytoin

P Procainamide

(Just for your info, know the commonest TRADE names for some of these drugs. I even heard LASIX is often substituted TOTALLY on the USMLE TESTS and in clinics for Furosemide. Just like KLEENEX (brand) is known better than tissue... and BAND AIDS (Brand name) is used more than "adhesive bandages". But these are exceptions...99% of the time the USMLE sticks to the generic names.

212

444.

Original Clinical Case Copyright ValueMD and Family:

We previously discussed cancers. Did you know you could be presented in clinics and USMLEs with a case addressing cancers and different organ types? KNOW that the most common cancer in any organ is metastatic and DO NOT say primary type. Now here it is:

Q) That said, you have an older gentleman named Crandle and he comes to you with spiking back pain. On exam, he has loss in his arms and legs, loss of motor function. You suspect metastatic cancer of the prostate to the SPINE. What are the classes of drugs you will be asked to master for this MOST COMMONLY diagnosed cancer in the US (LUNG Cancer is the most common death from cancer, but PROSTATE is most common in DIAGNOSIS)

A) There are several, and you may be asked all of them, the MOAs, and their side effects. They may give you like a drug series and asked the drug class missing. And their MOAs and Side effects/ SE may be described or in a pictorial form. So know that YOU HAVE to master the below information like your father's birthday.

Finasteride, KNOW it is anti androgen, KNOW it acts to block 5 alpha reductase. KNOW it blocks NOT testosterone directly, but the conversion of testosterone of testosterone to dihydrotestosterone (A critical fact). KNOW the SE involves liver failure and loss of libido and impotence.

Flutamide, KNOW it is a NON STEROIDAL, an antiandrogen confused with Finasteride. It is a MOA involving androgen binding and uptake (these MOAs sound the same but are drastically different especially on the USMLE and in "pimping?") All of these will be answer choices, and

213

you will forget Flutamide from Finasteride!

Leuprolide (Lupron is OH SO COMMON) ?KNOW that its MOA involves a synthetic nonapeptide analogue of GnRH that acts as a potent inhibitor of gonadotropin secretion, that is, LH and FSH is decreased.

Bisphosphonates, I told you he had bone pain, right? KNOW that they stop bone resorption via osteoclasts and NOT revving up osteoblasts MOA (another common answer pick mistake)

Paclitaxel (Taxol), Prostate cancer is hard to treat with chemo, but you will have to know that the MOA binds tubulin!

Prednisone ! I betcha you didn't know that, right? It helps via MOA of lowering PSA levels.

Hydrocortisone Cream—KNOW positively the MOA--which is blocking inguinal capillary permeability and inhibiting WBCs leaking and causing inflammation to the prostate. KNOW that you MUST ASK if the patient has thyroid issues..

Ketoconazole: EVEN AN ANTIFUNGAL IS GIVEN! KNOW that this MOA Produces responses similar to that of anti androgens. They block a variety of cytochrome P-450 enzymes, including 11beta-hydroxylase and 17alpha-hydroxylase, which in turn inhibit steroid synthesis. Could you remember all of that? If you don't get a question on this on STEP 1 you WILL be asked at some other time ALL of the ABOVE INFO.

Q) What is the market that doctors look for to see Prostate cancer?

A) PSA and alpha feto protein!!!!

Read the ABOVE POST MANY TIMES, it is confusing! At least thirty

214

times because there are 444 possible questions in the above post!

Q4) Also, I forgot to mention, what SPECIFICALLY DO YOU TELL TO a patient you are IDed as risks for PROSTATE CANCER?

Q5) Besides age, due to its genetic linkage, prostate cancer is more frequent in patients with a strong family history of prostate cancer. Likewise, people who smoke, African American males, and patients who consume a diet high in animal fat or high in chromium have increased incidence. DO NOT FORGET! I SHOULD HAVE BROKEN THIS POST INTO 44 posts, but I WANTED you guys to lump them together! For better recall!!!

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445.

Regarding PROSTATE CANCER and the previous case:

Q) You suspected prostate cancer in a second patient who comes in after the first one name Harold. Harold though, has an enlarged prostate from the famous M3 student consult anal exam, and you start anti androgen treatment. He does however describe his bone and sense pain with AN EXTREME EMPHASIS ON ABRUPT SUDDEN ONSET OF LEG PAIN too. You continue with prostate cancer tx. Then six months later, your senior attending got sued and lost his Mercedes and is bicycling to work.

What KEY mistake and dx did YOU MISS? VERY important!

A) You missed the easily and common mistake that the older guy with a large prostate actually had SPINAL CORD COMPRESSION/SLIPPED DISK which needed an Emergency surgery with an ORTHOPEDIST. Very common mistake...

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446.

Q) Still, another guy with another enlarged prostate...you are running short of surgical gloves...presents with the same symptoms as the first patient. You start prostate cancer therapy again but CHECK the CT to rule out spinal cord fracture to not repeat the same error. A new hematologist comes by and asks if you need her but you say no way...

But then...the replaced attending AGAIN is sued a year later and you see both of your past attendings losing their Mercedes and riding on a tandem bicycle to work together. What COMMON dx did you overlook and fail to rule out?

A) Many, many, leukemias and lymphomas can mimick the presentation clinically of prostate cancer patients. YOU HAVE BEEN WARNED BY VALUEMD AND ME! You had to have chosen a different treatment. Say goodbye to a good residency slot...sorry....

---

447.

NOW, you have seen a fourth patient name Jordan who is an older African American who smokes two packs a day for 40 years, eats only steak meat, has 6 children and wants no more kids or sexual relations in his life, and all his male ancestors had prostate cancer....he heard about your past two attendings and your mistakes...and he refuses all RADIATION THERAPY AND MEDS FROM YOU because of your common mistakes. But he still likes you and you are part of his limited HMO plan.

Q) You offer a surgical intervention, and he accepts... What is the NAME of the intervention and what did you do to him that WAS PROVEN IN MANY RESPECTED STUDIED TO LIMIT PROSTATE CANCER

216

IMMEDIATELY?

A) You did a radical prostatectomy and orchiectomy. That is, you castrated him. Not too pleasant, but very effective, and VERY ASKED!

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448.

OK, you get a fifth patient in who just saw Jordan your last pt limping out with pain from the surgery. The fifth guy KNOW everything now and was referred to your clinic and is only asking advice. His LOCAL PROSTATE CANCER is being controlled with meds, but his GLEASON score is 6. (KNOW the Gleason grading since PROSTATE cancer is oh so common)

Q) He asks what are the chances of METASTASIS (which is often fatal) for him?

Q) Also, what is the MOA the metastasis if he is good with his meds intake?

A1) After stage Gleason 6, metastatic cancer is expected. About half of all localized prostatic cancer WILL metastasize even with full med tx.

A2) The reason for this--even though aggressive meds are used--is due to resistance from ANTI ANDROGEN HORMONE REFRACTORY DISEASE FROM CANCER CLONES.

Now, you must remember your readings in PSYCH texts on how to "break bad news". Really, something like this happened to me. There are a lot of tears and it is VERY VERY awful. And I am serious.

---

217

449.

MANY MANY USMLE WRITERS want you to understand BASIC NUTRITION. For real...

So...

Q) A sixth patient a 15 y.o. high schooler, walks into your clinic named Siegfried. He had a father named Roy who just died of prostate cancer at 45. He has ALL the risk factors on history. You tell him because he asked, that IT IS TRUE THAT CASTRATED MALES LIVE LONGER. And he is so afraid also of meds, but he tells you to "put away the scissors, I am still a young boy who wants to date girls..and guys". So he asks you about how he can change his diet? You answer what?

A) Have him eat a lot of tomatoes, broccoli, Asian green tea, soy products, licorice root, selenium, and antioxidants and the vitamins. Seriously, the NBME will ask you to answer some basic dietary questions.

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450.

Q) Finishing up, what med can be asked and can be used if all the mentioned drugs fails? AND give me MOA?

Q) I forgot to ask, what is the difference in MOA of drugs Leuprolide and Abarelix? (YOU KNOW TO KNOW THIS FOR STEP 1).

A) You can give Suramin for refractory pts w/ MOA of INHIBITING GROWTH FACTOR for prostate cancer tx.

A2) Leuprolide is a GnRH analog and acts via competition so lowers LH and FSH.. BUT.... drugs like Abarelix are GnRH receptor antagonists...so be ready to know that the MOAs are different, but

218

both lower LH and FSH and thus .... dihydrotestosterone.

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451.

--If you see enzyme disorders, and are asked by your attending or NBME, what is the mode of inheritance (YOU HAVE TO KNOW ALL OF THEM COLD), then "usually" this is Autosomal Recessive

--If you see musculoskeletal, structural protein, endocrine thyroid pancreas, and neurofibromas, then guess Autosomal Dominant.

[Of course there are exceptions, but I am desperately trying to "lump" because it helps if all else fails]

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452.

Dear Future M.D.s,

I am now flooded with questions in my various mails. I love them because helping is fun! At least I think so. And the same questions come again and again..

1) For IMGs, a serious problem that just won't quit is the language barrier. I think this is the MOST difficult one to handle because time is a necessity. Please read one of the past posts which addresses this well.

2) Some of us IMGs are asking what books to study. This question is definitely one of the top five questions ever asked. I feel you need to start with [First Aid](#) and choose ONE or TWO of the series:

BRS, [Kaplan](#) Notes, HY series, or [Board Simulator Series](#) or Step-UP. AND two additions are Goljan's Notes and a GOOD PATHOLOGY/MRI atlas! (Here you could do Robbins or do Webpath). People do not

219

realize how visual the test is. They had 99.9999% of their tests in life without any pictures. But whoa, some of my students get image after image which all look like a case of pharyngitis.. or they get these abnormal HISTO PATH pics of lung diseases which all look the same. So, you must must pick a VISUAL source for your studying.

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Then, you must read and re read the same material. If you keep switching, I saw students getting confused and lost. Also, the ones who stuck to JUST ONE SOURCE like [BRS Biochemistry](#), ended up almost MEMORIZING the words and pictures. It happens to everyone. You pick up like [FA](#) over and over and then you for example....know that in the Pharm section the microbiology drugs are discussed first. It helps with memory skills.

3) Many asked about question banks. That IS critical. I have heard some say they passed with ONLY DOING QUESTIONS. I think this approach MAY work for some who have the basics DOWN but I do NOT recommend this for most. But if you lack the fundamentals, then doing [Kaplan QBank](#) or [BSS](#) is really just wasting your time. You are better off watching a movie or giving your significant other a backrub/backstrach. Because you will not retain the information. I saw students continue doing like QBook over and over and over and they were getting higher scores because EDUCATIONAL THEORY TELLS US THAT YOU ARE ONLY MEMORIZING QUESTIONS. It comes up again...you are like driving down the road to your work and there are vague stuff around that guide you. But you cannot stop and tell me the name of the road after "USMLE avenue?or the number of right turns after the gas station. The brain forms these patterns...and they will not repeat on your real exam. Especially if you are doing questions at random without linking subject material together in a "SPIDER WEB?like configuration in your brain.

220

SO...my advice is?YES, do ValueMD questions first, they are done by former Step 1ers who are constructing new novel material that will prepare you for the real exam because they understand the focus and theme and flavor of the exam to make you a better doctor. Also, when you sit for the real exam you will be amazed at the sheer VARIETY of the questions. Some are detailed, some are short answer, some are IMPOSSIBLE, some (just a couple) will actually just like point an arrow to the nose and ask, "What is this?"...meaning, a couple of the questions will be very easy. But the sheer variety is what makes me believe the NBME USMLE Step 1 is like the universe. Of my hundreds of students, hardly a single exact question repeated when they naturally discussed them over nights, lunches, etc. BUT, THE SAME CONCEPTS CAME UP IN ALMOST EVERY TEST. A perfect example of this is the second messenger concept. I tried to give a mnemonic that works miracles for me...but it may NOT work for you. Thus, I suggest doing questions (after the ValueMD ones) by grouping them. [Kaplan](#) is good, but then do [BSS](#) and Princeton Review, and then Board Review Series, then Pathology Review by Robbins (with pictures), then NMS. Naturally, you will run out of time, but at least you are using the right method. Again, do questions by system and subject or you will not retain anything. That is why in the OLD DAYS prior to the printing press people memorized by LUMPED stories, using rhyming techniques, timed repetitions, etc. to memorize texts as thick as the BIBLE. They had to...how do you think you got the present version of Homer's "The Iliad? If those same people just picked up the BIBLE at random, read a verse, shuffled the pages, read another verse, they may someday catch up, but the time for mastery is unacceptable. Admittedly, there are some, just a few of my students that blew me away in their capacity. They were only a couple out of thousands, and many never wanted to use their powers of memory for anything. So again, doing organized systemized questions in a formalized interval is the solution.

4) Many many questions are coming up about [QBank](#) analogies and the general time frame for preparation that I recommended for STEP 1. Mastery of [QBank](#) is only a rough measure of how you will score on the real exam.

After taking many polls, I found all sorts of statistical anomalies. One of my best friends matched in a competitive Radiology program and blew through 250/99 but was reportedly scoring about 65% on [QBank](#). NOW, before you get too excited, there were students beating 70%-75% on [QBank](#) but failing!!!!!!! Also, a close relative of mine was scoring consistently around 50% and barely passed. After many many statistical points, I would argue that if you are getting around 50% on [QBank](#), you are "close" to passing. But what examinees do not realize is the REAL STEP 1 is HIGHLY VARIABLE IN MATERIAL. Thus, good sources told me that some had deep emphasis on pharm, others on pathology, others on virology...one girl said she got mostly all immunology. And through a third party, I heard of a brilliant US med student who was aceing med school and doing 80% on [QBank](#) but failed his first time because he got a lot of questions on difficult new research in Molecular Biology. Thus, I recommend what I said before. I do think [QBank](#) is a VERY good source of questions, but you need varied question sources but you have to organize them properly. Also, many asked me for a solution and detailed their situation where time was a serious problem. Some had to prep for STEP 1 within a few days only, and some had the time for prep but the situation was too unwieldy...many, because we are the IMG family have unusual circumstances. Several desperate mails came because the students had children or sick parents. My heart breaks...because how will you push a square peg into a circle? YOU MUST RECEIVE THE TIME YOU NEED. Unlike a few tests that rely on math, or interpretation skills (LSAT comes to mind), life experience in literature (SAT I Verbal Section), the USMLE STEP 1 material cannot be gathered by life's chance or opportunity. For instance, you will see Xeroderma Pigmentosum because it is a wonderful concept involving DNA and

222

thymidine dimers and repair defects. But did you know that maybe if you are lucky you will find only 1000 people in the entire WORLD of 7 Billion with the disease? That is why THIS TEST NEEDS AND DEMANDS YOUR 100% ATTENTION and SUITABLE TIME that you need. Again, if you need maybe six months of 12 hours/day prep, do not feel that you are less smart than someone who studied in three months. Common society has determined that the second person is "twice?as smart, but that is NOT TRUE. There can be ONE MILLION reasons why one needs more time, but what I personally found was a "VARIABLE?related to reading speed and another "VARIABLE?related to processing speed. I knew of one of my friends who went to U of MICHIGAN and works at NASA. He was a super smart guy and blew everyone in my high school away (For you IMGs, U Michigan like U California, U Virginia, U Miami, U Washington, U Texas are among an ELITE GROUP OF COLLEGES EQUAL TO THE USA IV LEAGUES LIKE HARVARD.) Anyhow, my point is that some people are smart enough but I researched there are about a dozen measurable quantifiable "intelligence?points of reference. It is like a MACHINE that has many parts. All the machines can finish the work, but some take longer because maybe one part of the machine is not as efficient. BUT, that "slower?machine may produce higher quality products. Think of a HP PHOTOJET and HP LASERJET series #. The PHOTOJET makes BETTER CLEARER pictures, but the LASERJET is faster. But BOTH make copies and BOTH have value and BOTH are HELPFUL. So, you need to understand yourself and your limits and what exact time you need. If you do not approach this properly, then you WILL BE ONE OF FAILURE STATISTICS.

5) I will need to continue this thread of concepts because I note that there are additional questions in my mailbox. But please digest the above information. Oh, by the way, I believe my suggestion of notecards are effective. Make some up with say Pharm which are easier to develop. Then start front card #1 and move backwards. If you are getting say card #26 wrong, then move that card forward so

your repetition schedule for that question/concept will be seen more often. If say you mastered cards 40-46#, then they will end up toward the back of your index card box. Thus, you can start scientifically measuring your RETENTION LEVEL and READING SPEED LEVEL. There is a whole science to this that I feel I should tell you, but I need to go for a while. So, for the 2 Ross students and 4 East European students, etc. you SHOULD be worried about the time and scheduling.

6) Quickly, also know that the US students are NOT smarter than IMGs but they are better at the STEP 1 because of many reasons. Some include that they JUST FINISHED THE BASIC SCIENCES while some of the IMGs had them long ago. Also, many of them are "coached" by their schools from Day ONE with USMLE type questions (pics and all). Plus, the ones that write the test are mostly the ones that teach and test the US students. So, I believe that ALL IMGs and USAs are equally smart for the most part...Even if that was not true, it is NEVER a reason to give in.

LOVE Tommy

453.

Quickly, you are viewing an radiology report and seeing polyps in the colon--hundreds of them?

Q) What is this disease and the genetics and will this proceed to cancer?

A) After R/U IBD, this is Familial polyposis coli, which is AD and mostly becomes malignant!

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454.

ON CLINICS, USMLE STEP 1 you have to KNOW lead poisoning because...about 5% of all children have elevated blood lead and about 25% of all low income US children living in pre-1950 homes have elevated blood lead which can cause mental delay, anemic symptoms, bizarre behavior, GI upset. STEP 1 needs you to understand that LEAD POISONING IS SO COMMON BUT SINCE it is easy to miss (symptoms are non specific), you need to be aware because if you fail to order a blood lead level test on an at risk pt, you might as well become a city car ticket handler because you will lose your medical license:

Case: A boy named Donny Dosman comes in with nonspecific symptoms like hyperactivity, diarrhea, and occasional tired spells. YOU suspect Lead poisoning.

Q) What is the MOA of the medicine that you will pick as the DOC?

A) As we mentioned once, BAL or Dimercaprol works via chelation and is water soluble and rapidly crosses the blood-brain barrier. Forms a nonpolar compound with lead that is excreted in bile and urine. DOC in patients with acute lead encephalopathy, in whom first dose is given and then the second dose is given combined with calcium EDTA after a four hour interval. Remember that the Ca salts can also treat hyperkalemia! BTW, you found that Donny ate PAINT CHIPS from his old apartment.

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455.

You Lead intoxicated patient, Donny, then tells you from his history (he is an African American patient), that he has something called G 6 PD def.

225

Q) Do you continue with the BAL treatment?

A) NO! BIG legal mess. And you may kill the patient. BAL in G6PD def pts can hemolyze blood cells.!

---

456.

Q) Donny's mother then tells you she has a sister with a baby who is living with NO LEAD INTOXICATION RISK FACTORS in a new house. You see the baby named Shazam in your clinic at his one year birthday. Do you need to do a lead screen?

A) Yes, you still have to do one, and every 2 years thereafter on this low risk baby.

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457.

Your patient Shazam (recall, he is a baby), is 100% breast fed. His mommy asks you if she should give IRON supplements b/c she read it in a magazine.

A) NO, breast milk has enough iron. Give IRON supplements to formula fed patients unless the Formula can says "supplemented with IRON".

(This sounds advanced, but I KNOW it IS STEP 1 material)

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458.

226

Yow! Donny's Father then walks in for a quickie checkup. In his PE, you ask to see his tongue to test CN 12 but you note that you see something awful...he has ORAL HAIRY LEUKOPLAKIA. (Review picture) In such a case, what..

Q1) What two common patient populations will you get with this devastating dx?

A1) AIDS patients and heavy smokers and drinkers.

Q2) What virus if asked/pimped is involved? (Do you recall the viral structure and Family?)

A2) This is Epstein Barr Virus, EBV. IT is Double stranded, enveloped, linear, and part of the HERPES family DNA. It is also a cause of Burkitt's and mononucleosis!

Please do recall ALL the points here. The USMLE and attending may trick you and ask if the EBV is an RNA bug, which is wrong. And so you will have gotten so far but ended up short....

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459.

Your previous bad luck with all those prostate pts is forgotten, now Donny brings in three relatives with back pain (YOU WILL SEE THESE EVERY MINUTE DURING ROUNDS AND IT IS A CRITICAL CONCEPT)...

Q1) Donnycousin1 is 20 yo and is lifting heavy boxes for UPS as a job. You sent him on his way after ruling out deadly causes and confirming a "pulled deep back muscle". Did you do right by him?

A1) I KNOW I am sounding "picky" but you are mistaken. The NBME needs you to know that even a young man with a recent pulled back muscle should be advised to wear a "weight lifter" hip belt.

Q2) Donnycousin2 is 40 and has back pain with NO Hx of trauma or neoplasm. What may you see on Lumbar Puncture?

A3) In such a presentation, consider a bug that made its way into his spinal column!

Q3) Donnycousin3 is 65 (older cousin) and oh NO! He has lower back pain with INCONTINENCE and with CONSTITUTIONAL SYMPTOMS (Fever/chills/headache). Now what tests do you order, because you are fearing????

A3) As I said with questions, consider carefully the age, sex, ethnicity, diet, meds, etc. of the patient. Here is an older gent with the HINTS of incontinence from tumor pushing a local mass effect on the sacrococcygeal area and the CONSTITUTIONAL SYMPTOMS!

NOTE: The above cases are so common and tested and asked and pimped because back pain is so common. I saw more pts coming in with this than the flu!!!! So you MUST RULE OUT MALIGNANCY, even with younger patients...

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460.

'Case: Donny's cousins have a few second cousins, weird...all have

228

back pain with same BAD symptoms.....What I and NBME and your attending NEEDS you to KNOW are the slight differences in the bone producing tumors of the spine...b/c the tx's are different!!!!!! KNOW....

Osteoid osteoma - Benign and locally self limited

Osteoblastoma - Benign but locally expansile and aggressive

Osteosarcoma - Malignant spindle cell lesion which produces osteoid

Q2) Sorry, you must distinguish the bones and cartilage: KNOW the cartilage producing tumors of the spine which are...

Osteochondroma - Benign lesion with cartilaginous cap.

Chondrosarcoma - Malignant cartilage producing tumors that histologically demonstrate round cellular stroma in a chondroid matrix.

Whoa, look at this:

Q3) As I mentioned lymphomas can mimick simple back pain. It is exactly the kind of question USMLE needs you to KNOW how to differentiate...AND I KNOW THIS IS A VERY VERY HARD AREA....

Consider the Lymphoproliferative tumors...

Multiple myeloma and plasmacytoma are derived from plasma cell dyscrasias, which histologically appear as sheets of plasma cells, and remember lytic lesions and back pain?

Lymphoma - Associated with a large infiltrate of lymphoid cells

Q4) Sorry, we are NOT done yet!!!! Remember the

Tumors of notochordal origin?

Chordoma - Identified by the characteristic physaliferous cells.

Round cell tumor - best seen with a Webpath pic

Ewing sarcoma - Malignant tumor of childhood associated with large sheet of homogenous small, round, blue cells, and you KNOW we talked about this one.

EVERYONE THOUGHT THIS BACK PAIN AND ALL THESE TUMORS WERE IMPOSSIBLE TO GET STRAIGHT BECAUSE THE NAMES ALL SOUND THE SAME. I ALSO WAS SO STRAINED TO MEMORIZE THIS FOR STEP 1.

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461.

Q) YOU MUST KNOW: IF you get a case of a patient with COPD, ELDERLY, CHF, or sickle disease, you MUST give what specific vaccine that NBME/clinics will ask?

A) an annual INFLUENZA vaccine.

462.

Q) You have a sickle cell teenager (I SAW SOOO MANY) who had a splenectomy! WHAT 2 vaccines must they receive!!!? (HINT starts with letters m and p...)

A) THEY absolutely have to have meningococcal vaccines and one for pneumococcus!

463.

Q) All my students say HIV and such buggies are crawling their way into the TESTS and clinics. When you go into "patients" on the test, many will have a fever, the most common cause of fever is INFECTION. (Recall that even SLE can present with fever.) LUMPIN... You have a patient who is 3 months OLD. Which 2 vaccines do you reach for?

A) HBV 1 and 2 (fr Birth to 4 months)

464.

LUMPING along..

Q) Your patient Mickey is half a year old. You are asked by USMLE/ATTENDING what additional three vaccines should have been given? (HINT: mnemonic..HID) ... I "HID" the candy fr. my 6 month old.

A)

H..Hib!

I...IPV!

D...Diphtheria!

(anywhere from 2-6 months of age!)

465.

Q) Your patient Mickey is now grows to 1 1/2 old. Your USMLE/ATTENDING pimps you and asks which med will you grab next?

A) OPV...and you must be sure he received his THIRD HBV shot!!!  
OUCH!

466.

LUMPIN still.

Q) You have another patient named Minnie. She is exactly 1 year old. She is up to date with her immunos. But now, your attending/USMLE asks what TRIPLE vaccine do you grab? (BIG POINT!)

A) MMR (The first of two) (Remember, Measles, Mumps, Rubeola)

467.

Q) Oh NO! She comes back in one month. She is 13 months old. Your attending smacks you and you forgot to give a vaccine!!! (HINT: Starts with letter V) What is the bug assoc. with vaccine and structure?

A) LISTEN, at between 1 yr to 1 1/2 year, all patients must have their Varicella Vaccine! It is a HERPES virus family. IT is enveloped, double stranded, linear!

468.

A lot of people are asking about the "lumping" of some more MICRO. To answer "Big Concept":

The NBME must demand you understand the common things, what to do and not to do. Think, you have patients with an infection (Microbiology):

Case: A patient comes in with signs of a cold, but on the test and in life, you will be given more. Patient's name is Quentin a 11 year old and he has a fever after a raccoon bite. What vaccine should he have on his chart GIVEN STARTING IN HIS EXACT AGE that addresses the "bug". What is the family and structure of the bug?

A) He should have been given Tetanus vaccine that starts on age 10-11 and gets a "booster" every 10 YEARS. The bug we are worried about is rabies which is from family RNA Rhabdoviruses, SS, negative sense, square shaped. If the NBME tests you, know if you get even ONE of these data points wrong, you will miss the question. (Like thinking it is positive sense and not neg. sense).

469.

LISTEN, AS A HINT ON THE USMLEs AND IN LIFE, THINK VERY VERY CAREFULLY ABOUT THE PATIENT'S AGE, GENDER, ETHNICITY, TRAVEL, MEDICATIONS. This is often overlooked.

Q) Listen, a 2 month old named James Joyce comes into your clinic. He is CURRENTLY ON ANTIBIOTIC THERAPY. Your attending asks if you are still going to administer his vaccination schedule. Will you?

A) This is one of the most common mistakes. YOU STILL GIVE HIM HIS

233

VACCINATIONS...usually, (unless he is SO immunodeficient and deathly ill) and you are holding a syringe of live polio vaccine.

470.

Q) If I gave you a list of vaccines, and then told you the patient is allergic to egg proteins, what common vaccine should you be WARY of giving?

A) MMR

471.

Q) A patient comes in, he is 4 yo Mexican male with a POSITIVE PPD test!

Do you continue with his vaccine schedule?

A) YES YOU DO! Seriously!

472.

Q) So NOW YOU START FEELING GOOD ABOUT VACCINES....now a 14 year old pregnant girl named Nancy Voltaire comes in. She is missing her MMR and OPV (someone just email and asked what is OPV...it is the Oral Polio Vaccine). Can you give it to her?

A) NO, NO, NO! Including allergic reactions, pregnancy is contraindicated for Polio and MMR vaccines!

473.

Q) This is VERY important: Tell your attending about the difference between Sepsis and Bacteremia (most of my students think they are

234

the same--don't tell your attending that).

A) While you can bacteremia from just flossing your teeth (it is just bacteria in the blood), sepsis are those buggies crawling into the intravascular space, possibly causing septic shock, and killing the patient.

474.

Q) A patient named O Henry comes in, is 3 years old with a fever of 103, PE is normal otherwise. YOU are about to think it is a common cold virus going around, but there is a blood test that came back with tons of white cells on the smear! What is the commonest bug? What is the structure and in fact, tell me all you know here...?

A) Absent a "zebra" this is Strep. pneumoniae. This will look like "purple circles in chains or lancets (like Middle Age weaponry)" in stain. It is catalase negative. It is alpha hemolytic, has a POLYSACCHARIDE CAPSULE, is OPTOCHIN SENSITIVE, POS. QUELLUNG SWELLING. Don't confuse this with Strep. viridans or Strep pyogenes. NOT EVEN ONE CHARACTERISTIC. S. pneumoniae is SO IMPORTANT that it is like your "tongue" and "tasting". You can't live without seeing it daily in clinic/tests/etc.

475.

Q) Your subordinate M2 med student says "S. pneumo has streptolysin O for the alpha hemolysis!!!!" Is he right?

A) Critical point: NO! If you are getting these wrong, you are NOT RETAINING MAXIMALLY and missing concepts...IT IS STREP

235

PYOGENES, the bug that causes rheumatic fever, with streptolysin O!  
S. Pyogenes also has erythrogenic toxin...WHICH BINDS TO....MHC II  
as it is a SUPERANTIGEN TOXIN.

476.

EMERGENCY! Someone just emailed and asked a good question: Then  
is S. Pneumoniae the most common cause of sepsis?

Q) Can you answer him?

A) NO, as I said, S. Pneumoniae is related to commonly bacteremia...  
Sepsis is MUCH MORE SERIOUS, caused by endotoxin from gram neg.  
bug like probably E-coli.

477.

Q) So, your trusty med student says, "Let me go get a good third gen.  
ceph. for the bacteremic patient..." Is this a good drug of choice?

A) NO! As I said, bacteremia is S. pneumoniae, which is gram  
POSITIVE! So, since third gen. cephs. move into gram neg. coverage,  
pick PENICILLIN or AMOXICILLIN for the S. pneumoniae!!!!

Are you getting these right?

478.

Q) Your trusty med student asks, "S. Pneumoniae causes pneumonia,  
and you said we can give penicillin, and my friend Jon has "walking  
pneumonia" and a non productive cough. Can you write him a  
prescription for penicillin?" My question is, will you?????????

236

A) NO, NO, NO!!! The walking pneumonia is from *Mycoplasma pneumoniae*, NOT *Streptococcus Pneumoniae*. Use erythromycin....

Don't miss these!

479.

Q) Hey, now, you get another kid named CS Lewis who comes in with a fever...but he also has irritability and right ear pain. What is the likely dx, bug, and treatment?

A) PLEASE do not tell me you got this wrong. This is OTITIS MEDIA. (acute middle ear inflammation) This is as common as jokes about President Bush's grammar mistakes...(sorry Sir!). Most common bug is Strep. *Pneumoniae*, and again, the DOC is still Penicillin...

480.

Q) Now, another kid comes in with the same clinical presentation: irritability, fever pain, right ear pain. But his whole family has viral colds and HIS culture was NEG for *S. pneumoniae*. KNOW that *Hemophilus influenzae* can cause OTITIS, but due to immunizations, you may also see another bug...hard question....do you still give the penicillin for coverage?

A) NO! Recall H. Flu is GRAM NEGATIVE! NOT G-+. I am talking about the next most common bug, *Moraxella catarrhalis*, also GRAM NEGATIVE!!!!. This buggie has recently been shown to be both widespread and pathogenic, (This was ONE OF MY PERSONAL

237

UNKNOWN BUGS IN MY FINAL MICROBIO LAB TEST!). Several factors have been suggested as virulence factors, lipopolysaccharide (LPS) being one. Recent studies have shown the LPS to be without the O-chain, i.e. the polysaccharide part, and to have specific structural features corresponding to each of the three serogroups, A, B and C. The structures resemble in many respects those present in other Gram-negative nonenteric bacteria, with a galabiosyl element as a prominent common structure....take THAT! So, give GRAM NEG COVERAGE LIKE ceftriaxone. OH, THIS IS ALSO OXIDASE POSITIVE. Almost all of these buggies are beta lactamase producers, so penicillin will be cleaved. DO YOU REMEMBER EXACTLY WHERE IF I GAVE A DIAGRAM? OLDER CONCEPT!

481.

The following question/answer is how your brain will learn, by comparing/contrasting/analyzing/recalling.... here....

Q) Another child comes in with the same OTITIS MEDIA symptoms...but NOW, ALL THE USUAL SUSPECTS ARE RULED OUT! But, the recurrent, chronic suppurative OTITIS MEDIA is cultured and you smell grapes on blood agar. Plus, your attending says this bug also gave him EXTERNAL OTITIS while he was swimming. What is the bug? What drug? What structure for this SUPER IMPORTANT BUG?

A) This is *Pseudomonas aeruginosa*. For this, you usually MUST choose two drug combo like Ticarcillin/Gentamycin (A Penicillin and An Aminoglycoside). You can sub Aztreonam for the penicillin part of the combo. REMEMBER JEDI KNIGHT, this bug is OXIDASE POSITIVE and is a Gram negative bug. Review the types of pts. this bug bothers...

238

482.

Q) Oh, your call night is just NOT ENDING...another kid comes in presenting like OTITIS MEDIA, but the attending looks inside the ear and says, NO! This is OTITIS EXTERNA. What did he see?

A) OK, Sir Sherlock Holmes, or Lady Shirleylock Holmes...you usually see a NORMAL tympanic membrane and just touching the outer lobe of the ear is painful in OTITIS EXTERNA!

483.

Q) YUP, another patient with OTITIS MEDIA (ascertained by attending) comes waddling in. He KEEPS GETTING OTITIS MEDIA (MOA is DECREASED TONE of the EUSTACHIAN TUBE which drains that middle ear and so fluid comes right back up, I forgot to quiz you on that!) But, now, the mother was told of a common surgery to correct her son's problem. What is it?

A) The very common surgery for chronic OTITIS MEDIA is placement of a tympanostomy tube.

484.

Q) Cont. on with OTITIS MEDIA, there is possible hearing loss from rupture of the tympanic membrane. But what dx do you immediately worry about when the bugs move into the INNER EAR...(Hint: Starts with a "L")

A) Labyrinthitis...

485.

Q) CIA...Connections/Imaginations/Associations...let's look closer at *Streptococcus pyogenes*. (I won't ask, but do you recall ALL the structures of this bug?) This bug is the most common cause in clinics/tests for "strep throat". What is another common term for this dx which involves the anatomical region most affected? (hint: ans starts with a "p")

A) Pharyngitis. Students mix up *Strep pyogenes*, which IS THE SAME AS Group A beta-hemolytic streptococci. The clinics/board exams use BOTH NAMES...and then students mix this bug with *Strep pneumoniae*!!!! FATAL TEST AND CLINIC MISTAKE!

486.

The Group A beta hemolytic strep (*Strep pyogenes*), causes Strep pharyngitis. [students also mix this up...like thinking Strep pharyngitis is another different bug entirely instead of the disease that it IS!...like *Strep pneumo* is a BUG, not a dx... it is still *Strep pyogenes* that CAUSES Strep pharyngitis. ONCE MORE, the dx is Strep pharyngitis, the bug is *Strep pyogenes*.] We will come back to this, but think as simple as possible first...so think that pharyngitis=sore throat. Just start there first....

Q) If this bacterial pharyngitis is suppurative, what common very bad dx can it lead to? (starts with letters r... a.....)

A) Retropharyngeal Abscess!!!! (Remember this closed space, can you

240

ID it on a side view Radiograph?)

487.

Q) Now, we said *S. pyogenes* (what is the other name I JUST mentioned the boards exams uses?) causes the dx Strep pharyngitis. The suppurative consequence is retropharyngeal abscess. Very bad! But equally bad is the NON-suppurative consequences! Give me four VERY IMPORTANT dxs you will see in clinics and Step 1! This is hard, so look at the mnemonic below:

A) LISTEN UP! CAN YOU IMAGINE "PRetty SCarlet" O'Hara in the American movie classic *Gone With the Wind*? Look at the two words "PRetty SCarlet", NOW look at the first two letters... "PR... SC..." Now connect them like this:

P oststreptococcal glomerulonephritis (kidney damage)  
R heumatic fever (heart damage amongst other stuff)

S carlet fever  
C ellulitis

NOTE: The PR involves two organs that are lesioned...the kidneys and heart. The SC involves the organ called the skin! Link images like *Scarlet O'Hara* loving *Rhett (Rheumatic) Butler* (hero) with all her HEART, and *Rhett* replying "I do not give a damn." and urinating over her HEART with with his KIDNEYS which are emptying. Then SCARLET (heroine) feels terrible at being ignored and has a fever and faints and injures her skin which causes CELLULITIS of the skin. Repeat this often used, invented by me, so the copyright is ValueMD, mnemonic! Remember, the PR>>>SC is *Strep pyogenes* only. What a nasty bug!!!

488.

Q) Quickly, is Strep pyogenes Bacitracin sensitive or Optochin sensitive? Does Strep pyogenes have a capsule?

A) Remember, don't mix up these two bugs which are ALWAYS mixed up...Strep pneumoniae is sensitive to OPTOCHIN, and Strep pyogenes is sensitive to BACITRACIN! And Strep pyogenes has NO capsule like Strep pneumoniae!

489.

Q) Students on clinics and board tests confuse a typical VIRAL pharyngitis with Strep pharyngitis caused by Strep pyogenes. What is so UNIQUE and SPECIFIC for making the different diagnosis?

A) For bacterial Strep pyogenes, the pharyngitis pharynx is tender, and THERE ARE NO URI signs like coughing and rhinorrhea!!!! This is crucial and HY.

490.

Q) As the previous concept said, VIRUS PHARYNGITIS and Strep pyogenes pharyngitis is similar. ONE other bug causes similar symptoms. It is from EBV infection. The SUPER HYer is, "What does a confused intern order as a test to confirm Strep pyogenes?"

A) I must have ordered this on a thousand kids....you order a Rapid Strep Test which is an antigen detection test for Strep pyogenes/group A strep/Beta hemolytic non-group B strep. [HORRIFIC, I heard all

242

three names interchanged everywhere for this SUPERBUG] This test is awesome...it comes back in 30 minutes while a throat culture will take days while you wonder if it is a viral or bacterial cause. This way, you know right away if you need to administer antibiotics!

491.

Q) What is the tx for this Strep pyogenes pharyngitis? [REMEMBER, THE BUG THAT CAUSES THIS CAUSED THE FAMOUS EUROPEAN STORIES OF SCARLET FEVER WHICH IN EPIDEMICS KILLED ONE OF FIVE INFECTED PEOPLE. BAD. BAD. BUG...]

A) Pick up or pick out of answer choices: Penicillin G!!!

492.

Q) OH NO! For the Strep pyogenes, you found your patient is allergic to Penicillin G! What do you grab now?

A) Choose Erythromycin for pts. allergic to Penicillin here.

493.

Q) The Strep pyogenes pharyngitis pt comes back five weeks later with a sudden heart murmur, ECG shows prolonged PR interval, arthralgias, and blood in the urine. What happened to him/her?

243

A) For some unlucky ones, you still get the acute rheumatic fever and acute poststreptococcal glomerulonephritis (presents as above)! This is an IMMUNOLOGIC REACTION/PROCESS. That is why prophylaxis is needed. You may need diuretics to control the kidney dx!!!!!!!!!!!!!!

494.

Q) After the Strep pyogenes infection, you think you see scarlet fever from skin abruptions, but this time you get a clue that points in a different dx. You see RED conjunctiva on PE. What is this?

A) This is the much milder Rubeola/Rubella infection. Remember, are you retaining while studying? Can you tell me the EXACT structure of virus?

495.

Q) Another patient steps in for a follow up for Strep pyogenes pharyngitis follow up (6 weeks ago). She looks like she has scarlet fever...but a dermatologist attending comes in and says OH NO! We got tests positive for an exotoxin called SPEA, and her mortality rate for this is up to 70%!!!

What deadly disease is this? What tx?

A) Slightly different clinically presenting from scarlet fever is TSS or Toxic Shock Syndrome. It will crush multiple organ systems and is very violent and faster acting then the scarlet fever. There may be DIC, ARDS, Massive fluid loss, Terrible skin rashes, and so on. The NBME, if they ask, must describe a test coming back with an EXOTOXIN from the Strep. KNOW that Staph aureus can also cause

244

TSS from a woman who did not change her tampons inside her. The TSS from Staph is less deadly, but since you initially do not know, you must treat TSS for BOTH Strep and Staph with Penicillin and Nafcillin for the Staph coverage. Consider also giving Clindamycin as well. SERIOUS disease that you cannot afford to miss.....

496.

Q) OK OK, listen up.. Another patient walks in with the initial presentation of pharyngitis. But now all bacterial workup is NEGATIVE. And as I mentioned, you are seeing massive lymphocytosis, lymphadenopathy, malaise...what OTHER virus IN THE SAME FAMILY OF VIRUSES AS EBV causes this dx called INFECTIOUS MONO? And please give exact structure?

A) CMV virus may cause MONO, so do the serology with heterophil antibody tests. CMV and EBV are both from the HERPES virus family. And they are DNA, Double stranded, linear, WITH an envelope.

497.

Q) Another, I said another case of pharyngitis. This patient is a 12 yo boy named Toby who came in with his mom in the early summer. There is bad fever and the pharynx is so swollen, Toby does not wish to drink and has to be placed on IV fluids. Again, all cultures are negative for bacteria. Serological tests for viruses NOW exclude ALL Herpesviruses. Hmm.. you wonder as the PE reveals malaise, mild diarrhea, and lesions on the rear end, feet, and palms of the hand. What is the exact structure of the virus? The dx name? The virus family? Drug Tx? (This is a great connecting question)

245

A) The presentation variation of pharyngitis is known as Hand, Foot, and Mouth disease. Also known as HERPANGINA, this disease is caused by Coxsackie A virus (not the Coxsackie B=heart). This is part of family Picornavirus, an RNA virus, which is SQUARE, single strand positive sense, linear with NO envelope. The treatment is...NOTHING. Unless the airway is blocked by swelling, this very infective enterovirus comes and goes within a week. Did you get it???? Please say you did! We are LUMPING ALL THE PHARYNX inflammation dxs together to catch the subtle but DISTINCT differences...

498.

Q) YOU WILL SEE ON USMLE.....sinusitis (sinus pain, headaches) because it is so common. In this imaginary patient with sinusitis, there is NO INVOLVEMENT OF ALLERGENS THUS ELMINATING ALLERGIC RHINITIS FROM THE CHOICES/Differential. Also, serology is negative for viral etiology. Give me the usual common bacterial bugs that cause this dx (BIG HINT: We spoke of them before!) Drug tx?

A) Sinusitis is usually caused by the same bugs as those which cause OTITIS MEDIA! Use the same drugs.

499.

Continuing with sinusitis,

Q) As a newborn, you have the maxillary and ethmoid sinuses. What other sinus cavities develop? Do they develop at the same time as the maxillary and ethmoid sinuses?

246

A) NO! The frontal and sphenoid sinuses develop later in childhood. Watch out, you must KNOW that for this young sinus sufferers, you must be aware of possible orbital cellulitis!

500.

Q) You see coming into your clinic another child with a sore throat. Could it be again the pharyngitis? NO! Because here, you note the highly specific stridor (barking like a seal) sound. Three questions. What is a severe consequence of this dx and what can you give as treatment?

Also, TELL ME THE EXACT STRUCTURE OF THE BUG!???????????

A) This is Classic Croup, from parainfluenza virus. You may have to inject epinephrine if airway is blocked! And this virus for STEP 1 is... Family Paramyxovirus, Single stranded, HELIX shaped, negative sense, linear, WITH an envelope, and this virus is nonsegmented (which allows for better vaccines since segmentation increases the number of serotypes)!!!!!!!!!! YOU GOT IT! YOU KNOW IT ALL!!!!

501.

Q) HY Concept 500 spoke of croup from parainfluenza. NOW, there are exactly three other bugs/viruses within the same family. YOU MUST LUMP. What are they?

A) They are the same family, Paramyxovirus!! Measles, Mumps, and RSV. On my call last night, there were so many patients with RSV!! We

247

will discuss the subtle difference between RSV and croup from parainfluenza next time if I am still alive. I am on call TONIGHT TOO!  
Coffee, anyone???

502.

To answer Sanaray's Question about the pictures and diagrams, I found that [BRS Biochemistry](#), [FA](#), and [Kaplan](#) all have good diagrams. The key to remembering diagrams..I say it again..is to stick to one source. I FOUND THAT WRITING OUT MY OWN BIOCHEMISTRY CHART WAS THE MOST HELPFUL. After referring to BRS, etc. I wrote out this GIANT BIOCHEMISTRY CYCLE WHERE EVERY SINGLE RELEVANT CYCLE INTERTWINED WITH THE OTHER. This way, you REALLY remember because you created your own chart. I wonder if there is a way to paste my own chart onto this site, but THERE ARE MANY biochem books that have this information. REMEMBER, EVERYTHING FEEDS INTO THE SUPERHIGHWAY of energy metabolism, that is Glycolysis and the TCA cycle. Know where every cycle "FEEDS IN". They are NOT isolated, but interconnected. Come to think of it, Lippincott's Biochem Review has some good "linking" diagrams. If you really understand how all the pieces fit together, it is SO MUCH EASIER to remember come test time. Also, people are asking about an atlas, etc...Webpath is great, but if you really study Netter's Anatomy and understand some of the basic anatomical relationships (eg. If I ask you to draw a cross section at C8 spinal cord level from a scratch paper, can you draw the MAJOR ARTERIES, NERVES/GANGLIA/TRACHEA, and the relationship to the other?) Everyone needs to know this, in the ER, for eg. I needed to know where the retropharyngeal space was on radiograph. It is stuff like that the NBME will want you to understand. Tommy..

503.

Q) Again, you will see this on tests and in clinics every second...patient will come in with signs of a "cold". But what are you most afraid of? A common coronavirus (Structure? please? RNA or DNA?), will resolve

248

without duress in a immunocompetent person. So the NBME/attendings will "pimp" you on whether the person is immunocompromised (HIV), or has a BAD Bacterial/Fungal infection and also if the virus could compromise the airway. So, moving along: Case: You see a patient named Clarence Day who is a 6 year old female patient who looked like your previous patient with a "cold"...but you are ALARMED because in addition to tachycardia, she is leaning forward and slightly gasping for air..PLUS, she is DROOLING (Key!). Dx, and BUG, and Rx please?

A) Here, the disease is EPIGLOTTITIS, often caused by H. Flu, (but also S. pneumo and Group A Strep). This is a medical EMERGENCY b/c it can block the airway, so you call ETN and consider a CRICOTHYROTOMY and INTUBATE!

504.

Q) Case: You now have a patient named Edgar Poe who is six months old. His mother brings him in and your med student sees him. He comes out of the exam room and says that all the family had the "common cold" so he will send Edgar home with Tylenol only. But when YOU do your exam, you hear crackles and mild rhonci on lung exam, and the child seems to be gasping for breath. Your attending comes in and tells you this IS a virus, but in patients this young, it can cause deadly hypoxic events and infects the bronchioles and is VERY contagious. The month is December... Bug and Drug and Structure of Bug please?

A) This is classic RSV virus infection, part of PARAMYXOVIRUS family with RNA, HELIX shaped, Enveloped, single stranded negative polarity. The drugs are ONLY given for serious hypoxia. You may be asked to do a trial of albuterol to rule out asthma (similar presentation), and a rapid antigen test for RSV. Some like to tx with ribarvirin (MOA please)

249

and a monoclonal drug called synergin (an RSV antibody)

505.

Case: Similar presentation of a young child, female, named Edith Wharton. This patient has NOT had her immunization shots. Her mother is coming to you after her daughter has had fits of coughing that has waxed and waned for a year now. PE is notable for an extended stridor after taking a deep breath. Her CBC has marked elevated white count, and your attending tells you this is a serious NON-viral illness (you can R/O RSV) so that goes through phases. Bug and drug and dx please?

A) This is the famous "Whooping Cough". The INTERVALS AND PHASES of strong coughing differentiate it from the other common illnesses. Since she had no immunization shots, she did not get her Pertussis shot. The bug is *Bordetella pertussis* and is a gram neg bacteria. You need a two week course of ERYTHROMYCIN. (Note, a culture and fluorescent antibody staining can pinpoint your dx).

506.

Case: Well, I won't put one here except to say know the most common bugs of pneumonia, a favorite of clinics/boards due to its severity. But tell me, how can I tell the diff. between say, *S. pneumoniae* pneumonia and a viral pneumonia?

A) So key, LISTEN...both viral and bacterial bugs can cause pneumonia, but understand that a viral (or mycoplasma) source has USUALLY more BILATERAL, diffuse, crackles, rhonchi, wheezing of this LOWER respiratory tract infection(s). BUT...a bacterial source points to a more focal or UNILATERAL source, with dullness to percussion, absent breath sounds.

If you suspect a bacterial origin, treat with Penicillin or amoxicillin. But if you think you have *S. aureus* or *H. flu*, you need a second or third

250

generation cephalosporin. Recall that "walking pneumoniae" from Mycoplasma needs different coverage like Erythromycin.

507.

Case: Regarding the pneumonia cases, what outcome are you most afraid of (don't answer death..)? And what is the treatment?

A) The most common complication of the pneumonias is a pleural effusion bad enough to compromise respiration. If you "drain" their lungs with pleurocentesis (you could get out a 1 liter or MORE), you can help with this outcome.

508.

Case: A female woman, Mrs. Stevenson comes running into your office with twins with signs and symptoms of meningitis. Both twins are male and four years old. One male, named Robert, had a prodromal stage that was not specific and included fever chills nausea. The other male, named Louis, had a very high fever throughout and missed a prodromal phase. He also has some mild seizures. Which one has the bacterial origin? And what is the bug most common? What is the most specific test? What drug will you grab?

A) The bacterial meningitis is life threatening while the viral is usually not. The bug most common depends on the age of the patient.. but cover/choose S. pneumoniae as a common cause. The bacterial etiology usually has an absent prodromal phase. Do a lumbar puncture to pinpoint bug. Tx with a 3rd gen. ceph. like ceftriaxone which moves into the CSF easily.

509.

Q) Really quick, you see a young patient with diarrhea, vomiting, and low grade fever. All bacterial cultures come back negative. There is no history of travel and the patient is taking no medications. The time of

251

the year is February. What is the most likely VIRAL etiology?  
Structure?

A) Rotavirus. It is NOT enveloped, is square shaped, double stranded, and segmented.

510.

Case: You have a young patient who keeps returning to the clinics after all infectious etiologies for bugs are ruled out! Name a few NON-infectious causes of diarrhea in your young patient! VERY IMPORTANT!

A) IBD like ulcerative colitis, cystic fibrosis, anti bacterial meds, and conditions such as celiac sprue or gluten sensitive disease could do this.

Sincerely, tommy....

511.

Case: You see another young patient in your clinic. This time, she also has bad diarrhea. So after a history, your medical student grabs some Immodium (anti-diarrheal). There is blood and yellow sticky "goop" from the GI, and you see WBCs on wet mount.

1) Is this a good idea to give anti diarrheals?

2) Rapid Rotavirus Antigen Testing is Negative, and so is Clostridium difficile toxin detection for possible antibiotic use..NEGATIVE. Other bacterial cultures are negative. Hmm.. you sit there wondering... But then your attending says she she CYSTS in the stool sample. What bug and drug?

A1) No, do NOT give antidiarrheals here, treat instead with oral hydration and replace and manage the electrolytes as necessary.

A2) Most commonly, this is Giardia. Treat with Metronidazole.

512.

Case: A young patient of yours comes in with diarrhea. All common bacterial and viral tests come back negative. So your attending says consider a parasite like *Campylobacter jejuni*. What drug will you reach for?

A) Erythromycin

513.

Case: Still stickin' with diarrhea and stomach pains...now you see a young patient who was on Clindamycin therapy for a while...(what are your thoughts?)...your attending says he found *Clostridium difficile* TOXIN. DOC, please?

A) Metronidazole, given ORALLY

514.

Case: Now, you are still seeing diarrhea and stomach pains...but this time your patient is a young African American male who has associated symptoms of headache, fever, and muscle, and bone pain. What is the bug now?

A) Consider SALMONELLA.

515.

Case: Still going...another young patient with diarrhea and stomach pains. You get a good history and it does not seem like anything normally seen...there is some blood in the fecal material...he has isolated pockets of nerve damage, LOW platelets on a CBC, and hemolytic anemia. Bad, bad disease. Your attending hints this is

253

caused by a TOXIN spills by a couple of different bacteria. What is the disease, bugs?

A) This is the infamous HUS, or hemolytic uremic syndrome. Very deadly. Two bugs..E COLI 0157:H7 and Shigella dysenteriae are seen to cause this in young patients.

516.

Case: Still diarrhea is facing you....you see another young male age 10 with fever, some blood in feces, diarrhea. You are thinking the answer choices/differentials...E coli, Shigella, Salmonella, Entamoeba...Hmm..hard one but the GI attending stops by and hints this is NOT parasitic, and the patient has a history of taking H2 blockers and he loves eating raw pork hot dogs. The labs come back and the bug is oxidase negative, non lactose fermenting. What is the bug and drug?

A) You are on your way to becoming a doctor if you got this one right.. this is Yersinia enterocolitica (Y. pestis causes the PLAGUE!). As long as hypovolemic shock is avoided, you are in good shape. Give TMP-SMX as treatment since this bug is becoming resistant.

517.

Case: We move on briefly to hepatitis...since everyone in the US receives regular vaccines, you should not encounter HBV for example in your young patient population too often. But, please understand the HY facts which address when and where you see the different antigens and antibodies for each of the Hepatitis viruses...A, B, C, D and E. For Hep B, for example, understand that about 1 in 10 patients WILL have a chronic carrier state which IS INFECTIVE. They remain HBsAg (+), so they can infect others. Do you know the difference between HBV/HDV and HAV/HEV?

254

KNOW that anti-HB core antibodies are seen after HB surface antigen has been eradicated, and understand that this may occur before anti Hep B surface antigen antibodies appears! You must review Hep B core antigen/antibody detection!

518.

KNOW: That even though the attendings/NBME probably know that you are familiar that penicillin is the DOC for *TREPONEMA PALLIDUM*, and that you need VDRL and RPR for diagnosis, tell me...what is the specific test that is used for treponemal tests?

A) FTA-ABS test. Just understand that a patient who is young and has persistent jaundice, hepatosplenomegaly and lymphadenopathy is a classic presentation of syphilis obtained through "vertical" transmission, ie, from mother to child.

519.

Case: You have a young woman, say 21 years of age, which presents with a positive culture for *Chlamydia* and *Neisseria*. She is sadly...become infertile...

Q) What is the dx? What two bugs are commonly implicated? And what is the treatment? Can she have another common sequelae?

A) Since one in six or one in five with PID develop permanent infertility, you must be familiar with this. The two bugs (trick questions) ARE *Chlamydia* and *Neisseria*. The treatment for *Chlamydia* is Doxycycline or Azithromycin (Zithromax). For *Neisseria*, give a single dose of Ceftriaxone or a quinolone if you wish. The common bug *Neisseria* causes accompanied muscle pain in both males and females.

520.

Case: Your poor patient who has PID (pelvic inflammatory disease) is

255

now coming back to you after two years with the triad of arthritis, red conjunctiva, and inflammation of the urethra. What is the disease?

A) Untreated PID can progress to Reiter's syndrome.

521.

Case: You see a 23 year old female patient with painless growths on her vulva. She has a sexual history with multiple partners. Diagnostic tests demonstrate that this is a VIRAL etiology. What is the bug and tx?

A) Among the MOST COMMON of the sexually transmitted diseases, you must know and understand all about HPV or human pap. virus. They can often cause these painless chancres that you can treat with CO2 laser ablation, scalpel excision, or laser therapy. MEDICAL pharmacotherapy consists of interferon therapy, 5-FU, or Podophyllin (an anti mitotic). You must make this patient come for ANNUAL pap smears! Why??

522.

KNOW that for a young woman who comes to your clinic with vaginal itching, there are three USUAL SUSPECTS:

1) Bacterial Vaginosis from Gardnerella vaginitis, Mycoplasma hominis, and about 20 other vaginal flora. Sexual contact may or may not contribute... You will see these large "clue cells" on a slide. Tx is METRO.

2) Trichomonas...definitely you will see this, no question. This is easy to spot because you see these little oval creatures swimming around in wet mount...sexually transmitted. Treat with METRO.

3) Candida...you KNOW you will see this cottage cheese looking yeast

256

with pseudohyphae on wet mount. They are often seen increasingly with DIABETICS, PREGNANCY. Treat with NYSTATIN! KNOW this is NOT sexually transmitted.

523,

Case: ON NO!!! You have a patient with HIV, a young woman, who is with child!!! What drug will you give her for her baby since about 1/3 of the patients present with transmission eventually to their babies!

A) AZT can reduce the transmission to the fetus to less than 10%!!!!

524.

Q) Again, do you know what the most common HIV disease that progresses to AIDS is? And what is the tx? Do you know what it looks like under a microscopic slide?

A) This is PCP pneumoniae. TMP-SMX is the tx of choice.

525.

Case: You see a patient named Bram Stoker, who is a young patient who traveled to the Carolinas in the USA. He comes back with a tick bite which moves from the ankles and wrists to the PALMS and SOLES. What dx are you looking at?

A) You are looking at Rocky Mountain Spotted Fever. Consider the tick bite....tell me, now, how can we distinguish this from Measles,...a

257

paramyxovirus?

526.

Q) How can you distinguish Measles from Rocky Mountain Spotted Fever?

A) In Rocky Mountain spotted Fever, typically the soles of the feet and the palms are involved. In measles, you will see the main source which involves the rash which starts at the head and moves distally from there. If you are really lucky, you will see Koplik spots in the mouth.

527.

Case: You are seeing a young patient who looks so much like measles (which is a paramyxovirus). But your attending notes that instead of the lesions spreading from the head and on downwards, this patient has the rash/lesions on the trunk and spreading to the periphery. She has had an acute high fever before the development of the rash. What is the bug in question?

A) This is HHV6 or Roseola. NOT MEASLES which starts at the HEAD!

528.

Q) Quick review to see if you are getting all the concepts down. You have a patient say 30 year old female with Lyme Disease from a camping trip (bitten by a tick and showing physical signs of a bullet lesion on the leg).. What bug and drug?

A) This is classic from Spirochete *Borrelia burgdorferi*. The tick is *Ixodes scapularis*. here, you can give doxycycline or penicillin and ceftriaxone.

KNOW that arthritis symptoms may come back again later.

529.

258

IMMUNOLOGY WILL BE EVERYWHERE IN YOUR TESTS AND CLINICS:

Q) So, you have a patient with a case of a splenectomy and CLL,  
HINT: you know his HUMORAL IMMUNE def. is diminished. What type of bugs is he most susceptible to?

A) Bugs like Neisseria, Strep pneumoniae, and H. flu which are encapsulated can cause septic shock, osteomyelitis, pneumonia.

530.

Q) Case: Next you see a patient with a diminished CELLULAR IMMUNE def like HIV, leukemias, steroids. What bugs will attack him?

A) Think about a list including CMV, Candida, PCP, Toxoplasma, Cryptosporidium, HSV.

531.

Now, you see a patient with Neutropenia (remember this is different from Leukemia). KNOW that neutropenia has many causes, including bone marrow suppression, ALL, and chemotherapy. What bugs will you likely see?

A) You will see recurrent UTIs, septic shock, sinus inflammation and the usual fungus and parasites like Candida and Cryptococci.

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532.

Q) Through all your rotations and ALL exams, you must know the basics of genetics. So please forgive me as I quiz you. You have a pregnant female with bipolar disorder. She is taking LITHIUM. Is this a good idea? What effects can happen to the FETUS? (Don't be tricked, I asked about the fetus, not the mother).

A) The fetus can suffer heart anomalies like the congenital downward displacement of the tricuspid valve with the septal and posterior leaflets being attached to the wall of the right ventricle. Bad Bad...

533.

Q) Hard one: A neonate comes to your office with a deficiency in enamel matrix formation. BESIDES syphilis, critical infections, what antibiotic is known to affect the teeth in this way if given to the pregnant mom?

A) Tetracycline

534..

Q) A female patient with chronic anxiety who is also one month pregnant comes to your office. Your medical student grabs some thalidomide, which is an anxiolytic and sedative. But....you know better. What side effect can it cause?

A) To the unborn child this drug can result in seal flippers where the arms and legs attach to the body. Teratogen!

535.

Q) This is a good case. You have a pregnant woman G2P2, who comes to your office with recurrent UTIs. Your medical student respectfully grabs some aminoglycosides to cover the gram negs. You see him hand your patient a bottle of streptomycin. What is wrong with this picture????

A) All Aminoglycosides, which require Oxygen to be absorbed, are thus NOT effective on anaerobes like Bacteroides. But, here the streptomycin can cause the baby to lose her hearing. If you don't want a HUGE LAWSUIT, then remember this fact!!!

536.

260

Case: Regarding the previous two cases, galactosemia and fructose intolerance, what changes in the diet need to be made? What is the method of inheritance?

A) For fructose intolerance, eliminate fructose AND sucrose from the diet.

For galactosemia, eliminate galactose AND lactose from the diet.

Both the disease are inherited via autosomal recessive pattern.

537.

Q) What disease am I? I affect the proximal tubule of the kidney, I am congenital, I exhibit polyuria, polydipsia, and dehydration and hypokalemia and hypophosphatemia and interrelated manifestations of the syndrome. The MOA is from solute secretion accompanied by the loss of water. Despite the dehydration that ensues, the urine is often dilute, reflecting a concentration defect that is partially caused by hypokalemia. The bouts of dehydration may be associated with fever, particularly in infants. So, what dx am I?

A) Fanconi's Syndrome (HINT: Think of a lot of hypos..., and think of PROXIMAL TUBULE!)

538.

539.

Case: A 8 year old female named Virginia Woolfe comes to you with pain on urination. PE reveals sore throat and no blood in the urine, parasites on culture, or meds. The rash on the vulva is erysipelas and cellulitis is present. What two bugs are the likely suspects?

A) Beta hemolytic Group A streptococcus!!!! And maybe Staph aureus.

261

They cause cellulitis a lot!

540.

A 10 year old girl named Agatha Christie presents with fever, myalgias, stomach pain, and a rash LIMITED TO THE LOWER EXTREMITIES! (BIG HINT!) Labs show blood in the stool, RBC casts and mild proteinuria. What disease? (HINT: Is it Rocky Mountain or SLE or other?)

A) This is NOT Rocky Mountain or SLE. It is Henoch Schonlein vasculitis! The key words are LOWER EXTREMITIES!

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541.

Case: A mother is breast feeding her baby boy Ray Bradbury. She gets Staph aureus breast feeding, thus...mastitis. Can she continue breast feeding?

A) Yes.

542.

Case: A child named H.G. Wells comes into your office as his mother fed him only cow milk from Safeway stores. What effect does it have on the child?

A) The ultra high protein concentration slams the kidneys and dehydrates the baby with concomitant malaise.

543.

Case: A 5 y.o. boy named Isaac Asimov presents with recurrent right upper lobe pneumonia. His development milestones are normal. He had an ear infection at 1 year of age and rotavirus at 3 years of age

262

according to the chart. (Is the dx an immunodeficiency disorder or a foreign body aspiration or Chediak Higashi?)

A) Most common is foreign body aspiration. An IMMUNO deficiency would have A LOT more infection.

544.

Case: A kid named Frank Herbert comes for a routine visit. Frank can move an object from hand to hand, sit by himself, imitate speech, and he can hold an M&M candy easily between his thumb and forefinger. What age is he? (Pick either 4, 6, 8, 10 months)

A) 10 months

545.

A 5 year old girl named Joyce Carol Oates swallowed a bottle of her mom's prenatal vitamins. You are the ER attending. What do you do?

A) Prenatal vitamins have high iron. Give deferoxamine!

546

Case: A patient comes with Reye's syndrome. What caused this? PE is what?

A) Aspirin is responsible, and she has fever, chills and vomiting. Liver is palpable. A) Prenatal vitamins have high iron. Give deferoxamine!

547.

Case: You have a young patient named Ernest Hemingway who comes in with a high fever, rash, and spread downward to the palms and soles. Before this, Ernest had runny noses, red eyes, and red conjunctiva. He missed all his immunization shots. What does he have? What is the most common consequence?

263

A) He has the measles, and the most common consequence is otitis media.

548.

A young lady named Ayn Rand is breast feeding her baby. But she comes to you asking what the difference is between dairy milk and her own breast milk in terms of vitamins/nutrients? What do you say?

A) As you recall, we said human milk has LESS protein, BETTER iron absorption, much MORE vitamin C, and much less vitamin K than cow milk (so this is why many women eat vit K supplements).

549.

Case: You have a 5 day old baby named J.D. Salinger. On physical exam, you note he has an asymmetric Moro reflex. The biceps carry no reflex. PE demonstrates his left arm is slightly turned inward. Which 2 cord segments are affected? AND, what is the dx?

A) C5 and C6 are damaged...this is Erb Duchenne syndrome.

550.

Case: A young boy named Michael Chrichton comes in with a hand that appears looking like a "claw". BAM! You know the dx, now tell me which cord segments are involved?

A) The Klumpke's syndrome is C7, C8 and T1 lesions.

551.

Case: I, Tommy, actually saw and treated this case with my own hands a couple of days ago...a 10 year old boy came in with recurrent

UTIs. His mother said he had a congenital disease called "Prune Belly Syndrome".

Which organ system does this dx hit often?

A) Not to be confused with Potter's syndrome, PRUNE Belly Syndrome lesions the kidneys.

552.

Case: A twenty something couple walk into your clinic with a baby that has cyclical hypoxic events that are not very predictable. But, the baby is noted to choke and gag when he feeds. Then he stops for a moment, and breathing continues until the next "event." He otherwise has a normal PE and history of birth was non traumatic. What is this disease?

A) He has choanal atresia, where his nasal obstruction may cause death from asphyxia. During attempted inspiration, the tongue is pulled to the palate, and obstruction of the oral airway results. Especially during feeding, he must "close" his mouth on the nipple, and he can't breathe!

553.

Case: You see a 14 year old boy named Tom Clancy who is vigorously itching his scalp and losing tufts of hair. With a clinic fluorescent lamp, you see patches of blue green areas lighting up in the dark when shined on his head. What bug is this?

A) This is Tinea capitis. Give an anti fungal.

554.

Case: Let us say that you have a patient and you need to know the level of reducing sugars in the urine. Do you use the Clinitest or the methylene blue stain test?

265

A) The Clinitest. The Methylene blue test helps identify white cells in the feces.

555.

Case: You are asked which test starting with the letter "B" can help identify lesions in the LOWER intestinal tract like intussusception.

A) Barium enema test

556.

Case: A 12 year old boy named John Keats comes into your office with easy bruising and petechiae all over his body. It came on suddenly after a cold. There is no hepatosplenomegaly and he has a mild fever, other than that, his PE is normal. His CBC demonstrates thrombocytopenia. Does this look like ALL or something else?

Answer: This is ITP, which follows a viral infection usually. The disease should resolve on its own in two weeks.

557.

Case: For the previous patient, John Keats, if the symptoms don't resolve in 2 weeks, what can you give him?

A) You may consider giving steroids and gamma globulins.

558.

Case: You see two patients coming in with wheezes and both look like asthma. But your attending tells you one of the patients has bronchiolitis. How can you tell the difference?

A) These two diseases present so similar, but that is why HISTORY is so important. The family history and PMH should reveal prior episodes and a family history of asthma. So, make sure you always pay attention to history and not only the HPI.

559.

Case: You have a worried 30 year old lady named Erich Maria Remarque who brings her daughter in because the daughter is 3 years old and she can copy a circle, but NOT an square. Is she behind?

A) No, most 3 year olds cannot copy a square, but can copy a circle!

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560.

Q) Other than HIV infection, which conditions should make you give a two year old the pneumococcal vaccine?

A) Think and choose steroid use, splenectomy, sickle cell anemia, kidney failure, and SLE.

561.

Q) A 9 year old male named Stephen King wakes up in the middle of the night with a facial tick and twitch and when he goes to his parents' bedroom, his attack suddenly stops. Later in the ER, his EEG is normal. Do you start seizure meds?

A) No, this is benign partial childhood epilepsy, and he will outgrow it usually. Be careful, he stayed conscious during the attack and the attack was short in duration.

562.

Two baby children named Anne Tyler and Wallace Stegner come into your office. The mothers say that during feedings, the baby Anne drools and gags and coughs! After crying, the coughing does not stop! This sounds like choanal atresia, but both are NOT. The other baby Wallace has bilious vomiting....Which kid has a tracheoesophageal fistula and which one has Duodenal atresia?

A) Wallace, which has bilious vomiting, has duodenal atresia. Anne, with the gagging and coughing has tracheoesophageal fistula.

563.

Case: Your attending hints the next baby coming in has a face that is very round and soft. He is not retarded mentally but he is short in height. His liver and kidneys are slightly large. He has a defect in his clotting but the hypoglycemia is notable. What is the disease and missing enzyme?

A) The child has Von Gierke's disease and is missing an enzyme in gluconeogenesis called glucose 6 phosphatase.

564.

Q) Appearing in 1 in 4000 births, pyloric stenosis occurs when in childhood and tell me if it has bile in the vomit?

A) Pyloric stenosis occurs a few weeks, NOT HOURS, after birth. It does NOT have bile in the vomit.

565.

Case: A male name Jeff Wiley who is 32 years old confesses to you that he lies on his tax returns and embezzles money at work. Does Federal law say you must inform the federal authorities?

A) No.

566.

Case: A pregnant female comes in with Phenylketonuria. What exact enzyme is missing?

A) Phenylalanine hydroxylase

567.

Case) You WILL see this case a lot...A 5 year old kid with a week long fever also comes in with dry cracked lips, shedding of the skin, and edema and rash all over, and cervical lymphadenopathy. What is the disease? What body part(s) does it affect?

A) Kawasaki syndrome. This is a vasculitis of medium and large coronary vessels.

568.

Case: A young patient of your named Thomas Wolfe comes in before he is entering a US college. Oh, you give him MMR, diphtheria, tetanus, polio vaccines. But, do you HAVE to give him his Hep B shot? What about his H. flu B shot?

A) No, they are recommended, but NOT required.

569.

Case: Again, you will see BILLIONS of asthma patients. Other than albuterol, many use steroids. But additional meds include Ipratropium and Zileuton and Zafirlukast. What is the MOA of these THREE meds:

Ipratropium...antimuscarinic on receptors

Zileuton...blocks lipoxygenase

Zafirlukast...blocks leukotriene receptors

570.

Case: You examine a newborn child who presents with dark lower extremities but a light pink upper extremities. PE reveals a machine gun sound over the heart. What is happening?

270

A) Patent ductus arteriosus and a Coarctation of the aorta

571.

A 21 year old mother comes in with a question about breastfeeding since she has a vaginal yeast infection. Can she use the proper drug safely?

A) Yes, topical administration of nystatin for Candida is SAFE for breastfeeding.

572.

Case: The previous breastfeeding mother asks you if she can use benzodiazepines because she is anxious about being a newborn mom and methylphenidate for her ADD for graduate schooling. She still wishes to breastfeeding. What do you say?

A) CNS drugs, Steroids, PTU, and alcohol and SOME antibiotics like ciprofloxacin and tetracyclines should be avoided for breastfeeding moms.

573.

Case: A young child named Albert Einstein comes in with cystic fibrosis. He often develops respiratory infections as sequelae. The radiologist comes back and says the trachea is deviated to the left side and you had previously heard absent sounds on the right side. What is the pathophys?

271

A) These patients often develop pulmonary infections which lead to rupture from cysts caused by *S. aureus*. Thus, a PNEUMOTHORAX ensued.

574.

Case: A young boy named Issac Newton was eating a lot of frozen flavored ice cubes and holding them in his cheeks. Later, his mother brought him to the ED because his cheeks were slightly swollen, cool, and erythematous. What is the name of this condition?

A) Fat injury from the cold is PANNICULITIS.

575.

Case: A young child patient of yours is getting his immunization shots and reacts poorly to the DTaP shot. Which one component is likely to have caused the reaction (Choices: Diphtheria/Tetanus/Pertussis)?

A) The Pertussis part is usually culpable if there is an adverse reaction.

576.

Case: An eight year old boy named Johann Kepler was playing basketball when he noticed gradual pain, stiffness in the hip area through the playing season (three months). A radiograph showed femoral head necrosis. What dx is this?

272

A) This is Legg-Calve-Perthes disease.

577.

Case: Same clinical case presentation as HY Concept 576, but this boy basketball player is quite overweight. Other than Legg Calve Perthes dx, what is likely the problem?

a) Slipped capital femoral epiphysis.

578.

Case: Now, another member of the boys' basketball team named Ernest Rutherford started playing soccer in field sprayed with insecticides. He later started the typical cholinergic symptoms of "DUMBELS" or urination, pooping, sweating, salivating, etc. Other than Atropine, what ELSE can you often use that starts with the letter, "P"?

A) Pralidoxime, which reactivates acetylcholinesterase.

579.

Case: A neonate born named Alfred Hitchcock suffered from sepsis due to E-coli. There is a strong correlation between this bug and galactossemia, which we already studied (recall hepatomegaly, hypoglycemia, jaundice?). What is the exact enzyme that is missing?

A) Galactose 1 Phosphate Uridyltransferase

580.

KNOW that Fetal Alcohol Syndrome is EVERYWHERE. So understand exactly how it presents and what organs are involved. Understand it can "look" like Cerebral Palsy and/or Down's, so watch the demonstration of the history. You need to present these cases to Child Protective Services.

581.

Case: If I gave you a case of a cyanotic newborn baby and showed you a RADIOGRAPH with a "boot shaped heart" and slight pulmonary vascular markings, what common dx is that, and which specific finding is most important to determine if the baby will survive?

A) This is the Tetralogy of Fallot and the degree of pulmonary stenosis predicts the outcome of the baby.

582.

Case: When you take tests and go into clinics, you will see common things commonly. So, say I have six patients with one of the following:

1-PKU

2-Cleft Palate

3-Clubfoot

4-Hypospadias

5-Phocomelia

6-Myelo-meningocele

Which is the MOST common one you will see?

A) It is hypospadias, one in five hundred....

583.

HARD CASE: Listen, you see a child who is a GIRL in your clinic and she looks like she is autistic. She is 4 years old. Her mother said that she seemed fine until 1 1/2 years of age and then there was neurodevelopmental arrest and then sudden regression. Her PE resembles Cerebral Palsy with loss of motor functions. Two attendings come in and tell you that it is NOT cerebral palsy or autism. They say that it only happens to girls, the gene defect is MECP2, and she has short stature and an enlarged head as a hint to you. What is this dx that starts with an "R"?

A) Rett's syndrome

584.

Case: You will definitely see this on tests and clinics. You have a girl who is 12 years old and she is short for her age group at this time. BUT...labs reveal elevated FSH and LH signaling .... what? And you note a history of UTIs and hypertension. What is this common dx which occurs in 1 in 2000 women? Can she have children? What meds do you give?

A) You WILL see Turner's syndrome. They have ovarian failure and FSH and LH will be elevated. The coarctation of the aorta is related to the HTN. Due to ovarian failure, she sadly cannot have children. You need to give her GH or somatotropin. And estrogen at the later stages.

585.

Case: You see a woman in your clinic with seizures, mental retardation, and skin lesions. Your attending TELLS you this is classic tuberous sclerosis. What is the genetics here? AD, AR, XR, XD?

A) AD, or Autosomal Dominant

586.

Case: "Tyrosine is a precursor for what amino acid?" asks your attending.

A) Dopamine (VERY CRITICAL) Think "I married a man named TYler (Tyrosine) who became a DOPe! (Dopamine)."

587.

Case: We covered the fact that PKU is a def. what enzyme.... ? And in PKU, what primary food group must the patient NOT eat?

A) This AR dx (missing phenylalanine hydroxylase) must be treated with amino acid bars (among other Rx) and you must tell your patient to avoid meat, dairy, and nuts. Plus, tell them to be aware of some sodas and potato chips, which are high in aspartame and phenylalanine.

588.

A five year old boy patient of yours comes in with an overdose of a

276

common drug used for bed wetting (starts with letter "i"). How do you think he will present?

A) This is imipramine, a tricyclic. Overdose presents with lethargy, epilepsy, heart rhythm irregularities.

589.

Case: A 30 month old child presents with small bowel obstruction seen on x-ray. His PE has bad colicky abdominal pain with bloody diarrhea and vomiting. You feel a mass in the epigastrium. What is going on?

A) This is intussusception. Very common.

590.

Case: Recall what I said, to REALLY LEARN and RETAIN, you must study the differential diagnoses TOGETHER. So, the last case was intussusception. But there is another dx that is similar in presentation and the most common congenital dx of the ileum that involves the vitelline duct and ectopic pancreatic and gastric tissue with the GI bleeding. What is this???

A) Meckel's diverticulum (very diff to diagnose at birth)

591.

Case: HARD, but doable: You are in a city in Israel where the

277

incidence of this dx is 6 in 1000, very very common. A 5 year old boy is slowly starting to lose his protective reflexes, and becoming ataxic (demyelination). He is getting worsening respiratory problems. This disease name starts with the letter "K" and sounds like the word "Cab". What is the deficient enzyme here? What is the inheritance?

A) This is Krabbe dx. Krabbe disease is an autosomal recessive sphingolipidosis caused by deficient activity of the lysosomal hydrolase galactosylceramide beta-galactosidase (GALC). GALC degrades galactosylceramide, a major component of myelin. The elevated levels lead to widespread destruction of oligodendroglia in the CNS and to subsequent demyelination. Death from respiratory failure often results.

592.

Case: In the same "category" as the previous concept is this...you see a patient at 6 months of age with hepatosplenomegaly, lung problems, failure to thrive and psychomotor retardation. Your attending sadly tells you the patient will likely die by age 3. What is this dx that starts with the letter N.... and then P..... ? What enzyme is deficient?

A) This is Nieman Pick Disease and results from the deficient activity of sphingomyelinase, a lysosomal enzyme encoded by a gene located on chromosome bands 11p15.1-p15.4. The enzymatic defect results in pathologic accumulation of sphingomyelin (which is a ceramide phospholipid) and other lipids in the monocyte-macrophage system.

593.

Case: This HYer is so close but so far from the LAST HYer concept. So,

278

listen up...you have another patient who is an Ashkenazi Jew with hepatosplenomegaly, pancytopenia, and mild skeletal disease, and you are thinking Nieman Pick, but this is NOT it...this is Gaucher disease. OK, so what enzyme is missing?

A) Gaucher disease is a lipid storage disease, characterized by the deposition of glucocerebroside in cells of the macrophage-monocyte system. Deficiency of a specific lysosomal hydrolase, acid beta-glucocerebrosidase leads to the symptoms. Unlike Nieman Pick disease, there IS a medical treatment! You must get everything right for your patients!

594.

Another similar case! This time you got close enough to know the ataxia, lost reflexes, slurred speech is Metachromatic leukodystrophy. So close to Krabbe's and Gaucher's in presentation...what is the enzyme missing?

A) arylsulfatase A!!!

595.

Case: A young patient comes in with a triad of meningocele, spina bifida, and hydrocephalus. He is 8 months old with a large head. What is the name of the disease? And what is the Rx?

A) This is Arnold Chiari syndrome and you need to give acetazolamide.

596.

There are two distinct signs that a baby was shaken abusively...ie

"shaken baby syndrome". What are they? You MUST recognize them for the child's sake!

A) You may see a floppy baby with retinal hemorrhages and subdural hematoma.

597.

Case: You are seeing the delivery of a baby from a 18 year old young female with SLE. What is her baby most at risk for (name the organ system)?

A) SLE is assoc. with complete heart block towards the child.

598.

Case: An attending nephrologist comes in and explains to you that he has a patient with a defect in the proximal renal tubular reabsorption of phosphate. The patient is a young child and is short for his age. He tells you this is Vitamin D resistant rickets. What is the inheritance type?

A) X-linked dominant

599.

Case: ANOTHER child comes in with vitamin D resistant rickets. The most common rickets in the the USA. How will the child walk towards you?

A) The rickets causes bow leggedness and will result in a duck waddle.

600.

Case: I sadly saw this one myself....but let's say you see a deceased newborn infant with a prominent occiput and low set ears. His hands

are clenched with rocker bottom feet. Which trisomy is this? 13, 18, or 21???

A) This is Trisomy 18

601.

Case: Everyone in clinics and from all the USMLE tests are saying some of the versions heavily quiz physio and graphs and major homeostasis concepts. So...if you have any patient with V. cholera infection and they present with dehydration, OR if you have a patient with Diabetes IDDM with ketoacidosis, what will you initially do? Guess first before peeking at the answer!

A) Replace fluid and electrolytes first.

602.

281

Case: For the patients with dehydration, do a careful history to find out just why they are ill. Give me two classic findings on PE suggestive of dehydration.

A) Oliguria, (low urine output), and acute weight loss!

603.

Case: Regarding homeostasis and water balance, tell me some major causes that are CHRONIC which present with dehydration!

A) We already discussed diabetes, but also think of congenital adrenal hyperplasia, diabetes INSIPIDUS, severe sore throat (which prevents desire to swallow), cystic fibrosis. Did you get any of these?

604.

Case: As a patient of yours continues to LOSE fluid balance, he will present first with tachycardia, then his or her respiration will speed up. Why is this?

A) Often metabolic acidosis ensues, so you have compensatory respiratory alkalosis!

605.

Case: What is the most common form of dehydration (hyponatremic, hypernatremic, or isotonic)?

A) ISOtonic!! So this means that water losses roughly equal sodium losses.

606.

Case: You have a patient with severe fluid loss...what will the PE present like regarding his skin?

A) When you press his fingertips, capillary refill will be greater than 3 secs. Also, his or her mucous membranes will be dry (open their mouths and LOOK). If it is a baby, the fontanelles will be sunken!

607.

Case: Again, your patient is water deprived for a long time...what will the Urine osmolarity and specific gravity look like?

A) Both values will be severely ELEVATED. think why...and so will the BUN/Creatine ratio.

608.

Case: Again, lumpin along, what will your water deprived patient show on his PE for the bicarb level?

A) Secondary to acidosis, his bicarbonate will be decreased! BUT, KNOW that if he is VOMITING all the time, his body will face metabolic ALKALOSIS.

609.

Case: This concept is SO CRITICAL...tell me some differences between INCREASED vs. DECREASED anion gap! Be specific!

A) Increased anion gap includes: Hyperphosphatemia, HYPOkalemia, HYPOcalcemia, HYPOmagnesemia. Massive diarrhea, lactic acidosis, DKA, aspirin overdose, chronic kidney failure.

DECREASED anion gap includes: HYPERkalemia, HYPERcalcemia, HYPERmagnesemia, low albumin, Li overdose.

610.

Case: Say I give you a case study and tell you this is a prerenal failure. What exact LAB value threshold will you like to see to confirm

283

this?

A) A BUN/Creatinine ratio over 20.

611.

Case: Lumpin...let's say I present a case to you and the patient has a capillary finger refill of 1 sec and is very mildly dehydrated after a tough rugby match. Should I give him IV boluses?

A) No, for mild cases, just do ORT or oral rehydration therapy. Kinda sorta like giving GATORADE juice, you need to give approx. a solution with 90 mEq/L Na, 20 mEq/L of K, and 20 g/L of glucose. DO NOT give or pick just free water to rehydrate!

612.

Case: You have a patient with EDEMA from CHF. You do recall our discussion of the MOA (Mech. of Action)?

A) The CHF results in decreased renal blood flow. Thus, you retain Na and water, resulting in EDEMA

613.

Case: Lumpin along....another patient of yours comes in with EDEMA. He or she has LIVER DISEASE. What is the MOA?

A) Decreased albumin synthesis from liver dx results in edema here from decreased oncotic pressure.

614.

Case: Lumpin still. Another pt. with edema comes in with associated protein malabsorption syndromes. What is the MOA?

A) So much protein malabsorption results again in decreased plasma albumin...thus lower oncotic pressure...and ensuing EDEMA!

615.

Case: Another EDEMATOUS individual. This time he/she has one of the NEPHROTIC syndromes. What is the MOA of edema?

A) The loss of albumin and protein clotting factors results in again lowered albumin in the blood, thus lowered oncotic pressure...and thus EDEMA!

616.

Case: A sexually active female walks into your office. She has a lot of edema. What is the main thing you are thinking of?

A) PREGNANCY or OCPs!

617.

Case: Lumpin still...your physio prof comes in and tells you your edematous patient has a cardiac origin. HOW will his PE present?

A) SOB, cyanosis, sweating with eating suggest a congenital cardiac origin to the edema.

618.

Case: True or False: Can a severe allergic reaction mimick other forms of edema?

A) True or Yes...

619.

Case: Speaking of edema, there is a famous often tested disease that presents as a patient young or old that comes in with a deep rash that

285

started on the buttocks or lower legs. There is edema on the hands and feet. About half of the patients initially presented with Upper Respiratory Infections. What is the pathophys of this disease that we once spoke of? What meds should you give? (Hint: name starts with H... and is named after someone.)

A) This is Henoch Schonlein Purpura. About half the cases were preceded by a upper respiratory infection. The etiology of HSP involves the vascular deposition of IgA immune complexes. More specifically, the immune complexes are composed of IgA1 and IgA2 and are produced by peripheral B lymphocytes. The circulating complexes become insoluble, are deposited in the walls of small vessels (arteries, capillaries, venules), and activate complement, most likely by the alternative pathway. Thus, group A streptococci, varicella, hepatitis B, Epstein-Barr virus, parvovirus B19, Mycoplasma, Campylobacter, and Yersinia are often picked as inciting factors. Treat against the bug if persistent (after culture), and give prednisone for the inflammation. They also need pain relief like Tylenol or Ibuprofen for some pain.

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620.

Case: For the previously discussed cases of edema, which labs will help you differentiate between a liver, cardiac, renal, etc. source?

A) For the liver, do the liver function tests. For the cardiac, check for cardiomegaly and pulmonary edema on X-ray. Renal sources will point to inconsistent serum electrolytes and proteinuria.

621.

Case: We discussed patients with water deprivation from vomiting and diarrhea in a patient. What exact lab value for the Na indicates Hyponatremia?

286

A) Serum sodium under 130 mEq/L!

622.

Case: Because you will treat dehydrated patients all day long in clinics, you need to know what are the most serious consequences of protracted diarrhea/vomiting? What labs will the USMLE give you to assess the causes?

A) Beware of Hypovolemic shock as the patient's mental status worsens with decreased reflexes. The most serious consequence is seizures and cessation of breathing. Other than BMP, you need blood glucose, liver function tests, protein and lipid levels.

623.

KNOW every kind of hyponatremia and how the little arrows will point with respect to Urine Na, Urine specific gravity, etc. Here, let me ask just ONE question: For hyponatremia due to CHF, what is the value of the Urine Na and Urine specific gravity?

A) In CHF hyponatremia, urine Na is DOWN, and Urine specific gravity is UP. Remember the subtle differences...like KNOW that if you have ADDISON'S dx, the urine Na will be up and the urine specific gravity will be DOWN! Think why!

624.

Case: Since HYPERkalemia is so dangerous, tell me a few of the causes of hyperkalemia!

A) Addison's Disease, Acidosis, Dehydration (severe), Spironolactone drug therapy, and too much K infusion, tubular kidney damage leading to improper K excretion.

625.

Case: Despite warnings, your patient becomes Hyperkalemic. What does his/her EKG look like?

A) You will see T wave elevation, then muting of P waves, then QRS complex widening, and ST segment depression. Deadly V-fib can result!

626.

Hi brothers and sisters,

I need to address a vital question for everyone because it continues to be asked.

1) Many are asking about their personal assessments and how to gauge progress. This is an excellent question. This is SO VALUABLE because you will then KNOW if you are making progress or not. There is a LOOSE connection between say [QBank](#) performance and the actual exam. But, the material does correlate in the sense that if your [QBank](#) score is moving higher slowly and steadily, then you KNOW you are at least retaining some information. But, if you do not have [QBank](#) and/or cannot afford it, you can use Q-Book or another popular source. The most important issue is not really what you are getting in the absolute percentage, but whether that number is increasing over a set time. As an aside, I mentioned what I found to be accurate as to [QBank](#) scores... Most of the students scoring at least a 50% on [QBank](#) passed the exam. Now, the [QBank](#) leans heavily on Pathology and Pathophysiology, and asks questions with a slightly different slant than the actual test. So, if you are UNLUCKY and get say series of questions on Embryology and Immunology and you completely skipped these two subjects, then you will find that your 50% on [QBank](#) was not relevant. So, that is what a lot of repeat test takers are saying...that their second or third etc. test was ENTIRELY different from the initial

288

ones. Although I am not sure of this since I did not see their tests, I am certain that the NBME will not allow someone to receive the same or similar questions from his/her previous test. So much of the computer based testing is CENTERED on avoiding ANY POSSIBLE shortcuts like this.

Therefore...gauge your progress with weekly question banks like a [QBank](#) . Then, as I mentioned before, do the USMLE Sample Questions for ALL THREE STEPS that are found on the NBME website (These are free to download). Then, do the [Kaplan](#) Simulated CD that is found EVERYWHERE, your roommate probably has a copy. Also please complete the two NBME self assessment exams found on their website (There a cost to this that the NBME charges, but I think it is worth it to gauge your progress). Also, do the RETIRED NBME Step 1 questions. They can be found everywhere too for free. I think even ValueMD has a copy of it. You definitely WILL benefit from doing this to test yourself. For example, if the RETIRED questions have 100 biochemistry questions, then split them into 4 blocks of 25 questions each. Then do one block every weekend a month before the exam for each subject topic. Then graph your results to see if you are studying effectively. I know the question format is different, but the concepts tested were HIGHLY related to the actual exam in many cases. Also, try to do some of my questions too as a way to gauge your progress. Maybe you can make notecards of them and get a filebox. If you get the flashcard question right, then place the notecard in the end of the file so you will not see it again right away. If you get the item wrong, then place the notecard towards the front of the pack so you will see it again sooner. This is a highly effective way to study, and you need to keep up the repetitions daily. If you start skipping them, you will start forgetting them.

627. Dear Family,

Many are writing that they are suffering under severe anxiety and are asking me for advice. This is again a VERY good question.

289

1) The first thing to know is that this test is very "coachable" and "doable". Plus, unless you are pushing 50 years of age, you have time! Even if it takes one or two years to pass Step 1, 30 years from now, this event will seem like a distant dream.

But there needs to exist the right circumstances or it just cannot work. Still, many are shouldering the burden of work, family (kids), AND suffering some personal crisis. Then, they mention their test is in a month. My heart and everyone else's breaks upon hearing this, but we must ask God for the right TIME to PASS Step 1. If life events are not going to permit you the time to study, it is like trying to climb Mt. Everest tomorrow without any preparation or running the 26.3 mile marathon in a week. Both tasks are doable, but if you just broke your leg, you cannot run next week. We must all pray to get that necessary block of time required. Some are trying to lift their anxiety with serious alcohol and anti-anxiety and then sleeping all day long instead of studying. Not good. Although a few can use some anti anxiety medication in MODERATION, this often has the effect of putting you to sleep, which will make the anxiety 100 times worse after you awaken and lose a day of studying. Better again to WAIT until the right moment. If you are working and you cannot find anyone like a family member to live with and feed you while you are studying, please reconsider taking the test until the right time presents itself. You will only put more agony onto yourself if you do not pass...

628. Case: Although we glanced over this in Pharm, tell me what is the Rx for a first time HIV patient of yours and tell me the MOA of the drugs. Then, we will next quickly go over the MAIN dx of HIV and the Rx.

A) In clinics, we like to give 2 nucleoside analogs like AZT (Zidovudine) and Lamivudine....PLUS a protease inhibitor like Lopinavir or Rotinavir (These drugs usually end with suffix -avir). Recall that the nucleoside

analogs are THYMIDINE analogs which blocks virus replication via REVERSE TRANSCRIPTASE. The protease inhibitors work by blocking the modification of precursor polyproteins responsible for synthesis of reverse transcriptase and HIV-1 protease itself.

629.

Case: The next patient comes in with a positive ELISA and Western Blot for HIV. Do you recall at least TWO VIRAL ANTIGENS in the peripheral blood to also confirm HIV infection?

A) Look for GP41 and P24 antigen.

630.

Quick, what was the MOST COMMON worry you have with HIV patients (i.e. main dx)? What is the Rx? (Hint, this bug hits the lungs and can cause SPONTANEOUS PNEUMOTHORAX!)

A) PCP (Pneumocystis carinii). Give TMP/SMX as the Drug of choice. This can be LUMPED by thinking all have popular 3 letter abbreviations (HIV-PCP-TMP/SMX).

631.

Case: Now, the HIV positive person comes in with headaches and fever. A radiograph is shown that has ring lesions and midline shift. He was scratched by a cat last month. What COMMON bug starting with the letter "T" are you thinking of? What is the Rx? What are some side effects? THINK before you look at the answer below!

A) This is Toxoplasmosis gondii, give a folic acid antagonist like Pyrimethamine. Watch for his HTN in intracranium and possible seizure activity!

632.

291

Case: Now, another HIV positive pt comes with fever, and MILD headaches and a radiograph with hydrocephalus. What COMMON bug starting with the letter "C" am I looking at? What drug or Rx?

A) This is highly confused with PCP and Toxoplasma. The bug I am seeing here is Cryptococcus meningitis. MAKE SURE YOU KNOW THE DIFFERENCES COLD LIKE YOUR NAME, MOTHER'S NAME, etc. Treat with Amphotericin B. (Mnemonic: When you think of a "Crypt" (cemetery relation), think of a crazy band that is obsessed with the DEAD, and likes to play their AMPlified (Amp B) electric guitars inside the scary Crypt.)

633.

Case: Quick, the NEXT RELATED COMMON bug with HIV attacks the eyes, leading to loss of vision and retina detachment. What bug and drug? No hints here...except it starts again with the letter "C". Give morphology of bug...too.

A) This is CMV retinitis. YOU must give GANCICLOVIR! This is part of the HERPESVIRUS family. It is double stranded, linear, enveloped. (My mnemonic....think....CMV stands for California Motorcycle Vehicle GANG) [Think and imagine a CMV gang riding down the highway getting flies stuck in their eyes and slowly losing their eyesight!!!]

634.

Case: Now an ENT doctor refers to you a patient with HIV and oral thrush. What is this super common bug and drug in HIV? Can you identify IT PRECISELY under a microscope? If not, LOOK for it in a Microbiology book/atlas!

A) This is Candida. Treat with an "azole" like Ketoconazole, Clotrimazole or Nystatin.

635.

Case: OK, an HIV patient of yours has really BAD diarrhea. Your acid-fast staining of stool demonstrates red-stained round oocysts against a blue-green background. White and red blood cells should not be seen in the stool. What super common opportunistic bug starting with the letter "C" is here? Rx?

A) This is Cryptosporidiosis. Drug treatment is difficult, but the HAART treatment for HIV is helping a lot. Give them symptomatic treatment with LOPERAMIDE or Kaopectate.

636.

Case: You have a pregnant woman that asks you the difference if any between taking a teratogen in the first trimester or third trimester. What do you say?

A) Taking a teratogen in the first trimester usually damages organogenesis, while taking a teratogen in the third trimester often slows the CNS development and the growth of the baby!

637.

Case: Some pregnant women come to your office and asks what is the MOST COMMON maternal disorder that is teratogenic. What do you say?

A) Diabetes mellitus, one in ten diabetic females' babies have a birth defect.

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638.

Case: A woman who is African American with history of benign fibroids is pregnant. She asks if this could hurt the baby. What do you answer?

293

A) Unfortunately, the fibroids can "squeeze" the baby, and present with abnormal facies, club feet, or abnormal lie/breech presentation.

639.

Case: What is the inheritance pattern for the most common blood coagulation disorder named after a doctor with the name "V"?

A) So so common and heavily tested in clinic and exams, this is Von Willebrand's disease, and is AD or an Autosomal dominant disorder. Recall, you must tell the pt. she has a 50% chance of passing this to her children!

640.

Case: You have a patient with the classic triad of low intelligence, epilepsy, and raised tumorlike angiofibromas on the skin. What is the inheritance pattern of the dx? What is the name of the dx? What is the pathology exactly?

A) AD, this is Tuberous Sclerosis (we mentioned it long ago once), The protein tuberin is dys-regulated, leading to hamartomas and tumors in all organs of the body, notably on the skin.

641.

Case: This can be both autosomal dominant and recessive inheritance, but the common-"ness" of it demands a question. A patient comes in with a history of recurrent episodes of acute pancreatitis and eruptive xanthomas. He is at increased risk of coronary disease. He lives a sedentary life and eats only greasy hamburgers. Name SOME common causes of his condition, both genetic and non-genetic.

A) HyperTRIGlyceridemia, this is caused by uncontrolled Diabetes and obesity. Two genetic causes of high TG are lipoprotein lipase [LPL]

294

deficiency and apolipoprotein [apo] C-II deficiency leading to triglyceride (TG) elevations that are exceedingly elevated.

642.

OK, OK, you will be asked a lot of inheritance pattern questions. That is because you will have to explain to your patients their risks of passing their dx to their children. So, for all autosomal dominant disorders, we discussed they are STRUCTURAL PROTEIN disorders. They have 50% inheritance patterns.

Also, most autosomal recessive disorders are enzyme and "-emia" (blood stuff) related. Except for three common "-emias", that is: Hereditary spherocytosis, von Willebrand disease, Protein C deficiency (REMEMBER AT LEAST THESE THREE EXCEPTIONS), the enzyme and "-emia" diseases are autosomal recessive. The exceptions to the blood "emia" rule is initials H.P.V. as seen above. This is an abbreviation "mnemonic". AR is 25% passed on.

643.

Case: Anemia, jaundice, and splenomegaly. This class triad is seen with a patient who is young and has that classic palpable spleen. You see a slide which has these round RBCs. What is the disease?

A) We JUST spoke of it. Hereditary spherocytosis presents in this way. Don't forget this NBME/attending/resident favorite!!! Try to recall the MOA of the giant spleen.

644.

Q) I may have asked this long long ago, but what is the problem in Hereditary spherocytosis?

A) Alpha or Beta Spectrin def. (The alpha form is related to AR inheritance.) But know the Beta form is more common as is AD.

645.

Case: A nonsmoking patient of yours comes in and has panacinar emphysema. She also had chronic bronchitis. What protease is malfunctioning? What is the disease and Rx? What is the pathophysiology?

A) This is ALPHA 1 ANTITRYPSIN DEFICIENCY. The genetic defect of alpha 1 antitrypsin deficiency results in a molecule that cannot be released from its production site in hepatocytes. Low serum levels of the protein result in low alveolar concentrations, where the molecule normally would serve as protection against antiproteases. The resulting protease excess destroys alveolar walls and causes emphysema. Give a drug branded called Prolastin to replace the deficiency.

646.

Case: Couple A comes in and you note that the male is achondroplastic (dwarfism/extremely short stature). The female is pregnant, she asks what is the chance her baby is going to be have achondroplasia. What do you say?

A) This is AD, autosomal dominant inheritance, so the male will transmit the gene to half his offspring.

647.

Case: Couple B comes in and you note that BOTH are suffer from achondroplasia (dwarfism). They are asking about the inheritance to their children. What do you say?

A) Again, this is 50% BECAUSE the homozygous form usually does NOT survive to birth. So the 50% HETEROzygous form will have the phenotype of dwarfism, but half will be normal.

296

648.

Q) We are discussing achondroplasia. What is the MOA of this AD disease?

A) The MOA or pathophys is such that fibroblast growth factors are structurally related proteins affected...and are associated with cell growth, migration, wound healing, and angiogenesis. At the cellular level, their function is mediated by transmembrane tyrosine kinase receptors, known as fibroblast growth factor receptors (FGFR). Mutation in FGFR3 gene is responsible for the achondroplasia, or dwarfism.

649.

Case: You research 100 achondroplasia patients and only 10 had any history of the dx in the family... why???

A) Don't forget the mech. of SPONTANEOUS MUTATION. This disease is noted for 90% new mutations in the lineage.

650.

Case: You see a patient with hypertension, infections, hemorrhage and renal stones. You feel a large mass on one side of the body. What mode of inheritance is this dx? What is the dx?

A) AD inheritance, Adult Polycystic Kidney Disease presents as above with large cysts in the kidneys. Renal failure will usually result by age 60. Radiographs will show large circles or cysts.

Tommy's HY 651-700(skip 666,683)

651.

Case: A patient of yours with the previously mentioned ADPKD dies of a lesion around the vessels in the brain. Given a CT scan, could you point to the area that is lesioned?

A) Commonly, ADPKD patients die of a berry aneurysm, even if their renal failure is controlled.

652.

Case: You have a female patient who works in a pet shop who is 25 years old and sexually active. She just gave birth to a baby boy who is 7 weeks old. The baby comes in with otitis media, wheezing, and conjunctivitis. What is the drug you prescribe? What is the bug?

A) So common, this is Chlamydia pneumonia, caught by the baby through the birth canal. They may be asymptomatic for the first few weeks of life! Give tetracycline as drug of choice. (Mnemonic: Think and imagine...MY pet CLAM named RICK who swallowed my pet TETRA fish.) [ My-Mycoplams, CLAM- ChLAMydia, RICK-Rickettsia...and TETRA-Tetracycline ]

653.

Case: A 4 year old child pt. presents with slowly progressive difficulty walking. His eyes look like he has telangiectasias or conjunctivitis. There is some difficulty in respiration, and he is drooling a little. Histology shows that some Purkinje cells are being lost in his cerebellum.

1) What is happening to his Immunoglobulin levels?

- 2) What is the dx?
- 3) What is the primary pathophys defect?
- 4) Is this autosomal recessive or dominant?

A) 1) IgA is low, IgG and IgE are elevated.

2) He sadly has ataxia telangiectasia.

3) The primary defect is a problem with a DNA processing or repair protein.

4) AR

654.

Case: You have a male young patient coming over and over to your office with pulmonary infections. His stools are reported to be fatty and foul smelling. The question is, "Which vitamin (B1, C, or D) are you most concerned about supplementation (you need to give this)?" And the dx name please. True or false: The disease is X-linked recessive?

A) Due to exocrine pancreas lesions, the CYSTIC FIBROSIS patient has trouble digesting fat soluble vitamins like Vitamin D. False...the cystic fibrosis is autosomal recessive! KNOW THIS COLD!!!!!!!

655.

Case: For the previously discussed pt. with CYSTIC FIBROSIS, what is the most preferred and a specific test for diagnosis?

A) A Sweat Chloride test

656.

Q) Quickly, without pause, tell me the most common bug to affect our previously talked about CYSTIC FIBROSIS patient's LUNGS (that will cause pneumonia). What is the Rx? THIS BUG IS SO COMMON, so BE READY TO identify the morphology (gram stain, etc.) and the appearance on a petri dish.

A) Pseudomonas aeruginosa. Treat is varied and you can often use a penicillin type- Piperacillin/Tazobactam and combine it often with Gentamycin. Or you can pick Aztreonam. I have seen Imipenem and cilastatin work as well. Oh, also know it smells like grapes on a petri dish. Love you all my brothers and sisters!!!

657.

Case: Another boy enters your clinic with symptoms mimicking CYSTIC FIBROSIS like fatty stools, fat soluble vitamin deficiency, failure to thrive. But the boy DOES NOT have any history of respiratory infections. What is the dx that also starts with the letter "C" that is confused with cystic fibrosis?

A) Be aware of celiac disease, NO RECURRENT RESPIRATORY INFECTIONS distinguish it from cystic fibrosis!!!

658.

Case: Yet another patient walks into your clinic with symptoms

300

mimicking cystic fibrosis like the recurrent respiratory infections, failure to thrive. BUT, this time you note he has NO MALABSORPTION symptoms. Name one of a few type of dx that can cause this! (Hint: there is one with three words, the first letter is "c", second letter is "g")

A) This is Chronic Granulomatous Disease

659.

Q) There was a question on images and how a LOT of students just omit their studies of them. Some later say that you can answer the question without the image. BUT BEWARE!!! The NBME carefully and fairly puts in the images because a panel of experts KNEW the students HAD to have the image to carefully distinguish it from closely related diseases. SO, be familiar, and CLOSELY study the image the NBME gives you. It will make a difference over and over.

660.

Q) YOU HAVE TO understand that congenital syphilis infection presents in STAGES, so know ALL THE stages! If I gave you a case of LATE STAGE CONGENITAL SYPHILLIS, could you describe for me all that you know???

A) LATE: Breaks in the skin, saber shins, saddle nose, neuromuscular paralysis.

661.

Q) NOW, I just asked you late stage symptoms of congenital syphilis. Tell me the EARLY symptoms of congenital syphilis. You have to be able to spot this.

301

A) EARLY: Fever, hepatomegaly, failure to thrive, anemia, rash

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662.

Case: This is SO CLASSIC: A young girl comes into your office lacking her immunization shots. She had a rash that went from the face then progressed down to the toes. This should ALREADY BE FAMILIAR to you. What are some associated symptoms?? Tell me also the morphology IF it is a bug.

A) This is Rubella or German measles. Associated symptoms are fever, lymphadenopathy, and arthritis like symptoms. A TOGAVIRUS, it is enveloped, square shaped, SINGLE stranded positive sense.

663.

Case: A young girl comes in with a positive serological test for a bug that is the ONLY double stranded RNA virus you need to worry about for the Step 1 and in clinics. She is vomiting and has diarrhea. Every child in her day care has these symptoms. What is the exact morphology of said bug? What is the bug? What is the most serious sequelae? What is the drug?

A) This is ROTAVIRUS, so common and very contagious. This is Double stranded, NO envelope, square shaped, with segmentation. WATCH OUT for dehydration! And there is NO current medication for it. It resolves in about a week.

664.

Q) If asked, what are the 5 exact categories that determine the APGAR score?

A) Heart rate (2 max points if over 100), Respiration (crying?), Muscle tone (Flexion), Reflexes (can she/he cough), and Color (Blue or Pink)

665.

Q) KEY, when studying, try to keep things simple if possible...so let me ask you this--If you are trying to distinguish a lumbar puncture between viral or bacterial meningitis, what is the most easily seen lab result to differentiate?

A) A viral usually has NORMAL glucose levels.

667.

Case: A young female comes in with her 16 month old child who just had a tonic clonic seizure after an episode of otitis media and fever. She is very scared that he will have a lifelong illness. There is no history of seizures in the family. What do you tell her about the risks?

A) A FEBRILE SEIZURE usually does NOT progress to chronic epileptic seizure activity, esp. if there is no family history.

668.

Case: YOU will definitely see "skin" stuff in clinics and the USMLE. So,

303

let's say you see a one year old male with fever and chills and a RASH. He was OK until an ABRUPT onset of the fever lead straight into a fine erythematous maculopapular eruption rash that started on this stomach and waist and spread all over his body. His nose is runny. (His fever suddenly dropped as the rash started). Picture is given...and the dx starts with letter "R".

A) This is Roseola infection, a HHV 6 infection (herpes virus).

669.

Case: HINT: I am thinking of a TOGAVIRUS. Today we have a 3 year old without immunizations. He has a rash and pain behind the ears. He also has cervical lymph node enlargement. This picture "looks" like the Roseola infant but this is NOT the same. The rash started on the patient's face. What is the morphology? What dx is this?

A) This is clearly Rubella. The lack of immunizations and the "togavirus" hint should clinch your pick. We are square shaped, enveloped, single stranded, and linear.

670.

Case: A girl comes in with a rash that is viral in origin and started on the face bilaterally with a bright red appearance. She had a low temp for the last day. This virus is associated with fetal hydrops in utero. What is the bug, dx, and morphology?

A) This "slapped cheek" disease is Parvovirus B19, 5th Disease, and it is the ONLY Single stranded DNA virus. No envelope.

304

671.

As I have been doing, every time you see any question, think of ALL the secondaries of the bug like morphology, Rx, side effects of Rx, etc., how it looks like under scope, specific stain, etc. Do this automatically EVERY SINGLE TIME!

672.

Case: Another viral rash, this time you see the rash start on the face and spread down to the feet. AND you see these gray white dots on the mucal mucosa that has blue centers and red areolae. The young patient missed her immune shots. Bug and morphology? Name the OTHER bugs in the virus family.

A) This is Measles. The KOPLIK's spots are specific. It is an RNA virus, single stranded, neg. sense, linear. It is a helix shaped bug, and it is part of PARAMYXOVIRUS family. with Parainfluenza, RSV, and Mumps as part of the family.

673.

Case: This bug virus is in the same family as the rhinovirus. This virus causes a rash that unlike measles, starts in the mouth (oral ulcers) and hands and feet and spreads out. It is common in spring and summer. What is bug and morphology and dx name?

A) This is Coxsackie A virus causing "Hand foot mouth disease". It is a PICORNAVIRUS, NO envelope, single stranded, POSitive sense, square shaped.

305

674.

Case: A young girl's brother had the same illness that was a viral rash a month ago. Now, the sister has a similar lesion that is highly contagious, with pruritic rashes that have several stages of lesions at the same time. The rashes are "in patches" all around the body. The girl is considered infectious until all the vesicles are "crusted over", about a week after the onset. What is the viral culture test? Bug and morphology? Late sequelae?

A) This is Varicella. The "various stages present at the same time" is highly specific for the dx. People use a Tzanck prep for culture. It is a HERPESVIRUS! So, it is a DNA virus, yes to enveloped, double stranded linear morphology. Herpes zoster/shingles is a late reactivation sequelae that hits the dorsal root ganglion.

675.

Case: Of the viral rashes/eruptions, what is the Rx? What should be avoided?

A) Mainly supportive like Tylenol for the fever, and antihistamines for the itching. Don't give aspirin because you may get Reye's syndrome.

676.

Case: This is a good one. What are the two most common bacterial skin rashes? (Give the names of the two bugs and the two common clinical conditions).

A) Group A Beta hemolytic streptococcus and Staph aureus. Think of Bullous impetigo and Nonbullous impetigo. Please do an IMAGE SEARCH on the internet or look in an atlas to view the appearance if you cannot visualize them.

677.

Q) What is the pathophys of these bacterial infections of the skin, leading to rashes? Very important.

A) Listen, while intact skin commonly is resistant to colonization or infection by S aureus or Group A Beta Hemolytic Streptococcus (GABHS), these bacteria can be introduced from the environment and colonize the cutaneous surface only transiently. Experimental studies have shown that inoculation of multiple strains of GABHS onto the surface of volunteer subjects did not produce cutaneous disease unless skin disruption had occurred. The teichoic acid adhesins for GABHS and S aureus require the epithelial cell receptor component, FIBRONECTIN, for colonization. These fibronectin receptors are unavailable on intact skin; however, skin disruption may reveal fibronectin receptors and allow for colonization or invasion in these disrupted surfaces. Factors that can modify the usual skin flora and facilitate transient colonization by GABHS and S aureus include high temperature or humidity, preexisting cutaneous disease, young age, or recent antibiotic treatment, so don't forget the above!

678.

Q) As we recently discussed in a concept, impetigo is "usually" superficial cutaneous cuts and abrasions while cellulitis often is WARM and red and moves into deep dermis tissues. What two bugs are

307

responsible for cellulitis most commonly?

A) Same as impetigo, GABHS bacteria and Staph aureus. Now a distant third due to good immunizations in the US is H. flu.

679.

Q) What are a couple of Rx choices for cellulitis infection?

A) Again, treat mainly for the gram positive bugs like antistaphylococci drugs ox, clox, diclox, and nafcillin (from [Kaplan](#) review lectures), and cephalexin and or amoxicillin w/clavulanic acid (brand name augmentin).

680.

Case: Other common rashes are of course..."jock itch", scalp itch, "ringworm", "athlete's foot". What are the associated bugs? How do they look like? Rx?

A) These are the "MET" fungi Microsporum, Epidermophyton, Trichophyton and are part of the Tinea group of infections. Tinea versicolor, Tinea pedis, Tinea cruris (jockitch), Tinea capitis, Tinea corporis (body ringworm). These are often HYPOpigmented in color, not as red as the other rashes. Give the patient one of the azoles, like ketoconazole or terbinafine (both of which block ergosterol synthesis).

681.

Case: You have a another RASH, lasting at least 5 days, on a baby.

308

What is the likely bug?

A) This one is CANDIDA ALBICANS. Can you recall EXACTLY how it looks like under microscope?

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682.

Case: You see a patient who is INTENSELY ITCHING all over her body and has rashes too. She and her family recently traveled around the world. Called the "seven year itch" (not after the Marilyn Monroe movie), this bug shows eosinophilia and is highly contagious. This is NOT the previously mentioned bugs but is caused by ... ? Rx?

A) This is Scabies caused by Sarcoptes scabiei. The treatment is varied but you can pick an antiparasitic like IVERMECTIN.

683.

684.

Case: Everyone saw this sometime. Can be confused with impetigo, and other skin rashes that ITCH badly. This...is called LICE, you see it from a microscopic specimen looking like a little "tick". What is the pathophys of this?

A) Also called Pediculosis, Lice are buggie parasites that die of starvation within one and a half weeks of removal from their human host. Lice feed on human blood after piercing the skin and injecting saliva. A mature female lays 3-6 eggs, also called nits, per day. Nits are white and less than 1 mm long. Nits hatch in 8-10 days, reach

309

maturity in 12-15 days. Types of lice include pediculosis capitis (head lice), pediculosis corporis (body lice), and pediculosis pubis (pubic lice, sometimes called crabs).

685.

Case: What is the drug of choice for the just talked about LICE? What is the MOA?

A) Permethrin (Elimite) is preferred. It is very effective in killing adult lice and nymphs, but not as effective in killing nits (eggs). Permethrin is a neurotoxin that causes paralysis and death in parasites. It is available as 5% cream

686.

Case: Here we see another pruritic rash on a female teenage patient of yours. Your attending hints it is a bug that is associated with sebaceous glands. What is the bug, drugs and what test must you order to save yourself a possible lawsuit from side effects of the drug tetracycline? (sorry I gave one of the drugs away)

A) This is COMMON ACNE. It is associated with folliculitis type lesions and drugs range from benzoyl peroxide to Retin A cream to tetracycline. Give a pregnancy HCG test for the Retin A and tetracycline because they are teratogenic.

687.

Q) What is the treatment for diabetes insipidus? What is the difference between nephrogenic and non nephrogenic DI?

310

A) Desmopressin acetate (DDAVP) a vasopressin analogue. NON nephrogenic DI is associated commonly with head trauma, brain tumors, or craniopharyngomas. Nephrogenic DI is assoc. with collecting ducts not responding to ADH.

688.

Case: A patient comes in with symptoms of short stature. Name some causes of this...what are you thinking of? (Mnemonic: GRAPES)

A) Think of GRAPES when you see a short person in your clinic!  
Growth hormone, R enal disease (Vit D assoc), A chondroplasia (could be spontaneous mutation), P rimary hypothyroidism, E ating/absorption problems (eating disorders and absorption problems like celiac sprue), "S" for "S"ystic Fibrosis (actually cystic fibrosis)

689.

Case: Two other short stature young people come into your clinic. One boy is taking steroids for muscle growth and the other girl has an XO karyotype. What is the MOA of the short stature here?

A) So many things cause short stature. Here, we see steroids/hypercortisolism having short stature effect. And also know Turner's syndrome can cause short stature!!!!

690.

Case: A 22 year old pt named Cressida comes into your office with

311

bulging eyes and anxiety and tachycardia and anxiety. Does she have Papillary carcinoma or Graves disease?

A) Graves is the MCC (most common cause) of hyperthyroidism and the only one that presents with bilateral bulging eyes. The MOA is IgG binding to TSH, increasing release of TH. Papillary carcinoma is usually nonsecreting COLD nodules.

691.

Case: Why couldn't the previous case be Hashimoto's thyroiditis?

A) Because Hashimoto's = HYPOTHYROIDISM. It has a swollen thyroid and antimicrobial antibodies.

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692.

Q) Suppose you are asked by the NBME or an attending that the MOA of levodopa is the conversion to dopamine. In those neurons, does dopamine turn into tyrosine or norepinephrine?

A) Norepinephrine.

693.

Case: You are given a case of either a schizophrenic patient or Parkinson's patient. Tell ME IN GREAT EXACT DETAIL the MOA of norepinephrine synthesis involving dopamine...

A) First, you have the aa tyrosine. It is converted to DOPA thru HYDROXYLATION. After, DOPA turns into dopamine via DECARBOXYLATION. Next, dopamine is transported to the adrenal junction, where it turns into Norepinephrine via HYDROXYLATION. Finally, in the adrenal medulla in the kidney, the norepi changes to epinephrine via METHYLATION. REPEAT THE ABOVE LIKE 10,000 times really fast until you can write it from scratch!

694.

Q) The ureter's epithelium is derived from which of the following: ECTODERM, MESODERM, or ENDODERM?

Ans) Mesoderm

695.

Q) Which arteries supply the urinary bladder? Is it Internal pudendal?

A) NO! It is the internal iliacs.

696.

Q) T or F: The fasting serum gastrin is abnormal in patients infected with H. pylori.

A) False!! Duodenal ulcer patients have NORMAL FASTING serum gastrin. The gastrin moves up after MEALS.

697.

Case: A sickle cell patient has recurrent infections and a positive Quellung rxn and optochin sensitivity for the bug. What is the most common bug?

A) The above describes *S. pneumo*. The encapsulated bugs have a positive Quellung rxn.

698.

Case: A boy named Jack London comes in with recurrent pneumonia. What enzyme, NADPH oxidase or Glucose 6 phos dehydrogenase is lacking?

A) NADPH oxidase. He may have CGD.

699.

Case: Nut aspirations are oh so common. So tell us about the distal blood content of an almond nut lodged in the right lung lobe. Is it left shifted or does it have a lowered pH? What is the V/Q ratio?

A) The tissue is perfused but not ventilated so the V/Q hits zero. Thus, it has a LOWERED pH.

700.

Case: You see a patient with POLYCYTHEMIA VERA! You will see this at some point in your life!!! So, tell me the levels in the blood of

314

lymphocytes and neutrophils. Which is increased? or are both increased?

A) This is a MYELOPROLIFERATIVE DISORDER, so the myeloid lines are increased (neutrophils/RBCs/platelets), while the lymphocyte line is often NORMAL in lab values.

701.

Case: A girl named Catherine came in with a sore throat which then went away in a week. Then she started urinating RBC casts and "smoky" urine. What is the bug and histological finding?

A) This is streptococcal infection. First you have the pharyngitis then the poststrep glomerulonephritis. Commonly, you see subepithelial bumps on histo section.

702.

Case: A patient of yours has PERIPHERAL nerve demyelination. Are the oligodendrocytes affected? or is it the Schwann cells?

A) PERIPHERAL nerve demyelination is associated with SCHWANN cell lesions, oligodendrocytes lesions are associated with the CENTRAL myelination process.

703.

Case: Are axons preserved in Multiple Sclerosis?

315

A) Surprisingly, they ARE. The lesion is the DEMYELINATION.

704.

Case: A female patient has CNS symptoms like difficulty seeing, weakness and fatigue that present as attacks separated by time. The dx is progressive and deadly. What is the dx?

A) These ARE the CNS lesions present in MULTIPLE SCLEROSIS.

705.

Case: Patient of yours has Conn's syndrome and thus hypertension. Tell us the likely electrolyte problems (is renin up or down, etc.) and how this is different from SECONDARY hyperaldosteronism.

A) This aldosterone secreting tumor gives high blood Na, low K, and thus low RENIN. SECONDARY hyperaldosteronism has HIGH RENIN from too much stimulus by angiotensin II.

706.

Case: A patient of yours named Daige Kurosawa is Japanese-Korean. He lived in Japan most of his life. He has pain after eating large meals and coughs sometimes and feels "full" after eating only 2 mini sushi rolls and Korean dried squid mixed in preservatives. He has stomach cancer. Which area of the stomach is most likely to have the lesion?

A) the antropyloric region.

707.

Case: After a patient of yours had an MI, your med student asks if the first diagnostic change is proliferation of fibroblasts. Is he right?

A) He is wrong. The first change evident is wavy change of myocytes and intercellular edema. The fibroblasts come weeks afterwards!

708.

Case: After the MI (myocardial infarction) began, when will you see the infiltration of neutrophils to the site of infarction?

A) Within around 12 hours of the MI onset.

709.

Case: After how long will you see a soft yellow plaque on the endocardial section after an acute MI?

A) around one week.

710.

Q) If given a histo slide of a glomerulus, could you point precisely to the exact cells that release RENIN? If you are not sure, please look it up on a Histology atlas. What about the basement membrane? The macula densa?

317

711.

Case: You see a friend bleeding from a knife wound to the neck. The carotid artery (left) is lesioned. Which vertebrae can you push the carotid a. against the anterior tubercle to STOP the bleeding (C2, C3, C4, C5, C6, C7)?

A) C6

712.

Case: You see a baby having a hard time breast feeding and breathing. He gags every time he tries to drink milk. You see on X-ray the most common cause of tracheoophageal fistula. What is the MOA of the defect?

A) The tracheoesophageal septum failed to fuse in utero

713.

Case: A mother at risk failed to take her folic acid pills and gave birth to a child with myelomeningocele. Is this due to failure of spinal bone body or pedicle or what...that failed to form correctly?

A) Neither option given is correct. The correct answer is failure of fusion of the vertebral arches.

714.

318

Case: A patient named Aeschylus comes in with loss of sensation on his medial thigh area and one of his scrotums. What main nerve that starts with the letter "I" is lesioned?

A) Ilioinguinal n. NOT the Pudendal or Genitofemoral or Lateral cutaneous nerves.

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715.

Case: A man is lesioned in his brain such that his motor movements are hypertonic. Movements exhibit rigidity. What area of the brain is injured that starts with letter "B" and has two words?

A) Basal ganglia

716.

Case: An older man has an aneurysm that lesions the cerebral cortex at the interhemisphere fissure at the cross section of the central sulcus (sorry I don't have a picture, but look at an atlas). Is he having trouble moving what part of his body?

A) His legs. Review the homunculus of the brain.

717.

Case: You medical student says that serologic tests ARE useful for detection of Mycobacterium leprae and tuberculosis detection. Is she correct?

319

A) She is WRONG. You need clinical presentation, skin tests, and Ziehl Neelsen testing.

718.

Dear Family, As you continue doing the questions here, make sure to repeat the information and KEEP GOOD TRACK of what you are missing. If you are missing ALL the anatomy questions, you KNOW you need to review anatomy. If you are missing all the BIOCHEM questions, you know you need to review that. Assess the %correct manually so you know if you are RETAINING the information in these posts. IF one week later you are getting a higher percentage of questions correct, then you are golden. If not, you need to schedule shorter repetition schedules. That way, you will know if you are progressing in your knowledge.

719.

Case: A case of scarlet fever progressed to poststrept. glomerulonephritis. Tell us about the MOA and the likely pathogen.

A) Likely you are looking and GABHS or Group A beta hemolytic streptococcus. And the MOA is deposition of immune complexes and the attraction of complement, C3 and IgG to the site which triggers damage to the cells of the glomerulus.

720.

Case: What kind of glomerular lesion is caused by HIV and AIDS?

A) Focal Segmental Glomerulosclerosis, w/ HTN and proteinuria.

320

721.

Which two amino acids are ketogenic only? Glucogenic only?

A) Ketogenic: Leucine and Lysine.....Gluconeogenic only: Valine and Glutamate

722.

Give us three ways that an oncogenic virus can induce cancer.

A) Amplification (of proto-oncogenes leading to overexpression), translocation induction, and inactivation of suppression.

723.

T or F...Retroviruses integrate themselves without enzymes into the host's DNA.

A) False

724.

Q) Parathyroid cells are derived from WHAT ARCHES, and what are the main types of cells found?

A) 3rd and 4th arches. Dominant cell is the CHIEF cell. See it in a histo slide.

321

725.

Case: You see a forty year old with fatty tissue around the eyes (look sorta wrinkly and puffy). The LDL levels are ELEVATED. What is the dx? What is the MOA? What is at least ONE comorbid condition?

A) Xanthelasma. The LDL is high with foamy macrophages. Often associated with Primary Biliary Cirrhosis causes inability to excrete cholesterol.

726.

Case: What is the MOA of gout in alcoholics?

A) The associated ketoacid production and lactic acid production competitively blocks uric acid secretion, supporting gout with elevated serum urate.

727.

Case: You have a patient who cannot excrete ammonia. What happens to the acid and bicarb levels?

A) AMMONIA binds acid  $H^+$  and is the major way the body rids itself of  $H^+$ . Thus,  $H^+$  serum levels increase and bicarb. decreases.

728.

322

Case: VERY IMPORTANT: What is the difference between incidence and prevalence EXACTLY in biostatistics?

A) Incidence is the number of NEW cases of a disease within a given population in a year while prevalence is the number of people affected in a given number of people within a given year.

729.

Case: Regarding Alzheimer's disease, which of the 3 choices is the most strongly correlated with the MOA of the dx? (pick either thiamine def. or choline acetyltransferase def or acetylcholinesterase def.)

A) Although many causes are related to Alzheimer's dx, the lack of Ach from low levels of choline acetyltransferase are correlated highly.

730.

Case: Quick, what drug starting with the letter "f", blocks dihydrotestosterone synthesis?

A) Finasteride

731.

Case: Which drug, Tamoxifen or Mifepristone, blocks the stimulation of estrogen response genes in the nucleus?

323

A) Tamoxifen

732.

q) Which one, estrogen or mifepristone, blocks progesterone and causes menstruation? MOA?

A) Mifepristone, its inhibition of progesterone induces bleeding during the luteal phase and in early pregnancy by releasing endogenous prostaglandins from the endometrium.

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733.

Q) Which drug, flutamide (NOT to be confused with finasteride), cyproterone, or mifepristone is a NON-steroidal antagonist on androgen receptors?

A) The answer is FLUTAMIDE, cyproterone is steroidal. Finasteride, binds the 5 alpha reductase to achieve a similar reduction in dihydrotestosterone levels.

734.

Q) Does high serum levels of ketoconazole do ANYTHING to the synthesis of testosterone synthesis in the testes?

A) It inhibits the formation of testosterone in the testes

324

735.

Case: Given an experiment about osmotic gradients, which one of these three (glucose, Na, or BUN) affect the gradient the most? What about the least?

A) Na affects it the most, review the gradient equation. BUN has the least because it is considered a PERMEABLE solute and has NO effect on the water gradient.

736.

q) Does anything happen to PTH levels in 1 alpha hydroxylase deficiency?

A) Increases...due to decreased Ca levels in the blood from lack of 25 OH vit D activation.

737.

Q) Given a PTH level, what ion is most commonly seen to be elevated in tumorogenesis?

A) Calcium. HYPERcalcemia is most common ion elevated in cancers.

738.

Q) Is PTH severely elevated in the early stages of osteoporosis? Think

325

about the MOA.

A) Curiously, the PTH levels are normal and so is Calcium levels! The dx is GRADUAL in progression, so the labs are often NORMAL!

739.

Case: A young patient moves away from the mother by herself and then comes back by herself to the mother for hugs. What age milestone is she?

A) 16 months to 2 years. This is rapprochement.

740.

Q) This is a good one. The EXTERNAL urethral sphincter is located in which of the spaces (The Superficial Perineal Space or the Deep Perineal Space)?

A) DEEP Perineal Space.

741.

Case: A friend of yours asks how does a man prevent from peeing when he ejaculates? What do you say with respect to the SPHINCTERS, which one, the EXTERNAL or INTERNAL is closed during ejaculation?

A) INTERNAL sphincter is closed, the EXTERNAL is OPEN!

326

742.

Case: You see a person with Coccidiomycosis. How does this case differ from PARAcoccidiomycosis on physical exam? Recall where in the US you see each one the most.

A) The coccidiomycosis look like spheres in tissue, while paracoccidiomycosis has budding fungi that looks like spokes coming out of a wheel.

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743.

Case: Pt with HIV+ comes to you because of excessive bone marrow suppression with AZT. What new side effect is associated with his new drug regimen consisting of didanosine and zalcitabine he may encounter?

A) Several motor and sensory neuropathy and some get pancreatitis.

744.

Case: Which nerve provides innervation to the umbilicus (T5, T6, T10, or T12)?

A) T10, know ALL the major landmarks.

745.

Q) KNOW both the lac operon and the bacterial repressor protein

327

dogma for Molecular Bio. So...for the repressor protein, it binds to the operator region of DNA to regulate gene transcription in the bacteria. There is a sigma and rho factor in the bacterial RNA polymerase. Which one is involved in INITIATION VS. TERMINATION?

A) Sigma = INITIATION, Rho factor = TERMINATION

746.

Hard question in molecular bio: On the ribosomal binding sites, the A site is usually taken by aminoacyl t-RNA, NOT peptidyl t-RNA (for the P site). When is the ONLY time aminoacyl t RNA lands into the P site?

A) during PROTEIN initiation with fMet-tRNA.

747.

Q) Bacterial ribosomes bind to WHAT on their corresponding mRNA strands? Starts with letter "S" and is named after someone.

A) Shine Delgarno sequence.

748.

Q) Following up with...release factors are involved in chain termination when the ribosome encounters what codon?

A) NONSENSE codons...

749.

Q) Molecular bio: Is the ATTENUATOR involved in which--initiation, propagation, or termination--of bacterial proteins?

A) Termination, Think also about ENHANCERS, PROMOTORS, etc.

750.

Case: Two patients of yours come into your office. One has Hepatitis B and another has Tylenol overdose. What is the difference in the mech. of action of tissue damage?

A) Disorders such as Hep B involve viral antigen stim. and thus cytotoxic CD8 T-cells which damage tissue via perforin breakage of membranes. Tylenol or acetaminophen toxicity involve free radicals as the MOA for tissue damage. What other drugs/diseases work via the free radical MOA?

751.

Dear Family,

Please understand that RETENTION is everything. Please do NOT overestimate your retention but do NOT underestimate it either. After every one of these "concepts" and after EVERY single fact you read, "ASK yourself...what did I just read? Can I explain it to myself and others?" If you are simply reading and NOT retaining, you are learning but you will not be able to answer the USMLE questions. For example, you may understand a dx located on a specific chromosome number, but unless you repeat it to yourself often enough, you will not be able to answer the specific question on the USMLE. Love, Tommy.

329

752.

Pretend I showed you a figure of the kidney on a slide with arrows. Where exactly does gluconeogenesis take place (cortex or medulla)?

A) Cortex, beside the liver, the kidney can also undergo gluconeogenesis!

753.

Case: Can you describe for me what the heck "PHOSPHOCREATINE" is? What is it used for? What is the related MOA?

A) This is an alternative emergency energy storage in brain, heart and muscle tissue. Phosphocreatine kinase ADDS a phosphate group to ADP from creatine to make ATP!

754.

Dear Family, I have heard of some of us passing the Step 1 after 8 attempts. Seriously...Eight! So do not give up, unless you know that you have NO TIME to study. If you completely lack English skills, AND you do NOT have ANY time to study, you MUST either WAIT until God gives you the time or you must listen to His calling you to another way to serve Him. He may have other plans for you besides an MD. I am NOT saying give up at any time, but LISTEN to Him. If you find yourself without ANY POSSIBLE study time due to family issues, etc. then you need to pray and ask if He wants you to serve His Will in another way. Remember, with some of my close friends at schools where only 20% pass the Step 1 on a given block of time, there are a lot of tears...and know that you are NOT alone. As Helen Keller wrote..."The world is full of suffering, but is also full of the overcoming of it." Please trust God and keep a BROAD perspective. Think of others

330

and how to serve OTHERS, and the rest will follow. Love always,  
Tommy

755.

Speaking of the former PHOSPHOCREATINE, what is its exact relation as a test for heart damage indications? Give timing and levels too.

A) When you see an elevated CK-MB or phosphocreatinine MB, KNOW that after an MI, the blood levels start to elevate 6 hours after an MI, PEAK at around 25 hours, and then gradually decrease to normal after three days.

756.

What cycle am I thinking of?....The making of lactic acid during anaerobic glycolysis in RBCs, or via MUSCLE cells through oxygen debt, and its return to the LIVER and KIDNEY for conversion to glucose through gluconeogenesis is CALLED WHAT?

A) This often tested and pimped question in clinics is the CORI CYCLE.

757.

Case: Given a choice, which specific organ and cell has glycerol kinase activity?

A) LIVER HEPATOCYTES have glycerol kinase activity, which makes glycerol 3 phosphate from glycerol.

758.

331

True or False, Insulin is required for glucose uptake into cells, but is NOT required for fatty acid uptake into cells.

False! insulin is needed for fatty acid uptake into adipose tissue cells!

759.

T or F: Do brain neurons contain mitochondria?

A) True, they do, and rely on glucose primarily for energy.

760.

Case: As a general rule, which amino acid is likely to be on the INSIDE of a soluble protein molecule? (Arginine, Valine, or Serine)?

A) Look for NON-POLAR AA's on the inside, like VALINE. JUST KNOW THE BASICS, no structure memorization please.

761.

Just to test your understanding, what BUFFERING PAIR of MOLECULES constitutes the majority of total blood buffering capacity?

A)  $\text{HCO}_3^-/\text{H}_2\text{CO}_3$ ...remember the stuff we discussed on BICARB??

762.

Case: A woman comes to your office secondary amenorrhea. She has had MULTIPLE dilatation and curettages. This clue leads you to what dx cause?

A) This is associated with Asherman's syndrome, related to uterus scarring.

763.

Another patient comes in with secondary amenorrhea, but this time it is caused by HYPOPITUITARISM. What is the most common cause of the hypopituitarism? What else does the patient likely suffer from?

A) Ans: A pituitary ADENOMA. She would also have stuff assoc. with the pituitary malfunction like HYPOTHROIDISM and hypocortisolism, lowered ADH, oxytocin, etc.

764.

Case: Another secondary amenorrheic marathon runner comes into your clinic. Progesterone challenge test does not produce menses because the uterus is NOT primed by the estradiol from lowered GnRH. Are her back bones at any risk?

A) She is at risk for osteoporosis, due to loss of estradiol's prevention of bone resorption.

765.

I am steadily getting e-mails about some of you guys passing, and still some failing and feeling awful. This is a time when we need to pray and gain perspective and a broad outlook spiritually on life.

First, there is a book called, "Don't sweat the small stuff, and it is all just small stuff." Coming off of Zen-like material, it echoes the fact that there is SO much suffering in life, no matter what or who you talk to. I recall talking to some who passed Step 1 without any problems and THEY DO NOT APPRECIATE their jobs that much. But I recently talked to a guy yesterday when I was on call who after college parked

333

cars and did ODD jobs...then he went to a med school, and coming to the US, he had a difficult time passing step 1. BUT..after he succeeded, it all felt SO GOOD to him, like climbing a BIG MOUNTAIN.

I think this is TRUE. If you do NOT gain a inner peace and wisdom, you will feel sad no matter what, it is only a matter of time. If you pass step 1 and then fail Step 2, you will feel a deep sting. If you become a doctor and then get sued by on of your patients and you did nothing wrong, then you also will get slammed emotionally. I know this super smart plastic surgeon who is SO good, but because he did not TRIP up on the USMLEs, he does not know failure and NOW, one of his friends told me that he and his wife are arguing a lot. Someone else who PASSED easily may feel they HAVE to be the head of their department, and if they fail, then they will feel that sting, someone else who passed the steps will try to win the NOBEL prize, and they may likely fail... Life is gonna sting everyone, and we all need to pray together and work together to find our spiritual core and the peace. If not, this is only the beginning... a child who is not as bright, a child who becomes very sick, a husband/wife that is unfaithful, etc.

Thus, for those who are having so much trouble, understand that I heard the NBME is not raising the PASS level because even the USA students are now starting for unknown reasons to falter on the Step 1. Really. The test is nearly impossible and does not measure how smart you are. It only measure how well you can do on the Step 1 given your burdens in life. And IMGs HAVE a rougher time, without a doubt!

To conclude, I echo Cadusma and others who are helping us all out. Wait until you are really ready. You will know when you are. Use these concepts and other question bank sources to assess your progress. Even with the famous [First Aid](#), you need to make flashcards and/or have a friend QUIZ you while you are not looking. I even heard of a group who MADE UP A GAME LIKE TRIVIAL PURSUIT (A BOARD

GAME), and asked questions with each other.

There are SO many ways to go. A thousand different paths to take. And I CANNOT PROMISE THAT EVERYONE WILL MAKE IT this year...but you must listen to God and your circumstances.

With the competition SO TOUGH in the USA, the NBME has the ability to make the test so impossible. If we lived ONE HUNDRED YEARS AGO, I heard that there was NO NBME or board test. Even the MCAT was not born yet. Immigrants were swarming into the USA and doctors were in dire need! And science was not very advanced, and so much of the pharmacology was just primitive, like a mixture of heroin and alcohol for pain. Seriously.. It is just the weird time that we live in.

The step 1 may be "too extreme", of that I agree. But since now we must accept fate, we need to embrace a quiet hope that we will eventually find our way. Consider people who work all their lives to build something throughout history only to have it all wiped away without their control. We are still lucky to live now, in AD 2004.

So, to finish, sit back, continue emailing this board for suggestions, and try to find a way to tailor your understanding and "gaining" knowledge with your own situation and abilities. I absolutely believe those with determination WILL pass, but I cannot say WHEN. But, I do agree that God may have another plan for you, and if you cannot find the time to study (if you have a sick mother at home and/or you MUST work to feed your younger brother), God is calling you to a different path...at least for now. There is a great MYSTERY in life, and accepting it and still feeling hope and a "stillness" is what life is all about.

766.

Case: An older man with emphysema had intubation after ARDS. He

335

is on a ventilator set at 100% oxygen for a long time with a high RR. After 4 hours, what dangerous dx can happen to his lungs?

A) Pulmonary fibrosis is a risk with high dose O2 for ARDS patients.

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767.

Q) Does muscarinic receptor activation work via G protein receptors or ion channels?

A) principally by G-proteins, NOT ion channels.

768.

If I give you a picture, Are enzymes synthesized in cells bodies or axons?

A) cell bodies, b/c that is where the ribosomes are.

769.

T or F: Drugs that act on nicotinic sites are related to anesthesia before intubation. (if false, is the answer muscarinic?)

A) True..nicotinic.

770.

YOU HAVE to be able to DRAW out a cholinergic synapse from memory because it is so often tested everywhere. So True or False:

The ACh release is blocked by toxin of Clostridium tetanae.

False: BE AWARE: it is Clostridium botulism!!!

771.

True or False: The reuptake of ACh into the PRESynaptic receptor is the critical mech. in ACh termination signaling.

False!!! It is the acetylcholinesterase that is most crucial. CHOLINE is important for synthesis of NEW ACh.

772.

True or False: ChAT synthesizes ACh from the substrates acetyl coenzyme A and choline and it exists in BOTH the central and peripheral nervous systems.

A) True

773.

Which one, or BOTH, are found on a DISEASED individual?  
Fusobacterium and/or  
Pseudomonas

A) Pseudomonas

774.

Case: A patient of yours suffers from Clostridium difficle. What

337

mammalian protein do the TOXINS target? Are the C. difficile aerobic?  
T/F You can use a broad spectrum aminoglycoside to treat.

A) Rho protein

NO, they are anaerobic

False, aminoglycosides do NOT work on anaerobes.

775.

Case: Like the previous case, the Gram stain from C. difficile infected cells would reveal light pink rods. True or False?

False, they are Gram positive, so the stain is PURPLE.

776.

True or False: A release of a package of ACh causes a miniature end plate potential.

True

777.

1) T or F: Olfactory pathways are directly lined to gustatory pathways.

2) T or F: Olfactory pathways lack ANY DIRECT connections to the thalamus.

A1) False

A2) True

778.

1-True or False: Vasopressin secretion fr. Post. Pituitary is decreased by somatostatin release.

2-True or False: Vasopressin secretion is increased during long aerobic exercise.

A1) False!

A2) True!

779.

Case; A pt presents with symptoms of Wilson's dx (copper in liver). Symptoms include rigidity, chorea, tremor. WHERE is the lesion? What is diagnostic lesion in the eye? Liver?

A) Likely the Globus Pallidus and Putamen. Copper deposition in cornea gives Kayser Fleishcher rings (look at it on an atlas). Copper in liver leads to cirrhosis!

780.

Case: A pt of yours has a claw hand position. Plus, he is showing symptoms of Horner's syndrome (miosis, anhidrosis). What does he have (main cord roots affected)? What nerve root is responsible for the Horner's?

A) This ulner nerve lesion hits the C7, C8, T1 (Klumpke's palsy). The T1

339

lesion of the sympathetic fibers causes the Horner's signs.

781.

Q) The Klumpke's palsy we just discussed hits adduction or abduction of the brachial plexus?

A) ABduction.

782.

A 38 year old man waltzes into your clinic complaining of SOB and heart palpitations. You listen to his heart and hear a systolic ejection flow murmur, and a wide, split, FIXED S2 sound. You see an ECG showing atrial fibrillation and right ventricular hypertrophy. He is not cyanotic and his symptoms JUST evolved. What is this most likely? What direction is the "shunt"?

A) This is Atrial Septal Defect, a common left to right shunt.

783.

Which one is more worrisome in your 1 year old pt.?

Case1) Na=170, K=4, CO2=14

OR

Case2) Na=135, K=5, CO2=4 ???

Case 2 is worse. Usually, you need to really watch the CO<sub>2</sub> for signs of acidosis. You can breath fast to make yourself respiratory alkalotic to about 14, BUT you need bad METABOLIC ACIDOSIS to lower it all the way to 4 mEq/L. Reperfuse FAST.

784.

Q) What is material used for Tetanus shots? Intramuscular toxoid or Oral attenuated live virus?

A) Intramuscular toxoid

785.

What is the material used to make H. flu B shots (you even have to know stuff like this!)?

A) Intramuscular polysaccharide with adjuvant protein.

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786.

341

Q) Is MMR vaccine subcutaneous attenuated live virus or intramuscular whole killed bacilli?

A) subcutaneous attenuated live virus

787.

Case: A female patient of yours accidentally took phenytoin and "Depakote" during her pregnancy. What "commonly named disease" is her baby at risk for? Is it heart problems or CNS problems, etc.? Try to remember this often quizzed concept.

A) The risk is fetal hydantoin syndrome. This encompasses growth retardation, hypoplastic distal phalanges, and CNS malformations. WATCH OUT!

788.

Give a series of symptoms associated with maternal lupus erythematosus that can impact the baby? Think hard first before looking at the answer below!

A) Lupus patients which YOU WILL SEE, have a risk of giving their kids skin lesions and heart block (AV block), and septal defects, and BAD stuff like transposition of the great arteries. Look also for anti Ro antibodies among others to diagnose the neonate.

789.

Case: You are faced with an attending asking you what is the better med for a patient with edema secondary to nephrotic syndrome (25%

342

albumin w/ diuretic, packed red blood cells, or whole blood)?

A) Volume per Volume, the 25% albumin BOOSTS the oncotic pressure and will quickly relieve the edema.

790.

T or F: Your med student gives a fast injection of 5% Dextrose in saline thinking it may affect extracellular edema. Will it?

A) NO! This solution will GO STRAIGHT through the vessel walls (in and out) and have little effect on the relative pressures.

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791.

Case: QUICKLY, what vit def. is PELLAGRA associated w/?

A) NIACIN def.

792.

case: Pt of yours comes in with painful limp. He is 6 years old and of normal weight. Is he likely to have Legg-Calve Perthes dx. or Slipped Capital Femoral Epiphysis? MOA of dx please?

A) Answer is Legg Calve Perthes dx. Look at the age and weight. SCFE has older kids 10-15 years old and they are often obese. The MOA is

343

avascular necrosis of the femoral head.

793.

Case: Young patient with scoliosis of the spine. What age range is most common, 5-10 years old OR 10-16 years old?

A) LIKE the SCFE, it is more common in puberty ages, 10-16 years old. This is a minor emergency, they may need bracing of the back.

794.

Case: A patient of yours overdoses on phenothiazine. what is this drug used for? What antidote can you give that is SO COMMON that the family does not need a prescription for????

A) This drug is used for schizophrenia, and you can give BENADRYL, among other meds.

795.

Case: A jaundiced neonate...in front of you NOW! Which of the following contributed to this? (mother ingesting aspirin during pregnancy OR mother ingesting phenobarbital during pregnancy) close to term? THINK about mechanisms! MOA please?!

A) mother ingesting aspirin compete with binding sites on albumin with bilirubin! THUS, ingestion of aspirin can cause neonatal jaundice. However, phenobarbital may actually help in some cases of jaundice because it revs up the liver's ability to induce glucuronyl transferase.

344

796.

Case: A patient of yours, a 50 year old man, comes waltzing in with signs of ACOUSTIC SCHWANNOMA! Tell me his clinical presentation and tell me WHICH CRANIAL NERVE is lesioned commonly?

A) He may show cerebellar lesions, some hearing trouble, loss of sensory function on one side of the face. The lesion of the EIGHTH cranial nerve will yield a POSITIVE ROMBERG SIGN among others symptoms.

797.

Case: I read that even the great Dr. Sigmund Freud suffered this as he smoked a pipe. Others can get this dx. from chewing tobacco. A histo section shows squamous cell carcinoma of the lip. Typically, what is seen CLINCALLY and on CBC? What is dx?

A) Look for nonmotile mass on lips, swollen submental, submandibular, deep cervical, lymph nodes for the SQUAMOUS CELL CARCINOMA of the lip. Look for anemia on CBC in labs.

798.

Case: You are a family practice doctor who sees a neonate in the clinic. He has CONJUGATED hyperbilirubinemia (conjugated is always abnormal), and increased alk phos on labs, low blood albumin, NO urobilinogen in urine, and clay colored stools. What does he commonly have? Can we use PHOTOTHERAPY?

345

A) This is BILIARY ATRESIA, a very common cause of DIRECT elevated bilirubin. YOU CANNOT USE phototherapy for conjugated bilirubin.

799.

Case: So common, you will need to understand...a 24 year old male who was playing soccer got swiped laterally on his knee. What EXACT three structures are damaged??? can you point to them on x ray?

A) The unhappy triad consists of lesion of the ACL, medial meniscus, and medial collateral ligament!

800.

Case: You are faced with a week old neonate with bilious vomiting, NO stools, and abdominal distention. MOA of this dx please that starts with the letter H... in the formal name?

A) Hirschsprung's Disease. We see abnormal development of myenteric nerves with Meissner's and Auerbach's plexus ill formed. Thus, stools are stuck in the intestine. You may have to do a surgical excision of the aganglionic segments!

801.

Q) Other than CNS injuries at birth, which one (Hypomagnesemia or

346

Hypocalcemia) is likely to cause seizures in the neonate?

A) HYPOcalcemia.

802.

There are two stories I personally heard of of students facing Step 1. One guy was doing extremely well on his tests and then got AN IMPOSSIBLE version of step 1 and failed...barely. This reminds me of a lot of you e mailing me and letting me know how one time you got an easy version and then you got an IMPOSSIBLE version.

The other can happen too. Another guy in my school failed the first time, and then took it again. BUT...he said he did NOT study much for the second time and I believe him. BUT...HE PASSED easily. I think with a 216 or something. But when asked why, he said he "just happened" to get a version of the test which was more straightforward and less weird out of the world molecularbiophsiobiochem questions that MANY SAID THEY NEVER EVER SAW ANYWHERE IN ANY REVIEW BOOK OR HY CONCEPT BANK! So, you HAVE to assume there is A LOT of luck in this..

If you fail badly because a test was all about Immunoanatomy or some weirdo Microhistology (I can imagine this that the NBME "seems" to develop new science areas entirely)! then just stop and know that many have faced the same out of this world test form.

Just today, someone told me that even though they did ALL of QBank over and over I think 1.5 times, they did not get a SINGLE QUESTION that even resembled their actual TEST!!!! So, as I said these concepts and QBank and others are there to assess your retention since the material overlaps. But, the moral here is pray and be ready for

347

anything and everything!

After hearing about some of the exams some of the fellow family got, I and other higher scorers may have FAILED TOO! Even browsing other forums, I read of USA super students at the top of their class who are AOA suddenly FAILING the STEP 1!!! It seems impossible but you have to believe me that this is TRUE!

This Step 1 is unlike any other test I think on earth. There is SO MUCH VARIATION! Again, today some guy emailed me and PMd me that my HY concepts were so helpful and he saw so many concepts repeated...but then others said their test was something out of the twilight zone and could not even figure out what the questions were asking!!!

Tommy

803.

Dear family, I also know another student because HE happens to be a friend of mine. He FAILED a couple of times on the boards and did not know WHAT THE HECK to do. BUT, then, he got this SUPER HIGH SCORE. There is NO reason at all for me to lie to you, but I could not believe this...this happened a few years ago and he got a 255!! That is TRUE. A 255/99!!! He is now a radiology resident in New York City and cannot still believe it. And his scores on Step 2 and 3 were FAR FAR lower! And this guy was just a normal student! And he said his test Step 1, totally aimed right where he put his focus on. For example, he said he got SO MANY SIDE EFFECT questions. One after the other... And a lot of "Buzzwords" that are by nature, easily because the differential is narrow. Like Koplick spots...they are SO specific for measles... (I heard in the last couple of years the NBME started targeting their "buzzword" questions and erasing a lot of them. It does

348

not mean forget about them, but realize the NBME is always trying to make the test HARDER, not easier.

So the message that is so important is that you JUST DON'T KNOW what is gonna happen! That is the point of this impossible test. Just have faith that the impossible will become "possible".

Recall that even though some stats point to an average of I think 70% passing overall (notably higher for USA students). Know that his statistic figures in those that have taken the test a bunch of times and does not break it down state by state, country by country. THEREFORE, the test is much harder than the superficial stats suggest. A few of my CLOSE CLOSE friends at the Caribbean schools learned quietly that their pass level was around 20% at their school (I won't name which schools though). That means a whopping 80% are failing. Again and again. But I heard of one guy here now in the USA who finally passed, and I am not sure how many times he took the test, but he looks older like in his mid 40s when I met him in a rental building office.

Therefore, DO NOT get discouraged, but understand that you are FAR FROM ALONE!!!! This test is an impossible test that many who BUST themselves studying make possible the impossible. DO NOT lose hope! It can and will be done, but you have to study like mad ALL the subjects and sub subjects, and PRAY hard and then click that MOUSE PAD and hope that the questions coming up will give some mercy to you. Amen!

Tommy

804.  
Dear Family,

349

I forgot to mention another student who I knew PERSONALLY. He got a 39 on his MCAT and ended up at one of the most respected and competitive med schools in the country. But I heard that his USMLE Step 1 performance was not good, so much so he would not even mention it or speak of it except for giving a bunch of reasons why his score was low. But he had no trouble telling everyone his MCAT score. This guy is a nice smart guy but it is another reason why one can never know about what is "behind the Wizard of Oz" curtain. What will you find? No one knows, but the point here is that everyone is under pressure from the hardest test on earth.

Tommy

805.

Case: Patient presents with a infiltrating glioma in his brain. He is showing progressive right sided weakness of the limbs. His LEFT side of his tongue is weak though! The face is asymptomatic. He is also having trouble swallowing and talking clearly. WHERE IS THE LESION?

A) BRAINSTEM lesions are same sided (ipsilateral) cranial nerve palsy and contralateral hemiplegia, just like the above popular concept.

806.

Case) There is a thrombus..in the brain...it is the posterior cerebral artery. What kind of anopia (eye damage) do you get? What other structures does it supply? (pick it out on x ray)

A) You will see contrlateral hemianopia with macular sparing! It supplies midbrain structures like the THALAMUS, lateral, medial

350

geniculate bodies, and the occipital lobe. (FIND IT ON X RAY!)

807.

Q) Think now of occlusions: What foramen connects the Lateral and Third ventricle? Pick it out on x ray...the exact location!

A) Foramen of Monroe

808.

What structure connects the third and fourth ventricles?

A) CEREBRAL AQUEDUCT. Again, pick it out on X ray!

809.

Case: So common, a child's head is swollen. He also has myelomeningocele and syringomyelia. What is the name of the dx. and what is blocked?

A) Commonly, the cerebral aqueduct is blocked... in most cases. Also, this dx is commonly Arnold Chiari syndrome...w/ hydrocephalus.

810.

Q) Here, just understand that the fourth ventricle communicates with the subarachnoid space thru how many ventricles?

351

A) Three! Name them please. Point to them on X ray please...

811.

Q) How exactly do you define communicating hydrocephalus?

A) Here, it is NOT intra ventricular, but is due to CSF reabsorption FAILURE in the subarachnoid space.

812.

Case: Name a common bacterial dx. that causes the communicating hydrocephalus!

A) don't get non communicating confused with communicating. Meningitis has been implicated a lot in communicating hydrocephalus

813.

Q) Name the common nucleus that is lesioned in HUNTINGTON's Disease.

A) Caudate Nucleus

814.

Case: Given a coronal slice of the brain at the optic chiasm, pick out

352

the hypophysis, amygdala, and cavernous sinus, and NASOPHARYNX, and Caudate nucleus.

A) Sorry, please review on a X-ray atlas!!

815.

Given a circle of the cell phases, point to the part (M phase, G1, S, G2 phases) where Methotrexate works.

A) S or synthesis phase. Other drugs that work here are 5-FU, cytarabine, 6-mercaptopurine.

816.

Given a circle of the cell phases, point to the part (M phase, G1, S, G2 phases) where TAMOXIFEN works.

A) G1 phase, where RNA and protein synthesis occurs (S phase is where DNA synthesis forms)!

817.

Case: Given a circle of the cell phases, point to the part (M phase, G1, S, G2 phases) where Bleomycin works.

A) G2 phase.

818.

Case: You are doing a Lumbar puncture to test for a case of Lyme disease. Under which vertebra will you draw the fluid?

A) L4

819.

Case: If I gave you a picture of a man with Growth hormone hyperactivity (acromegaly), tell me, which two hormones regulates this hormone?

A) GHRH and SOMATOSTATIN!

820.

Case) Physio question renal: What is the net glomerular filtration pressure?

Bowman's capsule hydrostatic pressure = 10 mm Hg

Osmotic pressure of tubular fluid = 1 mm Hg

Osmotic pressure of plasma = 30 mm Hg

Glomerular hydrostatic pressure = 50 mm Hg

A) Remember: Take forces pushing out minus forces pushing in.

So, OUT PRESSURE FROM GLOMERULUS = 50 + 1 mm Hg MINUS

IN PRESSURE INTO GLOMERULUS = 10 + 30 mm Hg

Therefore,  $51 - 40 = 11$  mm Hg

821.

354

Case: A male pt. of yours who is 36 comes in with large bowel cancer. After it is cut out, a few months later the cancer returned with a vengeance. A serum decrease of which of the following is responsible for metastasis after removal? (Pick Endostatin or Platelet Derived Growth Factor)

A) HEY I said DECREASE...so Endostatin. This compound SUPPRESSES tumor growth while PDGF stimulates it.

822.

Case: A 25 year old woman becomes paraplegic after a spinal cord injury at T2. She is suffering from constipation. You advise her to distend her rectum with her finger to stimulate the defecation reflex. What MOA causes this? (Pick either Relaxation Of External Anal sphincter OR Increased peristaltic waves)

A) Increased peristaltic waves is the right ans. The stimulation sends afferent signals that move thru the myenteric plexus and starts peristaltic contractions which sends out the stool.

823.

The famous S curve shifts to the RIGHT during exercise and you do see higher 2,3 diphosphoglycerate which caused this. What is MOA?

A) Because GLYCOLYSIS is reved UP.

824.

True or False: Beta hydroxybutyrate and acetoacetate can be converted to glucose to supply the needs of the brain during a period of starvation.

FALSE! Although these Ketone bodies can supply energy, THEY are derived from ADIPOSE TISSUE and cannot be CONVERTED into glucose! Key concept here!

825.

Many many friendly words are coming to me to clarify my "scary" post on the USMLE Step 1. I wish to put to rest any possible confusion. What I really do not like about the USMLE Step 1 is the apparent variation different test takers face.

For instance, some told me they loved HY Goljian notes/audio and some only found it BARELY useful. Same as QBank . Some thought it helped them SO SO much, and others who felt they got a test full of transgenic mice and cell biology did poorly with QBank (which stresses Pathophysiology).

The point I am making even applies to First Aid. As some of you and others of my students said, DO NOT OVERESTIMATE OR UNDERESTIMATE First Aid. Some said they got like a large percentage of their test covering First Aid, and others said they recalled almost NOTHING in First Aid on their test.

That is why I really feel there is a "luck" element in the Step 1 which I do not particularly like. Long ago, before computers, the tests were all taken on the SAME day, like twice a year for TWO days. There were a

356

lot more short answer specific questions.

But today, because of the computer led variation, you have no idea what version out of an infinite set of possibilities you will receive.

So please remain hopeful. I do not mean to make anyone feel bad or hopeless. YOU MAY GET LUCKY like my friend in Radiology did! You may get a version of the test that addresses your STRONG points. Maybe you worked in an IMMUNO lab and your test is mostly IMMUNO. Or maybe your part time job was with a pathologist, and your test looks a lot like Robbin's Review of Pathology. OR, like one of the previous test takers, your previous career was a CHIROPRACTOR, and your test was filled up with ANATOMY. That DOES happen, but the problem is we all have no control over it. And that is why I think the test is "not perfect". If you failed long time ago, you know that basically everyone in the country taking the test that day also faced similar questions. But TODAY, if you GOT some WEIRDO set of questions (like a series of Radiographs), a block of Molecular Bio or Embryo which you totally did NOT study, and if you failed, that is not exactly fair because you knew SO much say Pharmacology and your test score did not and can not reflect that. Maybe the answer is to make SURE that all the topics are covered in a broader test that encompasses TWO days and more questions....I dunno. Any thoughts on my feeling bad about people struggling?

826.

Given a graph or table regarding systolic vs diastolic time, tell me...are the times equal or not?

A) They are unequal. Diastolic time is 2/3s and Systolic is usually 1/3 of the time.

357

827.

Describe what happens to a USMLE test taker who is tachycardiac from anxiety...I mean, compare the diastolic vs. systolic filling times as a ratio.

A) KNOW that as your heart races, the diastolic filling time decreases MUCH faster than systole. Crucial.

828.

Case: You have a pt. 41 year old man, who climbs up Mt. Kilimanjaro, way up high! After a week, he is slightly tachypneic with a decrease in arterial PCO<sub>2</sub>. Will you see increased kidney excretion of protons OR increased kidney excretion of bicarbonate?

A) INCREASED EXCRETION of BICARB! The initial respiratory alkalosis is compensated by metabolic acidosis. This is confusing but YOU NEED TO KNOW IT. The body will thus hold on to protons.

829.

In high altitudes, would you get alveolar hypoxic vasoconstriction or vasodilation?

A) PULMONARY vasoconstriction!

830.

T or F: You need to know the basics of receptors. Is the famous GABA-

358

A receptor a voltage gated CHLORIDE channel?

A) FALSE. Listen I am NOT being picky. It is a LIGAND-GATED CHLORIDE channel.

831.

The famous 5HT-3 receptor is a ligand gated Na<sup>+</sup> channel receptor. True or false?

A) TRUE!

832.

Case: The incredibly famous NMDA receptor is LIGAND gated and NOT voltage gated. (True or false statement?)

A) FALSE! The NMDA receptor is BOTH voltage and ligand gated. It requires BOTH a ligand and voltage/neuron depolarization for activation. Sodium AND calcium will move thru this channel.

833.

What ion is the "plug" for the resting NMDA receptor?

A) It is a magnesium ion.

834.

Case: A newborn baby suffers from lack of oxygen due to asphyxia. Would the baby have a sustained HIGH systemic blood pressure or a sustained HIGH pulmonary pressure? After picking, tell me why?

A) The baby would have sustain high PULMONARY pressure from RIGHT to LEFT shunting from either an open foramen ovale or PDA. Naturally, his systemic organs would all be damaged along with increased risk of DIC.

835.

Case: After delivering a 10 hour baby girl, you notice she produced a bloody meconium stool. What should you do for evaluation? Should you 1) Order an upper GI series test, or 2) Check an Apt test which determines fetal vs. maternal blood?

A) 2) You should check if the blood is from the mother or the fetus. If it is from the mother, no further workup is necessary.

836.

Case: A two and a half pound premature baby is brought to your office (this is a newborn nursery clinic you are working in). Her mother asks if her breast milk is good enough. T or F? And if False, what major ion does the premature infant desperately need (ans is not folate)?

A) F... Premature infants need extra doses of calcium, as breast milk will not provide nearly enough for the premature baby.

360

837.

Case: An infant weighing 4.5 lbs is born at 34 weeks. After delivery, in a few minutes, the baby will do which of the two? (Pick SHIVERING or RAPID BREATHING) And then tell me why?

A) The baby is not going to shiver, but his body temp will decline, and his metabolic acidosis will make him breath faster.

838.

This is some suggestions I gave to some who were SO BORED and SICK of studying....

Love you back. You absolutely have to find any way to KEEP your study schedule intact. The fact that this test is so long and awful subconsciously makes test takers nauseous. I know many who drank themselves silly because it hurt so much to study.

Thus, know that you are NOT alone at all. BUT, it seems you need a change of environment. One student I knew DID go to the library for a change of pace and it seemed to help. Another started studying OUTSIDE in the sun on his apartment complex rooftop overlooking the city. It somehow gave him peace.

Another female student I knew kept changing study places every few hours (coffee shop, bookstore, etc.) to stave off the boredom.

Still another, drank coffee and ate a LOT because it helped him rev up his sympathetic system and thus his depression over studying.

What else? Oh, one guy I knew would rent out a movie, and then

361

reward himself after a full day of studying. He would put the movie on his TV and that would be his motivation.. Let me know if this helps. I think I will put this in the ValueMD Concept Bank.

839.

Q) Does dopamine turn directly into tyrosine or norepinephrine in selective cells?

A) NOREPINEPHRINE. Remember, it goes Tyrosine to DOPA to Dopamine to Norepi to Epi.

840.

Case: DNA can be wrapped tightly because of HISTONES. What TWO amino acids are often found responsible for the tight fitting? They may give you a diagram.

A) Arginine and Lysine. Positively charged, they bind to the neg. phosphate groups on DNA.

841.

Case: Two patients, one with G6PD Deficiency and another with Pyruvate kinase def. How are these two diseases DIFFERENT in CLINICAL presentation from the Thalassemia dxs?

A) Both G6PD and Pyruvate Kinase def. present with INTERMITTENT hemolytic anemias (with normal periods), while the Thalassemias and other anemias are CHRONIC and CONSTANT in clinical presentation.

362

The triggers for dxs. like G6PD are the sulfa drugs, etc.

842.

Q) Many know that the EXTRACELLULAR compartment buffer is Bicarbonate. What about the INTRACELLULAR buffering compartment?

A) It is a PHOSPHATE BUFFERING system.

843.

A forty nine year old male has TYPE II Diabetes for 10 years. He dies suddenly at home. He is a nonsmoker. Which is more likely to have caused his death? (pick either MI, kidney failure, stroke, infection)?

A) MI is the most common cause of death for type II Diabetics!

844.

Case: Sadly, a patient of yours tries to kill himself by swallowing a jar of benzodiazepines with alcohol. Respiratory depression ensues. What will his LABS look like? give in terms of pH, PO<sub>2</sub>, PCO<sub>2</sub>?

A) His slow respirations causes respiratory acidosis. Thus, the pH is DOWN, the PO<sub>2</sub> is down, and the PCO<sub>2</sub> is up.

845.

Case: A female woman working in a dry cleaning facility gets heavy inhalation of carbon tetrachloride. Which organ is most likely to be

363

damaged the MOST? (pick one: heart, bladder, stomach, OR liver)

A) LIVER! recall the P450 system and free radicals generated when it tries to metabolize CCl<sub>4</sub>

846.

Case: A 71 year male with lymphadenopathy has recurrent infections and weakness. There is a M protein spike. There is BENCE JONES PROTEIN in urine. He has bone pain. BUT, he also has a hard time seeing now, bright eosinophilic plasma cells on bone marrow aspiration, and a cough.(EVERYONE IS THINKING MULTIPLE MYELOMA, but this is another dx.. so DON'T jump ahead.) Dx?

A) These are the symptoms of Waldenstrom Hypergammaglobulinemia...look for the cough, sight dysfunction, and "flame plasma cells".

847.

Case: A 73 year old female presented with a clinical picture and labs (w/ smudge cells) diagnostic of CLL. What are the relative levels of CD5, CD 22, and CD23?

A) Unlike other B cells disorders, CLL has HIGH CD5 and CD23, and low CD22.

848.

A 55 year old man has an ECG done. The QRS intervals are .15 secs

364

with atypical patterns. The second heart sound is SPLIT. What is the LIKELY conduction defect? Is it First degree AV heart block or Mobitz Type I AV block or Mobitz Type II AV block?

A) This picture is diagnostic for "bundle branch block". (QRS interval > .12 secs and S2 split). SO, know that Mobitz II AV block is common here.

849.

Case: You are examining a patient with First Degree AV block. What is the MOA here? Is the PR interval affected?

A) Here, we will see a PROLONGED PR interval (know it on ECG chart) over .22 seconds! The AV node is lesioned, so there is a conduction delay.

850.

Case: You see a male alcoholic patient with signs and symptoms of pancreatic carcinoma. (pain radiating to back, etc.). Which tumor marker helped you make the diagnosis on LABS? (pick either CEA elevated or alpha feto protein elevated)

A) Answer is CEA. Along with COLON cancer, pancreatic cancer has CEA as an active tumor marker.

851-900

366

Where are the cells that secrete PEPSINOGEN found in the stomach? Can you differentiate them on a histo slide from PARIETAL cells (which secrete HCl)? Do you also know there are enteroendocrine cells found alongside? What do they secrete and what is a common stain used?

-----Look for them in the fundus (chief cells). Examine a histo slide of the cells types there. The enteroendocrine cells secrete amines and polypeptides and is stained by a silver stain.

T or F: RNA polymerase is used in the initial step of DNA synthesis on the template.

-----FALSE! tricky...you need RNA PRIMER using dNTP substrates.

T or F: For DNA and RNA synthesis, mispaired nucleotides are removed by 3 to 5 prime exonuclease.

-----FALSE, RNA mispairings are NOT repaired.

T or F: The TEMPLATE strand is scanned in the 5' to 3' region.

-----FALSE! It is scanned in the 3' to 5' region!

What exactly is the function of SSB (single strand binding proteins) in DNA replication?

-----They bind to the DNA strand, block the strands from reassociating together and protecting them from degradation by nucleases..

Why EXACTLY is RNA primer needed in DNA replication? What makes it?

-----made by PRIMASE, RNA primer is crucial because DNA polymerases cannot initiate synthesis without a PREFORMED primer's 3' end already made.

Which DNA polymerase, I or III, is used to remove the RNA primer?

-----DNA polymerase I...they also fill in gaps by synthesizing DNA.

T or F? AFTER Helicase unwinds the DNA, DNA topoisomerase II inserts POSITIVE supercoils to RELIEVE the stress from the supercoils.

-----False. It inserts NEGATIVE supercoils.

You know drugs like Cipro block DNA topoisomerases in bacteria to kill them. But where else are they finding good use? Give an e.g. of a drug.

-----They are being used in anti neoplastic therapy. Etoposide is an example.

T or F: The majority of DNA repair occurs in the SYNTHESIS phase after replication.

-----It often occurs in the G1 phase.

What is the MOA of Hereditary Non Polyposis Colorectal cancer? What stage of the cell cycle is affected, and when is it repaired?

-----This is a common example of a mismatched base damage from a mutation of two genes. The repair process occurs in the G2 phase.

In Xeroderma Pigmentosum, what enzyme makes a nick in the damaged strand to remove the oligonucleotide?

-----Excision Endonuclease.

One of your patients is taking tetracycline (a A LOT OF SEs, know them ALL). It may cause a normal floral disturbance in the mouth and a growth of the bacteria ... (Pick either Candida or Nocardia)

-----CANDIDA...Nocardia is not normal flora

Autoimmune disorder in your patient. MOA question...it is believed that self antigen tolerance is due to chronically activated T suppressor cells. So, here is a T or F question:

T or F: T-helper cells that are revved up to antigen in the context of MHC class II is a major reason for autoimmune disease.

-----True! After exposure to interferon or cytokines, T-cells are activated, often against self in the context of MHC class II

Guillain-Barre syndrome. Infection causes molecular mimicry that targets an autoimmune process. MOA please?

-----Many viruses have antigens that resemble those in the human body. Thus, T-cell activation causes an immune response against self.

Explain why in MOA how an autoimmune response can be generated in the CNS/eye lens or spermatozoa after a surgical mistake.

-----The CNS and sperm usually have LITTLE contact with the immune system. If an accident or trauma releases those antigens into the blood, an autoimmune response can occur.

A woman in her mid thirties comes in with cold intolerance, malaise, and weight gain. You cannot figure out if the problem is in the HYPOTHALAMUS or the ANT. PITUITARY. What test pinpoints the diagnosis?

-----TRH test, or thyroid RELEASING hormone test.

---If an injection of TRH raises the TH level, then the problem is obviously in the HYPOTHALAMUS.

---But...if nothing happens, then the lesion is in the ANT. PITUITARY!

If you have a patient in whom you lesion the progesterone secretion with an anti progesterone drug. You do this on day 17-24 of a NORMAL menstrual cycle. What happens? (Pick either the cervical mucus will be thicker than normal OR LH secretion is higher than normal) WHY?

-----The LH secretion will be elevated because in the Luteal Phase, both the estrogen and progesterone is ANTI-LH release. Suppressing the progesterone will ELEVATE the LH during this Luteal

phase. (At the LH surge, the estrogens becomes PRO-LH and PRO-FSH).

T or F: PROGESTERONE is what is needed for endometrium maintenance during the ovarian luteal phase AND its INHIBITION in the luteal phase can result in EARLY menstruation. -----TRUE!

Quick, tell me at least THREE things that PROGESTERONE does...

-----Like the previous concept, it maintains the secretory endometrium during the ovarian luteal phase.  
-----It also, makes the cervical mucous thick  
-----AND decreases oviduct motility.-----

During the FOLLICULAR PHASE (Days 1-14, 1st half), what does the estrogen made by the GRANULOSA cells do? Name at least 3 things. Think hard before looking!

-----The steady rising estrogens causes proliferation and mitosis of the endometrium.  
Also, it causes the circulating estrogens to make the CERVIX's mucosal opening watery and THIN, so sperm may get through.  
Also, the CIRCULATING estrogens stimulate the female sex organs to develop.

The POPLITEUS muscle (identify its location on X-ray or diagram), in the knee does what? Answer true or false:

1) it is innervated by the tibial nerve

-----TRUE

2) it extends the knee -----FALSE, it FLEXES the knee

3) it rotates the femur bone medially----- FALSE, it LATERALLY rotates the knee unlocking it for extension

Other than BMS and hemorrhagic cystitis (of the bladder), will a patient's hair be affected with cyclophosphamide? Yes or No?

-----Yes, commonly, alopecia is a side effect of cyclophosphamide.

While on the subject, tell me which virus is linked with hemorrhagic cystitis? (Adenovirus or Echovirus)?

-----Adenovirus

Please review the histology of an ASHCHOFF body. it is common. especially b/c it follows strep infections which are everywhere. What dx is associated and what will I hear on heart exam? Due to what lesion?

-----This is diagnostic of Rheumatic Fever. You will hear an OPENING SNAP and murmur from mitral stenosis. Can you describe

how an ASHOFF body looks like?

OK, so you know mitral stenosis, but tell me the MOST COMMON CAUSE of AORTIC STENOSIS...usually...

-----Congenital bicuspid aortic valve.

You HAVE to know how to calculate the A-a gradient for either the USMLE or endless times in the clinics (A=alveolar, a=arterial). So, let's say I gave you a case of a COPD pt. with arterial PCO<sub>2</sub> of 80 mmHg, and arterial PO<sub>2</sub> of 40 mmHg. If the patient is on R<sub>mair</sub> (20% O<sub>2</sub>), is the A-a gradient abnormal? WATCH out, do not JUMP to conclusions because the PaO<sub>2</sub> is low.

-----Quick, the A-a gradient is PAO<sub>2</sub> - PaO<sub>2</sub>: so, PAO<sub>2</sub> = (% O<sub>2</sub>)(700) - PaCO<sub>2</sub>/.8.

Therefore, PAO<sub>2</sub> = .20(700 mmHg) - 80 mmHg/.8 = 50 mmHg

Thus, the A-a gradient is 50 - 40 = 10 mmHg. Anything under 25-30 mmHg is normal range, so this patient's A-a gradient is normal.

WHAT causes a very HIGH A-a gradient then? This is a CRITICAL concept.

-----The partial press in the alveoli rarely matches that of the arteries because there are V/Q mismatches and shunts. If you see a hypoxic person with super high A-a gradients, then that indicates there is a problem in V, , or Q, perfusion, and or diffusion.

T or F: A POSTIVE Romberg's sign points to a loss of unconscious proprioception.

-----FALSE, Tricky, A positive Romberg is CONSCIOUS proprioception lesions from the dorsal column medial lemniscus pathway.

What is the MOA behind spasticity, an UPPER motor neuron lesion? Key test used?

-----The spinal cord reflex is intact, BUT the cerebral cortical lesions from hypoxia or infection, etc. remove the inhibitory descending control.

Lots of Neuroanatomy for everyone: If given a picture of the brainstem and an arrow pointing to the nucleus cuneatus, tell me what tract is involved. What specifically is this nucleus associated with?

-----The nucleus cuneatus receives from the cuneatus fasciculus and are part of the DCML pathway. They are involved in 2 points touch and vibratory sensation from the UPPER part of the body.

T or F) The EXTRAPYRAMIDAL system consists of the descending motor pathways including the important corticospinal tracts? How does a pt. present if this tract is lesioned?

374

-----A1) FALSE. A2) If the tract is lesioned, a pt. presents with ataxia and posture and gait difficulties.

T or F: Diabetes mellitus is the most common cause of blindness in the U.S. today.

Secondary: What key finding differentiates diabetic retinopathy vs. hypertensive retinopathy? MOA?

-----A1) True A2) MICROANEURYSMS are common with diabetic retinopathy. They form because of osmotic injury to the pericytes circling the retinal vessels.

Case in Immuno: You need to know the BASICS of immunological markers...GIVEN a picture of an immature lymphocyte, which marker is found in both immature B and T lymphocytes, CYTOPLASMIC IgM or Terminal DEOXYNUCLEOTYDYL TRANSFERASE?

-----TdT or Terminal Deoxynucleotydyl transferase is one of the EARLIEST markers expressed.

T or F: BOTH FcR protein AND MHC class II are expressed during the early B-cell stage of lymphocyte development.

-----True.

True or False: You are seeing a small for dates baby (low birth weight). They are at increased risk for hyperglycemia and LOW

hematocrit.

-----False: They have HYPOglycemia and HIGH hematocrit, as well as increased risk for lung problems and malformations.

True or False: It is PROGESTERONE that is a precursor of mineralocorticoids and glucocorticoids in the adrenal cortex, of testosterone in the testis, and estradiol in the ovaries, AND is also the end product found in the luteal phase's corpus luteum.

-----True

What is the body's primary glucocorticoid and WHAT DOES IT DO? List at least three things...

-----In our molecular chem, CORTISOL revves up gluconeogenesis and causes catabolism of protein. This creates gluconeogenic precursors. Cortisol also possess anti inflammatory functions against PHOSPHOLIPASE A2.

True or False: A METHYL group is removed as TESTOSTERONE is converted to ESTRADIOL.----- True!

In a population of 10,000, in 2003, a total of 1,000 people died of a new alien virus. During 2003, 500 new cases of the viral illness was diagnosed. Calculate the INCIDENCE rate for 2003:

-----) YOU HAVE TO KNOW THIS. I.R. is calculated by dividing the

376

number of new cases by the population at risk within a said time period. So, here we have 500/10,000 or 5%.

Pretend you are shown a histo slide of a LYMPH NODE with areas everywhere (germinal follicle, paracortex, and sinus). Can you tell me the EXACT location of the most common area for the origin of malignant lymphomas?

-----Look on a histo atlas for a GERMINAL FOLLICLE, where B cells are sitting around.

Same histo slide of lymph node is shown...where does the antigenic stimulation of T cells in INFECTIOUS MONONUCLEOSIS occur exactly?

-----In the PARACORTEX (outside the germinal centers).

T or F: In DiGeorge's syn. patients, the paracortex of the lymph nodes are hyper proliferative with T-cells.

-----False, you will see an ABSENCE of T-cells due to absence of the thymus and parathyroids...but the germinal centers with B-cells are normal.

The famous disease Malignant Histiocytosis is found where in a histo slide of the lymph nodes?

-----There are found in the SINUSES of the lymph nodes.

377

Which is the most common site of metastasis of a cancer to lymph nodes (the lymph node paracortex, germinal center, or sinus area)?

-----Since the sinuses are most peripheral, choose SINUSES.

True or False: The internal laryngeal nerves innervates the CRICOTHYROID muscle (Famous HYer).

-----False, it is the EXTERNAL laryngeal nerve.

True or False: Giardiasis is caused by a bug that possesses EUKARYOTIC features like a true nucleus bounded by a membrane and inner mitochondria.

----- This is actually TRUE, as it is a PROTOZOA. Find out the EXACT appearance under a slide please.

Relating to the previous concept, does Ureaplasma urealyticum have a nuclear membrane and mitochondria like Giardia?

----- NO! It has a rigid cell wall and is a bacterial PROKARYOTE.

901.

Case: You have a patient who needs to use pseudoephedrine as a

378

nasal decongestant. He is an older gentleman with BPH, hypertension, hyperthyroidism, and coronary artery disease, and urinary incontinence. Which one of these symptoms are NOT made WORSE by the pseudoephedrine?

A) Urinary incontinence. Due to contraction, you may actually help the symptom of urinary incontinence.

902.

Q) First, tell ValueMD about the differences between extrinsic vs. intrinsic hemolytic anemia and extravascular vs. intravascular hemolysis. (People get these terribly confused at first).

A) Listen, EXTRINSIC hemolysis means something is wrong OUTSIDE the Red Blood Cell (RBC). INTRINSIC hemolysis means something is wrong INSIDE the RBC. Extravascular hemolysis occurs when MACROPHAGES eat up the RBCs and Intravascular hemolysis occurs when the hemolysis occurs by various mechanisms WITHIN the circulation.

903.

Q) What kind of hemolysis is PNH, or Paroxysmal nocturnal hemoglobinuria?

A) It is a stem cell disorder, acquired, by sensitivity of hematopoietic cells, which have a reduction of decay accelerating membrane factor, so they get destroyed by complement. So, PNH is an INTRINSIC, INTRAVASCULAR (they are NOT removed by macrophages) anemia!

904.

Q) Pt. comes in with WARM hemolytic anemia. What kind of hemolysis is this?

A) Its MOA is IgG/C3b deposited on RBCs with extravascular removal by MACROPHAGES, which have the Fc receptors for IgG and C3b! Thus, this is an EXTRINSIC and EXTRAVASCULAR hemolysis.

905.

Case: Pt with sickle cell anemia. Same question, what kind of anemia is this?

A) The sickled cells cannot escape the Billroth cords in the spleen. Thus, they are removed extravascularly by MACROPHAGES. Thus, this is an INTRINSIC hemolytic anemia with EXTRAVASCULAR hemolysis!

906.

Case: Middle aged man, smoker, received synthetic heart valve replacement.. later, anemia occurs. What kind of anemia?

A) Schistocytes (broken RBCs) are seen because they break apart after hitting the plastic heart valves. Thus, we see an EXTRINSIC (not inherent with RBC formation), INTRAVASCULAR (within the circulation) hemolysis.

907.

True or False: At a constant EDV (end diastolic volume), epinephrine RAISES stroke volume.

A) True.

908.

True or False: Vigorous exercise increases BOTH EDV and Stroke Volume!

A) TRUE!

909.

Case: Pt. on propranolol. Say ValueMD shows you a graph (which I don't know how to draw here). Does Stroke Volume decrease at CONSTANT EDV? Or will it change too?

A) Yes, EDV remains constant.

910.

Case: An older female with a pacemaker that malfunctions and speeds up while the patient is at rest. How will EDV and SV change?

A) Here, they BOTH decrease.

911.

Q) In which case will there be FOLATE deficiency and not vit B12 deficiency? (Pick either Crohn's disease, Chronic pancreatitis, or Pregnancy)

A) PREGNANCY! The other two result in vitamin B12 def. Think of the MOA of vit B12 uptake! (recall R factor in saliva, intrinsic factor in stomach, etc. )

912.

Q) relating to the previous concept, will small intestine bacterial overgrowth affect vit B12 absorption? HOW?

A) YES, by breaking up the intrinsic factor and vit B12 complex. (Note, folate is not affected)

913.

Q) The posterior cerebral artery distributes to the OCCIPITAL CORTEX

382

via the basilar artery...so will an embolism from the VERTEBRAL artery occlude such that someone's eyesight is lesioned? Yes or NO? The NBME stresses pictures of the brain, so be ready to identify all the main diseases and which blood vessels distribute to its different parts!

A) Yes. All is true here.

914.

Case: A man comes in with urinary stones...a history of them. Name THREE places which you should identify on radiograph or a diagram where a stone can likely get stuck along the ureter. Up to 10 percent of folks get stones!

A) LARGEST of them is at the ureteropelvic junction. Next is the area of the Pelvic brim. The last area is the ureterovesical junction, (area where the ureter passes through the bladder wall.

915.

Case: You see a 28 year old male with unknown reasons for lymphadenopathy, weight loss, and these weird raised skin lesions all over his chest. Which one, (Kaposi's sarcoma, a CD4 count of 220, or a positive antibody test for HIV), confirms AIDS?

A) Kaposi's sarcoma. The USA standards are that you need a CD4 count under 200, regardless of symptoms...BUT, if you see something like Kaposi's sarcoma, an AIDS defining illness, you can identify it as AIDS.

383

916.

Case: You see a person with bipolar disease on Lithium. He has Diabetes insipidus as a long term consequence from SIADH. True or False for each:

1..will he show HYPERnatremia?

2...intracellular compartment swelling?

3...increased plasma osmolality?

A1) YES, from loss of free water.

A2) NO (think of osmotic properties)

A3) YES...

917.

Cases: 4 different patients with gout...

First guy is on a drug that work by blocking the renal reabsorption of uric acid. Is it sulfinpydrazone, probeneidc, BOTH, or NEITHER?

A) BOTH, think MOA...

384

918.

Case: Next guy with ACUTE gout runs in limping. Will you give allopurinol or indomethacin for the ACUTE gout?

A) INDOMETHACIN, an NSAID which blocks prostaglandin synthesis.

919.

Case: Another guy with gout walks in. He needs meds for chronic gout. You give colchicine. What is the MOA against the gout?

A) It blocks leukocyte migration AND phagocytosis secondary inhibition of tubulin polymerization.

920.

Case: Another person waltzes in with chronic gout. He has a weak GI tract. Which med, probenecid or colchine, are you worried about giving?

A..colchine...it can cause serious GI side effects.

921.

Case: Which common bug, H. flu, S. pneumo, or Staph. aureus, LACKS IgA proteases which help a bug infect mucosal surfaces?

A) Staph aureus.

922.

Name two out of many enzymes that S. aureus makes which degrade human cells for colonization...what do they do?

A) Think about the Identifying traits like Catalase positive and Coagulase positive. These two enzymes of S. aureus work thus:

Coagulase clots plasma. And catalase converts cellular Hydrogen peroxide to water and oxygen, limiting the cellular killing of the bacteria.

923.

Case: A friendly friend comes into your office complaining of symptoms from an acoustic neuroma at the cerebellar-pontine angle. What symptoms is he likely to show? And what two nerves are likely to be affected?

A) Vertigo, Auditory stuff, and facial muscle paralysis are seen. CN VII

386

and VIII are often lesioned.

924.

Case: An aneurysm appears in the superior mesenteric artery at the level of LV2. Which is compressed, the left or right renal vein? Which is longer? Important since you will know which kidney is in danger.

A) The LEFT renal vein, which passes ANTERIOR to the aorta. The left renal vein is LONGER.

925.

Q) What is different about the drainage of the right ovarian vein and the left ovarian vein? Is there anything?

A) The RIGHT ovarian vein drains directly into the Inferior vena cava while the LEFT drains into the left renal vein first before the IVC.

926.

Q) Tell us about the MOA of the degradation of cortisol? Where does it occur?

A) It occurs in the liver, converted to tetrahydrocortisone. It is then converted into glucuronic acid via CONJUGATION. Now it is water soluble, and is then urinated out into the toilet or potty.

387

927.

Case: An older patient comes in with cataracts. Can it be due to sorbitol production in the lens? What common dx is associated with excess sorbitol production?

A) Yes, it is often due to diabetes mellitus.

928.

Case: A patient of yours named Jennifer Connolly steps on a nail in a house called "House of Sand and Fog." She suffers paralysis from Clostridia. Is there an exotoxin associated? What is the MOA?

A) Yes...the MOA is that an inhibitory neurotransmitter called GLYCINE is blocked from release from the CNS, causing tetanic paralysis.

929.

Case: A cases of a patient with a murmur...a diagram shows a crescendo-decrescendo, ejection type, diamond shaped figure between S1 and S2. What valve is lesioned?

A) This is AORTIC stenosis. Both pulmonary and aortic stenosis occurs during systole. The sound diagram is evident when the blood rushes out thru the narrow opening.

388

930.

Q) We just spoke of aortic stenosis and the sound diagram. What about MITRAL STENOSIS, in relation to S1 and S2?

A) Opening snap, crescendo, decrescendo, diamond/wedge shaped...you will see a crescendo wedge leading up to S1.

931.

Q) T/F. The aortic valve and pulmonic valve opens during diastole.

A) False, they CLOSE during diastole.

932.

Case: You are treating a patient with mitral regurg...how does the sound/time graph look like?

A) Try to imagine the mech of action (MOA), then you will not forget...this is a pansystolic or holosystolic murmur, so the graph will look like a rectangle, the line with zero slope, where the blood rushes back into the atria with a CONSTANT velocity.

933.

389

Q) Will tricuspid regurg look like mitral regurg on a sound vs. time graph?

A) Yes, both have the same MOA...think about it logically. They are coupled as are the pulmonary and aortic valves.

934.

Case: Another patient comes in with aortic REGURG...how will this sound/time graph look like? Please review in a cardio text, as my explanations are not the best without pics.

A) Think about what is happening...during DIASTOLE, there is an insufficient aortic valve, so there is regurg, so then there is a high pitched blowing murmur AFTER S2, when the aortic valve does not close right as the heart is trying to fill the ventricles. You will see a descending wedge/triangle after S2.

935.

True or False: An S4 heart sound is shown on a graph superimposed a cardiac cycle graph. Is it associated with atrial contraction OR ventricular contraction?

A) ATRIAL contraction or atrial systole...also seen with a hypertrophic ventricle...also maybe a heart attack.

936.

Case: You see a cardiac cycle graph. Point to the exact place where you may see an S3....what is the MOA?

A) Right after the mitral valve opens, you may see an S3 as you hear the blood slam into the walls of the ventricle during diastole (rapid ventricular filling).

937.

Case) (Hint, this is the most posterior chamber in the heart). A woman with rust colored sputum, difficulty swallowing, cough, and a hoarse voice comes in. What heart disease does she have that we recently discussed? What is the mech. of action?

A) This is MITRAL STENOSIS...greater pressure need to overcome the stenosis results in a hypertrophy of LEFT ATRIUM. As this is most posterior, enlargement compresses the esophagus (difficulty swallowing), the lungs (pulmonary edema and cough and hemoptysis), damage to the recurrent laryngeal nerve (horseness of the voice).

938.

Case: You hear a murmur radiating to the carotid arteries in a 65 year old smoker. He has angina and dizziness/syncope on doing gymnastics, and weak pulses on extremities. What is the MOA? What is the heart disease?

391

A) This is AORTIC STENOSIS. This results in left ventricular hypertrophy...as the heart must push against more resistance out of the heart. As a result, we see angina (due to lessened coronary artery refill), syncope because his exercise demands more oxygen and because the stenosis lessens the flow, and weak pulses for the same reason.

939.

Q) Quick, are you retaining? I repeat the angina case presentation with aortic stenosis. Quickly, what does the sound-pressure vs. time graph between S1 and S2 look like?

A) Remember the diamond shaped ejection murmur...

940.

Q) You see another diagram of ONLY the "rectangle shaped" sound/pressure vs. time graph between S1 and S2. This you recall is MITRAL REGURG. What is the MOST COMMON CAUSE of this dx?

A) Rheumatic fever from Group A beta hemolytic strep. Is this bug bacitracin sensitive??? Yes, it is.

941.

392

Q) Are Strep viridans partially or completely clear on hemolysis on blood agar? Are they susceptible to optochin?

A) They are alpha hemolytic (partially clear)...not beta hemolytic (which is completely clear). They are NOT susceptible to optochin.

942.

A patient presents with tertiary syphilis. You are shown a sound/pressure vs time graph where there is a decrescendo after S2 (a wedge or triangle with a negative slope). What dx and MOA of the heart disease is this?

A) This is commonly caused when the aortic valve closes INSUFFICIENTLY. The subsequent REGURG causes the syphilitic aortic aneurysm.

943.

A guy named Big MAC is very tall and has a heart defect from a chromosomal anomaly. He has Marfan's syndrome.. What other TWO common illnesses can cause this aortic valve insufficiency? (hint: M=Marfan's, A=?, C=?)

A) A=ankylosing spondylitis, and C=coarctation of the aorta.

944.

Q) T or F: Release of CCK results in contraction of the Sphincter of Oddi.

F) It results in its RELAXATION. It is the gallbladder that contracts.

945.

Q) True or False: CCK release will cause the secretin potentiation to release enzymes and BICARBONATE from the PANCREAS.

A) True.

946.

T or F: CCK is released by the presence of carbohydrates into the colon.

A) False, CCK is released by the presence of FATS and protein into the DUODENUM.

947.

T or F: CCK has no effect on the rate of gastric emptying.

A) False, CCK SLOWS the rate of gastric emptying by constricting the pyloric sphincter.

948.

Case: you are pimpled by the cardiology attending and shown a graph of the Jugular Venous Pulse with three peaks (a, c, v). What heart sound (S1, S2, S3, S4) does peak v represent. What is happening physiologically?

A) S3, The increased JVP is caused by the blood pressure against the closed tricuspid valve.

949.

Q) Same as the previous concept...what does peak c stand for in the JVP graph? When does it occur?

A) c=Right ventricular contraction, as the tricuspid valve pushes back into the atrium. Occurs right AFTER S1, when the mitral valve closes and the aortic valve opens.

950.

Q) Which aortic pressure is HIGHER as measured the the left ventricle,

395

the exact point when the aortic valve opens OR when the aortic valve closes? When?

A) Surprise...! It occurs at S2, when the aortic valve closes!

951.

Q) Very important in clinics/tests...you have a patient with angina. You need to DECREASE heart rate and cardiac contractility and block coronary vasospasm. Which drug, Verapamil or Nifedipine will do the work?

A) Verapamil will do it. Nifedipine, another Ca channel blocker, does not do this well.

952.

Q) Very important in clinics/tests: What is the rate limiting committed step in de novo purine synthesis? Is it:

- 1) Ribose 5 phosphate > PRPP or
- 2) PRPP > 5 phosphoribosylamine?

A) PRPP > 5 phosphoribosylamine, CONFUSING...but this is because Ribose 5 phosphate > PRPP is the FIRST step, but not the rate limiting one because PRPP is also utilized in PYRIMIDINE synthesis and in base salvage.

396

953.

Case on RBCs: If I present you with a mature RBC named George Bush, tell me, True or False:

In the RBC, lactate is converted to pyruvate for use in gluconeogenesis.

A) False, recall that gluconeogenesis occurs only in the liver and kidneys.

954.

Q) Another RBC floats to you named Richard Cheney. He asks you if inside himself/RBC, there is a glycolysis where there are 2 ATP made. The two reduced NADH are then used to convert pyruvate into lactate.

Is this true or false?

TRUE, some think it is acetyl CoA, but they are wrong.

955.

Q) A mature RBC named Condoleezza Rice asks if she uses the pentose phosphate pathway for the formation of NADPH. She asks why is this needed?

397

A) To maintain glutathione in a reduced state.

956.

OK, so Ms. "RBC" Rice asks you what is the reduced glutathione used for in the RBC? You say...

A) You need it to maintain the integrity of the cell membrane!

957.

Q) Let's say I show you a picture of a uterine lesion and tell you this is the most common benign soft tissue tumor in adults. What do you say?

A) LEIOMYOMA, do you know what a gross specimen looks like?

958.

Ahh, now I show you a picture of a skin lesion and tell you this is the most common soft tissue SARCOMA. What do you say?

A) Malignant fibrous histiocytoma.

959.

As we just discussed, malignant fibrous histiocytoma is found where and in whom usually. Do a google image search.

A) often in men, older, and involves the limb bones and retroperitoneum.

960.

True or False: Lipomas often will progress to liposarcomas, given enough years.

Also, where are they most often found?

A) False.

They are most often benign and found around the neck and torso!

961.

We discussed LIPOMAS (also known as uterine fibroids), are very common, but different from Leiomyomas. But what about leiomyoSARCOMAS? What are they?

A) They are malignant tumors of SMOOTH muscle origin. So, you will see lesions in the uterus, GI walls, and blood vessels.

962.

Remember this LUKE OR LEA SKYWALKER...what exactly is a rhabdomyoma? Benign or Malignant?

A) Benign, they are benign tumors of skeletal or cardiac muscle. IT is the Second most frequent tumor of the heart. Myxomas are the most common here.

963.

Hard Molecular Bio Q, tricky, but a good one...we discussed primase. What nucleotide cannot be a substrate of primase? (choices: ATP, TTP, UTP, GTP).

A) Think and recall that TTP has thymidine. Because primase makes RNA primers in DNA replication, only RIBOnucleotides can be used.

964.

You live in a house called "Sand and Fog". Again, your friend, Jennifer Connolly comes in and steps on a nail. You quickly give her tetanus immune globulin. Does this neutralize circulating toxin, toxoid, or fixed

400

toxin on nerve tissue?

A) Cirulating toxin.

965.

Case: Your attending pulmonologist walks in as asks YOU if a flowmeter tracing depicts the relationship between flow rate during a Forced Vital Capacity (FVC) and LV (Lung Volume). An FVC starts at the point of total lung capacity (TLC) and ends at Residual Volume (RV). Is all this true or false?

A) True, KNOW also that a restrictive lung disease will DECREASE BOTH TLC and RV.

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966.

T or F: You have a patient named Don Johnson who has partial seizures. He is refractory to phenytoin and carbamazepine. Your med student suggests ethosuximide. Is she correct?

A) NO! Ethosuximide works only for generalized absence seizures.

967.

401

Case: A previously healthy 7 year old girl suffers from a 2 week history of fever, fatigue, weight loss, muscle pain, and headache. He also has a heart murmur, petechiae, and splenomegaly. What dx does he have?

A) Endocarditis, with vegetations fr. Step. or Staph infection.

968.

case: You are seeing a 19 year old primiparous woman with toxemia in her last trimester of pregnancy treated with MgSO<sub>4</sub>. She delivers full term a 2 kg infant with poor Apgars. Labs have a persistent hematocrit of 80%, platelets of 110,000, glucose 40 mg/dL, Mag 2.5 mEq/L, and Calcium 10 mg/dL. Later this infant has a seizure. What is the cause?

A) Pt has polycythemia induced seizures. The Mg IMPLIES that she had PREGNANCY INDUCED HYPERTENSION. This results in nutritional deprivation and hypoxemia, and erythrocytosis. KNOW that a persistent hematocrit over 65% in a neonate baby results in HYPERVISCOSITY and seizures.

969.

Which bug more often causes congenital infections, Toxoplasma gondii, Mycobacterium tuberculosis, Trichomonas?

402

A) REMEMBER the TORCH! T=Toxoplasma...the others seldom are implicated.

970.

Case: Say I present you with a Webpath pic of a Turner's syndrome patient at infancy. (45, X,O). What lesion is predominant in the neck? What about in the heart?

A) In the neck, you will see redundant skin folds. In the heart, you often will see coarctation of the aorta, HTN, bicuspid aortic valve, and sometimes horseshoe kidney.

971.

Q) Failure to give vit K to a newborn patient will result in elevated prothrombin or thrombin time? Plus, what clotting factors are affected?

A) PROthrombin time, Factors II, VII, IX and X are affected.

972.

Your pregnant patient is 35 weeks. Which of the following should you NOT give to her (Pick from penicillin, phenytoin, heparin, and propranolol)?

A) Of these, propranolol is contraindicated at this 3rd trimester. Bradycardia and apnea can result. HOWEVER, phenytoin recall is contraindicated usually in the FIRST trimester. The other two are safe.

973.

Woman with no prenatal care delivers small for dates baby. She told you she had multiple sexual partners during her pregnancy and before. The PE of the baby has hepatosplenomegaly, noted lymphadenopathy, and nasal discharge like the snuffles. What test do you think of getting to confirm the dx?

A) FTA-ABS for syphilis. Choose PENICILLIN for Rx.

974.

A 7 month old pt. comes in with a resting HR of 50. PE reveals NO rash, and NO cardiomegaly. But electrocardiogram reveals d-looped ventricles. FH is significant for SLE. What is causing the bradycardia?

A) Most likely, a congenital complete heart block. Lyme disease can be ruled out because there is no tick bite, and cardiomyopathy can be ruled out because there is NO cardiomegaly on x-ray.

975.

Q) What and where is the anterior recess of the ischioanal fossa?

A) A fat filled space below the pelvic diaphragm, it is in between the inferior space of the pelvic diaphragm and the superior fascia of the urogenital diaphragm.

976.

A 37 y.o. male patient of yours has GI symptoms and feels high strung a LOT for no apparent reason, sweating AND dry mouth. Does he have panic disorder or Generalized anxiety disorder?

a) Generalized anxiety disorder...rule out panic disorder because panic disorder is usually triggered by a known cause. Give anxiolytics for meds.

977.

Someone, a 27 year old male goes to the Southern-Eastern states for a camping trip. He gets Rocky Mountain Spotted Fever. Except for the rashes and fever, what is a typical medication you would use to treat? What is the MOA of the bug? What test is helpful?

A) Use either doxycycline or tetracycline combined with chloramphenicol. The MOA of the bug is a vasculitis resulting from

405

endothelial invasion by Rickettsial buggies. The test of choice now is the indirect florescent antibody (IFA) test. OR you can use a Giemsa stain under light microscopy.

978.

Case: You see a 5 year old pt. with a history of a URI like symptoms that preceded a rash that started from his face and spread downward (there were no Koplick spots). Lymphadenopathy may OFTEN be present, particularly in the posterior auricular, posterior cervical, and suboccipital chains. What is the dx? What is the treatment?

A) This is Rubella. Treatment is supportive with Tylenol and Benadryl for the headaches and itching.

979.

A child patient of yours comes in. Your attending tells you this is NOT RUBELLA. He had a high fever for 3 days and the rash that followed started on the trunk and then spread from there but missed his face. The condition is an acute benign disease of childhood characterized by a history of a prodromal febrile illness lasting approximately 3 days, followed by defervescence and the appearance of a faint pink maculopapular rash. Bug please?

A) Roseola

980.

Case: Pt of yours comes in with crops of papular, vesicular, pustular

406

lesions starting on the trunk and spreading to the extremities. Lesions are asynchronous (happening at different times). What is this?

A) Varicella

981.

This time, you see a young patient with ulcers on his tongue and oral mucosa. You also see a maculopapular vesicular rash on the hands and the feet surfaces (key finding). What disease is this?

A) Hand foot and mouth disease

982.

983.

A 25 year old male patient of yours comes in with spironolactone overdose and HYPERKALEMIA. He gets muscle weakness and tetany. His potassium level is 7.4...no hemolysis. Which EKG change is NOT consistent with hyperkalemia? (pick between notched PR segment, ST depression, wide QRS complex, P wave loss, T wave elevation).

A) You WON'T see notched PR segments, but you WILL see all the others.

407

984.

Case: You see the same patient with Hyperkalemia. What are a few OTHER causes of this?

a) You'll see this in acute or chronic renal failure, especially in patients who are on dialysis.

Trauma, including crush injuries (rhabdomyolysis), or burns.

Ingestion of foods high in potassium (eg, bananas, oranges, high-protein diets, tomatoes, salt substitutes).

Meds - Potassium supplements, potassium-sparing diuretics, nonsteroidal anti-inflammatory drugs (NSAIDs), beta-blockers, digoxin, and digitalis glycoside.

985.

Case: Still looking at Hyperkalemia. We are dealing with a HYPERacute case of it. What med is better, Calcium gluconate or Kayexalate?

A) Calcium gluconate is better, its onset of action is as quick as 5 minutes while kayexalate may take 2-10 hours to take effect. HOWEVER, know that Calcium gluconate does not really affect TOTAL body K<sup>+</sup> stores, but rather is CARDIOprotective

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986.

408

Pt: A 6 year old child named Kill Bill presents with tachycardia at 230 beats per minute, no fever. The ECG shows a narrow complex tachycardia seen (no signs of atrial flutter). One dose of ADENOSINE makes the sinus rhythm normal with pre-excitation noted. There is NO cardiomegaly seen on radiograph. What is this? Could it be sinus tachycardia?

A) HARD HARD question. The pre excitation seen after conversion with adenosine is Wolff-Parkinson White syndrome. Sinus tachy is not likely because the patient is afebrile with no cardiomegaly.

987.

Case: Because this is so common, what is the difference in presentation between strabismus and amblyopia?

A) Strabismus is an eye that cannot align properly and amblyopia is the impairment of vision without detectable organic lesion of the eye.

988.

Case: A middle aged patient of yours tried to kill herself by injecting a bottle of antipsychotics with anticholinergic activity....can she acutely die from cardiac arrhythmias?

A) YES

409

989.

Case: True or False: Besides mental slowness, iron toxicity can cause seizures.

A) True

990.

Case: Which one, (CCK, secretin, or bile acid levels in the plasma), determine the rate of bile secretion by hepatocytes?

A) Plasma levels of bile acids...tricky tricky. Stuff like secretin and parasympathetic innervation works at the LEVEL of the biliary ducts...NOT the hepatocytes.

991.

case: You encounter a 34 y.o. patient screaming in pain because he has a kidney stone. You find that the stone is a struvite or staghorn stone. What bug does he likely have? Is the stone calcium? What minerals are part of the stone? Is the urine acidic or alkaline?

A) He likely has a Proteus infection producing urease. The stone is NOT the most common Calcium stones. The minerals are M.A.P. or Magnesium, Ammonia, and Phosphate. The urine is ALKALINE (think ammonia).

992.

Case: Oh darn! Your patient has cystathionine synthetase deficiency. What disease is this associated with? What Amino Acid is elevated? How do the patients present clinically? What do they need to remove from their diet?

A) Homocystinuria is the dx. The amino acid elevated is methionine since its conversion is impossible. The patients present as a Marfan's body w/ scoliosis, dislocated eye lenses, mild mental retardation, thrombosis. The restriction of proteins like sulfhydryl groups leads to very low protein, foul tasting diets.

993.

Oh boy, a patient of yours has galactose 1 phosphate uridyl transferase deficiency. What enzyme is missing? What is the clinical presentation? What is the treatment?

A) This dx is the most common error of carbohydrate metabolism, galactosemia. Glycolysis is affected, and you see evidence of liver

411

failure, direct hyperbilirubinemia, coag disorders, renal problems (acidosis, glycosuria), emesis, and sepsis. TREAT by eliminating all formulas and foods with galactose.

994.

Q) What is the enzyme disease associated with ornithine transcarbamylase deficiency? How is it inherited? What toxic metabolite forms? MOA? Clinical presentation? Treatment?

A) This...OTCD...is a urea cycle defect inherited in an X-linked fashion. Ornithine couples with carbamylphosphate to make citrulline. If the enzyme is def., ornithine builds up and then urea cannot be made and excreted. AMMONIA builds up instead, and within only 24 hours, the newborn baby will become lethargic and have seizures. DIAGNOSIS by measuring the orotic acid levels in the urine. TREAT with a low fat diet and alternate pathways to excrete nitrogen via benzoic acid and phenylacetate.

995.

Case: Your patient has a respiratory disorder and is cyanotic. He comes in with a normal arterial oxygen tension (PaO<sub>2</sub>) and a LOW arterial oxygen saturation (SaO<sub>2</sub>). Your med student rushes to give oxygen therapy and the patient is STILL cyanotic. What does he have? (Pick either Right to left SHUNT, Methemoglobinemia, Respiratory Acidosis). Why????????? How do you treat?

A) He has Methemoglobinemia. IRON needs to be in the ferrous form (+2) to be able to bind oxygen. In this dx, the IRON is in the ferric form (+3). So giving O<sub>2</sub> does not help. You must give methylene blue which aids in the conversion.

996.

CASE: Please refer to the previous HY Concept 995...why is the answer not right to left shunt? (This is a crucial point)

A) Because, while O<sub>2</sub> therapy has very little effect, BOTH oxygen tension (PaO<sub>2</sub>) AND oxygen saturation (SaO<sub>2</sub>) are LOW. Recall that in methemoglobinemia, the oxygen gas exchange is NOT affected in the lungs, so PaO<sub>2</sub> is NORMAL there!

997.

Q) Speaking of RBCs, a 14 month old male child presents with a hemoglobin of 7.6 and a hematocrit of 24%. The MCV is 65 and the adjusted reticulocyte count is 1.0. Is this ineffective erythropoiesis or not?

A) An ARC less than 2.0 is ineffective erythropoiesis for the anemia, an anemia with ARC more than 2.0 signals hemolysis or blood loss and decent erythropoiesis.

998.

Case: Everyone is going to have to do this procedure: Checking for the

413

red reflex...what happens though if you see a reflection from a white mass within the eye giving the appearance of a white pupil? What diseases can cause this?

A) Congenital cataracts, Retinoblastoma, Glaucoma...RECALL if you see signs of a retinal hemorrhage, think SHAKEN BABY SYNDROME and protect the baby!

999.

Case: One of your patients comes in with blood streaked feces. He is an 19 month old. Hemocult is positive. What diagnoses is MOST common here?

A) Anal fissure.

1000.

Refer to the previous HY concept 999. The 19 month kid with the bloody stool is sitting there while your inexperienced med student asked you "Why can't this be IBD?"

"Why can't this be Necrotizing enterocolitis?"

"Why can't this be a Mallory-Weiss tear?"

"What can't this be peptic ulcer disease?"

(So what do you say to each?)

A) Tell him that IBD (Chron's and Ulcerative Colitis) and necrotizing

enterocolitis appears later in childhood, and a Mallory Weiss tear and PUD will produce dark MELENA instead!

1001.

Case: You see a renal patient with edema, but NO other systemic diseases. You determine this is minimal change disease. The patient asks if she will have to take any medication. What do you answer?

A) Yes, you need to treat with steroids and salt restriction. In severe cases, use diuretics. This dx has a great prognosis.

1002.

Case: Your attending gives you a case of nephrotic syndrome. On biopsy, how will diffuse proliferative glomerulonephritis differ from poststreptococcal glomerulonephritis on microscopy?

a) glomerular basement membrane thickening = diffuse mesangial proliferative glomerulonephritis.  
with poststreptococcal glomerulonephritis, you will see inflammatory cell infiltrates.

1003.

Quick, recall the azygous veins? Where do they drain? Is it the Right Brachiocephalic vein?

A) NO,.. Superior Vena Cava

1004.

True or False: Your patient is going to an amusement park. She is worried of roller coasters. She needs an antihistamine for possible

416

nausea/motion sickness. You warn her that an overdose of diphenhydramine (Benadryl) will cause what?

First-generation H<sub>1</sub>-receptor antagonists, such as diphenhydramine, may be particularly dangerous because they may cause pronounced agitation leading to rhabdomyolysis and acidosis. Also, a quinidinelike sodium channel blocking effect may cause delayed conduction and contribute to ventricular dysrhythmias. This on top of blurry vision, dry mucous membranes, agitation, absence of sweating, GI non-motility. (anti cholinergic stuff)

1005.

Quickly, if you see a normal patient that walks unaided, is he 12 months old OR 15 months old?

A) 12 months

1006.

Most likely, a patient of yours who is NORMAL and is now using three word combo sentences is how old? (24 months or 36 months)?

A) 36 months

1007.

Quickly, in psychology experiments, how do you define the POWER of a study? Is high power good?

A) Power assesses the probability of rejecting the null hypothesis when it is false (that is a positive thing). High power is good, so you can raise it by increasing sample size.

1008.

Case: I show you a picture of a circuit of capillaries in "parallel". If I

417

ADD another circuit, will the Total Resistance increase or decrease?  
This IS within the scope of Step 1.

A) In parallel circuits, the addition of a resistor/circuit LOWERS the Total Resistance! Think of the way your house/apartment is hooked up!

1009.

A 52 year old female patient of yours is taking a drug treatment for a nervous system disorder. She is showing some bleeding gums, lip and nose broadening, and some signs of hirsutism. Which drug, phenytoin or amitriptyline, is she taking?

A) Phenytoin!

1010.

What is the diagnostic standard for pneumonia besides chest x ray for adults?

A) Get some Sputum.

1011.

Q) In the retina, are there more photoreceptors or ganglion cells?

A) Photoreceptors...the retina has a main function with convergence.

1012.

Q) In the retina, are there more RODS or CONES?

A) RODS!

1013.

Q) Nerve and blood vessels enter and exit the eye where?? are there photoreceptors there?

418

A) At the blind spot...which is the optic DISK. No photoreceptors reside there.

1014.

Q) What is a more likely cause of an infarct in tissue, (size of the blood vessel OR hemoglobin concentration)?

A) Size of the blood vessel. TRICKY...remember that tissue hypoxia assoc. with hemoglobin to a tissue is DIFFERENT from an infarct!!

1015.

Q) Point on a diagram where you will see an infarct occur...(a small vein or a medium sized muscular artery)?

A) Medium Sized Muscular Artery...think TURBULENCE.

1016.

Q) If shown a major organ, can you identify it on a histo slide? And the major cells?

A) please answer YES!

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1017.

True or False: AVOIDANCE is the best prevention against Strep. pneumo.

Plus, is there a brand name vaccine for it?

Name at least two populations that MUST receive it!

A) False, give Prevnar, a pneumococcal vaccine. Give at older patients, patients with splenectomy, HIV patients, Diabetics.

1018.

Q) Tell us EXACTLY, what effect do cardiac glycosides have on heart rate and contractility...if any?

A) They do not have a DIRECT effect on heart rate...however, they do increase CONTRACTILITY...with associated angiotensin-mediated vasoconstriction.

1019.

Q) Does Isoproterenol increase BOTH cardiac contractility AND heart rate or just one of them?

A) It increases BOTH! And has concomitant vasodilation.

1020.

Case) You are sitting around watching ER on television. A pt with renal disease is given dopamine. What is a slight difference between giving dopamine vs. dobutamine?

A) Dopamine increases renal blood flow a little more than dobutamine because of renal receptors. And dopamine increase vasoconstriction a bit more because of its alpha adrenergic activation.

1021.

Case: A coronary smoking patient is post MI. You are looking at two antiplatelet agents (aspirin and dipyridamole). What is the MOA differences?

A) You know that aspirin binds cyclooxygenase and stops thromboxane production. But dipyridamole acts at the level of platelet adhesion.

1022.

Case: Your inexperienced med student picks up a fibrinolytic like tPA (which revves up plasmin production). He administers it to a hemorrhagic stroke (in the Circle of Willis) patient. Is that right?

A) NO! You give fibrinolytics for THROMBOTIC strokes...giving them for hemorrhagic stroke victims could kill them. Does it make sense?

1023.

Case: To save your medical license, for the last pt in HY c 1022, you administer an antidote of what?

A) aminocaproic acid, which is a plasmin antagonist!

1024.

True or False: You are an ER doctor and you give a shot of epinephrine to a shock patient. His vessels vasoconstricts AND vasodilates plus BOTH his systolic and diastolic blood pressure increase. (Again, is this true or false?)

A) FALSE...Epinephrine does vasoconstrict and vasodilate...BUT only systolic blood pressure goes up, the diastolic BP goes down due to the vasodilation. Recall, BOTH contractility and heart rate increase.

1025.

Case: You administer a drug to a cardiac pt., ISOPROTERENOL. You know it increases HR and contractility, but does it VASODILATE? OR does it VASOCONSTRICT? WHY???

A) This drug vasodilates a LOT, but does NOT vasoconstrict. This is because ISOPROTERENOL is a nonselective B agonist, it has NO effect on the alpha 1 receptors, so would produce no vasoconstriction.

421

1026.

Case: Your patient has a cold and cannot breathe well. You give him inhaled phenylephrine. Does this directly affect the heart rate and contractility? Will it vasodilate? What exactly does it do and give a couple of clinical uses. (Be SURE to differentiate the sympathomimetics).

A) This sympathomimetic is primarily a VASOCONSTRICTOR. Thus, it can be used for glaucoma and for nasal congestion. It is an alpha 1 agonist which CONSTRICTS vessels, so you can even see it used in anesthesia, to elevate the Blood Pressure. It has LITTLE or NO effect on the Beta receptors of the heart, so heart rate and contractility is not affected!

1027.

Case: Your patient, a 69 year old female with a known brain tumor in the deep frontal cortex, is found lying unresponsive with dilated pupils. She has weak pulses and slow breathing. What is the likely lesion?

A) Uncal herniation is most likely. You will see the unreactive pupils to light and dilation of the pupils. The brain stem is compressed, so the nuclei responsible for heart rate and breathing are weak. This is an Emergency!

1028.

Case: Asthma...so common. What is the MOA of Ipratropium Bromide?

A) Also called Atrovent in clinics, this anti-cholinergic asthma drug works by acting at muscarinic receptors of the parasympathetic nervous system. Chemically related to atropine, it has antisecretory properties and, when applied locally, inhibits secretions from serous

422

and seromucous glands lining the nasal mucosa.

1029.

Case: Which pt. is more worrisome after a large meal that will secrete acid? (One with a Gastric ulcer or Duodenal ulcer)?

A) Duodenal ulcer (higher number of parietal cells are found)...strangely, a gastric ulcer is associated with lower numbers of parietal cells. A Gastric ulcer is often seen with mucosal breakdown in the stomach.

1030.

True or False: Pancuronium relaxes the skeletal muscle and ACh counteracts it.

True.

1031.

What drug is given for phenylephrine toxicity and why (Choose either prazosin or diphenhydramine)?

A) Prazosin, because it is a direct antagonist at the alpha 1 receptors which are turned on by phenylephrine.

1032.

Case: A pt. overdosed on an ACh boosting drug. His pupils are constricted. What prototypical drug can quickly reverse this exact effect?

A) Atropine

1033.

Case: Von Willebrand's Disease...other than Factor VIII def, what is happening at the level of the platlets to explain why the afflicted persons have DECREASED coronary artery disease?

A) The Von Willebrand Factor is responsible for adhering the platlets to the endothelium. No platlet clots...less coronary artery blockage.

1034.

Q) True or False: vascular endothelial cells make endothelin, angiotensin II, Neuropeptide Y, Vasopressin, which VASOCONSTRICTS. The vascular beds also make bradykinin, substance P, and ANP, and VIP (vasoactive intestinal peptide), which VASODILATES.

A) Of course True!

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1035.

What one, (reduced creatinine clearance OR reduced hepatic function) will precipitate digoxin toxicity?

A) Remember, digoxin is cleared by the KIDNEYS, so reduced creatinine clearance is dangerous.

1036.

The majority of CO<sub>2</sub> produced by the body is transported in the blood as (carbonic acid, bicarb, dissolved CO<sub>2</sub>, hemoglobin bound)?

A) Bicarb...recall the mech. AFTER the gas exchange, carbonic acid is made after the RBCs obtain O<sub>2</sub> from the lungs. Then, the O<sub>2</sub> increases oxygen tension, PO<sub>2</sub>, and displaces H<sup>+</sup> ions on hemoglobin. The

424

displaced H<sup>+</sup> ions combine with bicarbonate to form carbonic acid which then dissociates to CO<sub>2</sub> and water.

1037.

Pretend you are looking at a radiograph with an obstruction of the common bile duct. Tell us a few problems the patient may have...

A) Because of the loss of bile salts and absorption of fat soluble vitamins, he or she will bleed out (no vit K), have bone problems (no vit D), tetany for the same reason, and vit B12 def over the long term.

1038.

True or False: Rapid inhalation of CO<sub>2</sub> is very useful to diagnose PANIC disorder.

A) True

1039.

Case: Pt. comes in with a lesion of the deep peroneal nerve. How will he present? What main group of muscles are affected? Name a couple of the muscles.

A) FOOT DROP. Dorsiflexion is lost because the deep peroneal nerve innervates mostly the anterior compartment. The superficial peroneal nerve innervates mostly the lateral compartment. Muscles like the tibialis anterior, Peroneus tertius, extensor digitorum longus, are affected.

1040.

Q) What is a good sign that the bone marrow is working well? (i.e. effective erythropoiesis?)

A) High reticulocyte count and maybe the presence of "shift cells" (which have a bluish discoloration on Wright-Giemsa stain)

1041.

Case: Pt with anemia from pyruvate kinase def. The anemia is primarily mild because ???

A) This AR disease develops an intrinsic hemolytic anemia with extravascular hemolysis. Pyruvate kinase catalyzes to form ATP. Without the ATPase dependent sodium potassium pump, RBCs lose their shape. BUT, substrates proximal to the point pyruvate kinase works is built up, and 2,3 bisphosphoglycerate concentration shifts the O<sub>2</sub> dissociation curve to the right, making O<sub>2</sub> delivery better.

1042.

Q) What is the rationale for using an ALPHA 1 RECEPTOR BLOCKER for BPH in men? What is one typical drug with this MOA?

A) Alpha receptor blockage causes smooth muscles in the bladder neck and prostate to relax to allow peeing. A significant component of the BPH complex and its associated symptoms is believed to be related to the smooth muscle tension in the prostate stroma, urethra, and bladder neck. The smooth muscle tension in these areas is mediated by the alpha1-adrenergic receptors; therefore, alpha-adrenergic receptor-blocking agents should theoretically decrease resistance along the bladder neck, prostate, and urethra by relaxing the smooth muscle and allowing passage of urine. Phenoxybenzamine or Terazosin are two such agents...

1043.

Case: Pt. with migraine headaches. Should you give a Beta-agonist OR a Beta blocker? What other choices are in existence?

426

A) Commonly, Beta-BLOCKERS like atenolol are used. Some like to give amitriptyline, antihistamines, and valproic acid.

1044.

Case: You have to, on sight, recognize the main presentation of ALL in children (don't send these kids home with Tylenol)...so what do you remember (give a brief presentation)?

A) A young child comes in with fever, bloody noses, lymphadenopathy, bone pain, hepatosplenomegaly. Labs show normocytic anemia, thrombocytopenia, lymphocytosis.

1045.

Now, your attending asks which is more common, B-cell ALL in kids or T-cell ALL in kids?

A) B-cell ALL is 80% of all ALL in children.

1046.

Now your attending asks "name at least three B-cell ALL markers and one T-cell ALL marker". What do you say?

a) B-cell ALL has CD19, CD20, CD22, CD24, CD21, CD79. T-cell ALL has CD3

1047.

Even though prognosis for childhood ALL is improving, name two gene rearrangements that have POOR prognosis:

A) BCR-ABL and MLL translocations

1048.

427

Case: CAH, or Congenital Adrenal Hyperplasia. You see a case of 21-Hydroxylase deficiency (CYP21). Your patient is a male GENOTYPE. Is his genitalia normal? How do they typically present?

A) YES, their genitalia are normal! So it is hard to diagnose at birth. In a few weeks though, they have salt wasting, dehydration, hypotension, hyponatremia, hyperkalemia. HOWEVER, the male patient could present later in childhood too, with early pubic hair and accelerated linear growth.

1049.

Another CAH case, congenital adrenal hyperplasia. You see two similar cases with one a girl and one a boy. Both have CYP11 def. When do they usually present and how?

A) NOTE here the key point is the gradual HYPERTENSION as they come into the hospital after only a couple of weeks in life ironically with a salt loss crisis. Aldosterone production is inhibited, the mineralocorticoid deoxycorticosterone is boosted with Na and water retention.

1050.

Another case yet of CAH, congenital adrenal hyperplasia. Again we see CYP 21 or 21-Hydroxylase def. But this time we see a female genotype instead of a male genotype. Is her genitalia normal?

A) Unlike males, females with CYP 21 have AMBIGUOUS GENITALIA and are often thus diagnosed at birth. Recall this is a Salt Wasting syndrome from inadequate aldosterone synthesis.

1051.

YES, yet another case of CAH, congenital adrenal hyperplasia, but a

428

rarer form. This is 17-alpha hydroxylase def. Tell me the MOA and how a male genotype and female genotype will present!

A) Here, ONLY the mineralocorticoid line is produced. Therefore, the lack of androgens will make a male genotype present at birth with either totally female genitalia or mild ambiguity. Females will present normally. The male may actually be mistaken for a female and be raised as such until symptoms or puberty! Both the male and female will eventually present with serious HYPERTENSION from overproduction of aldosterone.

1052.

SO REMEMBER, as a general rule, the "teens", 11 B hydroxylase def and 17 A hydroxylase def result in hypertension, 21-B hydroxylase def is salt wasting.

Also, CYP 21 def. give females ambiguous genitals at birth.

Also, CYP 17 def. give males ambiguous genitals/no penis at birth

1053.

Again, the teens, 11 and 17 hydroxylase def., are like teen-agers who steal your car and give you headaches and HYPERTENSION.

Also, 21 is a good age for a mature MALE, so he does not have abnormal genitals at birth. but for females, 21 is BAD since genitals are abnormal.

Also, 17 is a good age for a lovely FEMALE, so she does not have abnormal genitals at birth.

429

The opposites are also true. This stuff is hard...hope my mnemonics help.

1054.

Serious hemorrhages in patients are most likely caused by which, coagulation factor def. or thrombocytopenia?

A) Coag. factor def. are more common.

1055.

Case: Traveler to S. China or Africa or Philippines. You see flukes under microscope which your attending says was caused by the patient swimming in FRESH water with SNAILS. What medicine is recommended?

A) Snails...equate with Schistosomiasis...S...goes with S...give praziquantel. Salt water is NOT infected with Schistosomiasis snails.

1056.

Pt case: A child pt. of yours is itching with pinworms after playing outside in the dirt. What med is often used? Tell me more about the bugs...

A) These pinworms are part of the NEMATODE PHYLUM, the most numerous multicellular animals on earth. Many are parasites found

430

EVERYWHERE on plants, animals, and many are free living. Although most are small, they can grow up to 1 to 8 meters! (Kidney worm and worms found in the gut of a sperm whale!). Give your patients Mebendazole for these helminths. (NOTE: Helminths are part of the NEMATODE PHYLUM and comprise Roundworms (like Ascaris), Tapeworms (like Echinococcus), and Hookworms, Pinworms, Whipworms.

1057.

Q) What is a second line agent if TMP-SMX fails upon treatment of PCP in AIDS? (Hint: this agent works by inhibiting growth of protozoa by blocking oxidative phosphorylation and inhibiting incorporation of nucleic acids into RNA and DNA, causing inhibition of protein and phospholipid synthesis.)

A) Pentamidine

1058.

Sometimes Clindamycin is given for PCP. What is its MOA?

A) We went over this a LONG time ago, but...Clinda inhibits bacterial protein synthesis by inhibiting peptide chain initiation at the bacterial ribosome where preferentially binds to the 50S ribosomal subunit, causing bacterial growth inhibition.

1059.

431

True or False: The vaccine against whooping cough is made from live bacteria.

A) False, it is made from KILLED bacteria.

1060.

REMEMBER: "MMR. P" {sounds like Mr. P is aLIVE} (Measles, Mumps, Rubella, and Polio) are made from LIVE viruses.

REMEMBER: "H.I.R. are made from KILLED viruses." (Hep B, Influenza, Rabies). Think of mnemonic, "I KILLED HIR yesterday."

REMEMBER: "d.D.T. is a toxin." (D=Diphtheriae, T=Tetanus, and D.T. is like the weed killer DDT, a toxin of sorts). These two are made from bacterial toxin.

1061.

In injury, what does endothelial secretions of TPA, Tissue Plasminogen Activator do? vWF? PGI2? Nitric oxide/NO?

A) TPA initiates the fibrinolytic system. vWF does platelet adhesion. PGI2 inhibits platelet aggregation and vasodilates (it is a prostacyclin). NO vasodilates upon sensing injury.

1062.

432

Case: An older woman, 53, hitting menopause with a history of hot flashes and thrombus formation. What do you treat with (estrogen, progesterone, both, or clonidine, or tamoxifen)?

A) CLONIDINE, the other choices either cause neoplasm/thrombus or CAUSE hot flashes in the case of tamoxifen.

1063.

Case: The anti-parkinson drugs are a HUGE concept. If I gave you a diagram of a synapse, with arrows everywhere, you need to point to the places where each of the anti Parkinson drugs has its action. What is the MOA and LOA (Location of Action) of:

Amantadine

—LOA, look for the arrow right after the neuron because it works to PUSH out more dopamine.

1064.

Case: The anti-parkinson drugs are a HUGE concept. If I gave you a diagram of a synapse, with arrows everywhere, you need to point to the places where each of the anti Parkinson drugs has its action. What is the MOA and LOA (Location of Action) of:

Benztropine

—it acts at the level of an ANTI Muscarinic at its receptor, thus lowering Ach levels.

1065.

Case: The anti-parkinson drugs are a HUGE concept. If I gave you a diagram of a synapse, with arrows everywhere, you need to point to the places where each of the anti Parkinson drugs has its action. What is the MOA and LOA (Location of Action) of:

Carbidopa

—Pick the arrow where it points to the periphery. It prevents dopamine from metabolizing in the body so it can move into the CNS.

1066.

Case: The anti-parkinson drugs are a HUGE concept. If I gave you a diagram of a synapse, with arrows everywhere, you need to point to the places where each of the anti Parkinson drugs has its action. What is the MOA and LOA (Location of Action) of:

Bromocriptine

—Acts as a dopamine analog/agonist at the post synaptic receptor for those with dopamine deficiency.

1067.

Case: The anti-parkinson drugs are a HUGE concept. If I gave you a diagram of a synapse, with arrows everywhere, you need to point to the places where each of the anti Parkinson drugs has its action. What is the MOA and LOA (Location of Action) of:

Selegilene

—This agent blocks the MAO-B, thus preventing the degradation of dopamine to homovanillic acid

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1068.

True or False: GIP, Gastric INHIBITORY peptide, augments insulin secretion after a meal.

A) TRUE, it sounds false, but that is what GIP does.

1069.

Q) If you destroy the trilineage myeloid stem cell in the bone marrow, will you get aplastic anemia OR Polycythemia OR T-cell deficiency?

A Aplastic anemia, at this early stage.

1070.

Q) After a meal, during the intestinal phase, what happens to the pH of the bile? This is due to what secretion? Is it somatostatin?

A) The pH will go UP because of secretin induced release of bicarbonate.

1071.

True/False: Gastrin stimulates D cells in the stomach to release acid thus lowering the pH. Also, the parietal cells indirectly secrete somatostatin, which acts in a paracrine fashion on G cells to stop producing gastrin.

False! Should read: Gastrin stimulates parietal cells in the stomach to release acid thus lowering the pH. Also, the D cells directly secrete somatostatin, which acts in a paracrine fashion on G cells to stop producing gastrin.

1072.

Q) Are small cyst lesions in the lenticular nucleus, internal capsule and thalamus consistent with a Hypertensive Crisis OR an embolism OR a demyelinating disease OR a thrombus formation?

A) Hypertensive crisis

436

1073.

Q) Your MD/PhD chief resident asks you in front of morning report: "Hey you see a case of Type III Hypersensitivity reaction, is the mech of tissue destruction:? pick one

1)the complement systems release of histamine and chemotactic agents that direct damage tissue..OR is it

2)NK cells that target tissue where immune complexes reside

3)Neutrophils that migrate to the site of immune complexes where chemotaxis directs.

A) Answer is 3. (Note, as an aside, the Arthus rxn is local, while serum sickness is SYSTEMIC)

1074.

Q)Review all the MAIN changes with respect to that famous graph of the cardiology stroke volume curve found everywhere like in [FA](#) and in [BRS Physiology](#) w/ Constanza. (For example, know how the graph changes if one moves from the standing to sitting and vice versa; and if someone is given an inotrope, exercising, etc.) This is super HY, but I don't know how to draw pictures here...yet...Tommy  
(There is no answer below)

1075.

Q) What of the DNA viruses have NO ENVELOPE?

437

a) A) Think "A" and "PA" (DNA that start with "A" and "PA" have NO envelope) Say that 10 times very fast. So, A-denoviruses and P-arvoviruses and Pa-povavrruses have NO envelope.. The others, Herpes, Hepadna, Poxvirus, come in an envelope.

1076.

Case: Very COMMON. You have a 22 year old woman who is in the ER for malaise and altered mental status. She has a 4 year history of SEVERE HTN. Her labs are 110 Na, 5.5 K+, Serum osm 240, Urine sodium 5 mEq/L. Which is likely making her hyponatremic? (ADH or aldosterone)

A) ADH...aldosterone hypersecretion would make her K+ low.

1077.

Q) The 7 membrane G protein coupled binding AND the PHOSPHOINOSITIDE pathways are EVERYWHERE in our bodies. The Phosphoinositide pathway begins with which? (Phospholipase A or Phospholipase C)? Then tell us the rest of the steps of activation. If you can do this, you cannot be tricked into an inferior answer choice.

A) Answer is Phospholipase C. After it is activated, the membrane releases IP3 and DAG by hydrolysis. The IP3 is the one that releases Ca +2 from the Sarcoplasmic Reticulum. The DAG activates protein kinase C (not A) to phosphorylate and produce cell effects.

438

1078.

Q) What substrate activates CYTOPLASMIC guanylyl cyclase (hint: it is a vasodilator often used in acute HTN? And what follows in the cell?

A) A) NO (Nitric Oxide) diffuses through the membranes and then activates cytoplasmic guanylyl cyclase to form cyclic guanosine monophosphate (cGMP). (cGMP) then relaxes smooth muscle.

1079.

Q) Think carefully, which protein increases adenylyl cyclase activity, (Gs, OR cAMP)? Tricky.

A) A) You have to get the steps down. The Gs cell surface proteins activate adenylyl cyclase which then activates cAMP.

1080.

Q) Sarcoma botryoides, those grape like lesions which comes out of the genitals of young boys and girls, are a cancer assoc. with a poor prognosis. What tumor markers will you find? (give 2)

A) Because they come from MUSCLE, they stain desmin and myoglobin.

1081.

439

Q) Which one, a seminoma or a yolk sac tumor is the most commonly seen childhood testicular tumor?

A) Yolk sac tumor. You will see the marker alpha fetoprotein. They have a good prognosis.

1082.

Q) Young kid with fever, chills, malaise. PE has gray membrane over the tonsils. This is diphtheria. Is this gram positive? What animals besides humans carry it?

A) Yes, it is gram positive. And this is a trick question. ONLY humans carry this. (Whenever ONLY humans are carriers, vaccination is MUCH easier...for obvious reasons.)

1083.

Q) You have an older gentleman with PAINLESS testicular mass. Which is it more likely, (testicular malignant lymphoma OR prostatic adenocarcinoma)?

Tricky!

A) Malignant lymphoma. The prognosis is POOR.

1084.

Q) Your patient is a child with Hypertension, a missing iris, unilateral

440

abdominal mass. Does he have (renal adenocarcinoma OR a Wilm's Tumor)?

A) Wilm's Tumor.

1085.

Q) Are any of the following statements about aldosterone false?

- 1) Aldosterone is stimulated (its release), by plasma potassium  $K^+$  concentration.
- 2) The way aldosterone is made is from Ang II which stimulates receptors on zona glom. Cells. Stimulation makes corticosterone turn into 18 hydroxycorticosterone which then turns into aldosterone.
- 3) Aldosterone attaches to a membrane receptor to stimulate  $Na^+$  release.

A) Statement 3 is false. Aldosterone Diffuses into the cytosol and attaches to a cytosolic receptor.

1086.

Q) Know how to read a Lineweaver Burke plot (double reciprocal plot). I cannot draw a diagram, but what does a competitive inhibitor look like? What about a noncompetitive inhibitor?

Recall that competitive inhibitors increase the  $K_m$  but do not affect the

441

V<sub>max</sub>! Noncompetitive inhibitors decrease the V<sub>max</sub> of an enzyme without affecting the K<sub>m</sub>. Know all the points, like 1/K<sub>m</sub> and 1/V<sub>max</sub> on that famous straight line graph. For example, which one crosses the line of the "no inhibitor?" Please look it up on a book like BRS or Lippincott! Don't forget!

1087.

Q) Flecainide...let's say you see this and I give you other antiarrhythmics like Quinidine, Sotalol. Which class is Flecainide and what does it do to phase 0 and phase 3 ?

A)It markedly prolongs phase 0 and has little effect on phase 3!

1088.

Q) Which class of a.a. is Quinidine in? How is phase 0 and 3 affected?

A)Quinidine is a Class 1a and prolongs both phase 0 and phase 3. As an aside, class III drugs like sotalol have NO effect on phase 0 but often prolongs phase 3 (Think Class III=Phase 3 only).

1089.

Q)Which is a better example of HYPERPLASIA?...

- 1)a man with a thickened bladder wall from urethral obstruction? OR
- 2)a man with difficulty urinating due to prostate enlargement?

A)The answer is 2) prostate enlargement. Answer 1 is wrong because it is like a heart that must pump against higher afterload....hypertrophy develops, the cells swell.

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1090.

You have a patient with severe cyanotic congenital heart disease. Does he or she have:

- 1) an INCREASED alveolar-arterial gradient
- 2) normal hemoglobin and hematocrit
- 3) will correct his or her hypoxemia on 100% oxygen?

A)INCREASED alveolar arterial gradient. This is because deoxygenated blood is mixing with oxygenated blood from the lungs. This increases the DIFFERENCE MEASURED between the alveolar and arterial oxygen!!!

1091.

Q) In the previous concept, why doesn't 100% O<sub>2</sub> by mask correct a cyanotic congenital heart disease patient?

A) Because there would be no effect on flow reduction of the Unoxygenated blood into the arterial system due to the R to L shunting.

443

1092.

Q) Your attending asks you if:

1) OSTEOLASTS have a dominant role in Metastatic Prostatic Cancer

OR

2) OSTEOLASTS have a dominant role in Multiple Myeloma?

A) 1) is the answer. Osteoblasts are assoc. with prostate cancer. They contain alk phos. (and PSA antigen). Multiple myeloma is assoc. with OSTEOLASTS. OSTEOLASTS are also responsible for osteopetrosis (via resorption of bone), Paget's Disease (early phase), and Primary hyperparathyroidism!

1093.

Q) GREAT CONCEPT Question: Say I present the Renin-Angiotensinogen-Angiotensin I-Angiotensin II-Aldosterone Pathway on a diagram. Say I administer then a loading dose of an ACE inhibitor. Which of the compounds in the pathway are ELEVATED or DECREASED?

A) Since ACE inhibitors block ACE (angiotensin-converting enzyme), which converts angiotensin I to angiotensin II, the decreased ANG II then decreases aldosterone (with higher K<sup>+</sup> serum levels). The resulting decrease in blood pressure will make INCREASED renin, which will then lead to HIGHER ANG I levels! Note: ACE inhibitors will NOT affect angiotensinogen levels! Cool!

1094.

Q) True or False: All the FOLLOWING dx are a direct result of portal hypertension: Ascites, Splenomegaly, Esophageal varices, Caput medusae.

A) True!

1095.

Q) What is the most common cause of PORTAL HYPERTENSION?

A) Alcoholic cirrhosis, which WILL cause obstruction of portal vein blood flow.

1096.

Q) What are the PREhepatic, HEPatic, POSThepatic causes of Portal Hypertension?

- A) Prehepatic: Possibly a thrombus or fibrosis of the portal vein.
- B) Hepatic: Cirrhosis...including sinusoidal system destruction, nodules, intrahepatic portal vein fibrosis.
- C) Posthepatic: These are the hepatic vein thrombosis like Budd Chiari syndrome and maybe right heart failure...

1097.

Q) Which factoid is correct about the genetic mechanism functions of

445

erythrocyte ABO group antigens?

(Choose either: 1) They involve genes that code for enzymes OR  
3) They also code for the antigens of the Rh.

A) ABO = They involve genes that code for ENZYMES that attach carbohydrates to the H antigen stem. KNOW that A and B antigens are ALSO present on endothelial cells, not just on RBCs. thus, ABO and Rh are different systems.

1098.

Q) One of your patients had a mastectomy after an estrogen sensitive breast cancer. What medicine do you give her? (Pick either Tamoxifen or Mifepristone)

A) Tamoxifen.

1099.

Q) A hirsute woman with PCOS can be treated with the following meds: Spironolactone AND Leuprolide...along with Oral Contraceptive Pills...what is the MOA of each?

WATCH THE SUBTLE DIFFERENCES!!!!

A) Spironolactone—This potassium sparing diuretic is also an antiandrogen.

B) Leuprolide—This suppresses steroidogenesis by decreasing LH and

446

FSH levels. GnRH agonist that DOWNREGULATES the pituitary!

1100.

Case: Hirsutism, besides the previous concepts and the drugs mentioned, understand some docs give Finasteride, which is a 5-alpha-reductase inhibitor approved for use in benign prostatic hypertrophy and in male-patterned alopecia. Blocks conversion of testosterone to its more active metabolite, dihydrotestosterone.

1101

Q) Regarding the previous concept of PCOS, some also are known to give Danazol, which is more commonly used for ENDOMETRIOSIS. What is this MOA please?

A) ) This, recall, is an Antigonadotropic agent which acts by inhibiting the midcycle FSH and LH surge and preventing steroidogenesis in the corpus luteum. This is an androgen and a partial agonist. Know the subtle differences. IF YOU HAVE TO CHOOSE, Danazol is PREFERRED over others for endometriosis

1102

Case: You see a 69 year old female with post op day 3. She had a left knee replacement for osteoarthritis. She has PMH of Diabetes I and glaucoma. Her sugars are under control. Meds are Oxycodone for pain. Also, she has PT at the hospital. What other med does she need most urgently? (pick either an NSAID or Oral Coumadin)

A) Answer is Oral Coumadin. Risk here are very high for a Pulmonary

447

Embolism. Aspirin and ACE inhibitors are good in the long run, but you need to address what may kill her first!

1103

Case: You are seeing a 20 year old white female with 5 months of crampy abdominal pain on the RLQ made worse after eating. There is an increase in stools to 5-6 times a day and she has lost 15% of her weight. PE is sig. for aching in her knees, two oral ulcers on her lip, tender RLQ, no masses, guaiac positive for stool. What does she HAVE, and what is the Rx? (is it appendicitis?)

A) This is a classic presentation of Crohn's Disease!! She has got the RLQ pain...WITH diarrhea, weight loss, APTHOUS ulcers and muscle aches. The location is such that it is in the terminal ileum, so a small bowel barium study is warranted.

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1104

For the previous case of Crohn's, what are 3 drugs of therapy?

A) Mesalamine, 5-aminosalicylic acid (5-ASA), Prednisone, and Hydrocortisone\_\_\_\_\_

1105

For HY concept 1103, we saw a case of ileal Crohn's. KNOW that a colonic Crohn's presents slightly differently! And use a barium enema or sigmoidoscopy to visual the diagnosis of COLONIC Crohn's instead of a small bowel series.

1106

Q) The 7 membrane G protein coupled binding AND the PHOSPHOINOSITIDE pathways are EVERYWHERE in our bodies. The Phosphoinositide pathway begins with which? (Phospholipase A or Phospholipase C)? Then tell us the rest of the steps of activation. If you can do this, you cannot be tricked into an inferior answer choice.

448

A) Answer is Phospholipase C. After it is activated, the membrane releases IP3 and DAG by hydrolysis. The IP3 is the one that releases  $Ca^{+2}$  from the Sarcoplasmic Reticulum. The DAG activates protein kinase C (not A) to phosphorylate and produce cell effects.

1107

Q) What substrate activates CYTOPLASMIC guanylyl cyclase? And what follows in the cell?

A) A) NO (Nitric Oxide) diffuses through the membranes and then activates cytoplasmic guanylyl cyclase to form cyclic guanosine monophosphate (cGMP). (cGMP) then relaxes smooth muscle.

1108

Q) Regarding the previous concepts on hirsute women, some also are known to give Danazol, which is more commonly used for ENDOMETRIOSIS. What is this MOA please?

A) This, recall, is an Antigonadotropic agent which acts by inhibiting the midcycle FSH and LH surge and preventing steroidogenesis in the corpus luteum. This is an androgen and a partial agonist

1109

Q) Which ANION (not cation) is most predominant in a Healthy normal individual's urine? Guess first...

A) Chlorine! But, note that if your body is alkalotic, you will start peeing out more  $HCO_3^-$  than  $Cl^-$ . Watch the pH numbers

1110

Q) A 40 year old overweight woman comes to you with diastolic hypertension. A PE reveals a fatty face, hirsutism, central obesity. Labs shows elevated morning and evening cortisol levels. You then do a low dose dexamthasone suppression test...there is no lowered cortisol. Then you do a HIGH dose dexamethasone test which DOES bring down her cortisol serum levels. The 24 hr. urine test has high cortisol levels. Is this condition from PCOS? Adrenal Hyperplasia? Or something else? Explain this often tested/pimped disease MOA?

449

A) The patient has Cushing disease. This is caused by a benign adenoma likely in the PITUITARY...which is secreting ACTH. Signs are the cortisol, 17 ketosteroids, lower mineralocorticoids. Too much cortisol raises glucagons and epinephrine for glycogenolysis, gluconeogenesis and lipolysis. You will then get hyperglycemia. Then you will get more insulin release, then you will get glucose UPTAKE in adipose tissue. Since gluconeogenesis is ENHANCED by the cortisol, muscle is broken down for the necessary amino acid precursors in the extremities. Thus, you get central obesity! WOW! Complex! Know also that cortisol revves up erythropoietin, giving polycythemia. KNOW if asked that the purple stria and veins the patients get is due to CORTISOL'S effect on the collagen in blood vessels. I wish I could make this easier and simpler, but it is a complex MOA! Finally, the hirsute features come from the weak androgens and the HTN comes from mineralocorticoid production and Na reaborption.

1111

Q)When you see such a patient previously discussed with Cushing-like symptoms, you need to figure out the source of the excess cortisol. What hormone is MOST important to measure here?

A) Either the just discussed 24 hr. serum cortisol test OR ACTH levels, a logical first step involves determining if the syndrome is ACTH-dependent or ACTH-independent. A plasma ACTH, measured by an immunoradiometric assay of less than 5 pg/mL, is suggestive of a primary ADRENAL tumor (NOT pituitary).. An ACTH level higher than 10-20 pg/mL is consistent with ACTH-dependent Cushing syndrome. As we discussed, a high dose dexamethasone test that SUPPRESSES cortisol serum levels suggests a....PITUITARY source of ACTH!! (Think about that). If not, it may be from the adrenals or an ECTOPIC source (like lung cancer).

1112

450

Q) While on the subject, what is the MOST common cause of the Cushing syndrome?

) EXOGENOUS excessive steroid use.

1113

Q) True or False: Does a loss or inactivation of an anti-tumor gene result in Burkitt lymphoma?

A) False. However, a loss or inactivation of an anti-tumor gene results in Retinoblastoma as an example. Memorize the main ones...Tumor suppressor vs. Oncogenic!

1114

Q) You have a pregnant pt. on 30 weeks gestation in active labor. You note she has diabetes type 1 for PMH. You need to give betamethasone and mag. sulfate. What else must you give here??

) The betamethasone is a CORTICOSTEROID and will lead to raised glucose in the diabetic pt. You must give INSULIN

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1115

Q) You are staring at a sympathomimetic pill. What is the difference between an amphetamine (like methylphenidate), cocaine, and MAO inhibitors on mech of action?

A) KNOW that stuff like methylphenidate and TYRAMINE revves up the release of stored catecholamines (thus they are INDIRECT). Cocaine (and imipramine) work by blocking the REUPTAKE. MAO inhabs. like tranylcypramine and phenelzine blocks the metabolism of catecholamines and thus increases stores.

1116

T or F: stimulation of alpha 1 receptors stimulates glycogenolysis in

451

the liver.

A) True, via the IP<sub>3</sub>, DAG, Ca<sup>2+</sup> path.

1117

Q) stimulation of B<sub>1</sub> receptors in the heart revves up heart rate and force...what does it do in the kidney?

A) In the kidney juxtaglomerular cells, renin is RELEASED

1118

Q) You are holding clonidine, (alpha 2 agonist) in your hand. It is used to lower BP, but what does it do,  
on the pancreas?  
on adipose cells?  
on platlets?

A1) INHIBITS insulin release

A2) INHIBITS lipolysis

A3) STIMULATES platlet aggregation

1119

1120

REMEMBER, alpha 2 stim. CONTRACTS smooth muscle and beta 2 stim. RELAXES smooth muscle. Don't forget this often asked by attendings question!!! (That is why some asthma meds are used for OB/GYN patients to stop contractions!!) See how all this fits together??

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1121

1122

Q) Think...ALWAYS watch the heart rate and blood pressure of drugs' side effects. What will PHENYLEPHRINE do to Heart rate? What will

452

Terbutaline do to heart rate?

A) REFLEXIVELY, very important, phenylephrine (alpha 1) will cause INCREASE BP and thus give REFLEX bradycardia!!!! Terbutaline (Beta 2) will reduce BP and thus give REFLEXIVE tachycardia! YOU MUST NOT FORGET

1123

Q) That said, which one (epinephrine, isoproterenol, or norepinephrine) will cause a REFLEX BRADYCARDIA? Think HARD about MOA, then you will recall better!

A) NORepinephrine! LISTEN carefully, by INCREASING blood pressure (you recall right?), you start up the vagal outflow and trigger the BARORECEPTOR REFLEX, and thus bradycardia! via alpha 1 agonist properties

1124

Q) THEN, which one (epinephrine, isoproterenol, or norepinephrine) will cause a TACHYCARDIA? Think first!

A) BOTH isoproterenol and epinephrine WILL via Beta agonism at the receptor!

1125

Q) OK, then, which one(s), epinephrine, isoproterenol, or norepinephrine...will have INCREASED systolic but DECREASED diastolic pressure?

A) EPInephrine.

1126

Q) Although epinephrine is dose dependent...tell me about isoproterenol. What does it do to blood pressure?

A) surprise! It lowers BOTH systolic and diastolic BP thus INCREASing heart rate!!!

1127

REVIEW it: Epinephrine RAISES the heart rate while NORepinephrine LOWERS the heart rate!

1128

Case: A 27 year old female coming into your office suffering from 2nd amenorrhea. Her last menstrual period was 6 months ago. There is associated milky nipple discharge. She has no PMH and is on no meds. Urine HCG is NEGATIVE. TSH is normal. Prolactin is high. What is the disease and give us one drug to treat.

A) Hyperprolactinoma .... give bromocriptine.

1129

A 30 year old woman with signs and symptoms of megaloblastic anemia from B12 def. She is also hypothyroid. Does she have:

--type A gastritis?

--type B gastritis?

After you pick, what is the cause of each of the gastritis?

A) answer is Type A, which is immunologically mediated, thus her thyroid issues and her antibodies to the PARIETAL cells (which secrete the intrinsic factor). TYPE B is assoc. with chronic NSAID use and Helicobacter pylori

1130

Case: Alrighty then...you have a 6 month old baby boy come to your office with oliguria of only .1 ml/kg/hr (no pee!) PE shows edema. BP is 100/50. HR is 140/min. BUN is 40, creatinine is 1.5. UA has spec. gravity of 1.010 and 3+ protein and granular casts in HPF. Fractional excretion of Na<sup>+</sup> is 3%. What disease does he have? How did you diagnose to your attending for SURE? Did you do a CT of the abdomen and pelvis OR a renal Ultrasound?

A) Baby has ATN, acute tubular necrosis. Renal ultrasound is what you did to confirm, because CT of the abdomen and pelvis requires contrast dye which can further injury the kidneys. Besides, the ultrasound by bedside is so easy

1131

454

Case: You see a 65 year old gentleman referred for neurologic consult. He has rapid progressive acute memory loss and his limbs have taken on a "jerking" motion. Neuro studies show cerebral atrophy and diffuse vacuolar changes of the gray matter with reactive astrocytosis. Does he likely have Alzheimer's? Glioma? Creutzfeldt-Jakob disease? or Pseudotumor cerebri?

A) Ans. is Creutzfeldt Jakob. This is more RAPID in course than Alzheimer's and you get those muscular jerking movements. It is caused by the mutation of the gene coding for prions. RECALL that GLIOMA is simply any brain cancer of glial origins, including astrocytoma and oligodendroglioma. You will see space occupying lesions. Finally Pseudotumor cerebri will present with HEADACHES from intracranial PRESSURE seen often in obese women

1132

Case: You have a 2 pack a day smoker who is 50 y.o. who comes to you with worsening COPD. On PE, there is rhonci in the left posterior lung. Labs shows an X-ray with a irregularly shaped lobar mass. Sadly, this is lung cancer. Which one, (increasing hoarseness in his voice OR bloody sputum that he regularly coughs up) is WORSE since it cannot be cured by surgery?

) Ans is HOARSENESS of the voice because it means the recurrent laryngeal nerve is lesioned out and metastasis has begun. The patient likely has a very poor prognosis...tell your patients to STOP SMOKING

1133

Case; You see your friend's mother in the clinic because she is 55 and fearful of osteoporosis. Which medicine do you prefer, (Calcium w/Vit D tablets OR Estrogen replacement therapy)? Or could it be something else that is preferred?

A) Give Estrogens. It is the mainstay of treatment. It also helps her

455

HDL too. Some like Fosamax, a bisphosphonate..but it can cause esophagitis.

!

1134

Case: You are examining a baby and you remove the diaper. You visualize erythematous, scaly red patches over the groin and buttocks. There are NO systemic symptoms. You tell the attending you think it is seborrheic dermatitis and she then slaps you silly. Where is seborrheic dermatitis usu. found?

A) Silly you, the pt. baby has a DIAPER RASH/Contact dermatitis. Seborrheic dermatitis is a red, scaly, itchy rash most commonly seen on the scalp, sides of the nose, eyebrows, eyelids, skin behind the ears, and middle of the chest

1135

Case: Another smoker...so depressing. He GRADUALLY is getting worsening dysphagia that first blocked him eating sausages, then later water too! He has massive weight loss. Will you choose a manometry OR barium swallow to confirm the location of the cancer?

A) You will choose a barium swallow for the likely SQUAMOUS CELL CANCER. Manometry is for motility disorders

1136

You are an ER doctor and someone in a horrible motorcycle accident is rushed in. Which is a BETTER measure of acute hemorrhage...(Low hemoglobin/hematocrit OR low blood pressure)?

A) Ans is low blood pressure. Sometimes the hematocrit takes hours to adjust because of fluid shifts

1137

Case: Two med students are known to study for finals and take massive doses of vit A, vit C, Vit E, Vit B-3, and Methylphenidate to "Pump" their brains up. One shows up in your clinic with sudden onset deathly painful right sided flank pain with hematuria. The other

456

roommate comes in with headaches, elevated LFTs and pruritis. Which substance caused what?

A) The one with flank pain got a kidney STONE from vit C toxicity. Effects may be renal colic (ie, nephrolithiasis), diarrhea, rebound scurvy in infants born to women taking high doses, hemolysis if G-6-PD deficiency is present, possible dental decalcification, and increased estrogen levels. The one with the headache and liver stuff had toxicity from vit B-3.

1138

Your clinic door opens and a retired circus clown walks in with claudication (limping with walking). He is finding it harder and more painful to walk. Due to cardiac reasons, he is taking diltiazem, hydrochlorothiazide, and propranolol. Which med should you discontinue for the leg pain?

A) Propranolol. REMEMBER the B2 receptor blockage will cause peripheral vasoconstriction of vessels. Stopping this will stop the leg pain.

1139

Case: Your clinic door swings open and sadly there is a child with mental retardation and difficulty walking. He also has telangiectasias on his face and recurrent lung infections. Which Immunoglobulins are missing??? What is the prognosis?

A) The child has ataxia-telangiectasia. Mainly, you will lack IgA and IgE levels. The disease is progressive, and half will die young from lung infections and the other half will die of lymphomas/leukemias

1140

Case: A twin pair with asthma and hypertension walks into your clinic. An inexperienced intern doctor gave one of them propranolol and the other enalapril for the hypertension. What are they likely to be

457

showing symptomwise?

A) Propanolol will cause wheezing from pulmonary bronchconstriction and enalapril will cause a cough

1141

Case: Another depressing case of a two pack a day smoker, 38, who has lung cancer. However, his labs are also showing signs of hypercalcemia at 14 mg/dL. What is causing this? What do you do? Can you give hydrochlorothiazide?

A) His lung tumor has a parathyroid related component, raising his Ca+. You need to give NOT hydrochlorothiazide, which will make him worse, but you should give FUROSEMIDE (LASIX) and IV fluids to lower his Ca+.

1142

Case: A 14 month old boy comes to your office below the 10% for height and weight. He has violent bilious vomiting and abdominal distention. PMH shows that he suffered a lot of constipation and did not pass meconium in the first 2 days. You push your finger into his anus, and stool rushes out. Is this (meconium ileus, duodenal atresia, Hirschsprung disease, or pyloric stenosis?)

A) It is Hirschsprung disease. The bilious and feculent vomiting are classic signs

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1143

Q) For hy c 1142, why wasn't it duodenal atresia?

A) In duodenal atresia you see a double bubble sign with small bowel

458

obstruction, and up to 50% of the cases are associated with Down syndrome.

1144

Case: AHH...a BIGGIE. A 31 year old man causally meanders into your clinic with some kidney disorder his "other" doctor looked at. He is has no rash, arthralgia, hematuria. Labs show BP at 100/70. He has edema, and a large palpable liver. Ulcers are seen on lower legs. Labs are BUN=21, Creatinine=1.7. U/A shows significant proteinuria. There is elevated cholesterol. Ultrasound shows enlarged kidneys. Tests you ordered show HIV neg., normal sugars, subepithelial granular immune complex deposits on ALL glomeruli. What is this dx?

Can it be Poststreptococcal glomerulonephritis? Why not?

A) NO! There is no hypercellular glomeruli, inflammatory cells, prodrome of strep throat, blood in the urine, and HTN

1145

Can it be IgA nephropathy/Berger's? Why not?

(A 31 year old man causally meanders into your clinic with some kidney disorder his "other" doctor looked at. He is has no rash, arthralgia, hematuria. Labs show BP at 100/70. He has edema, and a large palpable liver. Ulcers are seen on lower legs. Labs are BUN=21, Creatinine=1.7. U/A shows significant proteinuria. There is elevated cholesterol. Ultrasound shows enlarged kidneys. Tests you ordered show HIV neg., normal sugars, subepithelial granular immune complex deposits on ALL glomeruli.)

A) NO WAY! IgA nephropathy is the most common form of glomerulonephritis, and often is assoc. with hematuria. The presentation has a WIDE range from mild to very severe ESRD. Plus, think of the mech. IgA problems often are assoc. with respiratory and GI infections and will be exacerbated during and before the nephropathy. While IgA antibodies cannot activate complement through the classic pathway, studies have shown that complement can

459

be activated by the alternate pathway! Remember the [FA](#) concept ("G.M. makes classic cars"...IgG and IgM are classic pathway)

1146

Can it be Minimal Change Disease?

(A 31 year old man causally meanders into your clinic with some kidney disorder his "other" doctor looked at. He is has no rash, arthralgia, hematuria. Labs show BP at 100/70. He has edema, and a large palpable liver. Ulcers are seen on lower legs. Labs are BUN=21, Creatinine=1.7. U/A shows significant proteinuria. There is elevated cholesterol. Ultrasound shows enlarged kidneys. Tests you ordered show HIV neg., normal sugars, subepithelial granular immune complex deposits on ALL glomeruli. What is this dx?)

A) No, think here M.C.D. occurs in kids, by light/immunofluorescence, glomeruli look OK. And there is foot process fusion visible only with an electron microscope. Very treatable with steroids

1147

Can it be Goodpasture's?

A 31 year old man causally meanders into your clinic with some kidney disorder his "other" doctor looked at. He is has no rash, arthralgia, hematuria. Labs show BP at 100/70. He has edema, and a large palpable liver. Ulcers are seen on lower legs. Labs are BUN=21, Creatinine=1.7. U/A shows significant proteinuria. There is elevated cholesterol. Ultrasound shows enlarged kidneys. Tests you ordered show HIV neg., normal sugars, subepithelial granular immune complex deposits on ALL glomeruli. What is this dx?

A) No, in Goodpasture's, you will likely see pulmonary hemoptysis and a LINEAR pattern on immunofluorescence microscopy staining (not granular). The MOA is due to anti-GBM antibodies.

1148

Can it be Wegener's? If not, what specific test is often positive in Wegener's?

A 31 year old man causally meanders into your clinic with some kidney disorder his "other" doctor looked at. He is has no rash, arthralgia, hematuria. Labs show BP at 100/70. He has edema, and a large palpable liver. Ulcers are seen on lower legs. Labs are BUN=21, Creatinine=1.7. U/A shows significant proteinuria. There is elevated cholesterol. Ultrasound shows enlarged kidneys. Tests you ordered show HIV neg., normal sugars, subepithelial granular immune complex deposits on ALL glomeruli. What is this dx?

A) No, in Wegener's again, you will see pulmonary stuff. But you are likely to see URI symptoms. And likely a positive C-ANCA test.

1149

Can it be SLE lupus nephropathy?

A 31 year old man causally meanders into your clinic with some kidney disorder his "other" doctor looked at. He is has no rash, arthralgia, hematuria. Labs show BP at 100/70. He has edema, and a large palpable liver. Ulcers are seen on lower legs. Labs are BUN=21, Creatinine=1.7. U/A shows significant proteinuria. There is elevated cholesterol. Ultrasound shows enlarged kidneys. Tests you ordered show HIV neg., normal sugars, subepithelial granular immune complex deposits on ALL glomeruli. What is this dx?

A) NOOOO!! Unlikely b/c you are not given any other signs of lupus including fatigue, fever, rash, arthritis, and serositis or central nervous system (CNS) disease. HOWEVER, KNOW THAT SLE lupus nephritis IS SOMETIMES SEEN WITH PATIENTS WITH MEMBRANOUS NEPHROPATHY. There is no HTN here. HLA-DR2 and HLA-DR3 are associated with SLE, and there are again no such clues given in labs.

461

SLE nephritis disease activity can be evaluated with anti-dsDNA, complement determinations (C3, C4, and CH50), and erythrocyte sedimentation rate (ESR) or C-reactive protein (CRP), and again, nothing mentioned here.

1150

OK, now that you have ruled out most of the DIFFERENTIALS, what is this very common disease you will see over and over?

A 31 year old man causally meanders into your clinic with some kidney disorder his "other" doctor looked at. He has no rash, arthralgia, hematuria. Labs show BP at 100/70. He has edema, and a large palpable liver. Ulcers are seen on lower legs. Labs are BUN=21, Creatinine=1.7. U/A shows significant proteinuria. There is elevated cholesterol. Ultrasound shows enlarged kidneys. Tests you ordered show HIV neg., normal sugars, subepithelial granular immune complex deposits on ALL glomeruli. What is this dx?

A) This is Membranous Nephropathy, the most common cause of nephrosis. Note, in the early stages, a key finding is the LACK of HTN in many cases. He is a guy, and there is NO signs of SLE nephritis

1151-1200

1151.

T/F: Is Vit E a type of quinone derivative with a structure similar to coenzyme Q which serves as an ANTIoxidant by working together with reduced NADPH to keep glutathione in the reduced state, which is also stored in the liver?

Ans is FALSE!: You have to know some details, not just the trigger words "antioxidant". Vit E blocks cholesterol plaques on the arterial

462

walls by blocking the oxidation of unsaturated fatty acids in LDLs. You need to know oxidizing agents induce peroxidation of polyunsaturated fatty acids in membranes of the LDLs. Then, the macrophages engulf the oxidized LDLs because they have these scavenger receptors. Some weirdo "macrophages" keep eating LDLs and become fat and turn into foam cells. Vit E blocks all this, and thus the formation of plaques.

1152.

Oh, before I forget, why is it NOT Focal Segmental Glomerulosclerosis? Why not? Recall the case: . A 31 year old man causally meanders into your clinic with some kidney disorder his "other" doctor looked at. He has no rash, arthralgia, hematuria. Labs show BP at 100/70. He has edema, and a large palpable liver. Ulcers are seen on lower legs. Labs are BUN=21, Creatinine=1.7. U/A shows significant proteinuria. There is elevated cholesterol. Ultrasound shows enlarged kidneys. Tests you ordered show HIV neg., normal sugars, subepithelial granular immune complex deposits on ALL glomeruli.

A) Focal Segmental Glomerulonephritis, this involves only SOME (hence the name "focal") of the glomeruli. And You see hyalinization sclerosis and note that this FSG disease often involves HIV patients. This pt.'s HIV test was negative.

1153.

Q) What are some of the main causes of the discussed case MEMBRANOUS NEPHROPATHY? (part of the NEPHROTIC category, not the NEPHRITIC)

Here is a LONG LIST: (Just glance through it once, I just wanted the back of your minds to be mildly familiar.

- o Ankylosing spondylitis
- o Dermatomyositis

463

- o Graves disease
- o Hashimoto disease
- o Mixed connective-tissue disease
- o Rheumatoid arthritis
- o Sjögren syndrome
- o Systemic sclerosis
- Infectious diseases
- o Enterococcal endocarditis
- o Filariasis
- o Hepatitis B: This occurs in children in endemic areas.
- o Hepatitis C
- o Hydatid cyst
- o Leprosy
- o Malaria
- o Schistosomiasis
- o Syphilis
- Malignancy
- o Carcinoma (solid organ)
- o Leukemia
- o Lymphoma
- o Melanoma
- Drugs
- o Captopril
- o Gold
- o Lithium
- o Mercury-containing compounds
- o Penicillamine
- o Probenecid

1154.

Q) What are some of the meds to treat the most common membranous nephropathy? You have to know this.

Meds to Treat and some Mech of Actions:

Furosemide (Lasix) You know the mech..

Simvastatin (Zocor) – This HMG Coa reductase inhibitor decreases intracellular cholesterol pools and increases LDL receptors, which causes a decrease in LDL-C. Recall that often in nephrotic syndromes the serum lipids run higher!

Prednisone -- Exerts an anti-inflammatory effect via the inhibition of inflammatory mediator gene transcription.

Cyclophosphamide (Cytoxan, Neosar) -- Used for remission of nephrotic syndrome. Interferes with normal function of DNA by alkylation and cross-linking the strands of DNA and by possible protein modification.

Chlorambucil (Leukeran) -- For remission of proteinuria; given with prednisone (0.5 mg/kg/d) every other month. Steroids are given as 1 g methylprednisolone IV for 3 d. Interferes with DNA replication and RNA transcription by alkylation and cross-linking the strands of DNA.

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Cyclosporine A (Sandimmune) -- Inhibits production and release of IL-2, leading to inhibition of IL-2-mediated activation of T lymphocytes. You can give an ACE inhibitor like Enalapril also known as Vasotec OR Lisinopril (Zestril, Prinivil) -- Inhibition of ACE leads to decreased plasma angiotensin II, which, in turn, leads to decreased vasopressor activity and decreased aldosterone secretion. ACE inhibitors minimize secondary intraglomerular hypertension and hypertrophy, leading to decreased proteinuria in idiopathic membranous nephropathy.

Ibuprofen (Motrin, Ibuprin) -- Exerts its effects by inhibiting both constitutive and inducible isoforms of cyclooxygenase, which produces a mild-to-moderate anti-inflammatory and analgesic effect. NSAIDs decrease intraglomerular pressure and decrease proteinuria.

1155.

Which drug will likely cause neuroleptic malignant syndrome (Fever, etc.) in a psychotic patient? Haloperidol or Risperidol?

A) High potent ones like Haloperidol. Which receptors do Haloperidol act on?

1156.

What closes a PDA (patent ductus arteriosus in a cyanotic kid L-R shunt)?

A) INDOMETHACIN. But what keeps it OPEN?

1157.

Q) What keeps a PDA OPEN?

A) A Patent Ductus Arteriosus is kept OPEN with PGE. You need to leave it open if the kid has a Tetralogy of Fallot or Transposition of Great Vessels.

1158.

Q) Quick, the aortic valve OPENS, what heart sound is involved?

A) END of the FIRST heart sound!

1159.

Q) Quick! You hear the mitral valve CLOSING. What heart sound and when ?

A) BEGINNING of the FIRST heart sound!

1160.

Q) Quick! You hear the aortic valve now CLOSING? What heart sound?

A) START of the SECOND heart sound. S2

1161.

Q) Wow, you see that the VENTRICULAR volume is LOWEST... What part of the heart sounds is involved and what valve is opening/closing?

A) Occurs during the SECOND heart sound, recall here that aortic valve has just closed, meaning SYSTOLE or the squeezing of the ventricles and thus its volume is lowest here.

1162.

Will prostaglandins dilate the AFFERENT or EFFERENT renal arteriole?

A) AFFERENT. ANG II constricts the EFFERENTS! BOTH PGE and ANG II raises GFR! Think about that for a moment.

1163.

A man walks in post MI. There is cardiac tamponade....ventricular wall rupture....interventricular septum. Is this (about 7-10 days post MI OR 3-7 days post MI)?

A) 3-7 days post MI (very important)

1164.

Which one, CK-MB or Troponin I is the test of choice for MI? when do

467

levels peak?

A) Troponins are now considered the criterion standard in defining and diagnosing MI, according to the most recent American College of Cardiology. Troponin I serum levels increase within 3-12 hours from the onset of chest pain, peak at 24-48 hours, and return to baseline over 5-14 days.

1165.

A 73 year old gentleman goes for a ER visit follow up. He has a 2 yr PMH of CHF due to HTN. Yesterday he sought care at the ER for SOB and chest palpitations. He had a momentary atrial fib. His meds are thiazides and an ACE inhib. His PE is unremarkable. BP is 135/80. He has a grade 2 holosystolic murmur heard at the apex. JVP is 10 cm at 30 degrees. He has DIMINISHED LV function. What do you need to do? (Pick either Beta Blockers OR Digoxin)

Give digoxin to address the atrial rate control due to reduced LV function. Beta Blockers would be OK if not for the LV trouble. AF is associated with a 1.5- to 1.9-fold higher risk of death, which may be due to thromboembolic stroke. While patients can be asymptomatic, many experience a wide variety of symptoms including palpitations, dyspnea, fatigue, dizziness, angina, and congestive heart failure (CHF). In addition, the arrhythmia can be associated with hemodynamic dysfunction, a tachycardia-induced cardiomyopathy, and systemic embolism. Digoxin is indicated in patients with reduced LV function.

1166.

Q) For the previous patient with Atrial Fib., what is the mech of Action of digoxin in controlling rate?

A) Digoxin has positive inotropic and negative chronotropic (rate)

468

activity by working on the Na<sup>+</sup> channels which lowers intracellular Ca<sup>2+</sup> conc.

1167.

Q) For the patient with Atrial Fib, you need also to address thrombosis. What is the difference in mech of action between Heparin and Warfarin for thrombus prevention? VERY IMPORTANT.

A) Heparin (depending on whether it is low mol.weight or not), works to bind Factor X and/or increase APTT.) It also inactivates thrombin.

Warfarin, however, binds vit. K and lowers Factors 2,7,9,10 and increases PT.

Note that warfarin and not heparin can be given orally and warfarin is digested in the liver and heparin is digested by the kidneys.

1168.

A woman with striae and obesity and HTN and a POSITIVE dexamethasone suppression test comes in asking you if she will bruise easily too because she wants to take karate lessons. Yes or No?

A) Yes, people with Cushing's will bruise easily.

1169.

Case: There is a man, 40 years old, named George Washington coming in with palpitations, fluttering, fainting spells, weakness with activity, S.O.B. Your PE reveals a I/IV crescendo blowing diastolic murmur. ECG and Echo show mild LV enlargement. But he is not showing really bad symptoms like CHF worsening, post MI, coronary artery disease, etc. He reports a respiratory infection one year prior... what could this dx be and what is the bug? Treatment?

A) He is showing signs/symptoms of AORTIC INSUFFICIENCY from previous STREP infection. After some time, rheumatic heart disease can develop. You could do many things, including giving diuretics and maybe even surgically replacing the aortic valve.

1170.

A neonate named John Adams presents with acholic stools, jaundice, and hepatomegaly. What enzyme is often lacking in the liver?

A) Alpha-1-antitrypsin levels are often LOWERED.

1171.

A patient named Thomas Jefferson comes in with bipolar disorder. His treatment with Lithium may affect the thyroid...is this true or false?

A) True, thus you must monitor TSH serum levels.

1172.

A gentleman named James Madison comes into your ER because he was not restrained while sitting in the passenger seat in a head on collision. His knees look swollen after hitting the dashboard. What is more pressing, examining the hips OR the lumbar spine?

A) Look at the hips. A blow like this can cause the femurs to slip out of their sockets, an orthopedic emergency..

1173.

Case: A pt. named James Monroe comes into your clinic with mid abdominal pain, joint pain, rashes on his thighs and feet and buttocks, fever, and guaiac positive stools, and dehydration (mild). What is this, and what caused this?

A) This is classic for Henoch Schonlein Purpura. It often follows a strep

470

or URI infection and is thought to be an immune complex disease.

1174.

Pt, name John Adams, got a tick bite dx. while camping called Rocky Mountain spotted fever. What does this present like clinically?

A) RMSF presents with high fever, rash on the hands and feets which spreads centrally. They also have photophobia.

1175.

Q) Quick, I forgot to ask, what is the bug with Rock Mountain Spotted Fever? and a dangerous complication?

A) Rickettsia rickettsii are from the Ixodes tick and can cause vasculitis from endothelial seeding of the buggies.

1176.

A 24 year old programmer pt. named Andrew Jackson comes in complaining of alternating bouts of constipation and diarrhea. ALL PE findings and labs are normal. What is MOST likely dx?

A) Irritable Bowel Syndrome.

1177.

A pt. with numbers in his last name who goes by Martin Van Buren 1837-41 comes in asking about oral contraceptive pills. She is a heavy smoker. Is this bad? What other risk factors do you have to relay? (Oh, she was named a male name after her great great grandfather)

A) Contraindications for OCPs are hepatic disease, history of DVTs, thrombosis, smoking, migraine headaches, breast or endometrial cancer, and of course, known pregnancy.

471

1178.

Case: You suspect Hepatitis C in a patient. But your attending William Henry Harrison 1841 thinks it is autoimmune hepatitis. How can you tell who is right?

A) If she is HCV positive by HCV recombinant immunoblot assay (RIBA), and not the less specific HCV enzyme immunoassay, you can be more certain. Plus, autoimmune hepatitis presents with hypergammaglobulinemia, ANA autoantibodies, portal inflammation.

1179.

Case: A female, again named after her great great grandfather named John Tyler 1841-45, comes in with diagnosed PBC or Primary Biliary Cirrhosis. What labs are elevated? What is the most common presenting symptoms? Mech of Action?

A) You will see elevated ALT, AST, ALP (alkaline phos), GGTP, and IgM. The most common presenting symptoms are fatigue, pruritus, and xanthomas. Look for AMAs or anti mitochondrial antibodies. This is an autoimmune disease destroying the bile ducts.

1180.

A gentleman named James Polk was involved in a car accident and is in ICU. Slowly his condition deteriorated and his PHOSPHATE started to rise, eventually reaching 6.5 mg/dL (high). Which organ likely failed and why?

A) The kidneys. Advanced renal insufficiency is commonly the cause of hyperphosphatemia which inhibited phosphate excretion. You can also see elevated phosphate in acidosis DKA as the phosphate shifts to the extracellular space to balance charge. Rhabdomyolysis, Infections, and cancers can also be implicated.

472

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1181.

Zachary 1849-50 Taylor, a patient of yours, comes to the hospital for PE and chest CT. He has lung disease secondary to drug therapy for heart disease. PFTs show an advanced restrictive lung disease. What do you hear on lung exam? (pick either Fine inspiratory crackles at the bases OR Hyperresonance OR Diffuse expiratory wheezes OR Dullness to base percussion.)

A) Fine inspiratory crackles. The restrictive interstitial lung diseases often decrease lung compliance and recoil, so you will hear late inspiratory crackles like you may for CHF patients. DIFFUSE expiratory WHEEZES are heard in asthma or chronic bronchitis. DULLNESS to percussion at the bases are heard with pleural effusions.

1182.

Fillmore, Millard: A 65 year old gentleman who presents to you after an MI involving the inferior branches of the RIGHT coronary artery. What leads are changed? (pick either I, V5, V6 OR II, III, aVF)

A) II, III, aVF. I, V5, V6 is related to LEFT coronary circumflex artery infarct.

1183.

Pierce, Franklin 1853-57...A 50 year old patient of yours has KNOWN mitral valve prolapse. True or False: This dx. causes a MID-SYSTOLIC CLICK and is seen more often in patients with Ehlers-Danlos syndrome.

A) True, not only EDS syndrome pts., but other connective tissue disorder pts. have this commonly.

1184.

473

Buchanan, James med ID #1857-61. This pt of yours has rheumatic heart disease after GRP A B-hemolytic strep. This is a PAN-carditis...what are the FIVE major criteria for diagnosis?

A) Carditis, Polyarthritits, Chorea, Erythema marginatum (migratory rashes), Subcutaneous nodules. Recall, look for Ashoff and Anitschkow bodies on histo.

1185.

Study the LOG-DOSE curves in biochem and what agonists with similar efficacy but different potency look like.

1186.

KNOW you have a patient, Lincoln, Abraham Med ID #1861-65, who presents with Ehlers-Danlos VI syn. What extracellular matrix molecule is affected and how? Is the ans. laminin, fibronectin? Describe clinical findings. What enzyme is missing?

A) Molecules lesioned are Type I and Type III collagen, you'll see hyperextensible skin, joints, large vessel fragility, retinal detachment...skin bone vessel tendon stuff. This results from reduced lysyl hydroxylase activity which are critical to crosslinking collagen.

.....

Is it lasyl hydroxylase or oxidase? so what is affected, laminin or fibronectin?

.....

Thanks for asking. It is lysyl HYDROXLASE. And NEITHER laminin or fibronectin or affected. It is collagen. See ya.

1187.

Speaking of fibronectin, what is it?

A) Fibronectin is a macromolecule secreted by endothelial cells and fibroblasts. Fibronectins have multiple domains that confer the ability to interact with many extracellular substances such as collagen, fibrin and heparin and also with specific membrane receptors on responsive cells. Interaction of a cells fibronectin receptors (members of the integrin family) with fibronectin adsorbed to a surface results in adhesion and spreading of the cell.

1188.

Now, speaking of laminin, what is THAT?

A) It is THE most abundant glycoprotein in all basement membranes. Link proteins of basal lamina, consist of an A chain (400 kD) and two B chains (200 kD). Each subunit contains at least 12 repeats of the EGF like domain. Laminin induces adhesion and spreading of many cell types and promotes the outgrowth of neurites in culture.

1189.

Speaking of proteoglycans, what are they and what do they do?

A) These glycoproteins regulate connective tissue structure and permeability and have very high polysaccharide content.

1190.

Do you know how a papillary tumor, serous intermediate type, of the ovary looks like on a histo slide? Will it have a high likelihood of metastasis IF it is cystic and NOT of the adenocarcinoma type? Will it obstruct another organ?

A) These borderline tumors are usually benign, but they may obstruct the intestines as they slowly grow. Recall this is NOT a serous adenocarcinoma which is often bilateral and malignant, it is a serous

475

INTERMEDIATE cyst. You often will have to remove them.

1191.

Is the so common dermoid cyst a SEX CORD cell tumor?

A) NO! It is a Germ cell tumor which also includes yolk sac carcinomas and choriocarcinomas.

1192.

Speaking of Germ cell tumors, if you have a MALE patient, what is the most common testicular tumor? How do they look like on a histo slide?

A) Seminomas. They look like clear cells lumped into lobules encircled by lymphocytes.

1193.

Q) So, then, are Nonseminomatous germ cell tumors better to have than Seminomatous? Is there a marker?

A) NO! NSGCTs are much worse in malignancy. You will see mets to the nodes, liver, and lungs early on. They are RADIORESISTANT. AFP and hCG are detectable in the serum.

1194.

Listen, there is a child with blood in the stool, thrombocytopenic hemorrhages, eczema, recurrent sinopulmonary infections. What is the disease and common cause of early death?

A) This distinct triad is WAS or Wiskott Aldrich Syndrome. Think "All HE ever wanted W.A.S. H.E.R." initials W.A.S. for Wiskott Aldrich Syndrome, Initials H.E.R. for first letters in Hemorrhages, Eczema, RecurrentSinopulmonaryInfections. And it is a HE because WAS is an X

476

linked Recessive Disease! Recall, BOTH T and B cell function is lesioned.

1195.

Case: You have a 22 year old 1st year med student who is increasingly weak and lethargic for 4 months. She has no sig. PMH and she is on no meds. PE is: BP low at 80/55. Tachycardic. LABs show normal Na<sup>+</sup>, LOW potassium, bicarb is 45. What is going ON? (Pick chronic diarrhea, Cushing's, 1' aldosteronism, eating vomit, or bulimia).

A) Ans is bulimea. Her vomiting in her closet b/c she hates studying so much gives her the UNIQUE combo of alkalosis AND hypokalemia. The vomiting makes her ALKALOTIC. The body adjusts by grabbing protons from inside the cells to balance the charge, and thus pushing in K<sup>+</sup> INTO the cells through the antiport transporter H-K. This makes her serum K<sup>+</sup> LOW, causing her hypokalemia! The signs of HYPOTENSION is from the discharge of vomiting iinto her closet or windowsill plant.

1196.

Speaking of hyc 1195, if the med student was having diarrhea, would she be hypokalemic? We know that she is ACIDOTIC, but what about her K<sup>+</sup>?

A) Yes, she will diarrhea out her K<sup>+</sup> TOO, as well as her HCO<sub>3</sub><sup>-</sup>!!

1197.

For the case of hyc 1195, would the med student have been Cushing's?

A) NO! No signs of obesity, NO HTN involved, but Cushing's still has hypokalemia and alkalosis.

1198.

477

For the med student in hyc 1195, why couldn't she have primary aldosteronism?

A) The Na<sup>+</sup> reabsorption would cause release of potassium and H<sup>+</sup>, producing hypokalemia and alkalosis, and HYPERTension, not hypotension like the case we saw in hyc 1195.

1199.

Case: After a 24 year old 2nd year med student is facing her finals, she is SO stressed she develops a GRAND MAL SEIZURE. Next, she notes right shoulder pain and gets an anteroposterior (AP) x-ray films. Films are negative and the pain continues. She presents in tears to you, holding her arm close to her chest, her hand resting on the anterior chest wall. Does she likely have a (Posterior dislocation of the Shoulder OR an ANTERIOR dislocation of the shoulder OR an Acromioclavicular sep OR a TORN teres major and minor muscle)?

Answer is POSTERIOR dislocation, the MOA is massive contraction of all muscles and a missed diagnosis on a SINGLE AP view. Know that acromioclavicular sep. would HAVE been OBVIOUS on X ray. The ANTERIOR dislocation is much more common, but the arms would have been held close to the body with the forearm and hand rotated OUTWARD like they were going to shake hands. Finally Torn muscles of the rotator cuff are not common with seizures.

1200.

Case: A man, 48 years old, is in your office. You are called by the attending to do a hematocrit test...it is only 25%. Reticulocytes are DOWN. No significant PE findings...What is the likely disease and cause?

A) Iron def. anemia...from GI bleeds. Women get it during some menstrual periods.

478

hyc 1201 You have a 45 year old woman with a history of ulcer with diarrhea and duodenal ulcer disease, and you suspect Zollinger Ellison syndrome. You give secretin IV to test for gastrinoma. Pick which one would support gastrinoma's existence? (inhibition of gastric secretion) OR (increased blood levels of gastrin) TRICKY

A) Increased blood levels of gastrin. Those gastrinomas are gastrin secreting tumors in the pancreas. Know that although secretin blocks antral release of gastrin, it stimulates gastrin release from tumors. Know also that SECRETIN inhibits gastric emptying, inhibits gastric secretion, and stimulates pancreatic bicarb secretion.

1202

Let's say I show you a table with the ratio of urinary concentration to plasma concentration of inulin U/P is decreasing! Pick which of the following is true if the GFR is constant? HARD question (Pick either Inulin clearance has decreased OR Urine flow rate has increased)

A) Urine flow rate is increasing. Recall that Inulin is freely filtered by neither reabsorbed or secreted. Thus, since ALL the inulin filtered will show in the urine, the amount of water in the urine WILL give the inulin concentration, so inulin U/P will DECREASE if urine flow rate increases! This is a hard but essential concept.

1203

Given this list below, which is used to INDUCE abortion??  
(Pick from: PGG<sub>2</sub>, PGE<sub>2</sub>, PGH<sub>2</sub>, PGI<sub>2</sub>)

479

A) PGE<sub>2</sub>, know that PGI<sub>2</sub> is a prostacyclin, a potent INHIBITOR of platelet aggregation.

1204

A 70 year old man cannot urinate today, but could in previous days. PE has BP of 180/100. Labs show creatinine of 5 and BUN of 120. U/A has specific gravity of 1. What med will you give? (pick either Doxazosin OR Benazepril)

A) Doxazosin... BPH is very common and tested. Like Prazosin, Doxazosin is an alpha 1 blocker and will also help his high BP!

1205

You see a pt with a headache and nuchal rigidity. Labs show a lumbar puncture with bloody CSF and elevated pressure, high protein, and low glucose. Does she have (subdural hemorrhage OR berry aneurysm OR hypertensive vascular lesions OR amyloid angiopathy)?

A) berry aneurysm.

1206

You have an HIV positive 35 year old female pt. with possible B-cell lymphoma. What is most likely in the pt? (pick Very high IgG OR very high IgM or Reed Sternberg cells OR +EBV titer OR HTLV-1 infection)

A) Likely positive for EBV infection. KNOW that high IgG is a type of macroglobulinemia, usually NOT seen in B-cell lymphomas. And KNOW that high IgM is seen in Waldenstrom macroglobulinemia. KNOW Bence Jones protein is LIGHT chains in urine.

1207

480

You have a 30 year old drug addict with gonorrhoea and fever and SOB. Pulse-ox shows hypoxia. Chest x-ray shows bilateral interstitial infiltrates. What finding is diagnostic? (Pick acid fast bugs in sputum OR positive methenamine silver stain)

A) positive methenamine silver stain. With his risk factors, he is susceptible to *Pneumocystis carinii*!, most common.

1208

Listen, 2nd messengers are basically one type of SIGNALING MECHANISMS for neurotransmitter/drug effects. There are five types, but two distinct ones are famous...1) TRANSMEMBRANE DIFFUSION to a cytosolic receptor. and 2) G-protein coupled receptors (the so called 2nd messengers).

Listen, with respect to G-proteins, one of the most common are the sympathomimetic drugs, which activate or inhibit adenylyl cyclase by a multistep process. When you activate the receptor, you activate the G protein and either INHIBITS or STIMULATES the cyclase.

REMEMBER, there are basically TWO THREE G-PROTEINS, a Q, an S, and an I. Thus, Gq, Gs, and Gi. (3 G-PROTEIN classes are in existence for the USMLE)

YOU HAVE TO KNOW THE FOLLOWING!!!!!!!!!!!!:

Gq----Gq, stimulatory, works through Phospholipase C and either boosts IP3/Ca<sup>2+</sup> AND DAG/Protein Kinase C. How do you remember??? Well recall that Gi stands for inhibitory (thus the "i") and the others, q and s are thus stimulatory...like our Gq. And then think that Phospholipase "C" is linked with the "C" in Calcium and the "C" for protein kinase C. (Thus, link in your mind a bunch of "C"s together with Gq. {I THINK OF A GQ MAN AS "C"OOL and is Stimulating to a woman} This is a famous cool men's magazine. Therefore, the ideas of

481

"C"s being cool and stimulatory related to the "G"q magazine forever solidifies the connection. AGAIN, connect forever, Gq is the magazine and it is C for Cool Stimulating magazine (Phospholipase C and Protein kinase C).

Gs----As we said, the Gs is "stimulatory" (Think the initials Gs stands for "G"reat "s"ex which is stimulatory...so equate the G"s" with "s" in stimulatory. Now, think and equate the Gs with cAMP and Protein kinase A. HOW? First off, cAMP is always linked to a Protein kinase. A weirdo linko is to think of a GSA, "G"reat "S"ex "A"lways. So forever, you will link Gs with A, thus Gs with Protein kinase A in a STIMULATORY way.

Gi----HERE, it is easy because the "i" is inhibitory, and thus pushes down the cAMP and Protein kinase A. To LINK, Now, think of GiA, or "G"reat "i"nhibitory "A"nus (Since most of us have "no access" to our inhibitory anal openings.) So always link Gi with a inhibitory to Protein kinase A (A for anus). Remember, i stands for inhibitory for "A"nus.

hyc 1209 (guest)

Tired of all this? It is so hard to keep straight, I think I need to vomit right about now.

NOW we have to touch on what the activation of each receptor actually DOES in your body.

Start with alpha 1. OK, this is elementary, most know this is a VASOCONSTRICTOR.

Alpha 2 then? Unknown to many, this BLOCKS insulin release and is used as an antihypertensive.

B1 then? HERE, repeat to yourself over and over....this B1 boosts up everything! Think "Be number 1"...Be 1...B number 1! Be the best and rev up everything, esp in the heart.

482

B2 then? Remember, the 2 links that you have 2 lungs. So, B2 DILATES the lungs, and ALSO release pancreatic insulin! How do you recall this? Think "Be 2 in su (yours) lin" Think a salacious thought that you wish to be "inside" sexually your friend's sister Lin. Thus, B2-- insulin.

M1 then? Stimulation here targets the CNS and stimulates it. Recall that it is Gq, as most 1s are (like alpha 1, Beta 1, etc.) And since Gq is a "Cool" magazine, again take the first letter of Cool, "C" and link that it works with phospholipase C.

M2 then? Recall that most "2s" are inhibitory. So this DECREASES and inhibits heart rate.

M3 then? Stimulation here via Gq boosts up exocrine gland secretions. (NOW, if you remembered what we just talked about, that Gq is COOL and is STIMULATING from pictures of handsome man (not that I would be influenced), then you will know that SECONARILY, it works via the "C"s, Phospholipase C, Ca 2+, and Protein kinase C. See, the guys in Gq magazine are "C"ute. The C keeps coming back with Gq, the Men's Magazine.

D1 then? Again, this is stimulatory but this time with Gs, so you now have the A motif. You have to link the Gs with "A"--Gs is linked with ATP, c"A"MP, Protein kinase "A" Just start again with Gs with yet another link to "A", like GsA, like "G"oing "S"lowly up the "Anus" GsA, as you do a hemocult test. Again, equate Gs with A, GsA, GsA. D1 is associated with Gs and NOT Gq because D stands for a "dope" which is not as cool as a person with a Gq looks on their face.

Is this helping?

(Step 1) Beta 1 and 2 both are Gs and inc adenylyl cyclase --> inc cAMP

483

(tommyk)My previous mnemonic was the company AMD is SECOND (2) to the company Intel Corp in power. Thus, AMD Inc. is "inhibitory" to Intel's dominance. Then link the company's initials A.M.D. to the word inhibitory and the number 2. Finally link the fact tha A2, M2, D2 (and you are correct that B2 is NOT inhibitory), are all inhibitory. Again, Alpha 2, Muscarinic 2, Dopamine 2, A2, M2, D2 are all inhibitory.

1210

You have a 60 year old man, PAINLESS swelling on his neck. PE is splenomegaly. Biopsy of the neck reveals a neoplasm with small cleaved cells that recapitulate the normal follicle of lymph node. Is this (L-myc, OR p53, OR bcl-2 OR ras)?

A) Bcl-2...This is a case of non Hodgkin lymphoma, follicular type. Bcl-2 stops apoptosis. In most of B-cell lymphomas (esp. follicular), the gene is OVER expressed which causes other mutations like the lymphoma.

1211

If you are asked which of the following demonstrates AGING at the CELLULAR level, which is it? (pick hemosiderin, lipofuscin, or melanin spots).

A) Lipofuscin. This brown stuff accumulates with aging and is made from the PEROXIDATION of lipids inside the cell.

1212

A woman, 50 years old, is jaundiced. LABS=high CONJUGATED

484

hyperbilirubinemia. Urine bilirubin levels are WAY UP. Urine urobilinogen are WAY below normal. What is the MOA of her jaundice? (is it Blockage of the common bile duct OR deficiency of glucuronyl transferase OR hemolytic anemia OR hepatocellular damage)

A) Blockage of the common bile duct. Recall, it is CONJUGATED ALREADY.

1213

You see a 40 year old man with a vomiting of green stuff 45 minutes after eating. He is scheduled for a barium to evaluate the upper portion of the GI. There is no pain, but he is NOT jaundiced. What is the mech of action? (is he have annular pancreas OR esophageal atresia, or gallstones, or Meckel's)

A) The answer is annular pancreas, where a ring of pancreatic tissue that forms around the duodenum causing partial or complete obstruction of the duodenum. A complete duodenal obstruction may be detected. An annular pancreas may put pressure on the duodenum, a make the pt vomit bile. KNOW that esophageal atresia is found usu. in neonates. Gallstones cause indigestion, pain and jaundice. A Meckel's is the persistence of a portion of the embryonic vitelline duct or yolk stalk and is usu. asymptomatic.

1214

A football player gets hit from the lateral side. The THREE structure to be affected are:

485

A) Think of mnemonic, "Mam, that hurt!" M,A,M,...  
M..edial collateral ligaments  
A..nterior cruciate  
M..edial meniscus

1215

I am showing you a volume-pressure diagram of the left ventricle during one cardiac cycle. Where is the exactly part where systole starts.

A) on that rhomboid looking figure, it is the right most, lower right point. LOOK IT UP PLEASE!

(1216)In an experiment you did, radiolabeled ATP is injected into a muscle and stimulated for 10 seconds. Next, if you saw an audiogram from muscle biopsy, you will see radiolabeled ATP bound to what? (actin OR myosin OR tropomyosin OR troponin C)

A) the answer is MYOSIN. During the contraction, ATP binds to MYOSIN, causing the dissociation of myosin from action. KNOW then the actin forms cross bridges with myosin but there is no ATP binding. KNOW that Tropomyosin runs alongside actin. It blocks myosin binding sites.

KNOW that Troponin C is the calcium-binding SUBUNIT of the troponin complex and makes it shift to expose myosin binding sites.

hyc 1218 (Good question on physio/endo) You are a doctor who wanted to conduct a neurotransmitter experiment to assess extracellular neurotransmitter levels in the brain following electrical stimulation of the raphe nucleus. What will rise? (ACh, Dopamine,

486

GABA, Norepinephrine, Serotonin). Can you point to all the structures?

A) Serotonin. it is the main neurotransmitter in the raphe nuclei.  
KNOW that ACh is found mainly in the basal nucleus of Meynert.  
KNOW that dopamine is found mainly in the substantia nigra. Although I did not mention it, GABA is inhib. and found everywhere in the brain. Also, know that NOREPINEPHRINE is found in the locus ceruleus.

1219

You will likely face this concept if not on USMLE, then in clinic. The question is...What is the general ERPF or effective renal plasma flow for the average person? Do you know the simple equation? YOU HAVE TO KNOW THIS.

A) Around 635 mL/min. The equation is  $U_{pah}V/P_{pah}$ .

1220

Ah, good one. You have a father coming in with his son wondering if he is the TRUE biological father. What can you verify?

A) This is done a LOT, it is called RFLP...Here a blood sample is drawn and digested with restriction enzymes and you observe the distance of the fragments on the gel. Please look it up on a microbiology book!!!! We also use this test sadly for rape victims, etc.

1221

Hey, the molecular biology of pituitary hormones and pancreas are

487

which? (pick catecholamines, OR amino acid derivatives, OR peptides)

A) PEPTIDES, Recall that pancreatic glucagon and insulin are peptides!!!

1222

You have a pt with megaloblastic anemia with folate def. Erythropoiesis is lesioned due to a defect in what reaction? (pick Acyl transfer OR Carboxylation OR Decarboxylation OR Hydroxylation OR Methylation)

A) This is a toughie! Listen, ans is methylation. Recall the MOA is from TH4 in its reduced form. TH4 accepts methyl, methylene, carbons to transfer them! So the answer is methylation. KNOW that Acyl transfers occurs in Pantothenic acid/Acetyl CoA....KNOW that Carboxylations occurs in Biotin/Vitamin K....KNOW that Oxidative decarboxylations occur in thiamine rxns....KNOW that Hydroxylations occurs in Ascorbic Acid reactions!!!!!!!!!!!!!!

1223

You see a 60 year old alcoholic in the ER. You know you need to give thiamine. But your med students asks, "Why not Biotin, Niacin, Pyridoxine, Riboflavin?" How do you answer?

A) Biotin is a activated carboxyl carrier used to treat baldness, bowel inflammation, myalgias.

Niacin treats PELLAGRA (Diarrhea, Dermatitis, dementia).

Pyridoxine treats neuropathy and dermatitis.

488

Riboflavin treats skin lesions.!!!

1224

You are looking at a skin biopsy of malignant melanoma and see large visible nucleoli. Thus, the cells are making WHICH OF THE FOLLOWING? (Cell surface markers, Golgi apparatus, IgGs, DNA, Ribosomes)

A) Ribosomes!

1225

Tell us which enzyme is stimulated by glucagon? (acetyl CoA carboxylase, Glycogen phosphorylase, Glycogen synthase, OR pyruvate kinase)

A) Think about it, glucagon WILL be needed when glucose is needed...so, THINK we either need to break apart glucagon or create glucose (gluconeogenesis). THEREFORE, think we need glycogen phosphorylase to catalyze the first step in glycogenolysis!  
KNOW that acetyl Coa carboxylase is fatty acid synthesis, which is stimulated by INSULIN!!!!  
KNOW that you dont want to MAKE glycogen  
KNOW that pyruvate kinase catalyzes the LAST REACTION in glycolysis. Glucagon acts to INACTIVATE it, to STOP glucose consumption.

1226

You need to know HOW glycogen degradation and glycogen synthesis is different. Tell me, the glucose used in glycogen synthesis are bound

489

to WHAT KNOWN nucleotide???

A) UDP!!! When you cook up glycogen after eating too many fatty steaks, one high energy phosphate bond of uridine triphosphate is used by UDP glucose pyrophosphorylase to make UDP-glucose. THEN, this binds to glycogen primers to make glycogen. KNOW that if you chose GDP or GTP, you are thinking about the TCA cycle!

1227

A neonate comes in with vomit, diarrhea, stomach pain, hypoglycemia when the mom tries to feed. She has lactic acidosis, hyperuricemia, hyperphosphatemia. YOU are told this is fructose intolerance. The baby should also avoid WHICH other sugar?

A) SUCROSE. KNOW that You can be missin g either fructokinase OR aldolase B. If you do not have aldolase B, you are in BIG trouble because you lose intracellular phosphate to make ATP. YOU HAVE to stop eating sucrose because it IS FRUCTOSE and GLUCOSE!

1228

Lets say I give you a pic of a histo slide. Then I ask you to point to the thing that anchors an EPITHELIAL cell to the BASEMENT MEMBRANE? (is it adherent OR connexon OR hemidesmosome OR tight junction?????)

A) Hemidesmosomes! They are like spot welds between cells and hook onto an extracellular matrix like the basement membrane.

490

KNOW that adherences/zonula adherens are "attachments" and tight junctions are "seals".

1229

One of the previous posters said TCA cycles was high yield for his test. Let me ask then...succinate thiokinase cleaves to make a high energy compound. What can the resulting compound be used for INSIDE the cell?

A) YOU must know that GTP is synthesized here...so you need to know that GTP and NOT ATP is used to make proteins in ribosomes and they power tRNA binding!!!!

1230

Regarding amino acids, which one is involved in the BUFFERING capacity of hemoglobin? (pick arginine, aspartic acid, glutamic acid, histidine, OR lysine)

A) A buffer is good if it is close to pKa, pH wise. Thus, HISTIDINE is closest to physiologic pH and thus the right answer!!!!

1231

IF the USMLE or your attending asks, KNOW that 1 g of protein or carbohydrate makes 4 kcal of energy and 1 g of fat makes about 9 kcal of energy. You need to know this to calculate if someone goes on a crash diet or something.

Let's say an MD/PhD takes a culture plate of bugs which need almost no food, and then takes the buggies out and puts them on a plate full of bacterial goodies to eat. When the bacteria grow, each TYPE is isolated. The ones that cannot grow at all are called "THE MUTANTS",

491

and their genes are sequenced. In one case, the MD/PhD finds that a two nucleotide segment of DNA is deleted. This is what kind of MUTATION?

A) FRAMESHIFT. See, a long long vignette to ask a simple question...

1232

What DECREASES the fluidity of the plasma membrane?

(Pick either LOWERING the melting temp OR Increasing cholesterol OR increasing unsaturated fatty acids)

A) Increasing cholesterol!!! The more the cholesterol, the more tightly the phospholipids are packed up, resulting in a membrane with high rigidity and low fluidity. KNOW that if you decrease the membrane's long chain fatty acids, you increase fluidity because the molecules pack tighter than UNSaturated fatty acids.

1233 Ahhh..great one. I present to you an imaginary picture of a glucocorticoid receptor. What is the role of it? (TATA box, Enhancer, Cis element, Transcription factor)

A) Surprise, it is a TRANSCRIPTION factor. It stimulates the binding of RNA polymerase to promoter sites on DNA. KNOW a cis element regulates the expression of nearby genes. KNOW that an enhancer is a DNA sequence that itself stimulates promoters. KNOW that a promoter is where the RNA polymerase binds.

1234

492

Say someone is allergic to niacin. Which of the following can be a substitute? (Asparagine, Alanine, Proline, Tryptophan)

A) It is ... Tryptophan! A derivative can be used in NAD synthesis. Lots of tryptophan can replace a lack of niacin.

1235

You have a fetus (deceased) with a small head, eyes, cleft lip, palate, six fingers. Is he Trisomy 13 or Trisomy 18????

A) Trisomy 13! Remember to think of polydactyly (thirteen fingers) and CLEFT stuff like palate and lip. Trisomy 18 has the rocker bottom feet (18 year olds like to "rock n' roll") and have prominent occiput and low set ears. Both are mentally retarded.

1236

A patient has a PMH for multiple infections involving the lungs, liver, bones, granulomas, gingivitis, APHTHOUS ulcers. What enzyme is deficient?? (Good Question)

A) NADPH oxidase! Recall that this is results in Chronic granulomatous disease of childhood, thus, here, the neutrophils and phagocytic cells cannot make superoxides! Some people think the answer is MYELOPEROXIDASE but are wrong b/c this defect is usu. seen in diabetics with fungal infections.

1237

A cell that makes glycoproteins with 8-9 mannose residues per sugar

493

chain possesses a glycosylation enzyme defect in an organelle? Which one is it?

A) GOLGI APPARATUS! This step occurs in rough ER. The trimming of mannoses to 5 residues occurs in the Golgi PRIOR to complex sugar addition.

1238

You see a 18 year old with bilateral weakness with difficulty relaxing mostly the hands and feet. A muscle biopsy shows prominent ring fibers, central nuclei, nuclei chains. This disease is a mutation on which chromosome? What dx?

A) This is MYOTONIC DYSTROPHY, mutation on chromosome 19, Autosomal dominant. It also causes cataracts, testicular atrophy, heart trouble, dementia, baldness, and weakness. This is COMMON and systemic.

1239

You are asked to use DNA polymerase in the PCR test. This enzyme is resistant to which? (Pick Acid OR Base OR Heat OR high Na<sup>+</sup>)

A) HEAT! Recall PCR uses heat to separate the DNA strands to be used as templates. Thus, the DNA polymerase used MUST be resistant to the heat!

1240

Amongst your friends, the FREQUENCY of color blindness in males is 1

494

in 100. Assuming Hardy Weinberg equilibrium, the frequency of color blind females is what? (Hint: it is NOT zero)

A) Recall this is an X linked recessive for males. So the freq of it in males is EQUAL to the frequency of the allele in the population. So  $q = .01$  and  $p = .99$ . Then IF a female had BOTH copies of each gene, the rare case would show a frequency of  $q^2$  or .0001.

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1241

You see a man exercising. Aerobic glycolysis is used for the energy source. What exact COMPOUND will enter the TCA cycle?

A) Acetyl CoA...I wanted to know if you were paying attention.

1242

Let me guess, which of the amino acids is POST translationally HYDROXYLATED in the cytoplasm of fibroblasts? (Pick cysteine, glycine, proline, serine)

A) PROLINE!! The hydroxylation of proline in fibroblasts generates the modified amino acid hydroxyproline. This is used for stabilizing the three dimensional triple helix of collagen. KNOW that cysteine are part of the double disulfide bonds in the triple helix. KNOW that while GLYCINE is every third amino acid in collagen, it is NOT hydroxylated. KNOW that when SERINE is phosphorylated, it plays a role in signal transduction.

495

1243

1244

You have a pt running away from his angry wife in a short burst. You estimate that he will use .5 L O<sub>2</sub> aerobically. BUT, the metabolism of 15 L of O<sub>2</sub> needed to escape is mostly from anaerobic sources. SO, the majority of ATP generated is derived from what? (Creatine phosphate? OR Gluconeogenesis? Glycolysis?)

A) GLYCOLYSIS!!! KNOW that he will use up his ATP stores in only ONE second. Creatine is used up next for say 3 to 4 seconds. Then, to escape his angry wife, he will use glycolysis. KNOW that if his wife chases him all around the town for hours on end, gluconeogenesis and even lipolysis will KICK in.

1245

Which lipoprotein disorders is noted by an increase in chylomicrons and xanthomas INSTEAD of atherosclerosis? (is it Abetalipoproteinemia OR Familial hypercholesterolemia OR Familial Lipoprotein lipase def)

A) familial lipoprotein lipase deficiency.

1246

Your pt is a 28 year old with ORAL ULCERS. PMH is that she is a VEGETARIAN ONLY. LABS are severe for riboflavin def. Which ENZYME in the TCA cycle is most affected by the riboflavin def.?

A) Succinate dehydrogenase!!!! Riboflavin is used to MAKE FAD and succinate dehydrogenase uses FAD as a cofactor.

496

1247

Say I show you a figure of a DEOXY-nucleotide, does it block (cDNA synthesis? mRNA synthesis? poliovirus? )

A) cDNA synthesis. Because these babies lack the OH group, they can be seen as substrates by DNA polymerase, including reverse transcriptase (This IS ACTUALLY a RNA dependent DNA polymerase). RECALL that the RNA polymerases do not recognize deoxynucleotides as a substrate.

1248

True or False: Both sickle cell anemia AND Tay Sachs are autosomal recessive.

A) True!

1249

Your pt is a mommy AND her daughter. The girl suffers from a disorder where a sugar substitute called aspartame could really harm her. What dx does the daughter likely have? (Hyperuricemia? PKU? Hyper-valinemia?)

A) She has PKU. Phenylalanine to TYROSINE is lost.

1250

You are shown a picture of a cell. Given a list of AA, what is likely found on the outside SURFACE of the cell? (Alanine OR Arginine OR Leucine OR Tryptophan)

497

A) This is a typical TWO stepper. KNOW hydrophilic amino acids are likely to appear on the protein molecule surface exterior, and hydrophobic AA are interior. SO, what AAs are hydrophilic? Arginine is one, as it is a basic AA positively charged. The other AAs I gave are NEUTRAL!

1251

Given a picture of a retinoblastoma (can you spot one)? Tell me what chromosome is lesioned?

A) Usually this is a chromosome 13 lesion. They look like small masses of hyperchromatic cells with rosettes that form a circle.

1252

You have a 25 year old man in your clinic with pneumonia. Since age 5 months, he has had recurrent sinopulmonary infections from encapsulated bacteria. He has abnormal immune function of? (T-cells, B-cells, NK cells, Macrophages, Platelets)

A) B-cells, likely Common Variable Hypogammaglobulinemia, low serum levels of IgG at around 6 months of age when mommy's levels disappear from his blood.

1253

You have a female pt, 18, who tells her boyfriend that sex hurts. She also has to urinate a LOT. PE is high fever and no vaginal discharge or cervicitis. UA has 15 WBCs with Gram neg rods. What do you give her in meds? (More than one answer could be right)

A) You should try Ampicillin for her SIMPLE UTI. Some may think of Ceftriaxone, but reserve the "big gun" drugs for later. You can rule out Metronidazole since there is no discharge and there ARE gram negs in the pee.

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1254

You see a 30 year old female with allergic rhinitis who got hit in the face and stomach with a blunt object. Her spleen is lesioned. She is transfused with 4 units of ABO and Rh type blood. As the transfusion goes, she becomes hypotensive with airway edema. WHAT preexisting condition did she have? Pick either C1 esterase inhibitor def OR IgA def.

A) Likely she has IgA def. This is common with BLOOD TRANSFUSIONS and the combo with sinopulmonary infections! If you thought C1 esterase, you should have seen recurrent attacks of colic, WITHOUT pruritis or COLIC or allergic type reactions.

1255

Great question...A young woman at 35 weeks pregnancy comes in with urinary frequency and BURNING. PE has NO fever, chills, vomiting, nausea. LABS are positive for WBCs, PROTEIN, hematuria, gram neg buggies. What is the VIRULENCE FACTOR of the bug (is it HEAT STABLE toxin, HEAT LABILE toxin, P pili, Type 1 pili?)

A) This is E-coli most commonly and is P pili as the virulence factor in most cases.

499

1256

What cell surface marker is used to lyse IgG coated cells by NK (natural killer cells)? (CD3 or CD 19 or CD 16)

A) NK cells = CD 16 is an Fc receptor for NK cells. RECALL that CD3 is NONvariable part of the T cell receptor.. NK cells are CD3 negative. RECALL that CD19 is a B-cell marker! RECALL that CD 56 (if you thought about that) IS a NK cell marker, but is not involved with antibody dependent toxicity.

1257

You get a question/patient with a kidney stone made of STRUVITE (Magnesium Ammonium Phosphate). What bug is responsible (Proteus OR Ureaplasma urealyticum)?

A) Proteus. This buggie raises the pH. RECALL that Ureaplasma DOES made urease like Proteus, but causes urethritis.

1258

A young college dude comes in with fever, cough, blood in sputum. LABS show high BUN/creatinine. Microscopy shows LINEAR pattern of fluorescene along basement membranes. What HYPERSENSITIVITY TYPE IS THIS? (I, II, III, or IV)

A) II !!! Goodpasture's!! OK, when given stuff like autoimmune rxns, drug allergies, blood transfusions, hemolytic dx, think TYPE II. TYPE I, with the asthma, eczema, hives, are more obvious. TYPE III recall have IgG or IgM and activate complement. C3b, I repeat, C3b is made, and so is C3a and C5a. KNOW that TYPE III happens a week to two weeks AFTER exposure and classic TYPE III is serum sickness, and Arthus response and SLE and glomerulonephritis. TYPE IV is UNIQUE in that it is NOT mediated by antibody and tuberculin sensitivity and poison ivy/contact dermatitis are CLASSIC egs.

1259

Nasty dx. and bug...a man comes in with sickle cell dx. He has bad venous access, so a catheter is put in a subclavian vein. He later has arm pain and swelling and fever and chills. Bugs are cultured with gram positive cocci, catalase positive and gamma hemolytic. What is the BUGGIE? (Enterococcus OR Staph. epidermidis)

A) Don't be tricked by the gamma hemolytic and think of Enterococcus! Staph epidermidis is the right answer.

1260

Man, alcoholic, dental caries, pulmonary abscess, "treated with antibiotics". Days later he gets terrible diarrhea and GI pain. What antibiotic is more likely (Chloramphenicol OR Clindamycin)?

A) Clindamycin is likely here and he has C difficile.

1261

501

A middle aged man...chronic renal failure...gets new kidney...takes cyclosporine...7 MONTHS later his creatinine RISES. Your biopsy of his kidney shows what??? (Neutrophils?)

A) NOT neutrophils, which are part of HYPERACUTE rejection, but rather you will see INTIMAL FIBROSIS and TUBULAR ATROPHY from chronic rejection! KNOW the subtle differences. If he had rejection within say 4 months, you will see INTIMAL THICKENING, not fibrosis. ACUTE rejection often involves T-cells, interstitial edema, hemorrhage.

1262 KNOW that periplasmic space is found only in gram neg buggies.

1263

You have a boy, smoky urine, previous sore throat. PE has HTN (hypertension), edema. U/A has RBC casts. Is the buggies (Catalase +, Coagulase +, OR bacitracin sensitive)?

A) Bacitracin sensitive! The buggies are *S. pyogenes*. This is BETA hemolytic and BACITRACIN SENSITIVE

1264

What can you remember about the functions and production of IL-4?

A) It is produced by TH2 cells and mast cells. It induces cells to express MHC class II antigens and B-cell proliferation, induction of

502

atopic allergies, AND it helps class switching to IgG and IgE but not IgA. IL 5 does the class switching to IgA.

1265 We just said that IL-5 stimulates B-cell class switching to IgA. KNOW it is secreted by T helper cells and promotes B cell proliferation, production and eosinophils. What then does IL-6 do?

A) IL-6 recall stimulates acute phase reactants and Ig production.

1265 You see a child in your office with yellow stained teeth. Mother took antibiotics during the pregnancy...the one that caused this works how?

A) Think of Tetracycline. It works by binding to the 30S subunit and stopping aminoacyl tRNA attachment! (A common family member is Doxycycline)

1266 Someone takes a drug that is nephrotoxic and ototoxic. It requires O<sub>2</sub> for uptake, and prevents bacterial initiation complex formation. What drug is it?

A) Aminoglycoside.

1267 A young man gets a new kidney, etc. from a donor with blood type B. He has blood type A. Immediately he gets a horrible reaction w/ hemorrhage, fever, etc. Is this due to (hyperacute rejection fr. lymphocyte and macrophages OR hyperacute rejection fr. preformed ABO antibodies)

A) ABO antibodies...The preformed anti-B ABO antibody is causing this HYPERACUTE rxn., where complement reacts and kills the tissue. KNOW that preformed antibodies can also be found fr. previous grafts, blood transfusions, or pregnancy.  
1268 A man gets an abd. abscess and responds to NAFCILLIN but not cephalosporin. The bug hydrolyzes what bond if given a molecular diagram of cephalosporin?

A) Look for the arrow on the AMIDE bond. *S. aureus* is the likely bug here.  
1269 Year to year, the influenza A vaccine is not effective because???

A) Antigenic shift from reassortment.  
1270 present you with a case of sickle cell disease. The bug is motile, but does it ferment lactose?

A) Yes, *Salmonella* does.

1271 You are a clinician next to the Ohio River Valley. You see a young woman with headache, nonproductive cough, getting sick after cleaning a chicken coop. Is this *Cryptococcus*?

A) No, this is Histoplasma.

1272 Is the bladder supplied by the internal OR external iliacs?

A) INTERNAL iliacs

1273 What artery supplied the left lesser curvature of the stomach?  
What artery supplies the right half of the greater curvature of the stomach?

A) Left HALF of Lesser curvature = left gastric. (Right HALF of Lesser curvature = right gastric. Greater curvature = right gastroepiploic.

1274 The short gastric off the splenic artery/LEFT gastroepiploic, supplies what part of the stomach?

A) FUNDUS of the stomach.

1275 You have to know everywhere that the internal pudendal artery gives rise to. What are they?

A) It COMES from the anterior internal iliacs, and divides to the INFERIOR RECTAL, PERINEAL A., URETHRAL A., DEEP A. and DORSAL arter of penis/clitoris.

1276 You see a radiograph with an arrow pointing to a structure medial and deep to the uncus. What is it?

A) It is the AMYGDALA. If you thought Caudate nucleus, it lies LATERAL to the lateral ventricles. The putamen lies LATERAL to the caudate.

1277 A little boy, w/ blood in feces. +4 cm ileal outpouching 50 cm from ileocecal valve. What kind of ectopic tissue is here? What dx? (Hint: it is a persistence of the vitelline duct)

A) This is MECKEL'S Diverticulum. Very common. Causes ulceration, inflammation, bleeding because of ectopic gastric tissue.

1278 A woman, stabbed in the superolateral aspect of the thoracic wall at the third rib. No bleeding, no SOB. But, the medial border of the scapula on the injured side pulls away from the body wall when the arm is raised. Also, the arm cannot be abducted above the horizontal. What muscle is LESIONED? Innervation too please?

A) Serratus anterior! It holds the scapula against the body. You are seeing a "winging". It is innervated by the LONG THORACIC NERVE.

1279 For the previous concept case, why isn't the answer the supraspinatus? Give innervation.

A) It does NOT hold the scapula against the body wall, and a knife at the 3rd rib will not affect it. It is innervated by the suprascapular n.

1280 You are in lab looking at cells arrested at various stages of oogenesis. You see a follicle in the ovarian stroma that develops an antrum. This follicle is what? A Graafian follicle?

A) No, it is a primordial follicle. RECALL: Primordial follicle > Primary follicle > Secondary follicle > Graafian follicle

1281 A man with cirrhosis, portal obstruction in the liver. Portal blood could still reach the caval system through WHAT veins? More than one answer is possible, just give one...

A) Consider the azygous and hemiazygous veins. Because they anastomose with the left gastric vein, portal blood can go thru the superior vena cava via the azygous veins. Recall there are a couple of OTHERS like the superior rectal vein and the middle/inferior rectal veins. ALSO there is the paraumbilical veins with the epigastric veins (recall caput medusae?); ALSO recall the splenic and colic veins with the renal veins and those of the poster BODY wall.

1282 (Per reader request, on with second messengers..hyc 1208 )Now that we got that straightened (And you WILL be asked such stuff), we need to link that with the specific receptors.

As I said before, for:

Alpha 1 receptor, you MUST link it with G protein class "q" because that connection will connect alpha 1 with Phospholipase C and Protein

507

kinase C and  $Ca^{2+}$ . To do so, you need to think that the word "alpha" and the number "1" is first in every ordinal list. Then think of how a Gorgeous Queen "q" comes first in the priority list. Again, repeat, Alpha 1 is tops, and a Beautiful Queen is tops. Then you will recall that the Gq (GQ magazine) with good looking guys and gals) is stimulatory, and a boost of Calcium is always stimulatory, as is the Protein kinase "C". So again, going backwards, if you "C" (see) that Gq (magazine) features 1 Alpha males, you hopefully will link this. See??

For Alpha 2 receptors, just recall that it is the opposite of alpha 1 in that it is inhibitory in action  $G_i$ . This fact is easily remembered if you say it over and over that Alpha 1 and Alpha 2 are OPPOSITES of each other. Plus, know that ALL the subtype 2s like alpha 2, M2, D2 (except B2 for the lungs), are  $G_i$  proteins. Then, connect  $G_i$  with the letter A to form the word  $G_iA$ , who was a famous model (like those in Gq magazines). The letter A connects you to Protein kinase A. (except for B2), the  $G_i$  proteins which lower protein kinase A are inhibitory.

Beta 1 receptors...what are they? They are  $G_s$  or stimulatory. You may recall this from all that cardio stuff and the stimulation, but then think B1S, or Barf 1 sandwich for B1 and S. Again, B1 AND B2 are stimulatory via a  $G_s$  protein. Again, "B"e stimulatory. NOW, listen, the  $G_s$  protein is associated with c"AMP and "ATP and Protein kinase "A". Connect "Gs" and "A" with GSA. GSA, GSA, GSA, what can it stand for? Good Sex Alnight. G-S-A. Again, protein  $G_s$  stimulates protein kinase A via cAMP and ATP.

Beta 2 receptors..think here that you have ONE heart Beta one, and TWO lungs..for Beta 2. Both BETAS are stimulatory. Think Be-"T"otally "Awesome". Say over and over, Betas are STIMULATORY>

{Now for the Ms}

M1 is Gq and thus stimulatory (recall the magazine) via IP3 and

Calcium. AND Protein Kinase C.

M2 is Gi and thus inhibitory via cAMP and Protein kinase A

M3 is Gq and is thus stimulatory via IP3 and Calcium and Protein Kinase C

For the D1, think it is stimulatory because ALL 1's are stimulatory!  
Alpha 1, Beta 1, M1, D1, H1, are all stimulatory.. They are first, and thus stimulating. The 2's, Alpha 2, M2, D2, V2 are INHIBITORY!

So, D1 is Gs

D2 is Gi

H1 is Gq

H2 is Gq (an exception)

V1 is Gq

V2 is Gs (an exception) H2 and V2 are exceptions...again, say it again, H2 and V2 are exceptions. they are stimulatory and not inhibitory like the other 2s.. Say it again, H2V2, H2V2, H2V2, H2V2,...sick of it yet? Well I am not, you have to know they are exceptions. (This was helpful thanks!! But one lil correction, B2 is also stimulatory, it is an exception to the 2s being inhibitory - since it is in Gs class!) (All 2s', only alpa2 and D2 are inhibitory!) (Plus M2.) (But, I am just trying to generalize. You cannot know everything, and these second messengers are heavily tested guaranteed. So you need some weird NON perfect way to lump them...UNLESS you have a WORLD CLASS memory. I know I don't. .. sorry.)

1283 1282 (Remember, secondaries, secondaries) A man comes to you with gait problems, slow, slurred speech, cannot move items back and forth quickly, intention tremor, hypotonia, nystagmus. The lesion is a brain part that comes from which EMBRYONIC structure?????

A) Metencephalon. The man has a CEREBELLAR lesion. KNOW that the ANTERIOR end of the neural tube makes three parts (prosencephalon/forebrain, mesencephalon/midbrain, rhombencephalon/hindbrain). KNOW the cerebellum AND pons comes from the metencephalon.

hyc 1217 A 50 yo woman with CHF goes to the ER. PE shows resting O<sub>2</sub> of 200 ml/min, a peripheral arterial O<sub>2</sub> of .20 ml O<sub>2</sub>/ml of blood, and a mixed venous O<sub>2</sub> of .17 ml O<sub>2</sub>/ml serum. What is the cardiac output? YOU HAVE TO DO SIMPLE CALCULATION ON USMLE and in CLINICS!

A) Simply,  $CO = O_2 \text{ consumption} / (O_2 \text{ arterial} - O_2 \text{ venous})$ . So, we have  $200 \text{ ml/min} / (.20 \text{ ml O}_2/\text{mL blood} - .17 \text{ mL O}_2/\text{ml blood}) = 4000 \text{ mL/min} = 4.0 \text{ L/min}$

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1283 Regarding the previous concept, just KNOW that AFTER say a month, the PROSENCEPHALON develops further into the telencephalon and the diencephalon.

The TELENCEPHALON breaks up into the CEREBRAL HEMISPHERES (cerebral cortex, basal ganglia white matter).

The diencephalon becomes everything "thalamus"...thalamus, hypothalamus, subthalamus, incl. posterior pituitary and neural retina.

KNOW the mesencephalon/midbrain STAYS the mesencephalon.

1284 Hey, the ciliary body is deformed...this is due to malformation of what? Hard question...sorry.

A) NeuroECTODERM...of the optic cup, from the evaginations of the diencephalon.

1285 You likely recall that the PULMONARY valve is heard over the LEFT 2nd intercostal space. So, give me a children's common cause of such pulmonary stenosis? (more than one answer is possible)

A) TETRALOGY OF FALLOT.

1286 You attempt a study to increase the norepinephrine concentration in the cortex of an animal. He does this by electrically stimulating a nucleus in the brain. What nucleus is important for noradrenergic innervation to the cerebral cortex. ?

A) Locus coeruleus.

1287 KNOW that the Nucleus of Meynert are CHOLINERGIC neurons. It innervates the neocortex, hippocampus, amygdala. This degenerates in Alzheimers. Can you point to it on a CT scan of the head?

1288 KNOW the caudate nucleus (part of the basal ganglia) has GABA projection to the globus pallidus and substantia nigra. The GABA

511

neurons lesion in HUNTINGTON'S. Therefore, the nucleus, which is lateral side of lateral ventricles will atrophy so the ventricles will look bigger. Also, the caudate has CHOLINERGIC neurons which provide the ACh to the striatum.

1289 Recall, raphe nucleus = SEROTONIN. Can you point to it on a CT scan?

1290 Now, what about the SUBSTANTIA NIGRA pars compacta? What is the main substance here? Can you point to it?

A) Here we find DOPAMINE. This degenerates in Parkinson's disease or if you take MPTP. It is above the pons, posterior to the Nucleus Meynert. RECALL the Raphe nucleus and Locus Ceruleus are POSTERIOR on the brainstem. Be ready to point to them.

1291 KNOW that if you are asked by anyone in the future about the Ventral Tegmental Area, know that it has dopamine for the limbic and cortex. These area is also known as the mesolimbic neurons, which if overactive, leads to schizophrenia.

1292 You notice your attending tapping the side of the face of a patient who just had thyroid surgery. The attending is concerned about a lesion to WHAT vessels? Very hard question...

A) The attending is checking for tetany, which happens if the parathyroids are damaged and the superior and inferior thyroid arteries are accidentally lesioned during the surgery. If PTH is lessened, the pt. will contract HARD his masseter muscle from

512

hypocalcemia.

1293 A man comes to you, abd. pain, nausea, vomiting, afebrile. LABS show a loop of small intestine passed into the epiploic foramen into the omental bursa. If you try to FREE the intestine by cutting the epiploic foramen, what structures are you likely to damage?

A) You may cut parts of the portal triad: the hepatic artery, common bile duct, portal vein.

1294 A young man, stabbed in the left chest, comes in with decreased function of the LEFT arm. PE shows a WINGED scapula. What nerve was cut?

A) The long thoracic nerve was severed, and the serratus anterior m is not healthy.

1295 A young man is stabbed in his right fifth intercostal space at the midaxillary line. What is lesioned?

A) LIVER. Any wound usu. BELOW the fourth intercostal space likely hits the liver (recall midaxillary is NOT in midline) . If you thought R. atrium, KNOW it goes from the third costal cartilage to teh 6th costal cartilage just to the right of the sternum.

1296 The eustachian tubes and epithelial line of the tympanic membrane comes from which pharyngeal POUCH or ARCH?

513

A) Comes from the first pharyngeal pouch

1297 If I point to the AXILLARY nerve, what cord does it come from?  
What muscles associated with the rotator cuff are affected?

A) So important, it is from the posterior cord (C5, C6). Often comes from a break in the surgical neck of the humerus. The teres minor and deltoids can be lesioned so you lose arm abduction and sensation.

1298 Now I point to the lower subscapular nerve. What muscle does it innervate?

a) The teres MAJOR, is a branch of posterior cord C5, C6.

1299 What structure is most MEDIAL in a kidney if shown a histology slide? Is it the Renal pyramid?

A) No, it is the Renal pelvis, which is the dilated upper part of the ureter.

1300 A young man, falls while skating, lacerates a 4 cm gash on the lateral knee. You can see the head of the fibula sticking out. You see a foot drop while the pt. walks. What nerve is lesioned?

A) Common peroneal. If you thought Tibial, know the tibial nerve supplies the POSTERIOR compartment of the leg inc. the gastrocnemius and soleus and flexor digitorum LONGUS.

1301 You are doing an echo (cardiac). The anterior wall of the left ventricle is found ischemic. Is it the (left anterior descending OR left circumflex) that is lesioned?

A) Left anterior descending.

1302 What artery supplies the AV node AND the posterior wall of the LEFT ventricle?

A) Tricky. It is the Right coronary artery, which ALSO supplies the R ventricle

1303 Which famous nerve gives rise to the cremasteric reflex?

A) Genitofemoral nerve.

1304 YOU HAVE to know a few of the most famous nerves in anatomy. So, what nerve supplies the LATERAL side of the thigh?

A) Lateral cutaneous nerve.

1305 Again, famous nerves...What nerve supplies the anterior LOWER abdominal wall?

A) Iliohypogastric Nerve

1306 You will have to know how to calc. an ODDS RATIO for the USMLE. So, what is the formula and what's it for?

A) Odds ratio =  $(\text{TruePositives}/\text{TrueNegatives})/(\text{FalsePositives}/\text{FalseNegatives})$ .

1307 OH, I forgot to say, ODDS RATIO is used for CASE CONTROL studies to assess and approx. of the relative risk of disease if the PREVALENCE is low.

1308 You are going crazy studying for the USMLE because it is a torture to keep at it. You are given CHLORPROMAZINE. What are the side effects?

A) This has antimuscarinic effects, (DRY MOUTH, CONSTIPATION). And ORTHOSTATIC HYPOTENSION, SEDATION.

1309 Your friend is depressed studying for USMLE. You given her Imipramine. SEs? Just name a couple. You cannot know everything, ya

516

know...

A) This classic Tricyclic has anticholinergic, antihistamine effects, hypomania. And orthostatic hypotension.

1310 A classic MAO inhibitor is Phenelzine. What are a few of the classic side effects if given a case on the USMLE?

A) MAO inh. don't mix well with TYRAMINE, you get HTN. You can also face hypotension when getting up too fast.

1311 Tricky. There is a group of USMLE students, number is a quarter of a million. 10,000 have a disease called "I can't standing studying for USMLE." 1,000 new cases are diagnosed each year. 400 die from that PARTICULAR disease. Unfortunately, 2500 DIE from ALL causes every year. Give the PREVALENCE of the dx?

A) 0.04...Recall PREVALENCE is the # of cases of a dx at a single moment in time divided by the TOTAL population within a given span of time. so, 10K/250K.

1312 REFER to case/concept 1311. What if the USMLE question asked you to calculate disease specific mortality rate? WHAT is it?

517

A. It is the number of deaths per year from the dx in question DIVIDED by the population. So,  $400/250,000$ .

1313 Refer again to Case/Concept 1311, what is the RATE OF INCREASE of the disease?

A) Here, it is the number of NEW cases a year minus the number of deaths (or cures) per year....ALL divided by the total population. So, here,  $(1000-400)/250,000$ .

1314 True or False, a psychotic has tardive dyskinesia, can you substitute fluphenazine with CLOZAPINE to control for the side effects?

A) YES, but watch out for agranulocytosis...

1315 USMLE literature said you need to know Kubler Ross stages of dying. What are they?

A) Recall the word Dabsa, say it over and over. DABSA:

D enial

A nger

B argaining

S adness

518

A cceptance

It is SO depressing, but you will see this in patients over and over. Life is very very difficult. Keep a soft and loving heart in God.

1316 A man comes to your office unhappy with his past relations with women but HAPPY with his relations with men. He admits tremendous guilt. Is this (ego-dystonic or ego-syntonic)?

A) Ego-dystonic...due to his guilt.

1317 Which of the following will alter the pos. pred. value of a test? (PPV)  
(Pick Incidence, Odds ratio, Prevalence, Relative Risk...one of the previous is correct)

A) Prevalence (which is defined as the total number of disease cases in a specific period of time). This directly affects the PPV value (True Positives/Total Positives). RECALL that INCIDENCE is the number of NEW cases of a disease in a specific time period. There is a formula, but RR or Relative risk can define the incidence of a disease in a TREATMENT group divided by the incidence of a disease in a PLACEBO group.

1318 A kid comes in with BACTERIAL meningitis. What is released by the PREDOMINANT WBC present? Is it peroxidase????

519

A) No, it is LYSOZYME released by neutrophils. Peroxidase is released by eosinophils. Note that if it were a viral meningitis, there would be more LYMPHOCYTES.

1319 KNOW that if you are faced with a diabetic type I, GIP, (gastric inhibitory peptide) is released in response to HYPERGLYCEMIA.

1320 If I point to a cell on the pancreas and you figure out that it is an alpha cell, do you know it promotes glycogenolysis? Can you point to one if given a histo slide of the pancreas?

Recall that the alpha cells are on the outside periphery, beta cells fill the inside.

1321 True or False: The GFR can be calculated by determining the clearance of PAH?

A) False! PAH determines ERPF or Effective Renal Plasma Flow. Recall  $ERPF = UV/P$  for PAH. PAH is totally secreted in the proximal tubule and into the urine. You may have gotten confused if you said true because GFR is found by INULIN, which is filtered, not reabsorbed, and only slightly secreted into the urine. KNOW that in clinic, you approximate GFR though with CREATININE.

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1322 You will see liver disease and thus ascites on your test and in

520

clinic. What exactly causes this process? Decreased plasma volume??

A) NO! This is due to INCREASED hydrostatic pressure in the splanchnic beds secondary to portal hypertension! KNOW also that hypoalbuminemia and reduced oncotic pressure also play a part.

1323 Q) You have a woman who types 12 hours a day for years coming in with numbness on her hands. What deficit in sensation/action will she face?

A) YOU HAVE TO KNOW carpal tunnel syn. The damage to the median nerve makes the THUMB weak via the abductor pollicis brevis, flexor pollicis brevis, opponens pollicis. Distal to the carpal tunnel, you will lose control of the first and second lumbricals which flexes the digits two and three at the metacarpophalangeal joints and extension of the interphalangeal joints of the same digits. KNOW that the ADDUCTION of the thumb is the only short thumb muscle NOT innervated by the median nerve. KNOW that you will NOT lose sense in the lateral half of the dorsum of the hand because the area is supplied by the RADIAL n.

Gosh, this is CONFUSING, but HIGH HIGH YIELD. Look at the innervations of the hand on a diagram in [Netter's!](#)

1324 You have to know how to do this easy calculation/concept....there is an adult male weighing 75 kg. What is the volume of the Total Body Water, Intracellular Volume, Extracellular volume?

521

A) First, KNOW 60% of the weight is Total Body Water (so 45 Liters). Now, KNOW that of this 45 Liters, 2/3 is INTRACELLULAR and 1/3 is EXTRACELLULAR (people often get these mixed up). So Intracellular is 30 Liters, Extracellular is 15 Liters!

1325 Listen, your attending asks you how you will know if a spot of a patient's drop of bodily fluid is PLASMA or SERUM. A high level of what substance will identify the specimen as PLASMA? (pick Albumin OR Fibrinogen)

A) Fibrinogen. You have to UNDERSTAND that serum and plasma are DIFFERENT. Serum is DERIVED FROM plasma by extraction of fibrinogen and coag factors 2, 5, 8. KNOW that Albumin is present in BOTH serum and plasma.

1326 YOU HAVE TO KNOW THIS because your attending will ask, after he places a Swan Ganz catheter in an ARDS pt. with a pulmonary artery wedge pressure of 6 mmHg. The SAME pressure will be found in WHAT HEART CHAMBER?

A) Left Atrium

1327 A student is late to his USMLE test and is HYPERVENTILATING! And thus doubles his alveolar ventilation. Suppose his initial alveolar PACO<sub>2</sub> is 50 mmHg and his CO<sub>2</sub> production is constant. What is his NEW alveolar PCO<sub>2</sub> on HYPERVENTILATION????

A) 25 mmHg. It is HALVED.

1328 During the USMLE and in clinic meetings, you will have to read hundreds of FLOW VOLUME CURVES. Given a "typical one" what point on the curve represents RESIDUAL VOLUME? What about the "Effort Independent" part?

A) Residual volume is the LOWEST volume, usually all the way to the right of the graph. The effort independent part is the even downward sloping area.

1329 Students get Secretin and Somatostatin mixed up. What is the difference? Origins please?

A) KNOW secretin is secreted by the S cells of the duodenum and in response to a meal. It stimulates BICARB. fluid from the pancreas and bile ducts. This neutralization allows pancreatic enzyme activation.

KNOW that somatostatin is secreted by the D cells of the pancreatic islets. It is inhibitory to most things secreted....

1330 Q) ACTH promotes CORTISOL production by stimulating WHAT reaction?

A) Cholesterol to Pregnenolone (via enzyme desmolase). This is the FIRST step. The next step is Pregnenolone to PROGESTERONE. From there, it is converted to 17 hydroxyprogesterone, then 11 deoxycortisol (via 21 B Hydroxylase), then finally to cortisol.

1331 An OLDER pt. of yours has ONE SIDED hearing loss. What is lesioned? (pick Organ of Corti OR Medial Lemniscus or Inferior colliculus)

A) Organ of Corti. KNOW any lesion of a structure PROXIMAL to the superior olivary nucleus will give an ipsilateral deafness. Lesions DISTAL like the inferior colliculus to the medial geniculate nucleus to the primary auditory cortex/Hesch's gyrus will give BILATERAL deafness.