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Pharmacology Board Review 2005

This list of questions and topics is the result of going through about 10 years worth of old Board Exams in Pharmacology, cutting out all the questions, categorizing them into topic areas (e.g. antibiotics, local anesthetics, etc.), and then further grouping them into the type of information about a category of drugs that was being asked for. When you do this, you see that many exams repeat questions (sometimes they reword them a little bit to make them look different!), but in actuality it is possible to get a feel for the various facts that you are expected to know, and that there aren't that many of them. As you go through this handout, you will see that I point out to you the major facts that tend to get asked over and over again for the various major drug categories, and I also give you actual examples of questions (and the reworded versions), as well as the correct answer. In many cases, I have written out a detailed explanation of the answer, just to enlighten you further. So good luck and enjoy.

The downside is that these questions are from old Board exams. Some of the material is obviously dated, as drugs fall out of fashion, newer drugs get used instead of older drugs, etc. At the beginning of each section I will try to indicate some things that have changed and thus you may want to place less emphasis on some of the questions here.

On a positive note, there used to be a separate pharmacology section of 100 questions. Nowadays, you might see 25-30 in some versions, other versions less. Unfortunately, they still can draw from the realm of pharmacology so you gotta review it all. However, the good thing is that since they ask fewer questions, and since they are trying to ask more clinically relevant stuff, if you really focus your efforts on analgesics, antibiotics, and anesthetics, you should be covered for the majority of questions.

There are always going to be some random, unpredictable questions that means you have to review more if you want to do really well. Maybe you will luck out, and these will be the questions they are testing and they don't count.

Local Anesthetics

I. The largest category of LA questions focuses on your ability to distinguish amide LAs from esters: ***(This I hope is deemphasized, since amide local anesthetics are used almost exclusively now)***

esters = procaine, tetracaine, cocaine. All the rest are amides: lidocaine, mepivacaine, bupivacaine, prilocaine, dibucaine. They also require you to know that amides are metabolized in the liver, esters mainly by esterases in plasma. An infrequent question asks which class of drugs has the most consistency in structure . LAs are the drug group most consistent in drug structure, because LAs are either amides or esters, differing only in their structure in the intermediate chain (its either an amide or an ester) that connects the aromatic group to the secondary or tertiary amino terminus.

II. The next category of questions has to do with toxic reactions to local anesthetics, either due to high systemic levels of local anesthetics in general (cardiovascular collapse due to myocardial depression, hypotensive shock) or to a specific agent such as prilocaine, which causes methemoglobinemia.

III. A 3rd class of questions are aimed at your knowledge of the mechanism of action of local anesthetics: they prevent the generation of nerve impulses by interfering with sodium transport into the neuron.

IV. The last most frequent type of question regarding local anesthetics has to do with issues regarding absorption of local anesthetics. Remember, only the non-ionized (or free base form) form can penetrate tissue membranes. Inflamed tissue has a lower than normal pH, which decreases the amount of non-ionized form available to penetrate.

V. Usually at least one question comes up asking you to calculate how many mg of local anesthetic a patient has received, e.g. how many mg of lidocaine in 1.8 ml of a 2% lidocaine solution? 2% lidocaine is 20 gm/100 ml or 20 mg/1 ml, so 36 in 1.8 ml.

1. Which of the following is a local anesthetic subject to inactivation by plasma esterases?
   a. Procaine
   b. Lidocaine
   c. Prilocaine
   d. Mepivacaine
   e. Bupivacaine

   (a) Proccaine is the only ester listed -- all the rest are amides

2. Procaine differs from lidocaine in that
   a. Procaine is a p-aminobenzoic acid ester and lidocaine is not
   b. Lidocaine is a meta-aminobenzoic acid ester and procaine is not
   c. The duration of action of procaine is longer than that of an equal total dose of lidocaine
   d. Procaine hydrochloride is metabolized into diethylaminoethanol and benzoic acid.

   (a) this is basically a true-false type question. (a) is the only statement that is true

3. Which of the following local anesthetics would be expected to produce a sensitization reaction in a patient allergic to lidocaine?
   a. Mepivacaine
   b. Tetracaine
   c. Procaine
   d. Prilocaine
   e. Dibucaine

   i. (a), (b) and (c)
   ii. (a), (d) and (e)
   iii. (b) and (c) only
   iv. (b), (c) and (d)
   v. (b), (d) and (e)

   (ii) another ester vs. amide type identification question. Lidoccaine is an amide, thus other amides will be cross-allergenic - mepivacaine, prilocaine and dibucaine are the other amides listed. Procaine and tetracaine are esters and will not be cross-allergenic.

9. The hydrolysis of procaine occurs mainly in the
   a. Liver

b. Lungs
c. Plasma
d. Muscles
e. Kidneys

(c) procaine is an ester; esters are metabolized predominately by pseudocholinesterases in the plasma.

10. Which of the following is local anesthetic subject to inactivation by plasma esterases?
    a. Lidocaine
    b. Prilocaine
    c. Tetracaine
    d. Mepivacaine
    e. Bupivacaine

(c) esters are metabolized by plasma esterases - tetracaine is the only ester listed, all the rest are amides

11. The activity of procaine is terminated by
    a. Elimination by the kidney
    b. Storage in adipose tissue
    c. Metabolism in the liver only
    d. Metabolism in the liver and by pseudocholinesterase in the plasma

(d) remember #9 above? see the word "mainly"? same question, but worded a little differently to throw you off. Again, procaine is an ester; esters are metabolized predominately by pseudocholinesterases in the plasma, but also to some extent by esters in the liver.

12. All of the following factors are significant determinants of the duration of conduction block with amide-type local anesthetics EXCEPT the
    a. pH of tissues in the area of injection
    b. Degree of vasodilatation caused by the local anesthetic
    c. Blood plasma cholinesterase levels
    d. Blood flow through the area of conduction block
    e. Concentration of the injected anesthetic solution

(c) the word "EXCEPT" should alert you that this is basically a true-false type question with 4 true statements and 1 false statement; you just have to figure out which one! In this case, you just have to remember that plasma cholinesterase levels are only important for the duration of action of ester-type LAs, not amides, which are metabolized in the liver. All the other statements are variables which affect duration of the block, but apply to both esters and amides.

13. Which of the following is contraindicated for a patient who had an allergic reaction to procaine six months ago?
    a. Nerve block with lidocaine
    b. Topical application of lidocaine
    c. Topical application of tetracaine
    d. Infiltration with an antihistamine

(c) again, just another question that requires you to be able to pick out an ester or an amide from a list. Since procaine is an ester, only another ester LA would be cross-allergenic. In this list the only ester listed is tetracaine.

14. Bupivacaine (Marcaine) has all of the following properties relative to lidocaine (Xylocaine) EXCEPT bupivacaine
    a. Is more toxic
    b. Is an ester-type local anesthetic
    c. Has a slower onset of action
    d. Has a longer duration of action

(d) According to textbooks, local anesthetics fall into the following classes in terms of duration of action: <u>short</u>: procaine; <u>moderate</u>: prilocaine, mepivacaine, lidocaine; <u>long</u>: bupivacaine, tetracaine, etidocaine. Statements (a), 3, and 4 would be true if the question was comparing mepivacaine to bupivacaine, which are structurally similar; but the comparison is to lidocaine. The only difference that applies is duration of action ((d)), bupivacaine is longer. (b) is wrong, both are amides.

15. Amide-type local anesthetics are metabolized in the
    a. Serum
    b. Liver
    c. Spleen
    d. Kidney
    e. Axoplasm

(b) don't forget: esters in plasma; amides in liver

15. The duration of action of lidocaine would be increased in the presence of which of the following medications?
    a. Prazosin
    b. Propranolol
    c. Hydrochlorothiazide
    d. Lisinopril
    e. Digoxin

(b) this is an interaction I tested you on several times – now you know why! Propranolol interacts with lidocaine in two ways. By slowing down the heart via beta receptor blockade, blood delivery (and lidocaine) to the liver is reduced, thus lidocaine remains in the systemic circulation longer, and can potentially accumulate to toxic levels. Propranolol and lidocaine also compete for the same enzyme in the liver, thus metabolism of lidocaine can be reduced.

16. Severe liver disease least affects the biotransformation of which of the following?
    a. Lidocaine
    b. Procaine
    c. Prilocaine
    d. Mepivacaine

(b) Answer is (b)- You should be able to recognize that all of these drugs are local anesthetics. Local anesthetics are of one of two types, either esters or amides. Ester types are subject to hydrolysis in the plasma and thus have short half lives. Amides are metabolized primarily in the liver and have longer half lives. Thus the biotransformation (e.g., metabolism; again, the rats are using a different word to confuse you, even though they are asking the same basic question) of an amide type local anesthetic would be the most altered in the presence of sever liver disease. The key word here is "least". Of the drugs listed, only procaine is an ester. The rest are amides.

Questions regarding toxicity:

17. A patient has been given a large volume of a certain local anesthetic solution and subsequently develops cyanosis with methemoglobinemia. Which of the following drugs most likely was administered?
    a. Procaine
    b. Prilocaine
    c. Dibucaine
    d. Lidocaine
    e. Mepivacaine

(b) strictly memorization

18. Use of prilocaine carries the risk of which of the following adverse effects?
    a. Porphyria
    b. Renal toxicity
    c. Gastric bleeding
    d. Methemoglobinemia

(d) same as above but asked backwards. Methemoglobinemia may result from a toluidine metabolite of prilocaine, orthotoluidine.

19. The most probable cause for a serious toxic reaction to a local anesthetic is
    a. Psychogenic
    b. Deterioration of the anesthetic agent
    c. Hypersensitivity to the vasoconstrictor
    d. Hypersensitivity to the local anesthetic
    e. Excessive blood level of the local anesthetic

(e) Most toxic reactions of a serious nature are related to excessive blood levels arising from inadvertent intravascular injection. Hypersensitivity reactions (options b & c) are rare, but excessive blood levels will induce toxic reactions like CNS stimulation in most everyone. This is a case where option (e) is the "best" answer, because it is more likely than the other alternatives, which might be true, but are not as likely (e.g, "most probable") to happen.

20. High plasma levels of local anesthetics may cause
    a. Inhibition of peristalsis
    b. stimulation of baroreceptors resulting in severe hypotension
    c. Inhibition of the vagus nerve to the heart
    d. Depression of inhibitory neurons in the CNS

(d) Initially LAs inhibit central inhibitory neurons, which results in CNS stimulation, which can proceed to convulsions. At higher doses, they inhibit both inhibitory and excitatory neurons, leading to a generalized state of CNS depression which can result in respiratory depression and death.

20a. Unfortunately, you injected your lidocaine intra-arterially. The first sign of lidocaine toxicity that might be seen in the patient would be
    a. Elevated pulse rate
    b. Sweating
    c. CNS excitation
    d. Cardiovascular collapse
    e. CNS depression

(c) same question as above just worded differently. The intra-arterial injection would result in the high plasma levels mentioned in the previous question.

20. The first sign that your patient may be experiencing toxicity from too much epinephrine would be
    a. Cardiovascular collapse
    b. Convulsions
    c. Elevated pulse rate
    d. Slurred speech

(c) it is a sympathomimetic after all. All the other reactions are related to elevated lidocaine levels

20. Which disease condition would make the patient most sensitive to the epinephrine in the local anesthetic?
    a. Grave's disease
    b. Diabetes
    c. HIV
    d. Alcoholism
    e. Schizophrenia

(a) Grave's disease is an autoimmune disease that causes hyperthyroidism – the resulting high levels of circulating thyroid hormone result in a hypermetabolic state with heightened sympathetic activity, which combined with injected epinephrine could result in a hypertensive crisis.

21. Cardiovascular collapse elicited by a high circulating dose of a local anesthetic may be caused by
    a. Syncope
    b. Vagal stimulation
    c. Histamine release
    d. Myocardial depression
    e. Medullary stimulation

(d) Cardiovascular collapse is due to a direct action of the local anesthetic on the heart muscle itself (LA's in toxic doses depress membrane excitability and conduction velocity), thus (d) is the correct answer. All of the other alternatives are indirect ways to affect the heart.

22. The most serious consequence of systemic local anesthetic toxicity is
    a. Vertigo
    b. Hypertension
    c. Hyperventilation
    d. Post depressive central nervous system convulsions
    e. Postconvulsive central nervous system depression

(e) Of the options listed, this is the one that will kill the patient, which I guess makes it the most serious.

23. Hypotensive shock may result from excessive blood levels of each of the following local anesthetics EXCEPT
    a. Cocaine
    b. Procaine
    c. Lidocaine
    d. Tetracaine
    e. Mepivacaine

(a) All the listed local anesthetics except cocaine are vasodilators, especially ester-ctype drugs such as proccaine and the amide lidocaine. Cocaine is the only local anesthetic that predictably produces vasoconstriction. Cocaine is also the only local anesthetic to block the reuptake of NE into adrenergic neurons, and thus potentiate the NE that has been released from nerve endings

24. Which of the following anesthetic drugs produces powerful stimulation of the cerebral cortex?
    a. Cocaine
    b. Procaine
    c. Lidocaine
    d. Tetracaine
    e. Mepivacaine

    (a) see explanation above

Questions regarding mechanism of action:

25. Local anesthetics block nerve conduction by
    a. Depolarizing the nerve membrane to neutrality
    b. Increasing membrane permeability to K+
    c. Increasing membrane permeability to Na+
    d. Preventing an increase in membrane permeability to K+
    e. Preventing an increase in membrane permeability to Na+

    (e) didn't I make you memorize this? You should at keast remember Na+ ions are involved, which limits your choices to (c) and (e). (c) would increase or facilitate nervous impulse conduction, which is the opposite of what you want the local anesthetic to do, so pick (e).

26. Which of the following is true regarding the mechanism of action of local anesthetics?
    a. Usually maintain the nerve membrane in a state of hyperpolarization
    b. Prevent the generation of a nerve action potential
    c. Maintain the nerve membrane in a state of depolarization
    d. Prevent increased permeability of the nerve membrane to potassium ions
    e. Interfere with intracellular nerve metabolism

    (b) this should be really obvious!

27. Local anesthetic agents prevent the generation of nerve impulses by
    a. Decreasing threshold for stimulation
    b. Decreasing resting membrane potential
    c. Decreasing inward movement of sodium ion
    d. Increasing inward movement of potassium ion

    (c) Answer is (c)- straight memorization- nerve impulses are generated by the influx of sodium resulting in depolarization. repolarization and inactivity occurs when potassium moves out. (sodium-potassium pump). LAs act by blocking Na+ movement.

28. Local anesthetics interfere with the transport of which of the following ions during drug-receptor interaction
    a. Sodium
    b. Calcium
    c. Chloride
    d. Potassium
    e. Magnesium

    (a) see how many different ways they can ask the same question?

Questions regarding pH effects on absorption of local anesthetics

30. If the pH of an area is lower than normal body pH, the membrane theory of local anesthetic action predicts that the local anesthetic activity would be
    a. Greater, owing to an increase in the free-base form of the drug
    b. Greater, owing to an increase in the cationic form of the drug
    c. Less, owing to an increase in the free-base form of the drug
    d. Less, owing to a decrease in the free-base form of the drug
    e. None of the above

    (d) the next three or four questions are all versions of the same thing – see the explanation below

31. A local anesthetic injected into an inflamed area will NOT give maximum effects because
    a. The pH of inflamed tissue inhibits the release of the free base
    b. The drug will not be absorbed as rapidly because of the decreased blood supply
    c. The chemical mediators of inflammation will present a chemical antagonism to the anesthetic
    d. Prostaglandins stabilize the nerve membrane and diminish the effectiveness of the local anesthetic

    (a) while some of the other alternatives sound plausible, think about the factoids you were taught about local anesthetics and variables that affect their action. An important one was the role of pH and ionization factors. Remember, the free base or nonionized form is the form that passes through membranes, yet once inside the neuron only the ionized form is effective. Inflamed tissue has a lower pH than normal tissue and will shift the equilibrium of the LA solution such that most of it remains ionized and thus unavailable to penetrate

32. The penetration of a local anesthetic into nervous tissue is a function of the
    a. Length of the central alkyl chain
    b. Lipid solubility of the ionized form
    c. Lipid solubility of the unionized form
    d. Ester linkage between the aromatic nucleus and the alkyl chain
    e. Amide linkage between the aromatic nucleus and the alkyl chain

    (c) only options (b) and (c) are relevant here - the others have nothing to do with LA penetration into membranes. Membrane permeability is affected by whether or not the molecule is "charged" or ionized or not (e.g., unionized). Only the latter form passes readily through membranes. See, they're asking the same thing they asked in the previous question, just coming at it from another angle. Remember the fact and you can cover the angles.

35. At a pH of 7.8, lidocaine (pKa = 7.8) will exist in
    a. the ionized form
    b. the nonionized form
    c. an equal mixture of ionized and nonionized forms
    d. a mixture 10 times more ionized than nonionized forms

    (c) the ratio of ionized to unionized forms is given by the formula log A/AH= pH-pKa. In this instance the difference between pH and pKa is 0. Thus lidocaine will exist as an equal mixture (so (c) is correct). Most local anesthetics are weak bases with pKa ranging from 7.5 to 9.5. LA's intended for injection are usually prepared in salt form by addition of HCl. They penetrate as the unionized form into the neuron where they re-equilibrate to both charged and uncharged forms inside

the neuron - the positively charged ion blocks nerve conduction.

33. The more rapid onset of action of local anesthetics in small nerves is due to
    a. The slightly lower pH of small nerves
    b. The greater surface-volume ratio of small nerves
    c. The increased rate of penetration resulting from depolarization
    d. Smaller nerves usually having a higher threshold

Who knows? Who cares? probably the answer is (b) - the theory goes that there is a size dependent critical length of anesthetic exposure necessary to block a given nerve. Small fibers will be blocked first because the anesthetic concentration to h critical length in a small fiber will be reached faster than the critical length in a larger fiber. You have to block three nodes of ranvier, and they are farther apart in larger fibers than they are in small diameter fibers. Make sense?

34. Which of the following statements are true regarding onset, degree and duration of action of local anesthetics?
    a. The greater the drug concentration, the faster the onset and the greater the degree of effect
    b. Local anesthetics block only myelinated nerve fibers at the nodes of Ranvier
    c. The larger the diameter of the nerve fiber, the faster the onset of effect
    d. The faster the penetrance of the drug, the faster the onset of effect

    i. (a), (b), and (c)
    ii. (a), (b) and (d)
    iii. (a) and (c) only
    iv. (b), (c) and (d)

(ii) if you knew the fact above about small nerves, then this question basically becomes a true false type thing, and (c) is the false statement. (a) and (d) make logical sense so you are stuck picking between (b) and (c). You have your pick of memorizing the small nerve thing or the myelinated nerve nodes of ranvier thing.

And now, for those of you that complained in class "do we really have to know this stuff?"

35. A dentist administers 1.8 ml of a 2% solution of lidocaine. How many mg of lidocaine did the patient receive?
    a. 3.6
    b. 9
    c. 18
    d. 36
    e. 180

(d) 2% solution = 20 mg/ml X 1.8 ml = 36 mg lidocaine. And you thought you would never have to do this stuff again!

36. Three ml of a local anesthetic solution consisting of 2% lidocaine with 1:100,000 epinephrine contains how many milligrams of each?
    a. 6 mg. lidocaine, 0.3 mg. epinephrine
    b. 6 mg. lidocaine, 0.03 mg. epinephrine
    c. 60 mg. lidocaine 0.3 mg. epinephrine
    d. 60 mg. lidocaine 0.03 mg epinephrine
    e. 600 mg lidocaine, 0.3 mg. epinephrine
    f. 600 mg. lidocaine, 0.03 mg. epinephrine

(d) 2% lidocaine = 20 mg/ml x 3 = 60 mg lidocaine
1:100,000 epi = 0.01 mg/ml x 3 = 0.03 mg epi

37. The maximum allowable adult dose of mepivacaine is 300 mg. How many milliliters of 2% mepivacaine should be injected to attain the maximal dosage in an adult patient?
    a. 5
    b. 10
    c. 15
    d. 20
    e. 25

(c) 2% mepivacaine = 20 mg/ml, so 300 mg / 20 mg/ml = 15 ml

38. A recently introduced local anesthetic agent is claimed by the manufacturer to be several times as potent as procaine. The product is available in 0.05% buffered aqueous solution in 1.8 ml. cartridge. The maximum amount recommended for dental anesthesia over a 4-hour period is 30 mg. The amount is contained in approximately how many cartridges?
    a. 1-9
    b. 10-18
    c. 19-27
    d. 28-36
    e. Greater than 36

(d) 0.05% = 0.5 mg/ml . To give 30 mg, you have to give 30mg/0.5 mg/ml or 60 ml. 1 cartridge = 1.8 ml, thus 60ml /1.8ml = 33.3 cartridges. - first express the percentage of solution as a fraction of 100, then add the units gm/ml. 0.05% equals 0.5 or 1/2 gms per 100 ml. The cartridge is 1.8 ml which you can round off to almost 2 mls total. In this 2 ml you would have 1 gm of the local anesthetic. You need to give 30 gms, which would require 30 cartridges. The alternative that meets this answer is (d). Don't get tricked by the placement of the decimal point- many people read the 0.05% as being the same as 5 gms rather than 0.5 gms.

39. According to AHA guidelines, the maximum # of carpules of local anesthetic containing 1:200,000 epinephrine that can be used in the patient with cardiovascular disease is
    a. 1
    b. 2
    c. 3
    d. 4
    e. 11

(d) the AHA limit is 0.04 mg, compared to 0.2 mg in the healthy patient. 1:200,000 equals 0.005 mg/ml or 0.009 per 1.8 ml carpule. 4 carpules would thus contain 0.036 mg, which is just below the 0.04 mg limit

Antibiotics

1. The most frequently asked type of question requires you to be able to compare various penicillin antibiotics in terms of potency against certain bugs, allergenicity, drug of choice against certain conditions, etc. For example:

a. Penicillin V vs. penicillin G: the latter is more sensitive to acid degradation and thus is usually injected rather than taken orally **(Certainly no one in dentistry uses Pen G, so I would think they would not use too many of these questions)**

b. Which penicillin has the best gram-negative spectrum: ampicillin

c. Which drugs from a list are or are not cross-allergenic with penicillin: most usually asked about ones are: cephalosporins and ampicillin are, erythromycin isn't

d. Which penicillin is useful against penicillinase-producing bugs such as staphylococcus: dicloxacillin

e. Which is specific for Pseudomonas infections: an extended spectrum such as carbenicillin

f. Which combination of agents should be used prophylactically for patient with heart valve to prevent bacterial endocarditis: ampicillin and gentamycin (1988- according to latest recommendation of AHA and ADA, although use the latest guidelines that you have heard about)) **(here's a big change obviously, since combinations are no longer used, and neither are doses given before and after treatment – review your latest prophylaxis guidelines)**

Prophylaxis Regimens For SBE (AHA 1997 Guidelines)
1st choice: Amoxicillin: 2 g (4 X 500 mg), PO 1 hr before treatment. # of pills to be dispensed depends on # of appointments Children: 50 mg/kg 1 hr prior
For PCN allergic: Clindamycin: 600 mg (4 X 150 mg) PO 1 hr before treatment. . # of pills to be dispensed depends on # of appointments
non-oral: Ampicillin IV/IM 2 g, 1/2 hr before (Kids: 50 mg/kg) Clindamycin (for PCN-allergic) 600 mg IV 1/2 hr prior, kids (20 mg/kg)
Prophylaxis for the patient with a prosthetic joint
Keflex, 2 g, (4 X 500mg), PO 1 hr before treatment . # of pills to be dispensed depends on # of appointments

Examples of patient cardiovascular conditions that require prophylaxis and some that don't (AHA 1997 Guidelines)	
Prophylaxis Required	**Prophylaxis Not Required**
Prosthetic valves	Cardiac pacemakers
Previous endocarditis	Rheumatic fever without valvular dysfunction
Pulmonary shunts	Mitral valve prolapse without valvular regurgitation

Examples Of Dental Procedures That Require Prophylaxis And Some That Don't **(According to AHA 1997 Guidelines. Caveat: our clinic guidelines, should they differ from these, are also considered correct answers)**	
Required	**Not Required**
Extractions	Restorative Procedures
Periodontal Surgery	Intracanal endodontic treatment
Implants	Taking Of Impressions

Common Prescription Regimens For Treating An Infection:	
Penicillin VK	250-500mg, dispense 30, take 2 tablets at once*, then 1 tab every 6 hrs until gone (7 days) *some sources do not indicate loading dose, so dispense 28, take 1 q6h until gone Kids (less than 12 yrs): 20-50 mg/kg qid
Clindamycin	150-300 mg, dispense 21, take 1 capsule every 8 hrs until gone (7 days) Kids: 8-12 mg/kg tid or qid
Amoxicillin	500 mg, dispense, 21, take 1 capsule every 8 hrs until gone (7 days) Kids (under 20 kg): 20-40 mg/kg tid

2. The 2nd largest category expects you to know the mechanism of action of the various antibiotics:

   a. Bactericidal agents such as penicillin kill rapidity growing cells by inhibiting cell wall synthesis

   b. Bacteriostatic agents such as tetracycline limit population growth, but do not kill bugs by interfering with protein synthesis on bacterial ribosomes

   c. Antifungals such as nystatin bind to ergosterol in fungal cell walls to weaken the wall

   d. Bacteriostatic agents such as the sulfonamides compete with PABA in folic acid synthesis, thus resulting in folic acid deficiency

3. Many questions are asked regarding side effects or toxicities of penicillins, tetracyclines, clindamycin, etc:

   a. What are symptoms seen during allergic reactions to penicillins: dermatitis, stomatitis, bronchoconstriction and cardiovascular collapse

   b. What agent produces GI upset and pseudomonas colitis: clindamycin

   c. Which agents are most likely or least likely to cause superinfection: most: broad spectrum agents such as tetracyclines; least: narrow spectrum agents such as penicillin G

   d. Aplastic anemia is associated with chloramphenicol

   e. Liver damage or hepatotoxicity is associated with tetracycline

   f. Erythromycin estolate associated with allergic cholestatic

hepatitis

4. Questions involving interactions between antibiotics and other drugs:
   a. Tetracycline and penicillin (cidal-static interaction)cancel each other out due to opposing mechanisms of action
   b. Probenecid alters the rate of renal clearance of penicillin
   c. Effectiveness of tetracyclines is reduced by concurrent ingestion of antacids or dairy products
   d. Broad spectrum antibiotics enhance the action of coumarin anticoagulants because of the reduction of Vitamin K sources
   e. Antibiotics such as ampicillin decrease the effectiveness of oral contraceptives due to suppression of normal GI flora involved in the recycling of active steroids from bile conjugates, leading to more rapid excretion of the steroids from the body
   f. Macrolides such as erythromycin inhit the metabolism of drugs such as seldane, digoxin, etc.

5. More and more questions these days are being asked about antivirals and antifungals, so review

a. Acyclovir: an antiviral used for various forms of herpes

b. Fluconazole or ketoconazole: systemic-acting antifungals useful for treating candidiasis

Frequently asked questions on antibiotics:

5. For treating most oral infections, penicillin V is preferred to penicillin G because penicillin V
   a. Is less allergenic
   b. Is less sensitive to acid degradation
   c. Has a greater gram-negative spectrum
   d. Has a longer duration of action
   e. Is bactericidal, whereas penicillin G is not

   (b) memorization: basically the only difference

6. The sole therapeutic advantage of penicillin V over penicillin G is
   a. Greater resistance to penicillinase
   b. Broader antibacterial spectrum
   c. More reliable oral absorption
   d. Slower renal excretion
   e. None of the above

   (c) reworded version of the above

7. Which of the following penicillins is administered ONLY by deep intramuscular injection?
   a. Ampicillin
   b. Dicloxacillin sodium
   c. Penicillin G procaine
   d. Penicillin V potassium

   (c) Answer is (c)- (a), (b) and (d) are all used orally. Penicillin G is destroyed by acid in the stomach resulting in variable and irregular absorption. Penicillin V is acid stable and available for oral use. Penicillin G procaine is typically given intramuscularly in repository form, yielding a tissue depot from which the drug is absorbed over hours. In this form, it cannot be given IV or subcutaneously.

8 The principal difference among potassium, procaine and benzathine salts of penicillin G is their
   a. Potency
   b. Toxicity
   c. Duration of action
   d. Antibacterial spectrum
   e. Diffusion into the cerebrospinal fluid

   (c) again, just asking you to know something about the various forms of penicillin. Since in most cases you are going to use Pen VK orally, this question is an old one showing its age and probably not likely to appear anymore on board excams

11. Which of the following antibiotics is cross-allergenic with penicillin and should NOT be administered to the penicillin-sensitive patient?
   a. Ampicillin
   b. Erythromycin
   c. Clindamycin
   d. Lincomycin
   e. Tetracycline

   (a) ampicillin sort of sounds like penicillin so it must be the answer

12. Which of the following antibiotics may be cross-allergenic with penicillin?
   a. Neomycin
   b. Cephalexin
   c. Clindamycin
   d. Erythromycin
   e. All of the above

   (b) This is a memorization question, with (b) the correct answer. You have to remember that the cephalosporins (like cephalexin) are chemically related to the penicillins. The others are not chemically related and thus cross-allergenicity is unlikely

13. Which of the following antibiotics shows an incidence of approximately 8% cross-allergenicity with penicillins?
   a. neomycin
   b. cephalexin
   c. bacitracin
   d. vancomycin
   e. tetracycline

   (b) just slightly reworded version of the above question, but with some different alternatives thrown in. Obviously, if you can recognize whther or not a drug is a penicillin or a cephalosporin , and you remember that these are the classes that show cross-allergenicity, then you can handle any rewording of this question.

14. Which of the following groups of antibiotics is related both structurally and by mode of action to the penicillins?
   a. Polymyxins
   b. Cycloserines
   c. Cephalosporins
   d. Chloramphenicols

   (c) see above

13. For the dentist, the most reliable method of detecting a patient's allergy to penicillin is by

a. Injecting penicillin intradermally
b. Taking a thorough medical history
c. Placing a drop of penicillin on the eye
d. Having the patient inhale a penicillin aerosol
e. Injecting a small amount of penicillin intravenously

(b) all of the other methods involve unacceptable risk. Once sensitized, even a small amount can cause an allergic response. Remember, it is not a dose-related response that won't be problematic if you only inject a little bit.

14. Which of the following antibiotics is the substitute of choice for penicillin in the penicillin-sensitive patient?
a. Bacitracin
b. Erythromycin
c. Tetracycline
d. Chloramphenicol

(b) boy, if you haven't heard this a zillion times by now.. None of the alternatives listed would be a problem in terms of cross-allergenicity, but the reason (b) is the right answer is that the spectrum of activity of erthromycin is very similar to penicillin. The others offer a much broader spectrum of coverage than we usually require; always use the drug with the narrowest spectrum possible that includes the microbe in question. ***Standards have now changed such that clindamycin is the drug of choice in this situation. But if they don't include clindamycin, look for erythromycin, or for that matter Azithromycin***

15. Most anaphylactic reactions to penicillin occur
a. When the drug is administered orally
b. In patients who have already experienced an allergic reaction to the drug
c. In patients with a negative skin test to penicillin allergy
d. When the drug is administered parenterally
e. Within minutes after drug administration

i. (a), (b) and (d)
ii. (b), (c) and (d)
iii. (b), (d) and (e)
iv. (b) and (e) only
v. (c), (d) and (e)

(iii) memorize

16. Which of the following penicillins has a broader gram-negative spectrum than penicillin G?
a. Nafcillin
b. Ampicillin
c. Cephalexin
d. Methicillin
e. Penicillin V

(b) that's why it is considered an "extended-spectrum" form of penicillin

17. Which of the following penicillins has the best gram-negative spectrum?
a. Nafcillin
b. Ampicillin
c. Methicillin
d. Penicillin V
e. Phenethicillin

(b) didn't they just ask the same thing in the question above?

18. Which of the following antibiotics should be considered the drug of choice in the treatment of infection caused by a penicillinase-producing staphylococcus?
a. Neomycin
b. Ampicillin
c. Tetracycline
d. Penicillin V
e. Dicloxacillin

(e) that's really the only use for dicloxacillin

19. Oral infections caused by organisms that produce penicillinase should be treated with
a. Ampicillin
b. Dicloxacillin
c. Erythromycin
d. Any of the above
e. Only (a) or (c) above

(b) of those listed only (b) is penicillinase resistant. Ampicillin is an extended spectrum penicillin, and is not penicillinase resistant. Erythromycin shouldn't be affected by penicillinases, since it isn't a peniciilin, but it doesn't work against staph for other reasons.

20. Which of the following antibiotics is LEAST effective against penicillinase-producing microorganisms?
a. Ampicillin
b. Cephalexin
c. Methicillin
d. Clindamycin
e. Erythromycin

(a) same question asked backassward

21. Which of the following is a bactericidal antibiotic used specifically in the treatment of infections caused by *Pseudomonas* species and indole-positive *Proteus* species?
a. Ampicillin
b. Penicillin V
c. Tetracycline
d. Dicloxacillin
e. Carbenicillin

(e) Wow, I bet you didn't think they would ask something like this!. An extended spectrum agent is required. Ampicillin is ineffective, while Pen-V is too narrow in spectrum.

22. Penicillin's effectiveness against rapidly growing cells is primarily due to its effect on
a. Protein synthesis
b. Cell wall synthesis
c. Nucleic acid synthesis
d. Chelation of metal ions
e. Cell membrane permeability

(b) memorize, memorize

23. Chlortetracycline acts by interfering with
a. Cell wall synthesis
b. Nuclear acid synthesis
c. Protein synthesis on bacterial but not mammalian ribosomes

d. Protein synthesis on mammalian but not bacterial ribosomes

(c) that's why it is selectively toxic. Wouldn't you like it if your doctor prescribed a drug for you that did (d)?

24. The probable mechanism of the bacteriostatic action of sulfonamides involves
    a. Disruption of the cell membrane
    b. Coagulation of intracellular proteins
    c. Reduction in oxygen utilization by the cells
    d. Inhibition of metabolism by binding acetyl groups
    e. Competition with para-aminobenzoic acid in folic acid synthesis

    (e) memorize

25. The sulfonamides act by
    a. Suppressing bacterial protein synthesis
    b. Inhibiting the formation of the cytoplasmic bacterial membrane
    c. Inducing the formation of "lethal" bacterial proteins
    d. Inducing a deficiency of folic acid by competition with para-aminobenzoic acid

    (d) same as above worded differently

26. Which antibiotic is able to achieve a higher concentration in bone than in serum?
    a. penicillin
    b. erythromycin
    c. clindamycin
    d. metronidazole
    e. amoxicillin

    (c) that's why it is very useful for treating bone infections such as osteomyelitis. The question might have substituted gingival fluid for bone – that would make the answer tetracycline

27. Tetracycline reduces the effectiveness of concomitantly administered penicillin by
    a. Reducing absorption of penicillin
    b. Increasing metabolism of penicillin
    c. Increasing renal excretion of penicillin
    d. Increasing binding of penicillin to serum proteins
    e. None of the above

    (e) tetracycline is bacteriostatic and would slow the rapid growth of the microbial population that a bactericidal drug such as penicillin needs to be effective, sine only when rapidly dividing are the cells making cell walls

37. The action of which of the following drugs will most likely be impaired by concurrent administration of tetracycline?
    a. Clarithromycin
    b. Erythromycin
    c. Sulfonamide
    d. Penicillin
    e. Lincomycin

    (d) the classic cidal- static interaction! See above, since this is just a reworded version of the same fact

28. Which of the following antibiotics is most likely to cause liver damage?
    a. Streptomycin
    b. Penicillin G
    c. Tetracycline
    d. Cephalosporins
    e. Amphotericin B

    (c) (a) streptomycin can damage the eighth nerve, affecting both balance and hearing, but is not associated with liver damage. (b) other than allergic reactions, penicillins are extremely safe, with no effect on the liver. (d) the cephalosporins are chemically related to the penicillins and share their relatively nontoxic nature. (e) amphotericin B, is an antifungal agent that produces such adverse side effects as nephrotoxicity and hypokalemia, but not liver toxicity. Thus (c) is the correct answer. Tetracyclines have been shown to be hepatotoxic following high doses in pregnant patients with a history of renal disease.

29. Which of the following erythromycins associated with an allergic cholestatic hepatitis?
    a. Erythromycin base
    b. Erythromycin stearate
    c. Erythromycin estolate
    d. Erythromycin succinate

    (c) just because

30. Which of the following antibiotics is LEAST likely to cause superinfection?
    a. Gentamicin
    b. Tetracycline
    c. Penicillin G
    d. Streptomycin
    e. Chloramphenicol

    (c) superinfections are usually seen following the use of broad spectrum agents. Of those listed, all are wide spectrum except Pen-G

31. Gastrointestinal upset and pseudomembranous colitis has been prominently associated with
    a. Nystatin
    b. Cephalexin
    c. Clindamycin
    d. Polymyxin B
    e. Erythromycin

    (c) The only 2 possibilities that produce GI upset are (c) and (e). As for producing colitis, (b) and (c) are associated with this adverse side effect. (c) is the only drug which does both, therefore it's the right answer.

32. Symptoms that may be characterized as allergic manifestations during penicillin therapy are
    a. Deafness, dizziness and acute anemia
    b. Crystalluria, nausea, vomiting and anaphylactic shock
    c. Oliguria, hematuria, bronchoconstriction and cardiovascular collapse
    d. Dermatitis, stomatitis, bronchoconstriction and cardiovascular collapse

    (d)

33. Aplastic anemia is a serious toxic effect that occurs particularly after a course of treatment with which of the following antibiotics?
    a. Penicillin

b. Lincomycin
c. Tetracycline
d. Streptomycin
e. Chloramphenicol

(e) memorize

34. Each of the following is a side effect of prolonged tetracycline hydrochloride therapy EXCEPT:
a. Suprainfection
b. Photosensitivity
c. Vestibular disturbances
d. Discoloration of newly forming teeth
e. Gastrointestinal symptoms (when administered orally)

(c) memorize

36. Colitis that results following clindamycin therapy is caused by an overgrowth of
a. C. dificile
b. Staph aureus
c. Pseudomonas
d. Candida albicans

(a) memorize

Antibiotics, Miscellaneous

37. Which antibiotic is appropriate for premedication in the penicillin allergic patient?
a. Cephalexin
b. Clindamycin
c. Erythromycin
d. Amoxicillin
e. Ampiciilin

(b) clindamycin is the current recommendation. Erythromycin used to be, so if you get a question that doesn't include clindamycin as an answer, look for erythromycin. Cephalexin might be a choice, but there is the issue of cross-allergencicity, and it must certainly be avoided in the anaphylactic patient. Amoxicillin and ampicillin are penicillins!

38. Acyclovir is useful for treating
a. Candidiasis
b. Colitis
c. Herpes simplex
d. HIV
e. ANUG

(c) always think used for herpes as the first answer

38. A distinct advantage that tetracyclines have over penicillins is that tetracyclines
a. Have no side effects
b. Do not cause superinfections
c. Are safer to use during pregnancy
d. Have a wider range of antibacterial activity
e. Produce higher blood levels faster after oral administration

(d) broad spectrum vs. narrow spectrum. Tetracyclines certainly have more side effects than penicillin, and are certainly one of the antibiotics to avoid during pregnancy.

39. Which of the following has the broadest antimicrobial spectrum?
a. Vancomycin (Vancocin)
b. Clindamycin (Cleocin)
c. Erythromycin (Erythrocin)
d. Chlortetracycline (Aureomycin)
e. A third generation cephalosporin

(d) Answer is (d)- remember, tetracyclines are broad spectrum antibiotics effective against both gram-negative and gram-positive cocci and bacilli. Clindamycin has a spectrum of activity similar to erthyromycin and vancomycin, which is less than that of the tetracylines, mainly affecting gram-positive microorganisms. Ist generation cephalosporins are effective against both gram-negative and gram-positive organisms, but the third generation ones have increased activity against gram-negative but greatly decreased activity against gram-positive microorganisms.

40. Sulfonamides and trimethoprim are synergistic bacteriostatic agents because in bacteria they
a. Both inhibit folic acid synthesis
b. Interfere sequentially with folinic acid production
c. Are both antimetabolites of para-aminobenzoic
d. Are both inhibitors of dihydrofolic acid reductase
e. Are both transformed *in vivo* into a single active compound

(b)

41. Which of the following substances is the most effective agent against fungus infections of the mucous membrane?
a. Nystatin ointment
b. Undecylenic acid
c. Polymyxin ointment
d. Saturated magnesium sulfate
e. 10 per cent aluminum chloride solution

(a)

42. The most desirable property of an antibiotic when used to treat an odontogenic infection is
a. Rapid absorption
b. Little allergenicity
c. Ability to achieve and maintain adequate concentrations at the site of infection
d. Lack of significant binding to plasma proteins
e. No effects on drug metabolism

(c) if it can't do this it isn't going to be very effective.

14. Nystatin is of greatest clinical usefulness in treating
a. viral infections
b. fungal infections
c. spirochetal infections
d. Bacterroides infections
e. penicillin resistant gram positive infections

(b) Nystatin is the prototypic antifungal agent, thus (b) is the most obvious 1st choice, and eliminates (a). (d) & (e) require an antibiotic, not an antifungal

42. Which of the following drugs chelates with calcium?

a. Erythromycin
b. Polymyxin B
c. Tetracycline
d. Penicillin G
e. Chloramphenicol

(c)

43. Which of the following is NOT characteristic of tetracycline antibiotics?
a. Absorption is impaired when taken with antacids
b. They predispose to monilial superinfection
c. They form a stable complex with the developing tooth matrix
d. They have a low tendency for sensitization, but a high therapeutic index
e. They are effective substitutes for penicillin prophylaxis against infective endocarditis

*Answer is (e)- Again, the important phrase in the question is **not** (Hey, just Wayne and Garth). Obviously the fact that you will remember about tetracylines is that they can discolor teeth in the fetus when taken by the mother during pregnancy. But don't circle that answer because (a) is also characteristic of tetracyclines (they are the most likely of all the antibiotics to cause superinfection), and is an annoying side effect in adults resulting from alteration of the oral, gastric and intestinal flora. The real answer is (e). Tetracyclines are not the drug of choice for prophylaxis against infective endocarditis. This is due to streptococcal infection. 15-20% of group A streptococci are resistant to tetracyclines, but none are resistant to penicillin or erythromycin. Recently a non-streptococcal induced subacute bacterial endocarditis has been identified, especially in juvenile periodontitis patients. The causative bacterium is not susceptible to penicillin or erythromycin. It may be necessary to treat predisposed patients with tetracycline for a few weeks, and then follow this with a course of penicillin or erythromycin. Remember that these drugs are antagonistic to each other and thus can't be used concurrently. Penicillin is a bactericidal drug which kills or destroys microorganisms by interfering with the synthesis or function of the cell wall, cell membrane or both. Thus it is most effective against bacteria that are multiplying. Tetracycline is a bacteriostatic antibiotic that acts by inhibiting the growth and multiplication of organisms by inhibiting protein synthesis by binding reversibly to the 30 S subunit of the bacterial ribosome. When the two types are given together, their effectiveness is negated or reduced.*

Antibiotics, Drug Interactions

44. The concurrent administration of penicillin G and probenecid results in
a. Increased metabolism of penicillin G.
b. Increased renal excretion of probenecid
c. Decreased renal excretion of penicillin G
d. Decreased bactericidal effect of penicillin G
e. Increased excretion of probenecid in the feces

(c)

71. Interaction between penicillin and probenicid is best described by which of the following mechanisms?
a. competition at the receptor site
b. acceleration of drug biotransformation
c. alteration in the acid-base balance
d. alteration in the rate of renal clearance

Answer is (d)- penicillin is metabolized in the liver, but it rapidly disappears from the blood due to rapid clearance by the kidneys. 90% is excreted by tubular secretion. Thus patients with renal disease will show high blood levels of penicillin. Similarly, probenicid, a uricosuric agent (a drug which tends to enhance the excretion of uric acid by reducing renal tubular transport mechanisms), reduces the renal clearance of penicillins. And you wondered why we had those lectures on pharmacokinetics!

45. When broad-spectrum antibiotics are administered with coumarin anticoagulants, the anticoagulant action may be
a. Reduced because of enhanced hepatic drug metabolism
b. Reduced because of increased protein-binding
c. Increased because of reduction of vitamin K sources
d. Increased because of decreased renal excretion of the anticoagulant

(c)

46. The therapeutic effectiveness of which of the following drugs will be most affected by concomitant ingestion of antacids?
a. Cephalexin
b. Erythromycin
c. Tetracycline
d. Sulfisoxazole
e. Penicillin V

(c) hey, I asked you this on the exam!

47. Erythromycin should be avoided in the patient taking
a. Aspirin
b. Seldane
c. Benadryl
d. Ibuprofen
e. Propranolol

(b) remember the famous erythromycin –Seldane potentially lethal interaction, whereby erythromycin blocks the metabolism of Seldane to its antihistamine metabolite – it stays unmetabolized and causes cardiac arrthymias. Of course this question could have many other options listed, since erythromycin decreases the metabolism of so many other useful drugs, such as digoxin.

Cardiovascular Drugs

This category covers a lot of drugs and a lot of questions. They can be categorized as:

1. Questions asking about which drug from a list might be used to treat a certain condition:

hypertension:

1) Diuretics such as the high ceiling or loop-acting diuretic, furosemide;
2) Beta-blockers such as propranolol or the cardioselective beta blocker metoprololor atenolol
3) Alpha-1 blockers such as prazosin,
4) Centrally acting adrenergic drugs such as methyldopa or clonidine
5) Neuronal blockers such as guanethidine (reserved for severe hypertension)
6) Angiotensin converting enzyme inhibitors such as Captopril, lisinopril

angina: Nitroglycerin, sometimes propranolol, calcium channel blockers such as verapamil

arrhythmias:

1) Lidocaine (ventricular arrhythmias),
2) Phenytoin (to reverse digitalis induced arrhythmias),
3) Quinidine (supraventricular tachyarrhythmias, atrial fibrillation),
4) Verapamil (supraventricular tachyarrhythmias, paroxysmal tachycardia, atrial fibrillation),
5) Digitalis (atrial fibrillation, paroxysmal tachycardia)
6) Propranolol (paroxysmal tachycardia)

Congestive heart failure: Glycosides such as digitalis, digoxin, ACE inhibitors such as captopril

2. The second major category of questions concerns mechanism of action of the various agents:

Antiarrhythmics: Remember problem is that the heart beats irregularity

a. Type 1A agents such as quinidine: acts by increasing the refractory period of cardiac muscle
b. Type 1B agents such as lidocaine decrease cardiac excitability
c. When digitalis is used for atrial fibrillation it acts by decreasing the rate of A-V conduction

Antiangina drugs: problem is insufficient oxygen to meet demands of myocardium

a. Nitroglycerin increases oxygen supply to the heart by a direct vasodilatory action on the smooth muscle in coronary arteries
b. Propranolol reduces oxygen demand by preventing chronotropic responses to endogenous epinephrine, emotions and exercise.
c. Calcium channel blockers decrease oxygen demand by reducing afterload by reducing peripheral resistance via vasodilation

Antihypertensives: Remember, most drugs have the ultimate effect of reducing peripheral resistance via vasodilation

ACE inhibitors: Captopril blocks the enzyme which converts angiotensin I to angiotensin II. The latter is a potent vasoconstrictor (administration of angiotensin will result in an elevation of blood pressure).

Adrenergic Agents:

a. Prazosin: selective alpa-1 blocker, inhibits binding of nerve induced release of NE resulting in vasodilation
b. Methyldopa: acts centrally as a false neurotransmitter stimulating alpha receptors to reduce sympathetic outflow resulting in vasodilation
c. Clonidine: selective agonist stimulates alpha-2receptors in the CNS to reduce sympathetic outflow to peripheral vessels resulting in vasodilation
d. Propranolol: nonselective beta blocker reduces cardiac output and inhibits renin secretion
e. Metoprolol: selective beta-1 blocker, reduces cardiac output

Diuretics: decrease the renal absorption of sodium, thus resulting in fluid loss and a reduction in blood volume. This decreases the work the heart has to pump. Also have weak dilatory action. Types of diuretics which may be mentioned include:

a. Thiazides: chlorothiazide

b. High-ceiling or loop acting: furosemide

c. Potassium sparing: spironolactone

Congestive heart failure drugs:

a. Cardiac glycosides such as digitalis or digitoxin are effective because they have a positive inotropic effect, increasing the force of contraction of the myocardium. This is achieved by an inhibition of Na+, K+ ATPASE leading to increased calcium influx. Digitalis therapy reduces the compensatory changes that are associated with congestive heart failure such as increased heart size, rate, edema, etc.

Drug-condition questions

1. Quinidine is principally used to treat
   a. Hypertension
   b. Angina pectoris
   c. Congestive hear failure
   d. Supraventricular tachyarrhythmias

   (d) by elimination. Hypertension ((a)) is treated primarily with beta blockers such as propranolol. Angina is primarily treated with nitroglycerin, while digoxin (digitalis) is the drug of choice for congestive heart failure. Quinidine is classed as an antiarrthymic drug (Type I-blocks sodium channels). It reduces automaticity and responsiveness and increases refractoriness. It also has an antimuscarinic action preventing the bradycardia that follows vagal stimulation.

2. Quinidine is used to treat
   a. Hypertension
   b. Angina pectoris
   c. Atrial fibrillation
   d. Ventricular fibrillation
   e. Congestive hear failure

   (c) same question as above, just gave you a different type of

arrythmia

3. Verapamil is most efficacious in the treatment of
   a. Atrial fibrillation
   b. Atrial tachycardia
   c. Ventricular tachycardia
   d. Catecholamine-induced arrhythmias

   (a) memorize

4. Which of the following drugs is most useful in treating or preventing angina pectoris?
   a. Digitalis
   b. Quinidine
   c. Propranolol
   d. Procainamide
   e. Pentobarbital

   (c)

5. Each of the following drugs can be used in the prevention and treatment of angina pectoris EXCEPT
   a. Digitalis
   b. Propranolol
   c. Nitroglycerin
   d. Isosorbide dinitrate
   e. Pentaerythritol tetranitrate

   (a)

6. All of the following drugs are useful in the treatment of hypertension EXCEPT
   a. Ephedrine
   b. Reserpine
   c. Methyldopa
   d. Thiazide diuretics

   (a)

7. Digitalis is useful in the treatment of which of the following conditions?
   a. Atrial fibrillation
   b. Congestive heart failure
   c. Paroxysmal atrial tachycardia
   d. All of the above

   (d)

8. All of the following drugs are useful in the treatment of cardiac arrhythmias EXCEPT
   a. Digitalis
   b. Lidocaine
   c. Phenytoin
   d. Procainamide
   e. Aminophylline

   (e)

9. The drug of choice for initial therapy for mild hypertension is
   a. Reserpine
   b. Guanethidine
   c. Phenobarbital
   d. Chlorothiazide
   e. Alpha-methyldopa

   (d)

10. Which of the following antihypertensives are usually reserved for treatment of severe hypertension?
    a. Sedatives and reserpine
    b. Thiazide diuretics and reserpine
    c. Sedatives and thiazide diuretics
    d. Guanethidine and ganglionic blocking agents

    (d)

11. Which of the following beta-adrenergic receptor blocking agents is thought to be cardioselective?
    a. Nadolol
    b. Timolol
    c. Metoprolol
    d. Propranolol

    (c)

Mechanism of Action Questions

Antiarrhythmics

12. Antiarrhythmic drugs, such as quinidine, suppress certain cardiac arrhythmias by
    a. Stimulating the beta-adrenergic receptor
    b. Suppressing cardiac ATP-ase activity
    c. Increasing ectopic pacemaker activity
    d. Increasing the refractory period of cardiac muscle

    (d)

13. Most drugs useful in the treatment of cardiac arrhythmias act primarily by
    a. Blocking Purkinje fibers
    b. Blocking the alpha-adrenergic receptor
    c. Suppressing SA node impulse formation
    d. Causing a positive inotropic effect
    e. Increasing the refractory period of cardiac muscle

    (e)

14. The most important pharmacologic action of drugs that suppress cardiac arrhythmias is
    a. Blockade of the vagus nerve
    b. Stimulation of cardiac ATP-ase activity
    c. Blockade of the Beta-adrenergic receptor
    d. Stimulation of the beta-adrenergic receptor
    e. Increased refractory period of cardiac muscle

    (e)

15. Lidocaine produces its antiarrhythmic effects by
    a. Increasing AV conduction
    b. Decreasing cardiac excitability
    c. Increasing cardiac conduction velocity
    d. Increasing spontaneous pacemaker activity

    (b) arrhythmias are defined as any abnormality of the normal

sinus rhythm of the heart due to disease or injury induced damage to the impulse conducting systems. They also result from the development of ectopic pacemakers or abnormal pacemaker rhythms. Drugs such as lidocaine are used to normalize these rhythms. Lidocaine, a local anesthetic, depresses cardiac excitability, answer (b). The refractory period of cardiac muscle is increased, thus slowing the heart down. All of the other alternatives given would exacerbate the arrhythmia.

16. When digitalis is used in atrial fibrillation, the therapeutic objective is to
    a. Abolish cardiac decompensation
    b. Inhibit vagal impulses to the heart
    c. Decrease the rate of A-V conduction
    d. Increase the rate of cardiac repolarization
    e. Produce a decrease in the rate of atrial contraction

    (c)

Antiangina Drugs

17. Nitroglycerin dilates the coronary arteries in angina pectoris by
    a. Decreasing the heart rate reflexly
    b. Increasing the metabolic work of the myocardium
    c. Direct action on smooth muscle in the vessel walls
    d. Increasing the effective refractory period in the atrium
    e. Blocking beta-adrenergic receptors

    (c)

18. Propranolol is of value in treating angina pectoris because it
    a. Has a direct action on vascular smooth muscle
    b. Blocks autoregulatory mechanisms in the heart
    c. Inhibits oxygen metabolism in cardiac cells
    d. Provides relief within seconds of an acute anginal attack
    e. Prevents chronotropic responses to endogenous epinephrine emotions and exercise

    (e)

ACE Inhibitors

19. Administration of angiotensin results in
    a. Anti-inflammatory effects
    b. Antihistaminic effects
    c. Increased blood pressure
    d. Increased heart rate
    e. A sedative effect

    (c)

20. The primary antihypertensive effect of captopril (Capoten) is due to accumulation of
    a. Serotonin
    b. Angiotensin I
    c. Angiotensin III
    d. Bradykinin metabolites

    (b) Captopril is an angiotension-converting enzyme inhibitor that blocks the activation of angiotension I to angiotension II. The decreased blood concentration of angiotension II reduces blood pressure, because angiotension II is a potent vasoconstrictor. Thus (c) is wrong, accumulation of angiotension I is the usual effect. Captopril also maintains lowered BP by elevating bradykinin (which has potent vasodilatory action) in the blood by blocking its metabolism. Thus (d) is wrong, bradykinin metabolites do not accumulate.

21. Administration of angiotensin results in
    a. A sedative effect
    b. Increased heart rate
    c. Increased blood pressure
    d. Antihistaminic effects
    e. Anti-inflammatory effects

    (c) I guess because more angiotensin II would be formed, and that is a potent vasoconstrictor

Mechanism of Action

Diuretics

22. Which of the following is NOT characteristic of the thiazide diuretics?
    a. Increase renal excretion of sodium and chloride
    b. Increase renal excretion of potassium
    c. Increase the toxicity of digitalis
    d. Exacerbate existing diabetes
    e. Cause hypokalemia
    f. Cause hypoglycemia

    (f) first off, how can you have an option (f)?! (a) is how diuretics lower BP, (b) is why they can cause hypokalemia, which is conveniently option (e), and hypokalemia can potentiate digitalis induced arrythmias option(c). Theyy apparently can also cause hyperglycemia, which would relate to option (d). How the heck are you supposed to remember all of this?

23. The most useful diuretic drugs act by
    a. Increasing the glomerular filtration rate
    b. Decreasing the renal reabsorption of sodium
    c. Decreasing the renal excretion of chloride
    d. Increasing the renal reabsorption of potassium
    e. Increasing the secretion of antidiuretic hormone

    (b) people with high BP are always told to reduce salt intake, since high sodium levels cause fluid retention which can increase BP, so ipso facto, reducing renal reabsorption of sodium makes BP go down

24. Which of the following drugs act by inhibiting renal reabsorption of sodium?
    a. Urea
    b. Chlorothiazide
    c. Theophylline
    d. digitalis glycosides
    e. Procainamide

    (b) same question as above, just reversed.

Cardiac Glycosides

25. Digoxin exerts its positive inotropic effect by
    a. Activation of adenylcyclase
    b. Inhibition of phosphodiesterase
    c. An agonist effect of beta-receptors

d. Inhibition of Na+, K+ ATPASE leading to increased calcium influx
e. Decreasing the amount of calcium available for excitation-contraction coupling

Answer is (d)- Remember, cardiac glycosides such as digoxin are used in the treatment of congestive heart failure, which is the failure of the heart to function adequately as a pump and thus maintain an adequate circulation. Cardiac glycosides are thought to act by altering calcium ion movement, with a desired effect of increasing the force of contraction of the myocardium (e.g. the inotropic effect). While several of the alternatives involve calcium, the way digoxin does it is via (d), inhibition of Na+, K+ ATPase, resulting in an increase of calcium ion influx into the cardiac cells, and a subsequent enhancement of the contractile mechanism. (a) is the way epinephrine works.

26. Digitoxin is effective in the treatment of cardiac failure because it
a. Is primarily a diuretic
b. Reduces the ventricular rate
c. Decreases abnormal cardiac rhythms
d. Produces peripheral vasoconstriction
e. Has a positive cardiac inotropic action

(e)

27. The primary action of therapeutic doses of digitalis on cardiac muscle is an increase in
a. Force of contraction
b. Ventricular excitability
c. Refractory period of the atrial muscle
d. Refractory period of the ventricular muscle
e. Rate of conduction of impulse to the muscle

(a)

28. The beneficial effects of digitalis in congestive heart failure result in part from the fact that digitalis causes
a. A decrease in end-diastolic volume
b. A decrease in end-diastolic pressure
c. An increase in stroke volume and cardiac output
d. A decrease in central venous pressure
e. A decrease in rate of the hear where tachycardia exists

i. (a), (b) and (c)
ii. (a) and (c) only
iii. (c) and (d)
iv. (e) only
v. All of the above

(v)

29. The cardiac glycosides will increase the concentration of which ion in an active heart muscle?
a. Sodium
b. Bromide
c. Calcium
d. Chloride
e. Potassium

(c)

30. Which of the following ions augments the inotropic effect of digitalis?
a. Sodium
b. Lithium
c. Calcium
d. Chloride
e. Magnesium

(c)

31. In the treatment of congestive heart failure, digitalis glycosides generally decrease all of the following EXCEPT
a. Edema
b. Urine flow
c. Heart size
d. Heart rate
e. Residual diastolic volume

(b)

Adrenergic Agents

32. The mechanism of action of prazosin, an antihypertensive agent is to
a. Block beta-adrenergic receptors
b. Inhibit formation of angiotensin II
c. Inhibit nerve-induced release of norepinephrine
d. Stimulate central inhibitory alpha-adrenergic receptors
e. Inhibit the postsynaptic action of norepinephrine on vascular smooth muscle

(e)

33. Which of the following owes a significant amount of its antihypertensive effect to a central action?
a. Methyldopa
b. Metoprolol
c. Hydralazine
d. Propranolol
e. Guanethidine

(a) All of these drugs are used to treat hypertension, but act by different mechanisms. (a), methyldopa, is the drug with central action- it alters CNS control of blood pressure by acting on cardioregulatory and vasomotor systems of the brain by stimulating alpha2 receptors in the brain stem. Clonidine is the usual drug that is involved in this particular question. (b) metropolol is a selectively blocks beta-1 receptors in the heart to reduce cardiac output. (c) hydralazine has a direct action on vascular smooth muscle to reduce hypertension via vasodilation. (d) propranolol blocks beta receptors in the heart, while (e) guanethidine prevents the release and causes depletion of catecholamines taken up into storage vesicles and is released like a false transmitter. It does not cross the blood-brain barrier.

34. Which of the following drugs is thought to reduce arterial blood pressure by activating alpha receptors in the vasomotor center of the medulla?
a. Prazosin
b. Clonidine
c. Propranolol
d. Guanethidine
e. Chlorothiazide

(b)- see above explanation

35. Propranolol (Inderal) can be useful in the treatment of hypertension because it blocks
    a. Alpha-1 adrenergic receptors
    b. Sodium reabsorption in the kidney
    c. The release of renin from juxtaglomerular cells
    d. The release of norepinephrine from nerve terminals
    e. The reflex tachycardia seen with the use of other antihypertensives

        i. (a) and (b)
        ii. (a) and (d)
        iii. (b), (c) and (d)
        iv. (c), (d) and (e)
        v. (c) and (e) only

    (v) Answer is (v)- You should immediately recognize that propranolol is the prototypic beta-adrenergic receptor blocker, thus any answer with alternative a (i and ii) is wrong. Similarly, d is wrong as well-propranolol is a competitive beta- receptor blocker- it has no effect on NE release. Another drug used for hypertension, Clonidine, acts via this mechanism by stimulating alpha-2 autoreceptors. Thus ii, iii, and iv are wrong. This leaves (v) as the only possible right answer. Indeed, aside from blocking beta-1 receptors, blocking of renin release is thought to be the other mechanism whereby beta-blockers alter hypertension.

36. One of the proposed mechanisms of the antihypertensive effect of beta-adrenergic receptor blocking agents is
    a. Sedation
    b. A diuretic effect
    c. An antirenin effect
    d. A vagal blocking effect
    e. An increase in cardiac output

    (c)

37. Selective beta-1 adrenergic agonists will produce which of the following effects?
    a. Glycogenolysis
    b. Increased cardiac output
    c. Decreased diastolic pressure
    d. Decreased peripheral resistance
    e. Relaxation of bronchial smooth muscle

    (b)

Miscellaneous Side Effect Questions

38. Ototoxicity with deafness may encountered occasionally in patients taking which of the following diuretic agents?
    a. Osmotic
    b. Thiazide
    c. Mercurial
    d. High-ceiling

    (d) answer is (d)- straight memorization- deafness is typically associated with use of ethacrynic acid, a loop or high-ceiling diuretic. How the hell are you supposed to remember all of this stuff???

39. Symptoms of digitalis toxicity include all of the following EXCEPT
    a. Extrasystoles
    b. Nausea and vomiting
    c. Yellow-green vision
    d. A-V conduction block
    e. Decreased P-R interval

    (e)

40. Administration of which of the following drugs increases the likelihood of a toxic response to digitalis?
    a. Diazepam
    b. Lidocaine
    c. Spironolactone
    d. Chlorothiazide
    e. Acetylsalicylic acid

    (d) Chlorthiazide is a diuretic which causes potassium loss or hypokalemia. This results in greater penetration of digitalis into the myocardium, and thus potential toxicity.

41.

Analgesics- NSAIDS:

1. Mechanism of action questions regarding analgesic, antipyretic and effects on bleeding:

   Analgesic effects: aspirin inhibits the synthesis of prostaglandins

   Antipyretic effects: aspirin inhibits PG synthesis in the hypothalamic temperature regulation center

   Bleeding time: inhibit synthesis of thromboxane A2 preventing platelet synthesis

2. A 2nd type of question has to do with pharmacological or toxic effects of aspirin: you get to pick which of the list is or is not associated with aspirin. Therapeutic effects of aspirin include pain relief, antipyretic effects, antirheumatic and anti-inflammatory effects. Adverse or toxic effects include all of the following: occult bleeding from the GI tract, tinnitus, nausea and vomiting, acid-base disturbance or metabolic acidosis, decreased tubular reabsorption of uric acid, salicylism, delirium, hyperventilation, etc.

3. A third type of question focuses on the difference between 1) aspirin and acetaminophen, 2) aspirin and other anti-inflammatories like prednisone, and 3) between aspirin and ibuprofen:

   1) Acetaminophen lacks anti-inflammatory activity, is hepatotoxic, and does not cause GI upset
   2) Anti-inflammatories like prednisone, hydrocortisone, triamcinolone etc. are steroids and do not act primarily by PG inhibition
   3) Ibuprofen causes much less GI irritation
   4) Diflunisal (Dolobid) has a longer half-life than aspirin, acetaminophen and ibuprofen

4. Newer versions of the boards have questions about COX-2 inhibitors like vioxx. (Which of the following is a COX-2 inhibitor?)

5. These old questions focus a lot on aspirin. Nowadays, acetaminophen and ibuprofen are used much more commonly than aspirin, because of the many side effects of aspirin that turn up in these kinds of questions. So since aspirin is the comparator prototype drug, reviewing these questions are still useful.

1) But expect newer questions asking you to know:

a. Acetaminophen causes liver toxicity, especially when combined with alcohol or taken in excess of 4 gr/day.

b. Acetaminophen is the drug of choice for the feverish child (they usually ask the reverse, which is which drug should be avoided in the feverish child (aspirin- increased risk of Reye's syndrome)

Frequently asked questions on NSAIDS

4. The therapeutic effect of the salicylates is explained on the basis of the ability of the drug to
   a. Activate autonomic reflexes
   b. Uncouple oxidative phosphorylation
   c. Inhibit the synthesis of prostaglandins
   d. Competitively antagonize prostaglandins at the receptor site

(c)

5. The mechanism of the antipyretic action of salicylates probably results from
   a. Inhibition of prostaglandin synthesis in the CNS affecting hypothalamic temperature regulation
   b. Inhibition of bradykinin in the periphery leading to sweating
   c. Depression of oxidative enzymes leading to decreased heat production
   d. Suppression of cholinergic mediators in the hypothalamus
   e. Stimulation of norepinephrine in the hypothalamus

(a)

6. The antipyretic action of salicylates is explained in part by
   a. Analgesia leading to sedation
   b. Increased blood flow through the hypothalamus
   c. Cutaneous vasodilation leading to increased heat loss
   d. Depression of oxidative processes leading to decreased heat production

(c)

7. The locus of action of aspirin's central antipyretic effect is the
   a. Brain stem
   b. Hypothalamus
   c. Basal ganglia
   d. Limbic system
   e. Cerebral cortex

(b) memorization question- remember antipyresis means antifever. Temperature regulation center is in the hypothalamus.

8. A patient who has been taking large quantities of aspirin might show increased postoperative bleeding because aspirin inhibits
   a. Synthesis of thromboxane A2 and prevents platelet aggregation
   b. Synthesis of prostacyclin and prevents platelet aggregation
   c. Synthesis of prostaglandin and prevents production of blood platelets
   d. Thrombin and prevents formation of the fibrin network
   e. G.I. absorption of vitamin K and prevents synthesis of blood clotting factors

(a) The first fact you must remember is that aspirin prevents platelet aggregation- this limits your choices to (a) and (b). They hope to confuse you by using prostacylin, but of course you know that this is wrong immediately, the right word is prostaglandin, as in (c), but you have already eliminated that choice because it doesn't mention prevention of platelet aggregation. Thus, even if you didn't remember that thromboxane A2 induces platelet aggregation, and aspirin blocks this action, you could get the answer by elimination. (d) is how heparin works, while (e) is how coumarin works.

9. Anti-inflammatory agents, such as aspirin, interfere with hemostasis by
   a. Activating antithrombin
   b. Preventing vasoconstriction
   c. Inhibiting thrombin generation
   d. Inhibiting platelet aggregation
   e. Inhibiting polymerization of fibrin

(d)

10. Which of the following anti-inflammatory agents does NOT act primarily by inhibiting activity of prostaglandin synthetase?
    a. Diflunisal
    b. Ibuprofen
    c. Triamcinolone
    d. Oxyphenbutazone
    e. Acetylsalicylic acid

    (c) triamcinolone is a corticosteroid. Corticosteroids inhibit phospholipase A2, the enzymatic step that precedes prostaglandin synthetase. Diflunisal is a salicylate analgesic, like aspirin.

11. A nonsteroidal, anti-inflammatory agent that appears to produce fewer gastrointestinal disturbances than high does of aspirin is
    a. Ibuprofen
    b. Probenecid
    c. Pentazocine
    d. Acetaminophen
    e. Phenylbutazone

    (a) you might be tempted to answer acetaminophen, because it doesn't cause GI upset, but remember it is also not anti-inflammatory. The answer is ibuprofen. Tricky – you had to sort through two distinguishing characteristics. Good question!

12. Prolonged use of which of the following drugs does NOT cause a predisposition to gastric irritation and bleeding?
    a. Phenytoin
    b. Ibuprofen
    c. Indomethacin
    d. Phenylbutazone
    e. Acetylsalicylic acid

    (a) This is a straight drug identification question. Answers 2-5 are all non-steroidal antiinflammatory drugs which cause gastric irritation and bleeding due to their effects on prostaglandin synthesis in the mucosal wall of the gut. # , phenytoin, is an anti-convulsant-its major side effect that often appears as a question on boards is the production of gingival hyperplasia.

13. Each of the following agents has been associated with gastric irritation EXCEPT
    a. Aspirin
    b. Alcohol
    c. Ibuprofen
    d. Indomethacin
    e. Acetaminophen

    (e) note the difference in this question and #11 and 12. Ibuprofen was previously the answer to "shows reduced GI irritation", but it does cause some, which you have to remember to answer #12 and this question. So aspirin and ibuprofen are out. Indomethacin is a very strong NSAID that causes lots of GI irritation, so much that use is limited in humans, so it is out. What about alocohol vs. acetaminophen. Well, you should really know that acetaminophen is usually the answer to these types of analgesics questions, but if you didn't know that, perhaps you may know that alcohol also causes GI irritation, so it is out.

14. Which of the following is NOT produced by excessive doses of acetylsalicylic acid?
    a. Delirium
    b. Tinnitus
    c. Hypothermia
    d. Hyperventilation
    e. Metabolic acidosis

    (c) it only lowers your temperature if you have a fever,, taking aspirin does not have any effect on body temperature in the non-feverish patient, but high doses can cause all the other effects listed.

15. All of the following are pharmacologic and toxicologic properties of aspirin EXCEPT
    a. Tinnitus
    b. Analgesia
    c. Salicylism
    d. Antipyresis
    e. Suppression of the immune response

    (e)

16. Therapeutic effects of aspirin include
    a. Analgesia
    b. Tranquilization
    c. Pyretic action
    d. Anti-inflammatory action
    e. Antirheumatic action

    i. (a), (b) and (c)
    ii. (a), (c) and (d)
    iii. (a), (d) and (e)
    iv. (b), (c) and (d)
    v. (b), (d) and (e)

    (iii)

17. All of the following are pharmacologic or toxicologic properties of acetylsalicylic acid EXCEPT
    a. Tinnitus
    b. Analgesia
    c. Antipyresis
    d. Methemoglobinemia
    e. Inhibition of prostaglandin synthesis

    (d)

18. All of the following are possible effects of aspirin EXCEPT
    a. Reduction of fever
    b. Shortening of bleeding time
    c. Suppression of inflammatory response
    d. Bleeding from the gastronintestinal tract
    e. Increase in the renal excretion of uric acid at high doses

    (e)

19. Of the following, aspirin does NOT cause
    a. Occult bleeding
    b. Nausea and vomiting
    c. Acid-base disturbance
    d. Suppression of the cough reflex
    e. Decreased tubular reabsorption of uric acid

    (d) Answer is (d)- (a) & (b) are the major side effects of aspirin

(resulting from the inhibition of prostaglandin synthesis) for the majority of people, and one reason for the popularity of aspirin alternatives such as acetaminophen and ibuprofen, which produce these effects to a lesser extent. # 3 & 5 may also be seen following larger doses of aspirin. (d) is not seen with aspirin, but is a major therapeutic use of narcotic opiates such as codeine. I guess they are hoping that you will get the effects of codeine and aspirin mixed up, since the two are often compared and contrasted as moderate pain relievers.

20. Which of the following is NOT true about acetaminophen?
    a. Is a non-prescription drug
    b. Is cross-allergenic with aspirin
    c. Possesses both analgesic and antipyretic effects
    d. May induce methemoglobinemia at high doses
    e. May be the pharmacologically active form of acetophenetidin (phenacetin)

(b) because it ain't a salicylate, but the other statements are true

21. Which of the following is NOT true regarding acetaminophen?
    a. It has antipyretic properties
    b. It may induce methemoglobinemia
    c. It can be combined with codeine
    d. It has anti-inflammatory properties
    e. It is not cross-allergenic with aspirin

(d) reworded version of the preceding question, just asking what you know about acetaminophen. ***Nowadays, I think they would also put something about liver damage in the question, as below.***

22. The most prominent acute toxic effect associated with acetaminophen use is
    a. Hemorrhage
    b. Renal necrosis
    c. Hepatic necrosis
    d. Gastric ulceration
    e. Respiratory alkalosis

(c) Remember, acetaminophen (tylenol) is an aspirin alternative. Alternatives 1, 4, 5 are side effects of aspirin-type drugs. The popularity of acetaminophen as an aspirin alternative is because the incident of such effects with this drug is very low. However, because acetaminophen can undergo biotransformation to a toxic intermediate, hepatic and renal necrosis have been reported, especially after very high doses. (c), hepatic necrosis is the most prominent, especially when combined with alcohol consumption, since the alcohol induces the liver enzymes which make the hepatotoxic metabolites of acetaminophen

23. Which of the following anti-inflammatory agents does NOT act primarily by inhibiting the activity of cyclooxygenase?
    a. Ibuprofen
    b. Diflunisal
    c. Prednisone
    d. Indomethacin
    e. Phenylbutazone

Answer is (c)- (a), 2, 4, and 5 are NSAIDS that reduce inflammation by reducing prostaglandin synthesis by blocking the activity of cyclooxygenase. Prednisone is a corticosteroid. Corticosteroids are potent nonspecific inhibitors of the inflammatory process, acting at a variety of point throughout the inflammatory process. Although they do reduce prostaglandin production as well, they do this by a mechanism other then blocking cyclooxygenase, probably by inhibiting the release of the fatty acid substrate for prostaglandin synthesis.

24. Which of the following is the most appropriate drug to use to lower fever in a child under 12?
    a. Aspirin
    b. Ibuprofen
    c. Acetaminophen
    d. Salicylate
    e. Diflunisal

(c) acetaminophen is the best choice. Aspirin is contraindicated due to the potential for causing Reye's syndrome. Ibuprofen is approved, but usually not the #1 choice. The others would be inappropriate as well.

25. Which analgesic from the following list has the longest half-life?
    a. Acetaminophen
    b. Aspirin
    c. Diflunisal (Dolobid)
    d. Ibuprofen

(c) Diflunisal can be taken twice a day, the others three-four times a day is required.

Analgesics - Morphine

1. One of the most frequently asked questions concerns mixed-agonist-anatagonists (MAA) - they ask you to identify which drugs out of a list of 5 is an MAA. The one they usually expect you to know is pentazocine, but sometimes nalbuphine. ***Since these drugs have proved to be not very popular with patients, they have fallen out of use, so I would imagine that they don't ask somany questions on these drugs anymore.***

2. Additional drug identifications they always ask involve knowing that naloxone is an antagonist used to treat overdose, and that methadone is used in detoxification of morphine addicts.

3. Some questions give you a list of pharmacological effects and ask you to identify which is not an effect of morphine. Morphine produces respiratory depression, euphoria, sedation, dysphoria, analgesia, and constipation and urinary retention. The substitution they often make is diarrhea for constipation, or perhaps diuresis for urinary retention.

4. The last most frequently asked type of question regarding opiates concerns overdose of toxicity. In overdose morphine causes coma, miosis, and respiratory depression. Sometimes they ask the mechanism of respiratory depression: loss of sensitivity of the medullary respiratory center to carbon dioxide.

Frequently asked questions on morphine:

5. Occurrence of which of the following is LEAST characteristic of narcotic ingestion?
    a. Vomiting
    b. Diarrhea
    c. Urinary retention
    d. Bronchiolar constriction
    e. Increase in intracranial pressure

(b) Again, the key word is least. Narcotics, in the form of paregoric (tincture of opium), and Lomotil (loperamide) are over the counter oral preparations for the treatment of diarrhea. Opiates act on receptors in the gut to produce constipation. Thus (b) is obviously wrong. All of the other answers are side effects of opiate administration.

6. Therapeutic doses of morphine administered intramuscularly may produce
   a. Constipation
   b. Euphoria
   c. Dysphoria
   d. Mental clouding
   e. Decreased response to pain

   i. (a) and (b) only
   ii. (a), (b) and (d)
   iii. (a), (d) and (e)
   iv. (c), (d) and (e)
   v. All of the above

   (v) memorize

8. Which of the following are pharmacologic effects of morphine?
   a. Respiratory depression
   b. Euphoria
   c. Sedation
   d. Constipation
   e. Dysphoria

   i. (a), (b) and (c)
   ii. (a), (b) and (d)
   iii. (a) and (e)
   iv. (c), (d) and (e)
   v. All of the above

   (v) same question as above,, just reworded

7. Which of the following drugs acts to suppress the cough reflex?
   a. ASA
   b. Codeine
   c. Meperidine
   d. Acetaminophen
   e. Phenyibutazone

   (b) the only drugs that do this are opioids, and codeine and merperidine are the two opioids on the list. Of the two, codeine is much better at this than meperidine.

8. Morphine binds to which site to produce analgesia?
   a. By binding to specific receptors in the CNS
   b. By decreasing the influx of sodium
   c. By decreasing the synthesis of prostaglandins
   d. By decreasing nerve activatin at the site of injury

   (a) They might reword the question in a way that asks you to remember that the specific receptors are the mu receptors.

9. Morphine causes vomiting by
   a. A direct irritant action on the gastric mucosa
   b. Stimulation of the nodose ganglion of the vagus nerve
   c. Stimulation of the medullary chemoreceptor trigger zone
   d. Direct stimulation of the gastrointestinal musculature

   (c) is there such a thing as the nodose ganglion? Oh my god, I just googled it and there really such a thing – what a funny name – all these years I thought they just made this up. So it seems they want you to think that the emetic response to morphine is locally activated,, but actually it is an effect produced by morphine acting on the CTZ in the medulla.

10. The decrease in ventilation caused by morphine, meperidine and some of the related opioids depends chiefly upon
    a. Depression of cortical activity
    b. Peripheral blockade of chemoreceptor impulses
    c. An increase in carbon dioxide concentration in the blood
    d. Blockade of afferent autonomic impulses from the lungs
    e. Loss of sensitivity of the medullary respiratory center to carbon dioxide

    (e) memorization. Don't be fooled by (a)- these drugs are sedating, but this has nothing to do with the decrease in respiratory rate.

11. Small doses of barbiturates and morphine depress respiration primarily by
    a. A parasympathominetic action
    b. Inhibiting the Herine-Bueuer reflex
    c. Rendering the aortic chemoreceptor system insensitive to O2
    d. Rendering the respiratory centerin the brain stem less sensitive to changes in CO2
    e. A specific effect at myoneural junctions of phrenic and intercostal nerves

    (d) I told you this in my lecture on morphine-if you miss this you'll hurt my feelings.

12. Which of the following are pathognomonic symptoms of narcotic overdose?
    a. Miosis, coma and depressed respiration
    b. Mydriasis, coma and smooth muscle spasms
    c. Mydriasis, coma and depressed respiration
    d. Miosis, convulsions and depressed respiration
    e. Mydriasis, convulsions and depressed respiration

    (a) pinpoint pupils (miosis) and respiratory depression are hallmark opioid overdose effects, so the only distractor is coma or convulsions. If you can't breathe too well, I guess you might go into a coma!

13. The cause of death with opioid intoxication is
    a. Oxygen apnea
    b. Cardiac arrest
    c. Terminal convulsions
    d. Circulatory collapse
    e. Respiratory depression

    (e) - again, a memorization question. What happens is that opioids decrease the response of respiratory centers in the brainstem to the carbon dioxide tension of the blood, and also depresses pontine and medullary centers regulating respiratory frequency. Opioids do not cause oxygen apnea, ((a)), they can be convulsive, but not terminally so ((c)), they are stabilizing on the heart and some are actually used in open-heart surgery ((b)), and they do not cause circulatory collapse ((d)).

14. Which of the following is an opioid that has both agonistic and antagonistic activities?
    a. Codeine
    b. Methadone
    c. Naloxone
    d. Meperidine
    e. Pentazocine

    (e) This is an example of the type of question where the drug class is given. You are asked to not only identify a drug from the list as being from this class, but additionally that it has the properties that are given in the question that distinguish it from the other drugs of the class that are listed as alternatives. In this example, there is only one drug which meets this criterion. All are drugs which act via opiate receptors, but 3 are agonists ((a), (b), (d)), 1 is an antagonist only ((c)). (e) pentazocine is the only drug which has both types of action, and is the one drug left by the process of elimination.

15. A heroin-dependent patient should NOT be given nalbuphine (Nubain) for pain because
    a. It has no analgesic properties
    b. It may produce respiratory depression
    c. As a mixed agonist-antagonist, it can elicit withdrawal symptoms
    d. The high abuse potential of nalbuphine may add to the patient's problems

    (c) see above

16. A patient while not currently taking drugs has a history (6 months ago) of narcotic dependency. Which of the following analgesics should be avoided in this patient?
    a. Aspirin
    b. Pentazcine
    c. propoxyphene
    d. Indomethacin
    e. Acetaminophen
    f. None of the above

    (b) see above

17. Which of the following statements does NOT characterize pentazocine?
    a. It is equianalgesic with codeine
    b. It is a partial opioid antagonist
    c. Its abuse potential is less than that of heroin
    d. It may induce dysphoria and mental aberrations
    e. It is effective only on parenteral administration

    (e) lot of memorization required here for a drug that isn't used that much. I guess it was big news when these questions were written many years ago and they seemed hopeful, since statement (c) was true and was therapeutically an advantage, but it soon became apparent that (d) was also true

18. The antagonist of choice in the treatment of opioid overdosage is
    a. Naloxone
    b. Nalorphine
    c. Pentazocine
    d. Levallorphan
    e. Propoxyphene

    (a) nalorphine and pentazocine are mixed agonist-antagonists, levallorphan is an opioid agonist, as is propoxyphene

19. Which of the following is a complete antagonist of the opioid receptor and the agent of choice in the treatment of narcotic overdose?
    a. Naloxone
    b. Nalorphine
    c. Cyclazocine
    d. Levallorphan
    e. None of the above

    (a) reworded version of the preceding question

20. Methadone is used in detoxification (drug withdrawal) of patients physically dependent on morphine because methadone
    a. Precipitates withdrawal reactions
    b. Antagonizes the depressant actions of morphine
    c. Will not in itself produce physical dependence
    d. Withdrawal reactions are less intense and stressful than those of morphine

    (d) This is an example of the kind of question that requires that you have memorized a fact about a particular drug, in this case the fact is (d). Methadone you will remember is not an antagonist like naloxone- it is a full agonist with analgesic properties, just like morphine. When taken orally it is not euphoric in addicts, but acts just like morphine to produce tolerance and physical dependence. Withdrawal is less severe than with morphine because methadone has a much longer half life. Facts 1, 2, and 3 would be met by an antagonist such as naloxone, or perhaps even a mixed agonist-antagonist such as pentazocine.

21. Which of the following drugs is currently widely used in treating opioid-dependent individuals?
    a. Codeine
    b. Methadone
    c. Alphaprodine
    d. Pentazocine
    e. Meperidine

    (b) shortened version of the above question

22. Meperidine (Demerol) is
    a. An antidepressant
    b. An opioid analgesic
    c. A sedative
    d. A long-acting local anesthetic
    e. An antipsychotic

    (b)

Autonomics:

Cholinergics

1. Drug identification type questions that involve mechanism of action. You need to know the following types of facts:
    a. atropine, scopolamine, propantheline are competitive muscarinic receptor blockers which sometimes are used to control salivary secretions. An additional fact that often gets asked about atropine has to do with the fact that it blocks vagal reflexive

control of heart rate, resulting in tachycardia.
b. physostigmine and neostigmine are reversible anticholinesterases that differ in that physostigmine acts both centrally and peripherally, neostigmine only peripherally, but neostigmine also has some direct ACh like activity at the neuromuscular junction, in addition to prolonging the activity of endogenous ACh. They sometimes see use in treating xerostomia.
c. pilocarpine, methacholine, etc. are direct acting cholinergic agonists. May be used for xerostomia.
d. organophosphates and insecticides irreversibly inhibit cholinesterase
e. pralidoxime is an enzyme regenerator used in organophosphate toxicity
f. succinylcholine is a depolarizing neuromuscular junction blocker, subject to rapid inactivation by plasma pseudocholinesterase: it is used to prevent laryngospasm
g. d-tubocurarine is a non-depolarizing neuromuscular junction blocker
h. mecamylamine and hexamethonium are ganglionic blockers that produce orthostatic hypotension

2. The 2nd type of question has to do with physiological effects of cholinergic stimulation, blockade, or overdose toxicity situations (and what drug you would give to reverse the toxicity).
a. cholinergic crisis symptoms: bradycardia, lacrimation, salivation, voluntary muscle weakness, diarrhea, bronchoconstriction -treat by giving atropine
b. scopolamine overdose: disorientation, confusion, hallucinations, burning dry mouth, hyperthermia: treat with physostigmine
c. An additional fact that often gets asked about atropine has to do with the fact that it blocks vagal reflexive control of heart rate, resulting in tachycardia.

Frequently asked questions about Cholinergics:

Identification and mechanism of action questions:

1. Atropine and propantheline exert their effects on peripheral structures by
a. preventing release of acetylcholine
b. preventing synthesis of acetylcholine
c. enhancing destruction of acetylcholine
d. competeing with acetylcholine for receptor sites
e. producing physiologic effects opposite to those of acetylcholine

(d) - (a) is wrong- botulinum toxin does this. (b) is wrong- hemicholinium works this way. (c) is wrong- ACh is broken down almost instantaneously, so it is almost impossible to enhance its destruction. (e) is wrong-these drugs don't have any actions of their own, they just prevent ACh effects by blocking receptors: atropine and propantheline are postganglionic muscarinic receptor blockers-thus the answer is (d).

2. Neostigmine produces its effect by
a. depressing acetylcholinesterase release
b. inhibiting acetylcholinesterase activity
c. increasing the rate of acetylcholine synthesis
d. acting like acetylcholine at ganglionic sites
e. increasing the amount of acetylcholine released from nerve terminals

(b) – neostigmine is a cholinesterase inhibitor like physostigmine. These differ from the insecticides and nerve gases listed below in that they are reversible and can be used clinically; the latter are irreversible.

3. Organophosphate insecticides and nerve gases inhibit the action of which of the following enzymes?
a. adenylate cyclase
b. monoamine oxidase
c. phosphodiesterase
d. acetylcholinesterase
e. carbonic anhydrase

(d) memory – you and the bugs die from too much cholinergic stimulation

4. Drugs which are additive with or potentiate the effects of acetylcholine include

(a) methacholine
(b) scopolamine
(c) pralidoxime
(d) neostigmine
(e) pilocarpine

1. (a), (b), and (c)
2. (a), (c), and (d)
3. (a), (d) and (e)
4. (b), (d), and (e)
5. (c), (d) and (e)

(3) drugs which potentiate cholinergic stimulation can do so by being either direct acting cholinergic agonists, acting on the cholinergic receptor, or by indirectly increasing the duration of action Ach by preventing its enzymatic degradation. Methacholine and pilocarpine are direct-acting cholinergic agonists, whereas neostigmine acts indirectly. Scopolamine is a muscarinic antagonist like atropine, and will reduce or block cholinergic action via direct receptor antagonism. Pralidoxime is a chemical antidote used to regenerate AchE after nerve gas or insecticide exposure.

5. Which of the following drugs is best to administer after poisoning by an organophosphate cholinesterase inhibitor?
a. atropine
b. phenytoin
c. pralidoxime
d. propantheline
e. phenobarbital

(c) see above –this question must date back to world war II! What you need in this situation is a chemical that will help regenerate AchE that has been bound irreversibly by the organophosphate insecticide or nerve gas. Pralidoxime does just that – it knocks the insecticide off the enzyme, allowing it to again be able to break down Ach. As for the other alternatives: atropine is a competitive muscarinic cholinergic receptor blocker, phenytoin is an anticonvulsant, propantheline isProBanthine, an synthetic atropine like drug used to dry salivation and as an antispasmodic agent, and Phenobarbital is a barbiturate anticonvulsant. Maybe theylisted the the anticonvulsants to throw you off since you think that some one might experience convulsions after organophosphate exposure

6. Which of the following compounds is a ganglionic blocking agent?
a. curarine

b. edrophonium
c. mecamylamine
d. succinylcholine
e. gallamine triethodide

(c) this has got to be a really old question – ganglionic blockers, being so unspecific in their action (they would have both anticholinergic and antiadrenergic action) aren't used clinically anymore, so I wouldn't think they would still ask you about them, but if you must know… mecamylamine, hexamethonium, etc. are ganglionic blockers. Curarine is a nicotinic receptor blocker that causes muscular paralysis, edrophonium is an anticholinesterase used to treat myasthenia gravis, succinylcholine is a depolarizing neuromuscular junction blocker used for short term paralysis, gallamine is another long acting neuromuscular junction blocker for paralysis. Remember, these paralysis producing drugs all act via nicotinic receptor at the neuromuscular junction – some , like curare, are competitive receptor blockers, while the other class, like succinylcholine, are called depolarizing blockers – they don't themselves block the nicotinic receptor from being stimulated b y curare, but stimulate the recptor so much that it depolarizes, and while in this state it cannot be stimulated by the Ach and thus paralysis results.

7. Which of the following acts by antagonizing cholinesterase?
   a. atropine
   b. muscarine
   c. neostigmine
   d. pilocarpine
   e. acetylcholine

(c) this is the reverse of question 2 in the preceding column

8. When neostigmine is administered before acetylcholine, the action of acetylcholine will be
   a. blocked
   b. enhanced and prolonged
   c. less intense and of shorter duration
   d. none of the above. The action of acetylcholine is not affected by neostigmine

(b) the action will be prolonged because neostigmine prevents its breakdown by AchE! Its action would be blocked by atropine or scopolamine

9. Neostigmine can stimulate denervated skeletal muscle because it
   a. is a congener of acetylcholine
   b. is a competitive blocking agent
   c. has no effect on acetylcholinesterase
   d. is more potent than diisopropylfluorophosphate
   e. is capable of acting directly on the end-plate

(e)a strange and amazing truth! Denerevated means the skeletal muscle is not receiving neural input, and thus stimulating a cholinergic neuron to release Ach which would then stimulate the NMJ can't happen. But if you inject a drug which can stimulate the nicotinic receptors directly then you can see an effect on the muscle. Neostigmine is one of those anticholinesterases that can act like Ach at nicotinic receptors, in addition to prolonging the action of Ach itself by blocking the acetylcholinesterase that is trying to break the Ach down.

10. Which of the following is used to prevent laryngospasm?
    a. atropine
    b. epinephrine
    c. diazepam (Valium)
    d. neostigmine (Prostigmine)
    e. succinylcholine (Anectine)

(e) What is needed is a skeletal muscle relaxant. This requires a drug that acts at the neuromuscular junction. Of those listed, only succinylcholine (e) is in this category. (a) atropine is a cholinergic (Muscarinic) receptor blocker, (b) epinephrine is an adrenergic agonist, and (c) diazepam is a benzodiazepine, and (d) is an anticholinesterase.

11. In treating xerostomia, which of the following might be prescribed?
    a. atropine
    b. ephedrine
    c. neostigmine
    d. scopolamine
    e. mecamylamine

(c) salivation is typically considered to be a cholinergic response. Xerostomia is too little saliva and thus one could use a cholinergic agonist to stimulate more saliva secretion (assuming there is functional salivary gland tissue, which may not be the case in patients that have been subject to radiation therapy!) From the list, only neostigmine would produce a cholinergic effect, since it is an indirect acting cholinergic agonist. Of the others, atropine and scopolamine are cholinergic antagonists, and are actually used to reduce salivation, mecamylamine is a ganglionic blocker (not the action we desire, too non-specific, and a blocker at that!). Ephedrine is a mixed acting adrenergic agonist.

12. Which of the following drugs is most likely to dry secretions in the oral cavity?
    a. diazepam
    b. promethazine
    c. physostigmine
    d. propantheline
    e. diphenhydramine

(d) I guess since Dentists deal with so much spit, they think this kind of question should be on every test! Again, if you wish to reduce salivation, you want to give a drug that has an anticholinergic action. The usual suspects, atropine and scopolamine are not in this list, so it's not so easy. So what can you remove by elimination? Well, that depends that you know what some of the other drugs are, doesn't it? Diazepam is valium, and while it will reduce your patient's anxiety, it doesn't do much to make them stop salivating – might even make it worse like the barbiturate sedatives do! Promethazine is an antihistamine used for IM sedation. Looks to me so far that they have thrown in a lot of drugs used in a sedation context, and expect you to know which one is in the mix to control salivation. Physostigmine is an anticholinesteras – why is that in this list? Diphenhydramine is Benadryl, a very sedating antihistamine that is used as a sedation agent. In a previous question I discussed propantheline as Pro-Banthine, a synthetic atropine type drug – so this would work to dry up the oral cavity. What I don't like about this question is that the antihistamines are also anticholinergic and will have a drying action. I guess the distinction from propantheline is that it doesn't cause sedation, so is more selective for just drying up excessive salivation – so if you want only that effect and not the sedative action as well, a drug like propantheline is better.

13. The most useful drug to induce salivation is one which has properties that are

a. adrenergic
b. cholinergic
c. ganglionic blocking
d. adrenergic blocking
e. cholinergic blocking

(b) well, this is just the opposite way of asking what was asked in the other questions given here! If anticholinergic agents are useful to reduce salivation, only an idiot couldn't figure out that a cholinergic agent would be useful to induce salivation!

14. Drugs that are commonly used in the control of excessive salivation include
(a) meprobamate
(b) atropine
(c) methantheline
(d) codeine
(e) chlorpromazine

1. (a) and (b) only
2. (a), (b), and (c)
3. (b) and (c) only
4. (b), (c), and (d)
5. (d) and (e)

(3) you are probably thinking, who the hell ever heard of meprobamate and methantheline, or for that matter, chlorpromazine! Well, these are drugs that may have been useful in the olden days, but would be probably replace in this type of questions by more modern equivalents. But of course you should figure out that atropine (b), being the prototype anticholinergic drug has to be one of the answers. So option 5 has to bee incorrect, since it does not include atropine. Now only the most corrupt dentist would prescribe codeine to reduce salivation, so #4 should also be incorrect – that leaves 1, 2, or 3. So see, you didn't even have to recognize that chlorpromazine is an antipsychotic drug. So what is meprobamate – if we can eliminate that one then we are down to only option 3 as a possible answer. Meprobamate happens to be an antianxiety, skeletal muscle relaxant drug sometimes used by dentists to treat muscle spasms associated with TMD – also has use for external sphincter spasticity – imagine! But it doesn't seem to have anticholinergic activity that is significant enough to cause significant reduction of saliva. Methantheline, in contrast, is Banthine, a synthetic version of atropine! So option 3, atropine and methantheline are the drugs for this purpose.

Questions regarding physiological actions of cholinergic drugs:

<u>OK, here's an outline of cholinergic stimulation effects:</u>
Eye: miosis and reduction of intraocular pressure
CV: bradycardia; vasodilation (but only from injected cholinergic agents, since the muscarinic receptors on the vascualr smooth muscle has no neural input)
GI tract: increased spasmodic activity, increased salivation and acid secretion (overdose: nausea, vomiting, diarrhea)
Urinary tract: increased urination
Respiratory: bronchoconstriction
Glandular: lacrimation, sweating
Skeletal muscle: tremor and ataxia (overdose: muscle weakness, cramps and fasciculations)

<u>Anticholinergic (antimuscarinic) actions are the reverse of the above</u>:
Eye: mydriasis and loss of accomodation and increase of intraocular pressure
CV: increased heart rate (overdose: tachycardia)
GI tract: decreased spasmodic activity, decreased salivation and acid secretion (overdose:)
Urinary tract: decreased urination
Respiratory: bronchodilation
Glandular: decreased lacrimation, decreased sweating (overdose: hot, dry skin, hyperthermia)
Skeletal muscle: no effects, since they don't act on nicotinic receptors, only muscarinic
CNS: tertiary amines such as atropine get into the brain and cause restlessness, headache, excitement, hallucinations and delirium
Quaternary amines like methantheline and propantheline only have peripheral actions

15. Administration of ganglionic blocking agents will result in
a. miosis
b. diarrhea
c. copious salivation
d. orthostatic hypotension
e. enhanced activity of the parasympathetic nervous system

(d) a ganglionic blocker, since it acts by preventing cACh from stimulating nicotinic receptors at the ganglia level will have both anticholinergic and antiadrenergic effects. Options a, b, c, and e are symptoms of cholinergic stimulation, and thus can't be right. Option (d), the remaining answer, is an antiadrenergic effect, arising from decreases in sympathetic tone to the vasculature

16. Tachycardia in a patient administered with atropine or scopolamine results from
a. release of adrenal catecholamines
b. blockade of vagus nerve activity
c. blockade of the nicotinic cholinergic receptor
d. stimulation of the alpha adrenergic receptor
e. stimulation of the beta adrenergic receptor

(b) - Atropine and scopolamine are muscarinic cholinergic receptor blockers. Just knowing that eliminates all the alternatives except (b). But you should also remember that heart rate is kept under tight reflexive control: any sudden increase in HR usually stimulates baroreceptors to send a signal to the vagus nerve to stimulate the heart to slow it back down. This reflex is cholinergically mediated, and will be blocked by cholinergic blockers such as atropine. Even when given in the absence of higher than normal heart rate, atropine will block the normal cholinergic control over the heart, leaving the sympathetic system in charge with a resulting tachycardia.

17. All of the following are possible effects of cholinomimetic drugs except
a. mydriasis
b. bradycardia
c. increased peristalsis
d. stimulation of sweat glands
e. increased secretion by bronchial glands

(a) - The first thing you have to know is that a cholinomimetic

drug is one that mimics the action of acetylcholine, the endogenous neurotransmitter in the parasympathetic or cholinergic nervous system. The acronym for remembering the effects of cholinergic stimulation is SLUD, or increased salivation, lacrimation, defecation, and urination. The heart is the exception in that activity or heart rate is decreased (bradycardia)- thus since the question asks for an effect which does not occur with cholinergic stimulation, that leaves (a) as the only possibility. Miosis, not mydriasis, occurs with cholinergic stimulation.

18. A paralyzing dose of succinylcholine initially elicits
    a. CNS stimulation
    b. CNS depression
    c. decreased salivation
    d. muscle fasiculation
    e. extrapyramidal reactions

    (d) because succinylcholine (SUX) is an agonist at nicotinic receptors, so the initial response is muscle stimulation. But the NMJ rapidly depolarizes due to the inability of the plasma cholinesterase to break down the SUX, which isjust two molecules of acetylcholine fused together – the other information has no relevance unless you're just stuck thinking "gee, I know that SUX has something to do with autonomics but not sure exactly what!"

19. Based on its known mechanism and sites of action, scopolamine should theoretically be useful in
    (a) treatment of peptic ulcer
    (b) providing euphoria and amnesia prior to surgery
    (c.) relieving bronchoconstriction
    (d) relieving some of the symptoms of Parkinson disease
    (e) visualization of the retina

    1.. (a), (b), (d) and (e)
    2.. (a), (b), and (e) only
    3. (a),and (c)
    4. (b) and (e) only
    5. All of the above

    (5) scopolamine is an anticholinergic drug, and thus will have effects like atropine – effects opposite to those observed from cholinergic stimulation. Remembering SLUD doesn't work here, though, since none of the responses involve SLUD type reactions. Damn! So how do you fihutre this one out? You can memorize the list of therapeutic uses of anticholinergic drugs that I gave you in your syllabus (easiest way, since the list corresponds roughly to the list in this question, so therefore you should be preapared for any other type of question like this.

Questions regarding toxicity of cholinergic drugs:

20. Symptoms of poisoning by an organophosphate insecticide include all of the following except:
    a. skeletal muscle fasiculation
    b. excessive salivation
    c. bronchoconstriction
    d. hot, dry skin
    e. diarrhea

    (d) organophosphates kill you from too much cholinergic stimulation (SLUD). Option (a) is from nicotinic receptor stimulation, (b) and (c) and (e) are also cholinergic stimulation. Option (d) is an atropine, anticholinergic type reaction and thus doesn't fit the pattern of responses given.

21. All of the following symptoms are associated with neostigmine poisoning except
    a. diarrhea
    b. salivation
    c. convulsions
    d. bonchiolar constriction
    e. skeletal muscle paralysis

    (e) basically the same question as the preceding question, they just changed organophosphate insecticides to neostigmine. The difference is that neostigmine is a reversible anticholinesterase, whereas insecticides are irreversible. But again, the question just basically wants you to recognize two things – that neostigmine is an indirect acting cholinergic drug and then know what the symptoms of cholinergic stimulation are. But even if you didn't know those facts you might be able to get if you remember that neostigmine is a drug that is used to reverse the skeletal muscle paralysis produced by drugs of the curare class – the non-depolarizing neuromuscular junction blockers (you did remember this right?)

22. Symptoms of atropine poisoning in man include
    (a) decreased intraocular pressure
    (b) burning dry mouth
    (c) nausea, vomiting and diarrhea
    (d) hyperthermia
    (e) orthostatic hypotension

    1. (a) and (c)
    2. (b) and (d)
    3. (b), (d), and (e)
    4. (d) and (e)
    5. All of the above

    (2) glandular secretions are generally under cholinergic control, so sweating and salivation are greatly reduced by the anticholinergic drug atropine. So if you can't salivate or sweat, you very possible will show what kind of symptoms? How about a burning dry mouth and hyperthemia? Nausea and vomiting are cholinergic overdose, as is orthostatic hypotension.

23. The most likely signs or symptoms of overdosage with atropine are
    a. CNS excitation and tachycardia
    b. intestinal cramps and diarrhea
    c. skin rash and cutaneous itching
    d. ptyalism and increased sweating
    e. constriction of the pupils and blurring of vision

    (a) just when you thought you could get by with the anti-SLUD strategy, they then expect you to remember that atropine overdose causes CNS excitation and tachycardia? Actually, that is one of the interesting diffs between atropine and scopolamine, and the reason that scopolamine is used for sedation, while atropine isn't (b) and (e) are cholinergic stimulation I think, (c) is histamine produced, while I haven't got a clue what "ptyalism" is – do you? But I do know that if atropine causes dry hot skin because it prevents sweating then (d) can't be right!

24. Disorientation, confusion and hallucinations resulting from an overdose of scopolamine are most efficaciuosly treated by administering
    a. atropine
    b. levodopa
    c. acetylcholine
    d. physostigmine

(d) so ya gotta know scopolamine is anticholinergic, so ya need a cholinergic agonist, either direct or indirect to overcome its effects. The options in the list are c and (d). Acetylcholine won't work because it gets broken down way to rapidly by acetylcholinesterase, and thus is useless to inject. Physostigmine will work since it is an indirect acetylcholinesterase.

25. The immediate cause of death from irreversible cholinesterase inhibitors is
    a. shock
    b. convulsion
    c. cardiac arrhythmia
    d. respiratory paralysis
    e. dehydration from vomiting and diarrhea

(d) - while some of these are indeed associated with organophosphate toxicity, the immediate cause of death is due to (d), which results from the stimulation of nicotinic receptors at the neuromuscular junction resulting in paralysis of skeletal muscles.

26. Each of the following is a symptom of cholinergic crisis except
    a. bradycardia
    b. lacrimation
    c. vasoconstriction
    d. extreme salivation
    e. weakness of voluntary muscles

(c) this is an except question, don't miss that word! So looking at the list, we got the S (option d) and the L (option b) from SLUD, so we are left with (a), c, and (e) as possibles. (e) results from cholinergic stimulation of the NMJ, so that can't be it. (a) or bradycardia, can occur from too much cholinergic stimulation of the heart (that's why atropine is useful in surgery, to reverse the bradycardia that sometimes arises. So, by default, vasoconstriction is the exception we are looking for.

27. Succinylcholine is a short-acting neuromuscular junction blocking agent useful for providing a brief paralysis to aid in intubating patients. It is short-acting because
    a. it is subject to rapid metabolism in the liver
    b. it rapidly redistributes away from the NMJ
    c. it is subject to inactivation by plasma esterases
    d. it undergoes rapid inactivation in the GI tract

(c) that's why it is long-acting in patient's that have a deficiency in this enzyme

28. Atropine-like drugs are classed as
    a. anti-adrenergic
    b. cholinomimetic
    c. sympatholytic
    d. anti-cholinergic
    e. sympathomimetic

(d) again – don't you wish you had gotten comfortable with this type of terminology during the course? Imagine how many different drugs they might put into this kind of question! Anticholinergics, because they cause xerostomia, are obviously important to know for dentistry. TCAs, H1 antihistamines, opioid analgesics are all drugs that have potent anticholinergic activity, in addition to the prototypes atropine and scopolamine. All of these might turn up in the kind of question "Which of the following causes xerostomia?"

Adrenergics:

1. Drug identification type questions that involve mechanism of action. You need to know the following types of facts:

    a. Receptor blockers: alpha or beta adrenergic drugs such as prazosin or propranolol act by competitive inhibition of postjunctional adrenergic receptors
    b. Drugs that inhibit the action of adrenergic nerves:
        i. Reserpine: depletes NE by inhibiting reuptake
        ii. Guanethidine: inhibits the release of catecholamines
        iii. Alpha methyldopa: acts centrally as a false neurotransmitter which gets taken up into storage vesicles and released with NE, thus decreasing sympathetic activity
        iv. Clonidine: stimulates alpha2 receptors in CNS with a resulting decrease in sympathetic outflow
    c. Indirect acting sympathomimetic drugs:
        a. amphetamine, tyramine, and ephedrine act by stimulating the release of stored NE
        b. TCAs and cocaine block reuptake
        c. MAOIs block enzymatic destruction

2. Physiological action questions: Many of these questions involve actions of epinephrine in the presence of either an alpha or beta blocker, such as:

    a. "Epinephrine reversal": in the presence of an alpha blocker (usually they give prazosin, but drug such as chlorpromazine may also be given) epi causes decrease in blood pressure rather than increase because beta mediated vasodilation predominates
    b. vagal reflex: injection of a pressor dose of NE may result in decreased heart rate due to activation of baroreceptors which stimulate vagal reflex to reduce heart rate. Vagal reflex is blocked by atropine

Thus you must be familiar with the effects of alpha or beta receptor stimulation or block. The most important ones to remember are:

    a. Alpha-1 receptor stimulation: vasoconstriction, urinary retention, mydriasis
    b. beta receptor stimulation: increased heart rate (B1), bronchodilation (B2), vasodilation (B2)
    c. Alpha-1 block: vasodilation
    d. beta block: decreased heart rate (B1), bronchoconstriction (B2)

3. They usually throw in a question regarding the use of levodopa in the treatment of Parkinson's: remember, Parkinson's is a result of DA deficiency in brain. Remedy is to increase DA in brain. Injected DA doesn't cross BBB, but levodopa, a precursor to DA does cross BBB. Carbidopa is given with levodopa to block dopa decarboxylase activity in periphery, which in the absence of carbidopa, converts the levodopa to DA in the periphery, decreasing the amount of levodopa that ends up in the brain. You also need to remember that levodopa is sympathomimetic, and will produce sympathetic stimulation in the periphery. Development of abnormal facial movement, nausea and vomiting, cardiac arrthymias, and

mental disturbances are all associated with levodopa therapy.

Adrenergics - Mechanism of Action

4. Alpha or beta-adrenergic blocking drugs act by
   a. Inhibiting synthesis of norepinephrine.
   b. Increasing the metabolism of norepinephrine
   c. Competitive inhibition of postjunctional adrenergic receptors
   d. A local anesthetic effect on the adrenergic nerve terminal
   e. Depleting norepinephrine from adrenergic nerve terminals

   (c) don't you just love the way they obfuscate? Why can't they just say the block the receptors, then everybody could get it right! But then, I don't even feel that any of the other alternatives should even tempt you, although it all depends on your interpretation of what "blocking" means. In my mind, blocking means a neurotransmitter has actually been released, and now you want a drug that somehow blocks its activity. Taken this way, options (a) and(e) wouldn't work, since, although they would reduce sympathetic activity, or have a "sympatholytic" action, they don't actually block a neurotransmitter. Option (b) again is a sympatholytic action, but I don't really think there any clinically relevant drugs that work this way. Option (d) they just made up to fool you, since you guys work with local anesthetics, so you might be tempted to jump at this answer.

5. A mechanism for the antiadrenergic action of guanethidine is
   a. Inhibition of dopa decarboxylase
   b. Increased rate of metabolism of norepinephrine
   c. Depletion of norepinephrine from the nerve terminals
   d. Substitution for norepinephrine and subsequent action as a false transmitter
   e. Uncoupling of the action potential from the norepinephrine release mechanism

   (e) again, why can't they just say guanethidine reduces NE release! You might see these same options listed for several other drugs that could be substituted

6. Which of the following statements most accurately describes the effectiveness of action of methyldopa?
   a. It causes marked cardiac slowing
   b. It directly relaxes vascular smooth muscle
   c. It causes rapid depletion of norepinephrine from adrenergic nerve terminals
   d. It causes formation of a false transmitter that is released at vascular smooth muscle
   e. It produces a false transmitter, the effect of which is primarily at central nuclei

   (e) see, didn't I just tell you in the previous question that they could just change the drug and use some of the same definitions? But they are tricky, since they think you will speed reading the options, see "false" transmitter, and jump at option (d), and not read further to see that option (e) is the correct answer. Drugs like methyldopa and clonidine are the only centrally acting antihypertensives, reducing sympathetic outflow via alpha-1 agonist action.

7. The mechanism of action of reserpine is to
   a. Inhibit monoamine oxidase
   b. Inhibit catechol-O-methyltransferase
   c. Block the passage of the nerve action potential in the postganglionic nerve fibers
   d. Stabilize the axon terminal membrane thus preventing release of norepinephrine

   (d) I would be majorly surprised if they still ask questions about this drug since it hasn't been used clinically in the last 30 yrs, but (d) is the answer, making reserpine an example of an indirect-acting sympatholytic.

8. Amphetamine acts by
   a. Promoting storage of the mediator
   b. Causing a rapid release of the mediator
   c. Causing a slow depletion of the mediator
   d. Combining with a receptor substance on the effector cell
   e. Interfering with the response of the receptor to the mediator

   (b) amphetamine is one of the many indirect-acting sympathomimetics. It acts by causing the release of neurotransmitter.Just as a review: cocaine: reuptake inhibition and release, TCA antidepressant:, reuptake inhibition, ephedrine:causes release but also acts at receptor itself, MAOIs: block NT degradation.

9. Which of the following characterizes the mechanism of action on levodopa?
   a. It acts through a direct anticholinergic action
   b. It stimulates specific L-dopa receptors in basal ganglia
   c. It replenishes the otherwise deficient dopamine in patients with parkinsonism
   d. It increases concentrations of norepinephrine in the brain to counterbalance an otherwise overactive cholinergic system

   (c) this you just gotta memorize. Option (d) is true except for the fact that the imbalance is between dopamine and too much cholinergic activity, not norepinephrine. Since levodopa has dopa in its name, you could just narrow the question down to options (b) and (c). But you still have to know, Parkinson's is due to a deficiency in dopamine. So the treatment is to restore DA levels.

10. Which of the following combinations of agents would be necessary to block the cardiovascular effects produced by the injection of a sympathomimetic drug?
    a. Atropine and prazosin
    b. Atropine and propranolol
    c. Prazosin and propranolol
    d. Phenoxybenzamine and curare
    e. Amphetamine and propranolol

    (c) - The injection of a sympathomimetic (e.g. a drug that acts like NE) stimulates both alpha and beta-receptors. Alpha-receptor stimulation produces vasoconstriction, increased systolic and diastolic pressure and reflex tachycardia. Alpha and beta-receptor blockers can block these effects. the only alternative that lists both an alpha (prazosin) and beta (propranolol) blocker is (c). Atropine is a muscarinic (cholinergic) receptor blocker that would accelerate the heart, the opposite effect that you want, thus (a) and 2 are wrong. Phenoxybenzamine is an alpha-blocker, but curare is a nicotinic receptor blocker, not beta receptor- thus (d) is wrong. As for # 5, amphetamine is an indirect acting sympathomimetic, not a blocker, thus (e) is wrong.

11. Which of the following drugs competitively blocks the action of norepinephrine at beta-adrenergic receptors?
    a. Atropine
    b. Naloxone

c. Propranolol
d. Phentolamine
e. Hexamethonium

(c) picky memorization! They are all receptor blockers: atropine is muscarinic receptor blocker, naloxone is opioid receptor blocker, phentolamine is a non-specific alpha-blocker, and hexamethonium is a ganglionic blocker. Propranolol, is the only beta-blocker listed.

12. Pretreatment with reserpine prevents a response to which of the following agents?
    a. Amphetamine
    b. Epinephrine
    c. Phenylephrine
    d. Isoproterenol
    d. Norepinephrine

    (a) reserpine causes depletion of NE from storage sites, thus it cannot be released by amphetamine. All of the others listed act postsynaptically. Since this drug is no longer used clinically, I would be very surprised if they asked such a question! Wait, I think I said that a few questions ago!

13. Each of the following drugs is considered to be a direct-acting catecholamine EXCEPT
    a. Epinephrine
    b. Amphetamine
    c. Isoproterenol
    d. Norepinephrine

    (b) see question #8

Adrenergics - Physiological Effects

Okay, here's a quick review of the effects of adrenergic stimulation:

Alpha-1 agonists: increased smooth muscle tone, so vasoconstriction leading to increased blood pressure
Alpha-2 agonists: given orally they cause hypotension by reducing sympathetic outflow from the CNS
Beta-1 stimulation: increased cardiac rate and force of contraction, thus positive inotropic and chronotropic actions
Beta-2 agonists: dilation of skeletal muscle blood vessels and bronchi or relaxation of bronchiolar smooth muscle

Or to organize it another way:
Eye: mydriasis or relaxation of cilary muscle
Heart: acceleration, increased contractility
Vascular smooth muscle: vasoconstriction
Skeletal muscle vessels: relaxation or dilation
Bronchiolar smooth muscle: relaxation/bronchodilation
Sweat glands: sweating

14. After pretreatment with phentolamine, intravenous administration of epinephrine should result in
    a. Relaxation of bronchial smooth muscle
    b. Positive chronotropic and inotropic effects
    c. Splanchnic vasoconstriction
    d. Dilation of skeletal muscle vascular beds
    e. Secretion of a mucoid viscous saliva

    i. a, and b only
    ii. a, b and d
    iii. a, d and e
    iv c and d only
    v. c, d and e

    (ii) phentolamine is an alpha blocker, thus the epinephrine will stimulate beta receptors primarily, with the effects listed in option (ii): relaxation of bronchiolar smooth muscle (Beta-1), skeletal muscle vessel dilation (beta-2), and positiver chronotropic and inotropic action on the heart (b)

15. Which of the following changes produced by intravenous administration of epinephrine result from stimulation of beta-adrenergic receptors?
    a. Respiratory inhibition
    b. Cardiac acceleration
    c. Dilation of the pupil
    d. Increased systolic pressure
    e. Decreased diastolic pressure

    i. a, b and c
    ii. a, b and d
    iii. b and e
    iv. c, d and e
    v. c and e only

    (iv)well, beta receptor stimulation means stimulation of beta-1 receptors in the cardiac muscle,which will increase systolic bP, and beta-2 stimulation which will dilate vessels going to the liver and skeletal muscle, producing a decrease in diastolic BP. Mydriasis or papillary dilation is also a beta receptor response, so c is also right. While you might be tempted by "cardiac acceleration", none of the options including (b) work.

16. Which of the following is NOT an action of epinephrine when administered intravenously in a high dose?
    a. Increases liver glycogenolysis
    b. Causes bronchiolar constriction
    c. Produces a rise in blood pressure
    d. Evokes extrasystoles in the heart
    e. Produces restlessness and anxiety

    (b)all of the above are actions of epinephrine except bronchiolar constriction. Epinephrine would cause bronchodilation – that is why it is used to treat acute brochospasm. So option (b) has to be the exception, because it is just obviously wrong!

17. "Epinephrine reversal" of blood pressure can best be demonstrated by injecting epinephrine intravenously after pretreatment with
    a. Prazosin
    b. Atropine
    c. Propranolol
    d. Neostigmine
    e. Isoproterenol

    (a) epinephrine is a potent stimulator of both alpha and beta receptors. Injection of epi usually causes a rise in blood pressure due to 1) myocardial stimulation that increases ventricular contraction, 2) an increase in heart rate, and most important, 3) vasoconstriction due to alpha receptor stimulation. However, blood flow to skeletal muscles is increased due to powerful beta-2 receptor vasodilator action that is only partially counterbalanced by a vasoconstrictor action on the alpha receptors that are also present in the vascular bed. When given in the presence of an alpha blocker, beta-receptor mediated vasodilation is more pronounced, the total peripheral resistance is decreased and the mean blood pressure falls. This decrease

in blood pressure is called "epinephrine reversal". The only alpha-blocker listed is prazosin, answer (a). Atropine is a cholinergic muscarinic receptor blocker, propranolol is a beta-blocker, neostigmine is a cholinesterase inhibitor, and isoproterenol is a predominately beta receptor agonist.

18. Each of the following is a predictable adverse effect of drugs that block the sympathetic nervous system EXCEPT
    a. Gastrointestinal disturbance
    b. Postural hypotension
    c. Nasal congestion
    d. Urinary retention
    e. Miosis

19. Injection of a pressor dose of norepinephrine may result in a decreased heart rate because of
    a. Activation of baroreceptor reflexes
    b. Direct stimulation of alpha receptors
    c. Direct stimulation of beta-1 receptors
    d. Direct stimulation of beta-2receptors
    e. Direct stimulation of muscarinic receptors

    (a)- alternatives 2-4 all increase heart rate, while NE has no effect at muscarinic receptors (e), which are specific for cholinergic drugs.

20. Alpha-adrenergic agonists are used in combination with local anesthetics to
    a. Stimulate myocardial contraction
    b. Reduce vascular absorption of the local anesthetic
    c. Increase the rate of liver metabolism of the local anesthetic
    d. Increase the concentration of the local anesthetic at its receptor site
    e. Antagonize the vasodilating effects of the local anesthetic

        i. (a), (b) and (c)
        ii. (b), (c) and (d)
        iii. (b), (d) and (e)
        iv. (c), (d) and (e)
        v. (d) and (e) only

    (iii) Answer is (c)- Alpha-adrenergic agonists such as epinephrine produce vasoconstriction, which would accomplish both "b" and "e". # 3 is the only alternative that includes both b and e thus you don't have to know anything else. "a" and "c" are false. Vasoconstrictors are include in local anesthetic preparations to (1) prolong and increase the depth of anesthesia by retaining the anesthetic in the area injected, (2) reduce the toxic effect of the drug by delaying its absorption into the general circulation and (3) to render the area of injection less hemorrhagic.

21. Administration of an otherwise effective pressor dose of epinephrine could cause an "epinephrine reversal" in a patient taking which of the following drugs?
    a. Reserpine
    b. Propranolol
    c. Amphetamine
    d. Chlorpromazine
    e. Lithium carbonate

(d) CPZ is a potent alpha blocker like prazosin.

22. Of the following sympathomimetic agents, the most potent bronchodilator is
    a. Amphetamine
    b. Norepinephrine
    c. Phenylephrine
    d. Isoproterenol
    e. Methoxamine

    (d) What is needed for bronchodilation is relaxation of bronchial smooth muscles. This is accomplished with beta2 receptor stimulation. Isoproterenol is the only drug listed with potent beta2 action. (a) stimulates alpha receptors in the CNS, (b) NE stimulates alpha and beta1 receptors more than beta2, (c) phenylephrine is an alpha receptor agonist, while (e) methoxamine is a vasoconstrictor that stimulates alpha receptors preferentially.

23. Administration of which of the following drugs would produce vasoconstriction of the gingival vessels?
    a. Levonordefrin
    b. Phentolamine
    c. Epinephrine
    d. Propranolol
    e. Phenylephrine

        i. (a) and (b)
        ii. (a) and (c) only
        iii. (a), (c) and (e)
        iv. (b), (d) and (e)
        v. (b) and (d) only

    (iii) vasoconstriction is a result of stimulation of the smooth muscle of the peripheral vasculatire, which is an alpha-1 stimulatory action. Of the options, (a), (c) and (e) are alpha-1 agonists. Phentoalmine is a nonselective alpha blocker and thus would cause vasodilation. Propranolol is a non-specific beta-blocker.

24. Carbidopa, a dopa-decarboxylase inhibitor, is often used in the treatment of parkinsonism because it
    a. Potentiates the central action of dopamine
    b. Potentiates the central action of norepinephrine
    c. Decreases the peripheral metabolism of levodopa
    d. Inhibits the peripheral stimulatory fibers from the central nervous system
    e. Increases the permeability of the blood-brain barrier to levodopa

    (c) because that is just what it does!

25. Of the following, one of the most effective treatments currently available in the U.S. for most patients suffering from parkinsonism involves oral administration of
    a. Dopamine
    b. Amantadine
    c. Benztropine
    d. Levodopa alone
    e. Levodopa plus carbidopa

    (e) I guess this was true many years ago when this question was written, but I don't know if this would still be considered to be one of the most effective treatments, but that is beside the point. You should think levodopa here, since the problem with Parkinson's is not enough DA, so you can improve the disease by giving levodopa to increase DA levels in the brain. The problem with giving levosopa by itself is that it is rapidly metabolized in the periphery before it is able to cross the BBB,

However, the levodopa gets rapidly degraded by enzymes unless you block the enzyymatic degradation by giving carbidopa along with the levopdopa.

26. Levodopa therapy for Parkinson disease may result in each of the following effects EXCEPT:
    a. Development of abnormal, involuntary movements, especially in the face
    b. Extreme sensitivity to sympathomimetic drugs
    c. Exacerbation of an acute psychosis
    d. Nausea and vomiting
    e. Extreme sedation

(e) levodopa apparently overstimulates DA receptors in the basal ganglia and can cause visual and auditory hallucinations (option c), dyskinesia (option (a)), mood changes, depression and anxiety. It also can stimulate the emetic center, producing the effects given in (d). That leaves you with a choice between (b) and (e). Well, apparently, one of the first things you should know about levodopa therapy is that some people think that levodopa can sensitize the beta-1 receptors in the heart and this is a contraindication to using a local anesthetic containing epinephrine. So option (e) is all that is left to be the exception. How you are supposed to remember all this stuff about levodopa is way beyond me, but good luck!

27. Adverse effects of levodopa include:
    a. Arrhythmias
    b. Psychotic disturbances
    c. Nausea and vomiting
    d. Abnormal involuntary movements

    i. (a), (b), and (c)
    ii. (a), (b) and (d)
    iii. (a), (c) and (d)
    iv. (b), (c) and (d)
    v. All of the above

(v) Answer is (e)- Levodopa is the primary drug used to treat parkinson's disease, which results from abnormally low levels of dopamine in the brain. Levodopa, or L-DOPA is the direct precursor of dopamine (which can't be used because it doesn't cross the blood brain barrier- L-DOPA does and is converted to DOPA in the brain) is used to increase dopamine in the brain. All of the effects are established side effects of L-DOPA therapy, thus the answer is (e). Abnormal involuntary movements (AIMS) are the most prevalent and troublesome "extrapyramidal" side effects, typically involving the orofacial musculature. Nausea and vomiting are seen during the first phase of therapy but tolerance develops to these effects. The psychotic effects are much less prevalent, seen in only a small percentage of patients. Increased incidence of arrhythmias is also a problem. Levodopa also sensitizes the heart to epinephrine induced arrhythmias.

28. Which of the following drugs would be most likely taken by an asthmatic patient?
    a. Phenylephrine
    b. Albuterol
    c. Pseudoephedrine
    d. Prazosin
    e. Propranolol

(b) an asthmatic patient typically takes a drug that has bronchodilatory effects. For this action, you need a drug that is a beta-2 agonist (remember those charts?). Of the list, albuterol is the only beta-2 agonist listed. Phenylephrine is an alpha-1 agonist used for stuffy noses, pseudoephedrine might have distracted you, since it is a decongestant, prazosin and propranolol are antihypertensives.

General Principles of Drug Action

Remember this section of the course? This is where we defined a bunch of terms that described how drugs interact with receptors to produce their effects. Most of you yawned and thought "What the hell do we need to know this stuff for - just tell us about antibiotics!" Well the ***Great Board Gods*** want you to know some of this stuff - that's why I put it in the course. Your apologies are graciously accepted, as always!

1. A pharmacologic agonist is a chemical substance that
    a. Binds to a specific receptor and produces a response
    b. Elicits a pharmacologic response without binding to a receptor
    c. Possesses the property of affinity but not of intrinsic activity
    d. Exhibits no activity except to oppose the effect of an antagonist

    (a)

2. When comparing drugs with respect to intensity of response, the drug that produces the greatest maximum effect is the one with the highest
    a. Affinity
    b. Potency
    c. Efficacy
    d. Therapeutic index

    (c) obvious

3. If drug has a greater efficacy than drug B, then drug A
    a. Is more toxic than drug B
    b. Has a greater affinity for the receptor than drug B
    c. Has a greater margin of safety than drug B
    d. Is capable of producing a greater maximum effect than drug B

    (d) obvious

4. A drug with a high LD_{50} and a low ED_{50} has a
    a. High therapeutic index and is, therefore, very dangerous
    b. High therapeutic index and is, therefore, relatively safe
    c. Low therapeutic index and is, therefore, very dangerous
    d. Low therapeutic index and is, therefore, relatively safe

    (b) TI= LD50/ED50, never forget

5. The ratio of the median lethal dose (LD_{50}) to the median effective dose (ED_{50}) is the
    a. Morbidity index
    b. Mortality index
    c. Anesthetic ratio
    d. Therapeutic index

    (d) as I said above

6. The therapeutic index of a drug is the ratio of
    a. The effective dose to the toxic dose
    b. Half the toxic dose to half the effective dose
    c. The maximum tolerated dose to the minimum effective dose
    d. The lethal dose for 50% of animals to the effective dose for

50% of animals

(d) reworded version of above

7. The phenomenon in which two drugs produce opposite effects on a physiologic system but do not act at the same receptor site is
   a. Potentiation
   b. Chemical Antagonism
   c. Competitive antagonism
   d. Physiologic antagonism
   e. Noncompetitive antagonism

(d) and you wondered why I made you learn this!

8. Epinephrine antagonizes the effects of histamine by
   a. Preventing the release of histamine
   b. Acting on the central nervous system
   c. Producing physiologic actions opposite to that of histamine
   d. Competitively blocking histamine at the cellular receptor site

(c) epinephrine acts as a physiologic antagonist. It nonspecifically antagonizes histamine by exerting its own distinct effects, for example, vasoconstriction, bronchodilation, and decreased GI motility. It does not reverse the effect of histamine by blocking at a specific receptor ((d)), as do antihistamines. It does not prevent the release of histamine as does a drug such as cromolyn ((a), by preventing mast cell degranulation). Answer (b) is not relevant, while (a) is the mechanism of action for cromolyn, which inhibits mast cell degranulation..

9. Interaction between nitroglycerin and epinephrine is what type of antagonism?
   a. Allosteric
   b. Physiologic
   c. Biochemical
   d. Competitive
   e. Pharmacologic

(b) epinephrine would stimulate alpha adrenergic receptors to produce vasoconstriction, whereas nitroglycerin relaxes vascular smooth muscle. Thus the two drugs would have opposing actions. However, the actions are produced by the drugs acting on different mechanisms; nitroglycerin does not act at alpha receptors as does epinephrine. If it did, the interaction would be competitive. In this case the interaction is via competing physiological effects.

10. When the combined action of two drugs is greater than the sum of their individual actions, this is
    a. Induction
    b. Synergism
    c. Idiosyncrasy
    d. Hypersensitivity
    e. Cumulative action

(b) supposedly why Tylenol #3 was made

12. Which of the following responses is least predictable in occurrence?
    a. Toxicity
    b. Side-effects
    c. Idiosyncrasy
    d. Tachyphylaxis
    e. Therapeutic effects

(c) Idiosyncratic reactions are genetically determined abnormal responses to a drug. They are the most unpredictable in occurrence because the genetically-based difference responsible for such a reaction to a drug may not become evident until the drug is taken for the first time by the patient. Typically, the effect is one of abnormal sensitivity to a drug, such that a therapeutic effect is present at doses much lower than normally used, while the normal dose may result in a toxic reaction. An example is the response to succinylcholine in patients with atypical plasma cholinesterase. These patients don't metabolize succinylcholine at the same rate, and thus show a prolonged drug action and increased sensitivity to the drug. The other alternatives are typically related to the dose of the drug and because the majority of the population do not possess an atypical genetic basis for the response to the drug, the effects are predictable given the dose and knowledge of what the drug does.

13. Idiosyncrasies to drugs are related to
    a. Species
    b. Genetic factors
    c. Age of the patient
    d. All of the above

(b)

14. Two drugs, A and B, have the same mechanism of action. Drug A in a dose of 5 mg. produces the same magnitude of response as drug B in a dose of 500 mg. Which of the following statements is correct?
    a. Drug A is less toxic
    b. Drug A is more efficacious
    c. Drug A is 100 times as potent
    d. Drug A has a shorter duration of action
    e. Drug A is a better drug to use when a maximal response is desired

(c)

15. According to the theory that agonists and antagonists occupy the same receptor site, an effective antagonist should exhibit
    a. High intrinsic activity and high affinity
    b. Low intrinsic activity and low affinity
    c. High intrinsic activity and low affinity
    d. No intrinsic activity and high affinity

(d)

16. All of the following statements are true regarding the occupation theory of drug-receptor interaction EXCEPT:
    a. The affinity of a drug is dependent on its intrinsic activity
    b. The maximum effect of a drug occurs when all receptors are occupied
    c. An antagonist has affinity for the receptor but not intrinsic activity
    d. The magnitude of the effect of a drug is proportional to the number of receptors occupied
    e. It follows the law of mass action

(a)

17. The occupational theory of drug-receptor interaction states that
    a. The magnitude of the drug response is proportional to the number of receptors occupied

b. A partial agonist has intrinsic activity but no affinity for the receptor site
c. An antagonist drug has affinity but no intrinsic activity
d. The rate at which the drug-receptor complex associates and dissociates determines drug efficacy
e. The degree of drug action is dependent on the law of mass action

i. (a), (b) and (c)
ii. (a), (c) and (e)
iii. (b), (c) and (d)
iv. (b) and (e)
v. (c), (d) and (e)

(ii)

Sedatives

This category consists of mostly questions regarding benzodiazepines and barbiturates, and requires you to know the differences between the two types of drugs in terms of mechanism of action, therapeutic actions and adverse side effects and toxicities. Here's a capsule review:

Benzodiazepines:

1. Examples: diazepam, chlordiazepoxide, etc.
2. Mechanism of action: modulate the activity of the inhibitory neurotransmitter, GABA
3. Advantages vs. barbs: less addiction potential, less profound CNS depression, larger therapeutic index, less respiratory depression
4. Other points to remember: A) many BDZs form active metabolites; B) IV injection of diazepam can cause irritation such as thrombophlebitis due to the solvent the BDZ is dissolved in.

Barbiturates:

1. (a) question in occurrence about barbs is always regarding thiopental and redistribution. Remember, thiopental's action is terminated by redistribution of the drug out of the brain-it enters the brain rapidly and exits rapidly, thus quick onset and short duration of action.

2. (b) question wants you to remember that barbs are not analgesic - this is usually in the form of a list of actions and they want you to indicate which is not true.

3. Questions about toxicity -
   a. Barbiturate overdose kills you because of respiratory depression
   b. Barbs are contraindicated in a patient with intermittent porphyria - barbs enhance porphyrin synthesis and thus will aggravate the disease

Sedatives

1. Diazepam is preferred to a barbiturate as an antianxiety agent because diazepam
   a. Produces no sedation
   b. Has less addiction potential
   c. Is a very short-acting drug
   d. Is substantially less expensive
   e. Does not potentiate the action of CNS depressants

   (b)

2. Benzodiazepines produce their antianxiety effects by modulating which of the following neurohumors?
   a. GABA
   b. Glycine
   c. Dopamine
   d. Acetylcholine
   e. Norepinephrine

   (a)

3. Benzodiazepines exert their main effect on
   a. Neuromuscular junctions
   b. Peripheral reflex synapses
   c. Central GABAergic neurons
   d. Central serotonergic neurons
   e. Central adrenergic nerve endings

   (c) Answer is (c)- memorization- BDZ's bind to sites on the GABA receptor.

4. All of the following pertain to general anesthesia induced by thiopental EXCEPT:
   a. Fast induction
   b. Decreased secretions
   c. Low therapeutic index
   d. Short duration of anesthesia
   e. Predisposition to laryngospasm

   (b) Barbiturates are problematic as anesthetics because they often induce excessive salivation and bronchial secretion, usually requiring the use of an anticholinergic drug to be administered to reduce these secretions. Thus (b) has to be the false statement.

31. Which of the following factors contributes to the short duration of action of a single dose of thiopental?
   a. rapid biotransformation
   b. rapid accumulation in body fat
   c. high lipid solubility of the undissociated form
   d. ability to enter and leave the brain tissue rapidly

   (d) is correct- thiopental is the classic example always given of a drug whose duration of action is determined by redistribution away from its site of action in the brain to less well perfused tissues. They leave out the word redistribution from the answer to confuse you -they know this is the way you learned it.

5. Speed of recovery from short-acting anesthesia with thiopental depends chiefly on the rapidity of
   a. Renal tubular secretion
   b. Hepatic degradation of the thiopental group
   c. Redistribution from the brain to skeletal muscle

d. Reverse diffusion across the blood-brain barrier
e. None of the above

(c) reworded version of the question above

6. The action of the ultrashort-acting barbiturates is terminated primarily by the process of
   a. Oxidation
   b. Redistribution
   c. Renal excretion
   d. Plasma protein binding
   e. Conjugation with sulfate

(b) yet another reworded version

7. Which of the following is NOT characteristic of barbiturates?
   a. Possess anticonvulsant properties
   b. Possess significant analgesic properties
   c. Possess serious drug dependence potential
   d. Vary in degree of lipid solubility and hypnotic potency

(b)

8. The cause of death from acute barbiturate poisoning is
   a. Convulsions
   b. Liver damage
   c. Renal failure
   d. Respiratory failure
   e. Cardiovascular depression

(d)

9. Which of the following adverse effects is most commonly associated with administration of an intravenous barbiturate?
   a. Hypotension
   b. Renal failure
   c. Hepatic necrosis
   d. Nausea and vomiting
   e. Respiratory depression

(e)

10. Important steps in the treatment of barbiturate poisoning include:
   a. Maintaining an open airway
   b. Increasing the input of afferent stimuli
   c. Maintaining respiration
   d. Administering a narcotic antagonist
   e. Administering a central nervous system stimulant

   i. (a) and (b) only
   ii. (a), (b), (c) and (e)
   iii. (a) and (c) only
   iv. (b) and (e) only
   v. (d) only

(ii)

11. Which of the following are true regarding barbiturates?
   a. Significantly elevates pain thresholds
   b. Are metabolized by the liver
   c. Are classified according to duration of action
   d. Depress all levels of the CNS
   e. Cause death by cardiovascular depression

   i. (a), (b) and (c)
   ii. (a) and (d)
   iii. (b), (c) and (d)
   iv. (b) and (e)
   v. (c), (d) and (e)

(iii) Answer is (c)- Barbiturates are not analgesics, thus any answer with "a" such as (a) can be eliminated.. "b" is true, thus the answer must contain "b" as one of the alternatives, thus (d) and (e) are eliminated.. "c" is also true, barbiturates are always classified according to duration of action (thiopental- ultra-short acting; phenobarbital-long-acting, etc.). This eliminates # 3 so the answer must be (b). Of course, you should have been able to rapidly eliminate answers (c), 4, and 5 because barbiturates cause death by <u>respiratory</u> depression, not cardiovascular depression.

12. Barbiturates are contraindicated in a dental patient with:
   a. Emphysema
   b. Hypertension
   c. Undiagnosed severe pain
   d. Acute intermittent porphyria

   i. (a), (c) and (d)
   ii. (a) and (d) only
   iii. (b) and (c)
   iv. (b) only

(i) Answer is (a)- "d" is the absolute contraindication for barbiturate use, since these drugs stimulate the synthesis of enzymes involved in the synthesis of porphyrins and thus will aggravate this disease. Thus the answer must contain "d", eliminating (c) and (d). Since both (a) and (b) differ only by alternative c, that is the second fact you must know. Barbiturates are not analgesics, but sedatives. When pain is present, they may even make the pain worse, resulting in arousal, rage and perhaps delirium in the patient. Thus, "c" would seem to be a pretty strong contraindication, making (a) the right answer.

13. If diazepam (Valium) is to be given intravenously, it is recommended that a large vein be used in order to
   a. Hasten the onset of action
   b. Decrease the risk of thrombophlebitis
   c. Offset the vasoconstrictor qualities of diazepam
   d. None of the above

(b) is correct. This is one of the adverse side effects of IV diazepam. None of the other alternatives apply.

14. The most important therapeutic measure to be taken in a case of barbiturate poisoning is to
   a. Alkalinize the urine
   b. Aspirate stomach contents
   c. Administer a CNS stimulant
   d. Assure adequate respiration
   e. Administer osmotic diuretics

(d) barbs can be lethal due to respiratory depression, so keep them breathing!

15. For oral sedation in the dental setting, the most ideal group of agents is:
   a. Narcotics

b. Barbiturates
c. Antihistamines
d. Benzodiazepines
e. Anticholinergics

(d) while all have sedative actions, and have their uses given the appropriate setting, benzodiazepines are by far the most popular now given their safety compared to the other agents. The most popular oral BDZs has been diazepam (Valium), but there is a trend recently to the ultrashort acting versions such as triazolam (Halcion).

16. Which of the following is an advantage of midazolam over diazepam EXCEPT one. Which one is this EXCEPTION?
a. Lesser incidence of thrombophlebitis
b. Shorter elimination half-life
c. No significant active metabolites
d. Less potential for respiratory depression
e. More rapid and predictable onset of action when given intramuscularly

(d) BDZs in general are great for dental sedation precisely because they present less risk of respiratory depression in comparison to barbiturates and opioids. This is an advantage shared across all the BDZs, so if you know this you don't really have to know anything else. The others are true advantages of midazolam vs. diazepam. Midazolam is water soluble, and thus does not need to be dissolved in propylene glycol like valium – it is the solvent that cause the thrombophlebitis seen with valium injections. Midazolam also is short acting compared to valium because it does not have active metabolites like valium. Because of the above factors, statement (d) is also true.

17. Which of the following drugs best reverses the effects of benzodiazepines?
a. Naloxone
b. Flumazenil
c. Midazolam
d. Aminophylline
e. Physostigmine

(b) memorize, pure and simple. The only confusing one should be naloxone, since this is used to reverse the effects of Opioids. Midazolam is a BDZ, aminophylline is a theophylline derivative, and physostigmine is a cholinesterase inhibitor.

Psychopharmacology: Antidepressants and Antipsychotics

1. Antipsychotics questions are usually about phenothiazines and usually about chlorpromazine. **(Since we are now well into the era of 2nd generation antipsychotics, I would imagine that they ask more questions about those than they do about 1st generation drugs. And really, the only 1st generation drug still used to any extent is haloperidol (Haldol), a specific D2 receptor blocker, so focus on those questions**) They ask for:

a. Mechanism of antipsychotic action: blockade of dopaminergic sites in the brain

b. Major side effects: i) anticholinergic effects, ii) extrapyramidal stimulation resulting in tardive dyskinesia

c. 2nd generation drugs like clozapine:

a. Block dopamine receptors but also serotonin (5-HT) receptors

b. Treat negative as well as positive symptoms

c. Fewer extrapyramidal side effects like tardive dyskinesia

2. Antidepressant questions are usually about tricyclic antidepressants such as imipramine or amitriptyline-usually of the type ...which of the following is used to treat depression? ... TCAs are the most commonly used antidepressant medication, but remember that MAO inhibitors such as tranylcypromine or phenylene are also used. 2nd generation drugs are fluoxetine and trazodone. **(2nd generation antidepressants (SSRIs) like fluoxetine (Prozac) are much more commonly used than TCAs or MAOIs, so figure more questions regarding these)** The drug ID the questions may ask for:

a. **Mechanism of action**: blockade of amine reuptake or alterations of receptor number **(SSRIs block serotonin reuptake selectively)**

b. **Side effect**: anticholinergic or atropine side effects

3. The last type of question, again a drug ID asks that you remember that lithium is the drug of choice for the manic phase of manic depression.

Antipsychotics -
Mechanism of Action

1. Which of the following are pharmacologic properties of antipsychotic drugs?
a. They block the dopamine receptor
b. They affect the hypothalamic temperature regulation system
c. They cause emesis
d. They are synergistic with LSD
e. They cause hypertension

i. (a) and (b) only
ii. (a), (b) and (c)
iii. (b), (c) and (d)
iv. (c), (d) and (e)
v. (d) and (e) only

(i) You should immediately know that (a) has to be included as part of the answer, thus i or ii has to be right. If you also can dredge up from the recesses of your memory banks that antipsychotics are also effective antiemetics, and are often used clinically for this purpose, then you realize that (c) is wrong, thus limiting your answer to i.

2. The antipsychotic effects of the phenothiazines are probably the result of
a. Release of serotonin in the brain
b. Release of norepinephrine in the brain
c. Blockade of dopaminergic sites in the brain
d. Prevention of the release of norepinephrine from brain neuron terminals
e. Increase in the dopamine content of the cerebral cortex

(c) updated version of this question might use haloperidol instead of phenothiazines

3. The antipsychotic effects of phenothiazines result from
a. Release of serotonin in the brain
b. Release of norepinephrine in the brain

c. Blockade of dopaminergic sites in the brain
d. An increase in the dopamine content of the cerebral cortex
e. Prevention of the release of norepinephrine from brain neuron terminals

(c)

4. Chlorpromazine and related drugs are thought at act by blocking which of the following receptors?
a. Adrenergic
b. Muscarinic
c. Dopaminergic
d. Central serotonin

(c) Answer is (c)- Chlorpromazine is the prototypic phenothiazine, an antipsychotic drug used in the treatment of schizophrenia. Other antipsychotic drugs used for this purpose are haloperidol and thioridazine. These drugs act via dopaminergic receptors.

Side Effects

5. Which of the following is an irreversible side-effect resulting from long-term administration of phenothiazines?
a. Sedation
b. Xerostomia
c. Infertility
d. Parkinsonism
e. Tardive dyskinesia

(e) all are seen – key word here is irreversible

6. Tardive dyskinesia is a neurological side-effect of which of the following classes of drugs?
a. Alcohols
b. Tricyclic antidepressants
c. Barbiturate antiepileptics
d. Phenothiazine antipsychotics
e. Monoamine oxidase inhibitors

(d) Tardive dyskinesia is an irreversible condition that consists of involuntary movement of skeletal muscles, a condition which may be seen following prolonged use of drugs. This is typically a dopaminergic mediated effect. The phenothiazine antipsychotics are the only drugs listed which act via dopamine. The others, such as tricyclics and MAO inhibitors affect adrenergic transmission, barbiturates act via GABA, as does alcohol.

7. Which of the following drugs are most likely to cause extrapyramidal stimulation?
a. Antibiotics
b. Salicylates
c. Barbiturates
d. Phenothiazines
e. Benzodiazepines

(d) - Extrapyramidal side effects are the major side effects of antipsychotic medication and include Parkinson-like effects as well as tardive dyskinesia (from development of supersensitivity resulting from chronic blockade of dopamine receptors in basal ganglia)- abnormal rapid alternating movements of tongue and perioral areas, facial grimacing, etc. Phenothiazines are the only antipsychotic drugs listed.

8. Phenothiazine derivatives do NOT produce
a. Jaundice
b. Xerostomia
c. Gingival hyperplasia
d. Postural hypotension
e. Symptoms of parkinsonism

(c) Answer is (c)- (c) should stand out immediately because this is almost always mentioned as a side effect of dilantin, which is not a phenothiazine, but an anti-convulsant. Phenothiazine derivatives are antipsychotic drugs such as haloperidol (Halcyon) or chlorpromazine used in the treatment of disorders such as schizophrenia. You should remember that the most troubling side effect of these drugs is the production of tardive dyskinesia and the parkinsonian-like extrapyramidal disorders. Thus # 5 is eliminated. As a rule these drugs have anticholinergic and anti-alpha-adrenergic side effects, which would eliminate xerostomia, and postural hypotension (due to an anti-adrenergic depressant effect on both vasomotor centers and the autonomic nervous system) as possible answers. Jaundice is a less frequent side effect than the extrapyramidal symptoms, and often results from an allergic reaction to these drugs. Thus the answer is (c).

9. All of the following actions are associated with the use of chlorpromazine EXCEPT:
a. Jaundice
b. Photosensitivity
c. Excessive salivation
d. Anticholinergic effects
e. Antiadrenergic effects

(c)

Antidepressants

10. The drug most commonly used to treat severe mental depression is
a. Sodium
b. Imipramine
c. Chlorpromazine
d. Tranylcypromine
e. Dextroamphetamine

(b) this question is obviously from the early 80's; you would see a different list of drugs today, so review your syllabus on antidepressants. Today you would probably see something like Prozac. Tranylcypromine is the only other antidepressant listed but is an MAO inhibitor. These are 2nd choice drugs due to side effects. Chlorpromazine is an antipsychotic used to treat schizophrenia.

11. Which of the following drugs is most effective as an antidepressant?
a. Diazepam
b. Reserpine
c. Amitriptyline
d. Chlorpromazine
e. Hydroxyzine

(c) drug list, drug list

Mechanism of action

12. Which of the following is most likely to be the major mechanism of action of the tricyclic antidepressants?
a. Enhanced release of acetylcholine

b. Inhibition of neuronal synthesis of norepinephrine
c. Potentiation of serotonin synthesis in nervous tissue
d. Stimulation of reuptake of norepinephrine from the synaptic cleft
e. Blockade of the reuptake of amine neurotransmitters released into the synaptic cleft

(e) a long winded way of saying reuptake blockers

Side Effects

13. Tricyclic antidepressants have a prominent side effect that most nearly resembles the usual pharmacological action of
a. Codeine
b. Atropine
c. Ephedrine
d. Propranolol
e. Methacholine

(b) TCAs are strong anticholinergics, and atropine is the only anticholinergic drug listed

Antimanics

14. Which of the following drugs has its primary use in the treatment of the manic phase of depressive psychosis?
a. Lithium
b. Reserpine
c. Imipramine
d. Amphetamine
e. Chlorpromazine

(a)

15. Lithium carbonate is particularly effective in treating
a. Parkinsonism
b. Hypertension
c. Schizophrenia
d. Acute anxiety
e. Manic-depressive psychosis

(e)

16. The current drug of choice for treatment of the manic phase of manic-depressive psychosis is
a. Lithium
b. Caffeine
c. Reserpine
d. Imipramine
e. Amphetamine

(a) three versions of a question asking the same factoid – what if they said "bipolar disorder?"

Anti-inflammatory Drugs:

In general, you need to remember that

1. These are corticosteroids or glucocorticoids and that they suppress the immune system in addition to their antiinflammatory activity. Thus latent infections such as tuberculosis may go systemic or opportunistic infections such as Candidiasis may become more of a problem

2. Side effect profile: gastric ulcers, immunosuppression, acute adrenal insufficiency, osteoporosis, hyperglycemia, redistribution of body fat

Frequently asked questions on anti-inflammatory drugs;

1. Which of the following statements is NOT true regarding the adrenal corticosteroids?
a. Cause retention of sodium and fluid
b. Decrease activity in lymphoid tissue
c. Heighten the immune response to antigens
d. Can produce a diabetes-like syndrome with high blood levels
e. Are therapeutically beneficial when administered orally, parenterally or topically

(c)

2. Which of the following conditions contraindicate use of corticosteroids in a dental patient?
a. AIDS
b. Candidiasis
c. Tuberculosis
d. Peptic ulcers

i. (a), (b) and (c)
ii. (a), (b) and (d)
iii. (b), (c) and (d)
iv. (c) and (d) only
v. All of the above

(v) Answer is (e)- Corticosteroids are antiinflammatory drugs used topically, orally and parenterally. However, they suppress the immune system of the body. They have been known to cause peptic ulcers, as well as mask the symptoms of an ulcer, and perforation and hemorrhage may result. Thus "d" has to be in the answer, eliminating (a). Because they are immunosuppressive, they would obvious make an AIDS patient, who already has a compromised immune system, worse. Similarly, latent tuberculosis could also be reactivated. Thus # 3 and (d) can be eliminated. Use of corticosteroids in inhalers for asthma, while advantageous in reducing the side effects resulting from systemic administration, has led to an increase in problems with Candidiasis, so "b" has to be in the answer. (e) is the only answer that meets all these requirements.

3. Which of the following does NOT result from prolonged treatment with steroids?
a. Gastric ulcer
b. Osteoporosis
c. Hyperglycemia
d. Myocardial atrophy
e. Redistribution of body fat

(d)

4. Glucocorticosteroids are useful as secondary treatment of anaphylaxis because they
a. Inhibit the production of antibodies
b. Prevent the union of antigen with antibody
c. Prevent the release of histamine from sensitized cells
d. Suppress the inflammatory response to cell injury

e. Inhibit the release of serotonin from vascular storage sites

(d) Answer is (d)- Glucocorticoids such as hydrocortisone are classed as antiinflammatories, inhibiting every step of the inflammatory process.- thus (d) is the correct answer. The other alternatives are single steps along the pathway, that are handled by other drugs that are more selective than glucocorticoids.

5. Adrenal steroids are used successfully to treat all of the following conditions EXCEPT
   a. Gastric ulcers
   b. Addison disease
   c. Lupus erythematosus
   d. Rheumatoid arthritis
   e. Aphthous stomatitis

(a) adrenal steroids, otherwise known as corticosteroids, actually cause gastric ulcers! All the rest are therapeutic uses.

General Anesthetics:

1. Questions always come up regarding factors that influence the rate of induction. Remember that onset of anesthesia is inversely proportional to solubility of the anesthetic in the blood. The more soluble the agent is in blood, the more must be given to reach critical tension in the brain.

2. A second set of questions has to do with adverse side effects of various general anesthetics. Halothane is associated with hepatotoxicity.

3. Some questions are based on the progressive depression of CNS function leading to anesthesia that characterized older anesthetic agents. Remember the 4 stages of anesthesia:

   Stage I: analgesia
   Stage II: delirium
   Stage III: surgical anesthesia
   Stage IV: medullary paralysis

Frequently asked questions:

1. Signs and stages of anesthesia are most likely to be seen with a general anesthetic that has a
   a. Low potency
   b. Slow rate of induction
   c. Low Ostwald coefficient
   d. High oil-water solubility coefficient
   e. High tissue-blood partition coefficient

   (b)

2. All of the following influence the rate of induction during anesthesia EXCEPT:
   a. Pulmonary ventilation
   b. Blood supply to the lungs
   c. Hemoglobin content of the blood
   d. Concentration of the anesthetic in the inspired mixture
   e. Solubility of the anesthetic in blood (blood-gas partition coefficient, Ostwald coefficient).

   (c)

3. The rapidity of onset of anesthesia with an inhalation anesthetic agent is primarily related to its
   a. Molecular weight
   b. Degree of blood solubility
   c. Temperature in the gas phrase
   d. Interaction with preoperative drugs

   (b) the rule is, the more insoluble the agent is, the faster the onset and offset of effect. That's why nitrous oxide, which is very insoluble in blood works so fast and leaves the body so quickly once you stop administration.

4. Which of the following forms of drug toxicity is associated with the halogenated hydrocarbon general anesthetics?
   a. Liver damage
   b. Myocardial atrophy
   c. Peripheral neuritis
   d. Severe hypertension

   (a) just because

5. The behavior of patients under general anesthesia suggests that the most resistant part of the central nervous system is the
   a. Spinal cord
   b. Medulla oblongata
   c. Cerebral cortex (motor area)
   d. Cerebral cortex (sensor area)

   (b)

6. General anesthetics can do all of the following EXCEPT:
   a. Produce delirium
   b. Stimulate medullary centers
   c. Produce a state of unconsciousness
   d. Reduce perception of painful stimuli
   e. Decrease excitability of the motor cortex

   (b) maybe cuz it's the base of the brain? They will eventually depress medullary centers (Stage IV), patient will stop breathing and die. (c), (d), and (e) are desirable actions, (a) is stage II of anesthesia

7. In general anesthesia, the last part of the CNS to be depressed is the
   a. Medulla
   b. Cerebrum
   c. Midbrain
   d. Cerebellum
   e. Spinal cord

   (a) that's Stage IV. maybe cuz it's the base of the brain?

49. General anesthesia with halothane is commonly preceded by administration of atropine to
   a. inhibit vagal overactivity commonly caused by halothane
   b. induce muscular relaxation by blocking cholinergic receptors
   c. reduce salivation and bronchial secretions caused by halothane
   d. all of the above.

Answer is (c)- this is the classic clinical use of atropine and one you should have committed to memory. Atropine does not induce muscular relaxation- that would be a neuromuscular junction blocker such as curare, thus (b) and (d) are wrong. #! might confuse you. Atropine is used to override vagal activity, but that is not the reason it is given before halothane.

Antihistamines:

Questions regarding antihistamines generally want you to know what H_1 and H_2 antihistamines are used for:

H_1 antihistamines are useful for:

1. Treating dermatologic manifestations of an allergic response. (ex. chlorpheniramine)
2. Preoperative medication for sedation, antiemetic properties, anticholinergic effects. (ex. promethazine)
3. For controlling the symptoms of parkinsonism (ex. diphenhydramine)

H_2 antihistamines such as cimetidine are used to reduce gastric acid secretion (ex. cimetidine). **Now available OTC for heartburn**

1. Which of the following drugs is useful in treating dermatologic manifestations of an allergic response?
   a. Diazepam
   b. Atropine
   c. Hexylresorcinol
   d. Chlorpheniramine
   e. Phenoxybenzamine

   (d) Answer is (e)- The implied drug class is antihistamines. Of the drugs given only 2, (c), and (e) are antihistamines. There are two classes of antihistamines, H1 and H2. H2, represented by cimetidine, are used to treat ulcers not skin conditions. H1 antihistamines are used to treat allergic reactions, and chlorpheniramine is an H1 drug. Thus the answer is (e).

2. Cimetidine is used therapeutically to
   a. Stimulate respiration
   b. Protect against anaphylaxis
   c. Decrease gastric acid secretion
   d. Hasten excretion of barbiturates
   e. Dilate smooth muscles of the bronchioles

   (c)

3. Gastric acid secretion has been shown to be most effectively reduced with the use of
   a. Adrenal steroids
   b. Anticholinergic drugs
   c. Serotonin antagonists
   d. H_1-histamine receptor antagonists
   e. H_2-histamine receptor antagonists

   (e)

4. Drug-mediated inhibition of H_2-histamine receptors is most useful in treating
   a. Asthma
   b. Anaphylaxis
   c. Contact dermatitis
   d. Gastric hyperacidity
   e. Localized allergic reactions

   (d) same question repeated three different ways

5. Which of the following antihistamines is most commonly used as preoperative medication?
   a. Meclizine
   b. Cyclizine
   c. Promethazine
   d. Dimenhydrinate
   e. Chlorpheniramine

   (c) meclizine is used for vertigo, the others for allergies

6. Use of diphenhydramine (Benadryl) in controlling the symptoms of parkinsonism is based upon which of the following effects?
   a. Anticholinergic
   b. Local anesthetic
   c. Adrenergic-blocking
   d. CNS depressant on the midbrain
   e. Stimulant to dopaminergic nerves in the basal ganglia

   (a) Remember that Parkinsonism is a due to a deficiency of DA in the brain, and is currently treated with levodopa and carbidopa. However, prior to these, anticholinergic drugs were the first drugs found to be somewhat effective for treatment of this disease, in that cholinergic and dopaminergic tracts interact in the brain, and thus reducing cholinergic activity via anticholinergic drugs improves or enhances dopaminergic function, suggesting one is inhibitory to the other. Drugs with anticholinergic activity are often still the first drug tried. Antihistamine drugs such as diphenhydramine often have strong anticholinergic activity, which accounts for their effectiveness in drying nasal secretions associated with a cold. Therefore, the answer is (a). (e) is there to confuse you, but don't be. Diphenhydramine does not stimulate dopaminergic nerves in the basal ganglia. Adrenergic blockers ((c)) do see some use in the treatment of Parkinson's, but diphenhydramine has no adrenergic blocking activity. Diphenhydramine does have the actions given in (b) and (d), but these are not responsible for its efficacy in Parkinson's..

41. The mechanism of action of H1 antihistamines is
   a. MAO enzyme inhibition
   b. competitive antagonism
   c. physiologic antagonism
   d. noncompetitive antagonism
   e. inhibition of release of bound histamine

   (b) Answer is (b) - memorization - H1 antihistamines are competitive histamine receptor blockers. Many students answer (e), but this is the mechanism of action of cromolyn. (c) also draws some answers, but is wrong-epinephrine is the physiological antagonist of histamine.

Miscellaneous questions from a variety of categories that are not asked quite as frequently as the ones above:

24. Which of the following drugs is often used to treat trigeminal neuralgia?
a. clonazepam
b. carbamazepine
c. acetazolamine
d. succinylcholine

Answer is (b)- This is a memorization question. Carbamazepine, as well as phenytoin, are the main drugs used to treat trigeminal neuralgia. Clonazepam is a benzodiazepine, succinylcholine is a depolarizing neuromuscular blocking agent, while acetazolamine is an anticonvulsant like carbamazepine.

1. The use of epinephrine for local hemostasis during surgery might result in
a. hypoglycemia
b. cardiac arrthymia
c. an acute asthma attack
d. a drastic drop in blood pressure
e. any of the above

The answer is (b), Cardiac arrthymias are the main bad side effect of epinephrine as a vasoconstrictor. Epi stimulates both alpha and beta receptors. Beta receptors are found in the heart and stimulation of beta receptors increases heart rate, force of contraction, cardiac output and oxygen utilization. (a) is wrong, epi elevates blood glucose. (c) is wrong- beta stimulation in smooth muscle and bronchi causes bronchodilation, and is thus a drug of choice for acute asthmatic attacks. (d) is obviously wrong- a decrease in blood pressure would not result from adrenergic stimulation, increased blood pressure is the rule.

4. The highest risk associated with use of oral contraceptives is
a. hepatic necrosis
b. permanent sterility
c. cancer of the breast
d. cancer of the uterus
e. thromboembolic disorders

The two major side effects are suspected carcinogenicity and the tendency to produce thromboembolisms. Liver function can be altered but this is reversible. Research has failed to prove the link between estrogen and breast cancer, and reproductive function eventually returns upon cessation of therapy. The answer according to the gods of the testing board is (e). Amen.

#9. Factors common to all forms of drug abuse include
a. miosis
b. tolerance
c. physical dependence
d. psychological dependence
e. any of the above

The answer is (d). Miosis ((a)) is a cholinergic effect typically observed with opiates. Cocaine doesn't cause this kind of effect for example. (b), tolerance, is common to many drugs such as opiates, barbiturates and sedatives, but has not been clearly demonstrated for all abused drugs. (c), physical dependence, is usually thought to be true only for morphine, alcohol, caffeine, and perhaps cocaine.

27. Alcoholic euphoria results from
a. increased activity of the cerebellum
b. increased activity of the cerebral areas
c. increased activity of the spinal synapses
d. decreased activity of the medullary centers
e. removal of inhibitory activity of the cortex

Answer is (e)- Alcohol inhibits the CNS, thus eliminating (a), (b), and (c), leaving the choice between 4 and 5. Alcohol has been postulated to inhibit GABA effects, the major inhibitory transmitter in the CNS, especially in the cortex. Thus (e) is the answer.

28. Sulfonyl ureas cause insulin secretion by
a. adrenergic simulation
b. cholinergic stimulation
c. direct stimulation of pancreatic beta cells

Answer is (c)- memorization- alpha agonists decrease insulin release, beta agonists increase it. Thus because both of these are adrenergic stimulation, (a) can't be the answer. (b)- muscarinic agonists increase insulin release, but this is not the mechanism of sulfonylureas. They cause insulin release by (c), and are the primary oral antidiabetic agents used therapeutically.

37. If a patient requiring an extraction reports that he is on dicoumarol therapy, the laboratory test most valuable in evaluating the surgical risk is
a. clotting time
b. bleeding time
c. sedimentation rate
d. complete blood cell count
e. plasma prothrombin time

Answer is (e)- Dicoumarol is an oral anticoagulant. Thus the risk in an extraction is that the patient may bleed excessively, or that a serious interaction with a drug that you might require in your management of the patient, such as barbiturates or salicylates may occur. You may need to adjust the anticoagulant activity to within a safe range for surgical procedures. Since dicoumarol prevents blood clotting by preventing the conversion of Vitamin K to prothrombin, (e) is the only appropriate test.

42. A drug has a half-life of 4 hours. If 2 gms are given every 4 hrs what will be the amount in gms in the body immediately after the third dose?
a. 1.5
b. 2.0
c. 3.5
d. 4.0
e. 6.0

Answer is (c)- 2 gm at 0 hr, at 4 hrs 1/2 from first injection is gone, leaving 1 gm in the body and you inject 2 more for a total after the second injection of 3. At the third dose, 1/2 of 3 is gone, leaving 1.5 gms, you inject 2 more for a total of 3.5 immediately after the third dose.

43. Which of the following combinations represents acceptable agonist-antagonist pairs in antidotal therapy?
a. morphine-naloxone
b. dicoumarol-protamine
c. warfarin-phenylbutazone
d. carbon monoxide-carbon dioxide

(a) should be immediately obvious- go no further, do not pass go, do not collect $200. (b) is wrong-Protamine is an antagonist of heparin, not dicoumarol. (c): phenylbutazone enhances the toxicity of warfarin by displacing it from plasma protein binding sites. (d): hyperbaric oxygen would be a useful treatment for carbon monoxide poisoning, not CO2

46. The various insulin preparations useful in the treatment of diabetes mellitus differ primarily in
a. locus of action
b. mechanism of action
c. mode of transformation
d. onset and duration of action
e. none of the above

Answer is (d)- remember, diabetes medications can be organized into 3 groups based on their onset and duration of action: 1)fast-acting: insulin injection, 2) intermediate acting: Isophane insulin suspension, and 3) long acting: protamine zinc insulin suspension

48. Displacement of a drug from protein binding sites is expected to increase the
a. drug effect observed
b. duration of drug effect
c. dose required for a given effect
d. none of the above

answer is (a)- most people answer 3, confusing protein binding sites from active receptor sites where drugs exert their effects. Protein binding sites are just uptake sites that take up a drug and keep it from getting to its real receptor site of action. Some drugs are extensively protein bound, for example coumarin is 97% protein bound and only 3% is left to reach effective sites. Administration with another drug that is also extensively plasma protein bound will displace the drug that is already on these sites via competition, and thus effectively increase the level of drug that can now get to an active receptor site.

52. In Fig. 1, three different doses of drug A are tested for activity. In Fig. 2, three doses of drug B are tested for activity in the same test system. In Fig. 3, three doses of drug A are tested in the presence of the high dose of drug B. Based upon the responses seen, which of the following statements best describe drugs A and B?
a. drug A is a partial agonist; drug B is an antagonist
b. drug A is an antagonist; drug B is a partial agonist
c. drug A is an agonist; drug B is an antagonist
d. drug A is a agonist; drug B an partial agonist
e. drug A is an agonist; drug B is neither an agonist or an antagonist

(5) sorry can't reproduce the figure. But I think they give you a copy of this exam, don't they? If not, look this up in your course syllabus. You still have your course syllabus, don't you? Answer is (e)- drug A depicts a dose response curve for a full agonist- the typical sigmoidal dose response curve going from 0-100% response, thus (b) is eliminated from further consideration. The dose response curve for drug B is flat-no response at all. Drug B thus can't be a partial agonist, which means (d) is wrong. It must either be an antagonist or neither an agonist or antagonist in this system. The fact that it doesn't seem to alter the dose response curve for Drug A in Fig. 3 indicates it can't be an antagonist- the curve would be similar in shape except shifted to the right. The answer is thus (e).

56. In an addisonian crisis (hypoaldosteronism) resulting from stress from a minor dental procedure, the patient should be treated immediately with
a. 0.5 ml norepinephrine
b. 5 mg. prednisolone acetate
c. 1% triamcinolone acetonide
d. 0.5 ml, 1:1000 epinephrine
e. 2 ml (100 mg) hydrocortisone hemisuccinate

Answer is (e)- Addison's disease results from failure of the adrenal cortices to produce adrenocortical hormones such as aldosterone. Aldosterone is a mineralocorticoid that controls sodium retention and potassium excretion. Lack of aldosterone results in electrolyte imbalances, with the major problem being hyponatremia (sodium loss). Similar symptoms may also be seen when a patient is withdrawn from chronic adrenal steroid therapy. Due to depressed adrenal function, patients can't respond to stressful situations (such as dental procedures) adequately, and an adrenal crisis may occur. The recommended treatment is (e), 100 mg of hydrocortisone hemisuccinate. Of the other corticosteroids given, he dose given for prednisolone ((b)) is too low, while triamcinolone lacks any effects on sodium retention. NE and Epi would not be used in this situation- they might be used in a case of adrenal medulla insufficiency.

59. Drug A inhibits the biotransformation of Drug B. The duration of action of drug B in the presence of drug A will usually be
a. shortened
b. prolonged
c. unchanged

Answer is (b)- usually biotransformation results in a more water soluble, more readily excreted form of the parent drug. In most cases this inactivates the drug, but there are some exceptions which involve the formation of metabolites with activity (diazepam) or when an inactive prodrug is given (levodopa) which becomes active after the first step in the biotransformation pathway (DOPA) sometimes are asked in questions of this form.

61. In which of the following groups of drugs is there the most consistency in chemical structure?
a. diuretics
b. antiepileptics
c. local anesthetics
d. general anesthetics
e. nonbarbiturate sedatives

Answer is (c)- Remember that local anesthetics are either esters or amides. All of the other alternatives diverge widely in their structures.

64. Cimetidine is administered to
a. aid in sleeping
b. relieve asthma
c. inhibit gastric secretion
d. relieve cold and flu symptoms

Answer is (c)- Remember, cimetidine or "Tagamet" is an H2 antihistamine are used therapeutically to inhibit gastric secretion ((c)) in cases of peptic ulcer. This is its only clinical use. You might see it in a question regarding drug metabolism- it is also a potent inhibitor of the mixed function oxidase drug metabolizing enzyme system in the liver. The other alternatives are to confuse you because you probably at least remember that it is an antihistamine, but you don't know the difference between H1 and H2 antihistamines.

65. Which of the following anticancer drugs can be classified as an antimetabolite?
a. cisplatin
b. lomustine
c. vincristine
d. methotrexate

Answer is (d) - I would guess (d) because its the only one I've ever heard of, and since this wasn't covered in class you might be one to guess it also. Wow, we got it right! Actually, if you look the others up, (a) & (b) are alkylating agents, (c) is an alkaloid derived from plants. (d) is a folate antagonist, which acts as an antimetabolite. Just for your future edification, most cancer chemotherapy drugs cause cell death by affecting the ability of cells to divide. The drugs thus inhibit one or more phases of the cell cycle or prevent a cell in G_O (the nondividing phase) from entering into the cycle of cell division. Antimetabolites may act in 2 ways (1) by incorporation into a metabolic pathway and formation of a false metabolite which is nonfunctional or (2) by inhibition of the catalytic function of an enzyme or enzyme system. Methotrexate is an example of a cell cycle specific antimetabolite that inhibits DNA synthesis during the S phase. Vincristine acts during the mitotic phase

66. Which of the following hormones acts to elevate blood concentration of ionic calcium?
a. glucagon
b. parathyroid
c. aldosterone
d. thyrotropin
e. thyrocalcitonin

Answer is (b)- maintaining the concentration of Ca++ in extracellular fluid by regulating the deposition and mobilization of calcium from bone, absorption from the GI tract, excretion etc. is the main function of parathyroid hormone. Thyrocalcitonin is another name for calcitonin. They hope to confuse you because there is a correlation between calcitonin and calcium, except that calcium concentrations regulate the synthesis and release of calcitonin. Glucagon is a pancreatic hormone that stimulates glucose production, thyrotropin is there to confuse you with parathyroid hormone, and aldosterone regulates Na+ levels not Ca++.

67. Disorientation, confusion, and hallucinations resulting from an overdose of scopolamine are most efficaciously treated by administering
a. atropine
b. levodopa
c. acetylcholine
d. physostigmine

Answer is (d)- scopolamine is a muscarinic receptor blocker similar to atropine, thus (a) is wrong. Levodopa has nothing to do with this question. To counteract a competitive muscarinic receptor block, you need to increase the levels of agonist, in this case acetylcholine. However, you can't give ACh because it is broken down almost instantaneously by acetylcholinesterase. The answer is to give a drug which knocks out the acetylcholinesterase, allowing endogenously released ACh to accumulate to overcome the action of scopolamine. The drug which will do this is physostigmine.

69. Developed hyporeactivity to a drug is
a. tolerance
b. antagonism
c. detoxification
d. desensitization

Answer is (a)- your choices are between tolerance and desensitization, (a) and (d). The latter refers to some alteration of receptors that leads to diminished response to the drug, and is really a mechanism whereby tolerance may occur. Thus the Gods of the Board questions have decreed that the answer shall be (a).

77. Absorption of a drug from the intramuscular site of administration may be slowed by
a. excercise
b. vasoconstriction
c. the presence of congestive heart failure
d. administering the drug as an insoluble complex

1. a and b
2. b only
3. b, c, and d
4. b and d
5. all of the above

Answer is (c)- "a" can't be right because exercise increases blood flow through muscles and thus improves absorption. Thus # 1 is wrong. "b" is obviously right for the same reason. "c" would also result in reduced blood flow so slower absorption would also be a problem. "d" is often used for prolonged and steady drug release so it also is true. The answer must thus be (c).

78. Salicylism includes which of the following?
a. nausea
b. tinnitus
c. vomiting
d. gastrointestinal bleeding

1. a, b, and c
2. a, b, and d
3. a and c only
4. b, c, and d
5. all of the above

Answer is (e)- "Salicylism" is a mild toxic reaction to aspirin (acetylsalicylic acid), usually occurring after prolonged treatment with large doses. Nausea, tinnitus, vomiting and GI bleeding are all symptoms of salicylism. Other notable side effects of aspirin which result from ingestion of a single large dose are disturbances of acid-base imbalance (acidosis or alkalosis), fever, hypoglycemia. Remember, aspirin, is contraindicated in children suffering from influenza or chicken pox: aspirin has been implicated in the development of Renee's syndrome.

82. Which of the following are important criteria for the adequate clinical evaluation of a new drug?
a. comparison with a placebo
b. evaluation of side effects
c. utilization of control groups
d. comparison with a standard drug
e. double blind experimental design

1. a, b, c, and d
2. a, b, d, and e
3. a, c, d, and e
4. b, c, and e
5. b, d and e only
6. all of the above

Answer is #6- A gimme-this is just common sense.

84. Each of the following agents has a long duration of action due to the presence of liver generated active metabolites EXCEPT
    a. diazepam
    b. oxazepam
    c. flurazepam
    d. chlordiazepoxide

Answer is (b)- This is a pure memorization question. All of these drugs are benzodiazepines, which typically have a long duration of action because they are converted to pharmacologically active metabolites with long half-lives. Oxazepam, midazolam, and lorazepam are exceptions that are not converted to active metabolites. The answer is thus (b).

86. Each of the following statements relates to the general aspects of toxicology EXCEPT
    a. most drugs exert a single action
    b. toxicity is both time and dose dependent
    c. toxicity can be due to overdosage of a drug
    d. symptoms of toxicity can be anything ranging from nausea to death
    e. for some drugs, even a minimal concentration can be harmful

Answer is (a)- If you learned anything from pharmacology, you should instantly be able to identify (a) as the correct answer. The constant quest of pharmacology as a science is to design or identify drugs that have as narrow or specific range of action as possible, but not to many single action drugs have been identified. Alternatives (c) and (b) should remind you of phrases such as dose-response curves and therapeutic index (the ratio of a toxic or lethal dose to a therapeutic dose, LD50/ED50). A toxic effect can be broadly defined as any undesired effect of the drug, thus # 4 can't be the exception. To rule out # 5, think of nerve gas.

92. The central actions of ethyl alcohol are not synergistic with which of the following?
    a. diazepam
    b. meperidine
    c. pentobarbital
    d. chlorpromazine
    e. methylphenidate

Answer is (e)- The central actions of alcohol are depressant. Thus the choice of correct answer comes down to knowing which of the drugs listed is not a CNS depressant. Diazepam is a benzodiazepine, pentobarbital is a barbiturate. Both are sedating. Meperidine is an opiate, while chlorpromazine is a phenothiazine antipsychotic. Both of these classes of drugs are also typically sedating. The correct answer is (e), methylphenidate (Ritalin). Ritalin is an indirect acting sympathomimetic, and acts similar to amphetamine in the CNS- it is a stimulant.

97. A heroin dependent patient should not be given nalbuphine (Nubain) for pain because
    a. it has no analgesic properties
    b. it may produce respiratory depression
    c. as a mixed agonist-antagonist, it can elicit withdrawal symptoms
    d. the high abuse potential of nalbuphine may add to the patient's problems

Answer is (d)- Nalbuphine is a mixed agonist-antagonist that is analgesically equipotent with morphine (thus # 1 is ruled out). However, analgesia is produced by its agonistic effects at kappa opioid receptors. It has pronounced antagonistic effects at the mu receptor, and can be used clinically to reverse respiratory depression (by blocking mu receptors) without a loss of analgesic effects (by stimulating kappa receptors). Thus, # 2 is ruled out. # 4 is not likely. The mixed agonist-antagonists were designed to combine analgesia with enough antagonistic properties to prevent their abuse. Thus, # 3 is the right answer. Typically, these drugs can mimic the effect of morphine in a drug free patient, but antagonize opiate action in a dependent patient, thus precipitating withdrawal. Of this class of drugs, butorphanol is the exception- it will not precipitate withdrawal because it is only a weak mu antagonist.

99. All of the following methods of drug biotransformation are classified as synthetic <u>except</u>:
    a. N-alkylation
    b. O-dealkylation
    c. sulfate conjugation
    d. glucuronide conjugation

Answer is (b)- Nonsynthetic reactions (phase I reactions) include the various transformations of molecular structure: oxidation, reduction, and hydrolysis; they represent the first stage of biotransformation. Synthetic (phase II) reactions consist of the <u>conjugation</u> of drugs or their metabolites with functional groups. Of the alternatives, (c) and (d) are conjugation reactions, and thus are wrong because they are synthetic reactions. Alternative (a) is tricky. It is made to sound like N-dealkylation, which is a nonsynthetic or phase I oxidative reaction. There is no reaction called N-alkylation. Thus (b), an oxidative phase I reaction is left as the right answer

Board Exam Review Addendum

More board questions that were released after I prepared the original review:

52. Which of the following drugs is often administered to treat life-threatening arrhythmias?
a. quinidine
b. lidocaine
c. verapamil
d. propranolol

(b) ventricular arrythmias are the life threatening ones, and lidocaine is particularly effective for this kind of arrhythmia when given parenterally in an emergency situation. It has a very rapid onset of action when given IV, which is obviously important in an emergency. The others all also have antiarrythmic action, mostly used for supraventricular tachyarrythmias

58. A male patient who is receiving Coumadin therapy presents for an elective extraction. His protrhombin time (PT) is prolonged. Which of the following methods is preferred for reducing the PT to an acceptable level?
a. administering vitamin K (Aqua Mephyton)
b. Withdrawing Coumadin for two days
c. reducing Coumadin to one half the usual dose for two days
d. administering a Coumadin antagonist, such as heparin
e. administering a platelet transfusion to enhance coagulability

(b) the issue is obviously that the patient may bleed excessively, a situation that would cause problems during dental surgical procedures. Dentist should consult with the patient's physician about stopping the Coumadin for a few days. Options (a) and (e) might be appropriate in an emergency situation, but that is not what we are dealing with here. Option (c) would take longer to reduce (PT) than just stopping the Coumadin. Finally, heparin is not a Coumadin antagonist, but an anticoagulant in its own right. ***Guidelines into what adjustments to make in Coumadin therapy prior to dental treatment have changed since this type of question was written – the safest bet is to consult physician – depending on patient's status, it could be that no adjustment should be made at all, certainly not for minor surgery anyways.***

62. Each of the following drugs has a significant anti-inflammatory property except one. Which one is the exception?
a. aspirin
b. cortisol
c. acetaminophen
d. ibuprofen
e. indomethacin

(c) geez, they keep repeating this question year after year after year - if you don't immediately know that the answer is acetaminophen, I will call Mr. Fleming and change your grade in Pharmacology to an "F"!

72. A patient has a history of significant cardiovascular impairment. The maximum safe dose of epinephrine that can be administered to this patient is:
a. 1 cc, 1: 50,000
b. 2 cc, 1: 50,000
c. 1 cc, 1: 100,000
d. 2 cc, 1: 100,000

(b) the rule is ..04 mg of epi max in CV patients. The easiest way to figure this one out is to remember you shouldn't give more than 2.2 carpules of xylocaine with 1:100,000 epi - your usual choice as a local anesthetic. Two carpules is 3.6 cc. This eliminates options (c) and (d), since they would be safe but not maximal. Since 1:50,000 is twice as concentrated as 1:100,000, 1 cc of 1:50,000 (option (a)) is the same as 2 cc of 1:100,000 so still not close to max, so (b) has to be the right answer. Of course, you could have just remembered, 1:100,000 equals .01 mg per cc, so 1:50,000 equals .02 mg per cc, so (b) would equal the max of .04 mg.

75. Which of the following is the current drug of choice for status-epilepticus?
a. diazepam (Valium)
b. phenytoin (Dilantin)
c. chlorpromazine (Thorazine)
d. carbamazepine (Tegretol)
e. chlordiazepoxide (Librium)

(a) memorize, memorize. Most are anticonvulsants, but diazepam is drug of choice. Chlorpromazine is an antipsychotic, not an anticonvulsant. Phenytoin, is an anticonvulsant, and is one of the answers to the question "which of the following causes gingival hyperplasia?" Don't forget diazepam is also given for antianxiety and sedation.

83. Which of the following is an example of an enteral route of administration?
a. oral
b. submucosal
c. inhalation
d. subcutaneous
e. intramuscular

(a) enteral means via GI tract-only route listed that goes directly into GI tract is (a) oral.

93. Propranolol (Inderal) exerts its major antianginal effect by
a. dilating coronary arteries
b. dilating systemic blood vessels
c. increasing cardiac contractility
d. stimulating vagal slowing of the heart
e. blocking beta-adrenergic receptors of the heart

(*e) my god, if you don't remember propranolol as the prototype nonspecific beta adrenergic receptor blocker, you shouldn't be taking this test. In angina, the goal is to reduce the oxygen demands of the heart, since it can't get enough. (a) and (b) are useful actions in angina, but are the way that nitroglycerin and calcium channel blockers work, not propranolol, which acts to decrease cardiac output and contractility (so (c) is obviously wrong- this is an action that is needed in heart failure patients, not angina)*

99. Bradycardia is MOST commonly treated with which of the following drugs?
a. atropine
b. epinephrine
c. a diuretic
d. a potent vasodilator

(a) bradycardia is a reflexive slowed heart rate, controlled by vagal

input to the heart, and is cholinergically mediated, which means you need a cholinergic receptor blocker to reduce the vagal effect. Atropine is the only drug listed which is a cholinergic agent. They threw in epinephrine as a tease, because if you knew bradycardia was cardiac slowing, you might be tempted to think epi, which usually speeds up the heart, would be the right answer- but you've got to block the vagal input, epi won't work.

103. Which of the following best explains why drugs that are highly ionized tend to be more rapidly excreted than those that are less ionized? The highly ionized are
a. less lipid soluble
b. less water soluble
c. more rapidly metabolized
d. more extensively bound to tissue

(a) nonionized forms of drugs cross membranes more readily and are highly lipid soluble, and tend to get stored in fat tissue from where they are only slowly released. Thus highly ionized drugs, which are less lipid soluble don't get stored in fatty tissue and are subject to more rapid excretion.

110. Which of the following groups of drugs is CONTRAINDICATED for patients who have glaucoma?
a. adrenergic
b. cholinergic
c. anticholinergic
d. adrenergic blocking

(c) one of the things you had to memorize about atropine, the prototype anticholinergic drug was don't use it with glaucoma patients, as it will increase intraocular pressure, which is already the problem with glaucoma patients. Drugs in categories (b) and (d) are actually used to treat glaucoma, so they are obviously not contraindicated. Obviously, (a) wouldn't help either.

125. Low dose aspirin therapy prevents the formation of thromboemboli by preferentially inhibiting which of the following?
a. phospholipase A2 in the blood vessel walls
b. prostacyclin synthetase in the blood vessel walls
c. thromboxane synthetase in the platelets
d. vitamin K in the liver

(c) platelet aggregation is controlled by two factors, prostacyclin, which decreases it, and thromboxane, which enhances it. Low dose aspirin blocks the latter, so (c) is right, (a) is the mechanism of action of corticosteroid drugs, (d) is the mechanism of action of anticoagulant drugs like coumadin.

128. A patient who is receiving an IV diazepam sedation has upper eye-lid ptosis (Verill's sign). The dentist should
a. assist respiration immediately
b. consider the patient to be adequately sedated
c. place the patient in the Trendelenberg position
d. administer one more increment of diazepam and proceed with the treatment

(b) memorize - this is the sign you look for to tell the patient is adequately sedated.

132. A dentist is considering the use of nitrous-oxide conscious sedation for a patient. However, this type of sedation will be CONTRAINDICATED, should the patient have a history of which of the following?
a. dental anxiety
b. psychotic care
c. controlled hypertension

(b) even if you didn't know that nitrous can alter the patient's sense of reality (that's why you don't give it alone in your office, they might fantasize that you had your way with them when you didn't!) by elimination (b) has to be the right answer since (a) and (c) are indications for the use of nitrous

137. The correct total liter flow of nitrous oxide- oxygen is determined by
a. a standard 6 liter per minute flow
b. the patient's metabolic oxygen requirements
c. the amount necessary to keep the reservoir bag 1/3 to 2/3 full
d. the largest volume that the patient can exchange within one minunte

(c) I didn't know this- I hope you did! I hope someone teaches this somewhere in the curriculum!

139. Which of the following sympathomimetic agents is the most potent bronchodilator?
a. amphetamine
b. norepinephrine
c. phenylephrine
d. isoproterenol
e. methoxamine

(d) for bronchodilation you want a potent beta-2 adrenergic receptor agonist- of the drugs listed (d) is the best. Isoproterenol is a non-specific beta agonist. Methoxamine and phenylephrine are alpha-1 agonists, amphetamine is an indirect acting agonist that causes the release of norepinephrine, which is less potent at beta receptors than isoproterenol.

148. Succinylcholine blocks neuromuscular transmission by
a. inhibiting cholinesterase
b. inhibiting the central nervous system
c. depolarizing the motor endplate of skeletal muscle
d. inducing the formation of cholinesterase at the endplate
e. blocking release of acetylcholine at the endplate

(c) See- you knew those drug list definitions I made you memorize would come in useful! Neuromuscular transmission requires the action of acetylcholine at the nicotinic receptors on the neuromuscular junction endplate. The two drugs used clinically to do this are curare and succinylcholine. Curare is a nicotinic receptor blocker, succinylcholine acts to overstimulate the receptor, thereby causing its subsequent depolarization of the neuron and a block of nuscle activity. (e) is how spider venoms and snake toxins work, not succinylcholine. (a) would enhance neuromuscular action, and is actually made use of clinically with myasthenia gravis patients. (d) isn't possible.

153. Which of the following effects are common to pentobarbital, diazepam, and meperidine?
a. anticonvulsant and hypnotic
b. analgesia and relief of anxiety
c. sedation and ability to produce dependence
d. amnesia and skeletal muscle relaxation

(c) the only definition which covers all three drugs. (a) applies only to pentobarbital, (b) applies only to meperidine, and (d) applies only to diazepam.

154. The onset of action of a drug is primarily determined by the rate of
a. excretion
b. absorption
c. distribution
d. biotransformation

(b) obviously - drug has to be absorbed before any of the other actions can take place. The others would determine duration of action, not onset.

168. Injection of a local anesthetic into an inflamed area usually has a less than optimal result. Which of the following best explains why?
a. the prostaglandins stabilize the nerve membrane
b. inflammation reduces the availability of the free base
c. the drug will be absorbed more rapidly because of the increased blood supply
d. the chemical mediators of inflammation will present a chemical antagonism to the anesthetic

(b) they ask a variant of this question every single year, so I would suggest you look at the many different wordings of this question and know that they are just testing the factoid that inflamed tissue has a lower pH, which results in reduced availability of the free base form needed to cross the neural membrane.

174. The major effect of a drug is produced by the amount of the drug that is
a. free in plasma
b. excreted by the kidney
c. detoxified in the liver
d. bound to plasma protein

(a) if its excreted or detoxified, it ain't going to get to its active site to have an effect. Same if it is bound to plasma proteins – it has to be free to get distributed to active site

176. A 4 yr old child is shy, timid, and fearful. Which of the following will be MOST appropriate for the restorative appointments for this child?
a. Naloxone
b. Nitrous oxide/oxygen
c. Promethazine
d. Hydroxyzine hydrochloride (atarax) or hydroxyzine pamoate (Vistaril) in divided doses
e. Meperidine (Demerol), promethazine (Phenergan) and chlorpromazine (Thorazine) combined

(b) most likely because it is titratable and thus can be used to provide just enough of an effect to calm the anxious child. (c), (d), and (e) are sedative regimens that would probably induce deeper sedation than nitrous, and that much sedation doesn't seem to be called for here. Naloxone is a drug used to reverse the effects of too much morphine.

177. A primary advantage of intravenous sedation is which of the following?
a. fewer side effects from the sedation
b. slower biotransformation for prolonged action
c. the ability to titrate individualized dosage
d. a smooth and more gradual onset of sedation

(c) hey – I asked this on the exam! Titratability is the primary advantage of iv drug administration. Onset is very fast, not gradual,since it doesn't have to be absorbed, so (d) is out. (a) and (b) are not possible.

179. Each of the following is true regarding drug biotransformation EXCEPT one. Which one is the EXCEPTION?
a. the rate may differ significantly in various animal species
b. it primarily occurs in the liver microsomal enzyme system
c. it usually converts a drug to its lipid soluble, nonionized form
d. it generally involves alterations of the chemical structure of the drug

(c) this is the exception, since the purpose of drug metabolism is to change the drug into an excretable form, which is water soluble, ionized. Making it lipid soluble and nonionized would make it stay longer in the body. The other statements are true.

191. Which of the following drugs causes the LEAST CNS depression and impairment of psychomotor skills?
a. diazepam
b. buspirone
c. alprazolam
d. chloral hydrate

(b) memorize, memorize. Buspirone is used to relieve anxiety – it is not a benzodiazepine like diazepam and alprazolam (BDZs may not be that sedating, but can cause impairment of psychomotor skills), and not a strong sedative like chloral hydrate

16. A patient who has which of the following conditions is most likely to have postoperative bleeding after multiple extractions?
a. angina
b. diabetes
c. cirrhosis
d. rheumatic fever
e. chronic bronchitis

(c) vitamin K is stored in the liver – this is decreased by cirrhosis – the result is deficiencies in prothrombin dependent coagulation factors

42. Local anesthetics aid in reducing the flow of saliva during operative procedures by
a. blocking the cholinergic nerve endings
b. blocking innervation to major salivary glands
c. blocking efferent parasympathetic nerve pathways
d. reducing sensitivity and anxiety during tooth preparation

(d) locals don't have actions (a), (b), or (c)! Nervous patients, however, do salivate more.

49. Which of the following combinations of agents would be necessary to block the cardiovascular effects produced by the injection of a sympathomimetic drug?
a. atropine and prazosin
b. atropine and propranolol
c. prazosin and propranolol
d. phenoxybenzamine and curare
e. amphetamine and propranolol

(c) a sympathomimetic drug is going to potentially activate both alpha and beta receptors, so you would need a pairing of drugs which blocks those receptors. Prazosin, an alpha-1 blocker,, and propranolol, a non-specific beta-blocker, are the only pair that block both types sympathetic/adrenergic receptors. Atropine is a muscarinic blocker (anticholinergic), phenoxybenzamine is an alpha-blocker, but curare is a

neuromuscular junction blocker. Amphetamine is a sympathomimetic drug, not a blocker or sympatholytic drug.

75. When compared therapeutically to penicillin G, penicillin V has a
    a. slower renal excretion
    b. more reliable oral absorption
    c. broader antibacterial spectrum
    d. greater resistance to penicillinase
    e. lower potential for allergic reaction

    (b) same drugs just different salts, which primarily affects absorption from GI tract. Pen G, due to lousy absorption when taken orally, is usually injected. ***Pen G is hardly used in dentistry anymore, so I would expect these kinds of questions to disappear.***

89. Each of the following is a common side effect of prolonged tetracycline therapy EXCEPT one. Which one is the EXCEPTION?
    a. diarrhea
    b. superinfection
    c. photosensitivity
    d. visual disturbance
    e. discoloration of newly forming teeth

    (d) since you use tetracycline everyday, you have committed all this stuff to memory, right? ***I would hope, since hardly anyone outside periodontists hardly uses tetracyclines anymore in dentistry that they would not ask you a lot of questions about tetracyclines, but if they do, you're ready, right?***

110. To reduce a patient's salivary flow, a dentist has prescribed atropine. As a result of this medication, the patient might experience which of the following side effects?
    a. sedation
    b. diarrhea
    c. bradycardia
    d. blurred vision
    e. stomach cramping

    (d) because it will also cause the anticholinergic effect of mydriasis, or papillary dilation

112. Of the following local anesthetics, which has intrinsic vasoconstrictive actions?
    a. cocaine
    b. procaine
    c. xylocaine
    d. bupivacaine

    (a) ah, the classic cocaine is a local anesthetic question! I told you they asked this question all the time – I think they like to justify keeping some cocaine in their office ("But Officer, it's an excellent local anesthetic!"). So once again, all locals except for cocaine are vasodilatory, that's why most require the addition of epinephrine.

118. Which of the following antibiotics is found at much higher concentrations in crevicular fluid than in serum?
    a. clindamycin
    b. penicillin
    c. metronidazole
    d. tetracycline

    (d) what the heck is "crevicular fluid" and where is it? If you need to get some antibiotic in there, tetracycline is apparently the one – must be something to do with gums,since periodontists use tetracyclines a lot.

124. When administered as oral centrally acting analgesics, which of the following is considered to have the highest dependence liability?
    a. codeine
    b. oxycodone (in Percodan)
    c. propoxyphene (Darvon)
    d. pentazocine (Talwin)

    (b) memorize that list of opioid analgesic potencies. It would go propoxyphene<codeine<pentazocine<oxycodone

135. Antibiotics are useful in the treatment of which of the following?
    a. herpangina
    b. angina pectoris
    c. recurrent apthous stomatitis
    d. necrotizing ulcerative gingivitis

    (d) by process of elimination: herpangina is a viral condition, angina pectoris is heart pain caused by lack of oxygen, apthous stomatitus is canker sores – these are not the result of a bacterial infection, so that leaves (d) gingivitis

136. Auditory nerve deafness is associated with the use of
    a. polymixin B
    b. chloramphenicol
    c. amphotericin B
    d. gentamycin

    (d) each one of these drugs has a side effect that they may use in the stem of this question, and give you the same list of drugs: the effects are polymixin- renal necrosis, chloramphenicol- bone marrow depression, amphotericin B –nephrotoxicity

151. The maximal or ceiling effect of a drug is also correctly referred to as the drug's
    a. agonism
    b. potency
    c. efficacy
    d. specificity

    (c) efficacy sounds like it might have something to do with effect, doesn't it? Didn't I make you memorize potency vs. efficacy? Potency is how much drug does it take to produce an effect, and efficacy is now much of an effect is a drug capable of producing.

168. Which of the following agents found in tobacco products cause addiction?
    a. tar
    b. formaldehyde
    c. nicotine
    d. carbon monoxide

    (c) if you are addicted to formaldehyde, you are dead! Same with the others. How dumb do they think you guys are?

172. Allergic reactions to local anesthetics are caused by
a. rapid absorption
b. slow detoxification
c. an antigen-antibody reaction
d. improper administration technique

(c) another duh! They must throw these in there to inflate your scores.

184. Which drug group is the LEAST likely to cause xerostomia?
a. opioids
b. antidepressants
c. antihistamines
d. benzodiazepines
e. anticholinergics

(a) xerostomia is an anticholinergic effect. All the drugs listed except opioids are unfortunately strongly anticholinergic, in addition to their desired mechanism of action.

187. Currently, the BEST oral sedative drugs for dentistry fall into the class of
a. narcotics
b. barbiturates
c. phenothiazines
d. benzodiazepines

(d) BDZs are better than narcotics or barbiturates because they don't cause anywhere near the problem with respiratory depression that those two do.. Phenothiazines have more side effects.

189. Which of the following best describes the drug-receptor activity of naloxone?

	Affinity	Intrinsic Activity
a.	high	high
b.	low	high
c.	high	none
d.	none	low

(c) naloxone is the example I made you learn of a "pure" competitive receptor antagonist – such a drug binds to a receptor with high affinity, but has no intrinsic activity. If it did it would be an agonist. They could also have reworded this question by substituting competitive antagonist for naloxone.

200. Which of the following types of chemical bonding is the least likely to be involved in a drug-receptor interaction?
a. covalent bonding
b. hydrogen bonding
c. dipole-dipole bonding
d. electrostatic bonding
e. van der Waal's forces

(a) why would you care which one is the least likely? Actually, because if it binds covalently, that is a non-reversible situation that can't be overcome – you get a long-lasting drug effect that can only be overcome by making new receptors. Some antagonists work this way.

11. After a threshold stimulus, the cell membrane becomes permanently altered. The liberation of which of the following transmitter substances causes this alteration?
a. acetylcholine
b. cholinesterase
c. hydroxycholine
d. acetylsalicylic acid

(a) easy – the only "transmitter" listed is acetylcholine. You don't have to have any idea what the question is about (I don't!) as long as you recognize that there is only one transmitter listed – the others kinda sound the same – cholinesterase is the enzyme that breaks acetylcholine down, acetylsalicylic acid is aspirin, have no idea what hydroxycholine is

23. Which of the following compounds is used as an antiviral agent?
a. amantadine (Symmetrel)
b. novobiocin
c. miconazole (monistat)
d. amphotericin B

(a) miconazole and amphotericin B you should recognize, I hope, as antifungals, so your choice is between (a) and (b). Novobiocin sounds like an antibiotic, which it actually is, so guess (a)

36. With an overdose of a cholinergic drug, one would expect to see each of the following signs EXCEPT one. Which one is the EXCEPTION?
a. sweating
b. urination
c. mydriasis
d. bradycardia
e. copious serous saliva

(c) mydriasis is papillary dilation, which is a hallmark effect of anticholinergic drugs, and why they are used in eye exams

42. Cephalosporins are definitely CONTRAINDICATED for penicillin-allergic patients who exhibit
a. immediate-type reactions
b. nausea and vomiting with erythromycin
c. any type of reaction to the penicillins

(a) see the words "definitely" and CONTRAINDICATED – why do you think they worded it that way? This means worst case scenario, which would be someone who has had an anaphylactic reaction to penicillin, which is an immediate type reaction

46. Which of the following is a beta-adrenergic receptor blocking agent used for the treatment of hypertension?
a. prazosin (Minipress)
b. clonidine (Catapress)
c. atenolol (Tenormin)
d. hydralazine (Aprezoline)
e. verapamil (Calan)

(c) this is one of those questions where they give you five drugs as possible answers, and can create 5 different questions just by changing the question regarding mechanism of action. So (a) is an alpha-1 blocker, (b) alpha-2 agonist that decreases sympathetic outfloe from CNS, (d) is a direct acting vasodilator, and (e) is a type I calcium channel blocker

50. In which of the following categories are ephedrine, tyramine, and amphetamine classified?
    a. anticholinesterases
    b. alpha-adrenergic blocking agents
    c. indirect-acting sympathomimetics
    d. direct-acting parasympathomimetics

    (c) hey, this is the question everybody missed on the exam! Don't you wish you had learned it then?

57. Thiazides, which are used in the treatment of hypertension, may require supplemental administration of
    a. sodium
    b. chloride
    c. calcium
    d. potassium

    (d) so they don't get hypokalemia – duh!

76. Which of the following adverse reactions of oral contraceptives is the most common and the most serious?
    a. hypotension
    b. hepatotoxicity
    c. uterine neoplasia
    d. thromboembolic disorder
    e. decreased resistance to infection

    (d) they all sound pretty serious, don't they. I mean hypotensive shock, liver damage, cancer, stroke, immunosuppression – fortunately only (d) is a real risk with oral contraceptive therapy – would you take one if you were gonna get all of those bad things?

80. The supraspinal analgesic activity of morphine is mediated primarily through its influence upon which opioid receptor subtype?
    a. mu
    b. kappa
    c. delta
    d. sigma
    e. epsilon

    (a) these are all opioid receptors – only mu and kappa are involved in analgesia, mu at the supraspinal level and kappa at the spinal level (the receptor that pentazocine (Talwin), the mixed acting agonist/antagonist acts on

85. Which pair of anesthetics is most likely to show cross-allergenicity?
    a. lidocaine-mepivacaine
    b. prilocaine-tetracaine
    c. procaine-mepivacaine
    d. procaine-lidocaine
    e. lidocaine-benzocaine

    (a) you are looking for an ester - ester pairing. Only (a) meets this criterion. procaine, benzocaine and tetracaine are esters, lidocaine, prilocaine and mepivacaine are amides. Amides, unlike esters are not by rule cross-allergenic

107. Thrombophlebitis, which occurs after intravenous administration of diazepam, is usually attributed to which of the following substances in the mixture?
    a. benzoic acid
    b. ethyl alcohol
    c. propylene glycol
    d. sodium metabisulfite

    (c) memorize this picky little fact please!

110. Each of the following is a pharmacologic effect of phenothiazines EXCEPT one. Which one is the EXCEPTION?
    a. sedation
    b. an antiemetic effect
    c. alpha-adrenergic effect
    d. potentiation of the action of narcotics
    e. an anticonvulsant

    (e) see, they do ask you questions about drugs that you don't normally use every day. Actions a, c, and d are all clinically useful actions of the phenothiazines, which you might remember were discussed under the category of antipsychotic drugs. But wait- Promethazine (Phenergan) is used in dentistry as a sedative, often in combination with Demerol because it reduces the nausea associated with the use of the opioid and also potentiates its analgesic effect, allowing lower dose to be used, thus again reducing the potential for adverse side effects. Alpha adrenergic effects are an adverse side effect.

119. Epinephrine antagonizes the effects of histamine by
    a. preventing the release of histamine
    b. acting on the central nervous system
    c. producing physiologic actions opposite to that of histamine
    d. competitively blocking histamine at the cellular receptor site

    (c) exactly why I kept asking you on every exam to give me an example of physiological antagonism as a drug-drug interaction question. It does not act via (a), which is how cromolyn (Intal) works, or by (d) which is how antihistamines work.

121. Which of the following represents the drug-of-choice in the treatment of candidiasis for an HIV-infected patient?
    a. acyclovir
    b. nystatin
    c. AZT
    d. chlorhexidine

    (b) candidiasis is a fungal infection that needs to be treated with an antifungal agent like nystatin. (a) and (c) are antiviral drugs used to treat HIV, while chlorhexidine is a antimicrobial mouthwash

132. A patient presents for treatment of a large fluctuant mass in the submandibular space as a result of extension of odontogenic infection. He has a temperature of 38.5 degrees C (101 degrees F). Initially, the dentist should treat this patient with which of the following?
    a. salicylate therapy to reduce the temperature
    b. alternate application of heat and cold to the area to improve circulation
    c. incision and drainage and a culture for antibiotic sensitivity
    d. antibiotic therapy to reduce the swelling and infection

    (c) antibiotic therapy won't be very effective if you don't incise and drain first. (a) and (b) are palliative actions the patient can take at home, not actions for the dentist.

146. The only local anesthetic that increases the pressor activity of both epinephrine and norepinephrine is
a. cocaine
b. procaine
c. dibucaine
d. lidocaine
e. mepivacaine

(a) hey, why do they always ask some dumb question like this about cocaine-like you guys use it every day in your office (you better not!!). Any way, remember, cocaine is an indirect acting adrenergic agonist, acting by causing the release of adrenergic neurotransmitters as well as blocking their reuptake, thereby prolonging their activity. The other drugs listed are just local anesthetics that don't have this action - they just block sodium influx into the neuron.

155. Corticosteroid therapy for arthritis is contraindicated for a patient who also has which of the following conditions?
a. anemia
b. nephritis
c. alcoholism
d. peptic ulcer
e. rheumatic heart disease

(d) Patients using corticosteroids for arthritis often develop ulcers because these drug block prostaglandin action in the stomach, thereby increasing acid secretion while decreasing the protective mucosal barrier of the stomach.

160. Which of the following is the first symptom that is usually perceived by the patient being administered nitrous oxide?
a. nausea
b. euphoria
c. giddiness
d. tingling of the hands

(d) the first three are all signs that the patient is getting too much nitrous.

176. Each of the following, EXCEPT one, is a good reason for using sedation. Which one is this EXCEPTION?
a. to allay apprehension, anxiety or fear
b. to decrease the amount of local anesthesia that is required for a given procedure
c. to alleviate stress in a severely medically compromised patient
d. to accomplish certain procedures that a practitioner would not normally be able to do on an anxious patient

(b) sedation does not decrease the local anesthetic requirement, the patient will still feel pain. Sedatives, with the exception of nitrous oxide, have no analgesic effect -barbiturate sedatives may make the patient more sensitive to pain.

179. Which of the following is classified as an antianxiety drug?
a. methohexital
b. lorazepam
c. haloperidol
d. pentazocine
e. phenylpropanolamine

(b) only benzodiazepines are actually classified as antianxiety drugs, although other drugs, such as opioids have antianxiety actions in addition to their other clinically useful actions. (b) lorazepam is the only BDZ listed. Methohexital is a barbiturate - these are classified as seatives. Haloperidol is an antipsychotic, pentazocine an opioid, and phenylpropanolamine is a decongestant used in cold medications.

Alcohol abuse occurs when someone

A. drinks more than the legal limit of alcohol.
B. experiences withdrawal symptoms when not drinking.
C. gets seriously intoxicated on a regular basis.
D. drinks enough that it puts their relationships, job, or health in jeopardy.

Answer (d)

For oral sedation in the dental setting, the most ideal group of agents is

A. narcotics.
B. barbiturates.
C. antihistamines.
D. benzodiazepines.
E. anticholinergics.

Answer (d)

122. In most cases, which of the following is the accepted dose schedule of penicillin in the treatment of actinomycosis of the jaw?

A. 250 mg. oral tablets q.i.d., 7 days
B. 500 mg. oral tablets q.i.d., 7 days
C. 600,000 units intramusculary twice daily for two weeks followed by oral medication
D. 10 million units intravenously per day for 10-14 days followed by long-term oral medication.

Answer (d)

Pharma

ANTIBIOTICS

- L.A- esters- procaine,cocaine,tetracaine,benzocaine
- Amides- rest everything
- Short acting-procaine
- Moderate- prilocaine, lidocaine, mepivacaine
- Long - bupivacaine, tetracaine, etidocaine
- Esters- plasma & amides- liver
- Prilocaine- methemoglobinemia
- 2% lido= 20gm/100ml or 20mg/1ml
- High plasma LA levels, toxicity, injected into artery- CNS excitation.(lidocaine effect)
- Increased pulse rate- too much epinephrine
- Powerful CNS stimulation/vasoconstriction - cocaine
- Penicillin gram -ve- ampicillin
- PenicillinG - acid resistant /injected
- Against penicillinase bugs- dicloxacillin
- Pseudomonas- carbencillin
- Endocarditis- ampicillin/gentamycin
- Prophy - endocarditis,shunts, valves
- Bactericidal- inhibiting cell wall synthesis
- Static- interfering w/ protein synthesis
- Nystatin binds to ergosterol
- Sulfonamides- bind to PABA in folic acid synthesis
- GI upset and pseudomonas colitis- clindamycin(overgrowth of c.dificile)
- Aplastic anemia- chloramphenicol
- Liver damage- tetracycline
- Allergic cholestatic- erythromycin estolate
- Tetracycline and penicillin cancel each other
- Probenicid alters penicillin renal clearance(decreases)
- BSA enhance coumarin action due to reduction of vit K
- Ampicillin decrease effectiveness of contraceptives
- Erythromycin inhibit metabolism of sedane,digoxin.
- Substitute for penicillin- clindamycin, erythromycin or azithromycin
- More conc. in bone than serum- clindamycin
- More in GCF than bone- tetracycline
- aplastic anemia- chloramphenicol
- streptomycin-affect 8th nerve(balance & hearing)
- amphotericinB- nephrotoxicity, hypokalemia
- tetracycline/1st gen cephalo- gram +ve & -ve
- clinda/erythro/vanco- gram +ve
- 3rd gen cephalo- gram -ve
- tetracycline-chelates with Ca

CARDIO

HYPERTENSION
•highceiling/loop acting diuretic-furosemide
•Alpha1-prazosin
•centrally acting adrenergic-clonidine
•neuronal blockers-guanethidine(severe hypertension)
•ACE inhibitor-captopril,lisinopril(Inc.BP)
•angina- nitroglycerin, verapamil, propranolol
•ARRHYTHMIAS also procainamide
Lidocaine- ventricular(decrease cardiac excitability)
Phenytoin- digitalis induced
Quinidine/verapamil- supraventricular, atrial fibrillation (Quinidine increase refractory period of cardiac muscle)
Propranolol/digitalis/verapamil- paroxysmal tachycardia
•congestive heart failure- glycosides (digitalis,digoxin),ACE inhibitors(captopril)
•type 1A agents- quinidine increase refractory period
Type 1B lidocaine decrease cardiac excitability
• digitalis- decreasing the rate of A-V conduction

ANTIANGINA
•nitroglycerin- increase O2 by vasodilation of smooth muscle
•propranolol- reduces O2 demand
•Ca channel blockers- decrease O2 demand by reducing peripheral resistance
ANTIHYPERTENSIVE
•ACE inhibitor-captopril blocks enzyme converting angio1 to angio2, reserpine/ methydopa/ thiazide diuretics.
Hydralazine(direct action on vascular smooth muscle),guanethedine (depletes cathecholamines)
Propranolol-release renin from jxt & reflex tachycardia seen with other antihypertensive drugs.

ADRENERGIC AGENTS
•Prazosin-alpha1,
Methydopa-alpha(central action) ,
clonidine-alpha2- vasodilation(vasomotor centre in medulla)
•propranolol- nonselective beta blocker(prevents chronotropic responses to endogenous epi emotions and exercise)
Beta adrenergic- increase cardiac output and antirenin effect
metoprolol -beta1- reduce cardiac output
DIURETICS- decrease renal Na absorption, fluid loss, reduce blood volume.

• thiazides- chlorothiazide(low BP)
• high ceiling/loop acting- furosemide(deafness)
• K sparing - spironolactone
•guanethidine &ganglionic blocking agents. (high BP)
CONGESTIVE HEART FAILURE
• digitalis,digitoxin- increase force of contraction of myocardium,ionotropic action.
Chlorothiazide causes hypokalemia and increases digitalis toxicity.

NSAIDS
•Aspirin-analgesic (inhibits synthesis of PG)
antipyretic-inhibits PG in hypothalamus
Bleeding time- inhibit thromboxane A2 preventing platelet synthesis.
•Aspirin v/s acetaminophen(not anti inflammatory, is hepatotoxic & doesn't cause GI upset)
•prednisone(doesn't inhibit cyclooxygenase),hydrocortisone, triamcinolone are steroids
• ibuprofen much less GI irritation
•diflunisal(dolobid) longer half life than aspirin,acetaminophen and ibuprofen.
•acetaminophen -liver toxicity w/ alcohol or >4gm/day
•avoid aspirin in fever child- reyes syndrome use acetaminophen
•salicylates inhibit PG synthesis
•heparin-inhibits thrombin & prevents formation of fibrin network
•coumarin-inhibits GI absorption of vit K & prevents synthesis of blood clotting factors
•aspirin- prevents platelet aggregation
•triamcinolone- is a corticosteroid inhibits phospholipase A2
•phenytoin- prolonged use doesn't cause gastric irritation.
•Ibuprofen less GI irritation ... acetaminophen no
•acetaminophen not cross allergenic with aspirin also causes hepatic necrosis

ANALGESICS-MORPHINE

•naloxone- antagonist to treat overdose
•Methadone - detoxification of morphome addicts cz withdrawal symptoms are less
•morphine overdose- loss of sensitivity of medullary resp. center to CO2
•codeine and meperidine suppress cough
•morphine- binds to mu receptors in CNS ,causes vomitting by stimulation of medullary chemoreceptor trigger zone
•pentazocine & nalbuphine-agonist & antagonist, avoided in heroin pts cz it causes withdrawal symptoms
•levallorphan/propoxyphene- opioid agonists
•meperidine(demerol)- opioid analgesic

AUTONOMICS

Cholinergics- •atropine,scopolamine,prpantheline- muscarinic receptor blockers controls salivary secretion.
•atropine- causes tachycardia by blocking vagal reflex control of HR
•physostigmine(peripherally & centrally)
Neostigmine(peripherally)-reversible anticholinesterases- treat xerostomia
•pilocarpine,metacholine-cholinergic agonists treat xerostomia
•organophosphates & insecticides- irreversibly inhibit colinesterase
•pralidoxime- enzyme regenerator used in organophosphate toxicity
•succinylcholine- NMJ blocker, rapid inactivation by plasma pseudocholinesterase, prevents laryngospasm
•d-tubocurarine- non depolarising NMJ blocker
•methamylamine & hexamethonium- ganglionic blockers producer orthostatic hypotension.
•cholinergic crisis- give atropine
•scopolamine overdose- give Physiostigmine
• botulinum toxin-preventing Ach release
•hemicholinium- prevents Ach synthesis
•curarine-(competitive)nicotine receptor blocker muscle paralysis
•edrophonium-used for myasthenia gravis
•succinylcholine(depolarising)/gallamine-paralysis
•neostigmine-stimulates denervated muscle at end plate
•methacholine,neostigmine &pilocarpine increase Ach duration
•neostigmine-given in xerostomia
•propantheline/atropine/methantheline- given when excess saliva
•ganglionic blocking agents- orthostatic hypotension
•tachycardia- vagus nerve blocked
•succinylcholine- muscle fasciculation an agonist at nicotinic receptors,short acting cz inactivated by plasma esterases
•neostigmine-helps reverse skeletal muscle paralysis
•atropine overdose-Cns excitation and tachycardia
•scopolamine overdose is treated by physostigmine
•irreversible cholinesterase inhibitor- death due to respiratory paralysis

ADRENERGICS

•prazosin or propranolol-competitive inhibition
•reserpine-depletes NE by inhibiting reuptake
•guanethidine-inhibits release of cathecholamines
•alpha methyldopa-false neurotransmitter taken into storage vesicle & released with NE decreasing sympathetic activity at central nuclei
•clonidine-stimulates alpha2 receptors in CNS with decrease in sympathetic activity

•amphetamine,tyramine,ephidrine-stimulating release of stored NE
•TCA's and cocaine- block reuptake
•MAOI's block enzymatic destruction
•alpha blocker prazosin/chlorprazine - epi causes vasodilation
•vagal reflex due to pressor dose of NE- blocked by atropine
•Parkinson's- levodopa+carbidopa
•alpha1- vasocomstriction-inc.BP
•alpha2- hypotension,reducing sympathetic outflow
•beta1-inc.cardiac rate and force of contraction
•beta2-dilation of skeleyal muscles and bronchi
OR
•eye-mydriasis
•heart-acceleration,Inc.contractility
•vascular smooth muscle-vasoconstriction
•skeletaluscle vessels-relaxation/dilation
•bronchodilation &sweating
•phentolamine(a blocker)+epi- bronchodilation,+ve chronotropic and ionotropic effects,dilation of skeletal muscle vascular beds
•epi-bronchodilation,liver glycogenolysis,rise in BP,extrasystoles in heart,restlessness and anxiety
•prazosin and chlorprazine(a blocker),atropine cholinergic muscarinic blocker,propranolol beta blocker,neostigmine cholinesterase inhibitor,isoproterenol is beta receptor against.
•phenylephrine,levonordephrine-alpha receptor agonist
•methoxamine- stimulates alpha receptors and is a vasoconstrictor
•NE -stimulates alpha and beta 1 >beta 2
•phentolamine- nonselective alpha blocker
•levodopa can stimulate beta 1 receptor in heart and contraindicated with epi
•albuterol-beta 2 agonist

SEDATIVES
Benzodiazepines- diazepam, chlordiazepoxide
Benzodiazepines converted to active metabolites thus short acting are-diazepam, flurazepam,chlordiazepoxide
Not converted are- oxazepam,midazolam,lorazepam

•modulate the activity of inhibitory neurotransmitter,GABA
Barbiturates- thiopental-enters and exists brain rapidly,increase secretions
•Barbiturate overdose can kill you cz of respiratory depression
• enhance porphyrin synthesis
•Barbiturates- metabolised in liver, classified according to duration of action and depress all levels of CNS.
•diazepam-thrombophebitis (select large vein)
•benzodiazepine- diazepam(Valium)

Recent one is triazolam(halcion)
•midazolam over diazepam- less resp. depression
•Flumazenil reverses benzodiazepine
•Naloxone reverses opioids

PSYCHOTICS-used for schizophrenia
•clozapine-block dopamine and affect hypothalamus temp regulation. serotonin receptors,few sides effects like tardive dyskinesia,reduces salivation
•phenothiazine(extrapyridal side effects) & chlorprazine also haloperidol & thioridazine
•dilantin is anticonvulsant

ANTIDEPRESSANT
•tricyclic -imipramine or amitriptyline
•MAO inhibitors- tranylcypromine or phenylene
•2nd gen- fluoxetine(prozac) and trazodone
Mechanism- blockade of amine reuptake at synaptic cleft
Side effects-anticholinergic or atropine
•lithium -manic depression

ANTI-INFLAMMATORY

• they are corticosteroid or glucocorticoids- also suppress immunity

GA
•onset of anaesthesia inversely proportional to solubility
•halothane-hepatotoxicity
I-analgesia,II-delirium,III-surgical anaesthesia,IV-medullary paralysis
•Hb doesn't influence induction
•halogenated hydrocarbon-liver damage
•medulla oblongata-most resistant
•atropine given before halothane to reduce salivation and bronchial secretions

ANTIHISTAMINES
•H1(competitive antagonism) given for derma related- chlorpromazine, chlorpheniramine
-preop for sedation,antiemetic, anticholinergic effects-promethazine
-controlling symptoms of parkinsonism -diphenhydramine
•H2 cimetidine- reduce gastric acid secretion
•diphenhydramine has anticholinergic activity in parkinsonism NOT DOPAMINERGIC
• carbamazepine,phenytoin- trigeminal neuralgia
•cardiac arrhythmia - main side effect of epi
•thromboembolism main side effect of oral contraceptives
•alcohol inhibits GABA effects in cortex of CNS

•sulfonyl ureas cause insulin secretion by direct stimulation of pancreatic beta cells
•dicoumarol prevents blood clotting
Agonist-antagonist
•morphine-naloxone
•protamine-heparin
•carbon monoxide-O2
Insulin preps differ in onset & duration- fast-insulin, intermediate-isophane insulin, long acting- protamine zinc insulin
•adrenal insufficiency/Addison disease- 2ml(100mg) of hydrocortisone hemisuccinate given for hyponatremia
•methotrexate-antimetabolite(inhibits DNA synthesis during S phase)
Antimetabolite acts 2 ways by either forming false metabolites that are nonfunctional or inhibition of catalytic function of enzyme
•physostigmine treats scopolamine overdose
•ethyl alcohol-methylphenidate(Ritalin) not synergistic to it cz its CNS stimulant
•nalbuphine- shouldn't be given for pain in heroin addicts
•lidocaine-arrythmias
•anticholinergic- glaucoma
•buspirone-least CNS depression
•propoxyphene<codeine<pentazocine<oxycodone for dependence
•polymixin-renal necrosis , chloramphenicol-bome marrow depression, amphotericinB-nephrotoxicity, gentamycin-deafness
•opioids- least cause xerostomia
•ephedrine,tyramine,amphetamine- indirect acting sympathomimetic
•mu-supraspinal & kappa-spinal analgesia
•propylene glycol-thrombophebitis

Quizlet Tufts Pharmacology NBDE Part II

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1st generation antipsychotic drugs	Phenothiazine or Haloperidol, specific D2 (dopamine) receptor blocker
2nd generation antidepressant drugs	Fluoxetine or Trazadone, much more commonly used now, block amine reuptake or alterations of receptor number
2nd generation antipsychotic drugs	Clozapine, block dopamine receptors and serotonin 5HT receptors, treat negative and positive symptoms, have fewer extrapyramidal side effects
Acetaminophen	no anti-inflammatory activity, is hepatotoxic, does not cause GI upset, liver toxicity esp when combined with alcohol or taking 4g/day, is choice for feverish kid, may induce methemoglobinemia at high doses
AHA limit of epinephrine that pt with CV disease can have	0.04mg normal pt is 0.2mg
Albuterol	beta2 agonist for bronchodilatory effects
Allergic reactions to penicillins	dermatitis, stomatitis, bronchoconstriction, cardiovascular collapse
Alpha1 receptor stimulation	vasoconstriction, urinary retention, mydriasis
Alpha2 receptor stimulation	hypotention, reduces sympathetic outflow from CNS
Alpha methyldopa	acts centrally as false neurotransmitter which gets taken up into storage vesicles and is released with norepinephrine, decreases sympathetic activity, reduces sympathetic outflow via alpha agonist action
Amide anesthetics	lidocaine, mepivacaine, bupivacaine, prilocaine, dibucaine, metabolized in the liver
Amphetamine	stimulates release of stored norepinephrine and stimulates alpha receptors in CNS
Amphotericin B adverse effects	nephrotoxicity and hypokalemia
Ampicillin	best gram negative spectrum
Anesthetics mechanism of action	prevent generation of nerve impulses by interfering with sodium transport into neuron, only non-ionized form can penetrate tissue membranes because inflamed tissue has a lower than normal pH, the amount of non-ionized form available to penetrate is decreased
Anesthetics that are vasodilators	procaine, lidocaine, tetracaine, mepivacaine
Antibiotics interaction with coumarin	deplete vitK sources so will enhance coumarin anticoagulants
Antibiotics interaction with oral conctraceptives	suppress normal flora involved in active steroids from bile conjugates => more rapid excretion of steroid from body
Antipsychotics	mostly dopaminergic receptor blockers, are often used as antiemetic drugs
Aspirin adverse/toxic effects	occult bleeding from GI tract, tinnitus, nausea and vomiting, acid-base disturbance, metabolic acidosis, decreased tubular resorption of uric acid, salicylism, delirium, hyperventilation
Aspirin mechanisms of action	analgesic effects: inhibits synthesis of prostaglandins antipyretic effects: inhibits synthesis of prostaglandins in hypothalamus, cutaneous vasodilation bleeding time: inhibits thromboxane A2 synthesis and thus platelet aggregation slows
Aspirin therapeutic effects	pain relief, antipyretic effects, antirheumatic, anti-inflammatory effects
Atropine	competitive muscarinic receptor blocker blocks vagal reflexive control of heart rate => results in tachycardia
Barbiturate contraindications	intermittent porphyria: will enhance porphyrin synthesis and will aggravate the disease undiagnosed severe pain: may make the pain worse and result in arousal, rage, delirium emphysema
Barbiturates	CNS depressants, will depress all levels of CNS, are not analgesic, will often induce excessive salivation and bronchial secretion and require use of anticholinergic drug to reduce these, are metabolized by the liver, are classified according to duration of action
Barbiturate toxicity	overdose kills you because of respiratory depression
Barbiturate toxicity treatment	need to maintain open airway, increase input of afferent stimuli, maintain respiration, administer CNS stimulant
Benzodiazepine adverse effects	IV injection of diazepam can cause irritation like thrombophletbitis due to solvent (propylene glycol)

29. **Benzodiazepines** — modulates the action of inhibitory neurotransmitter GABA, many form active metabolites, is most common drug group given for oral sedation ex: diazepam, chlordiazepoxide
30. **Benzodiazepines > barbiturates** — less addiction potential, less profound CNS depression, larger therapeutic index, less resp depression
31. **Beta1 receptor stimulation** — increased heart rate, increased force of contraction, positive inotropic and chronotropic actions
32. **Beta2 receptor stimulation** — bronchodilation, vasodilation, dilation of skeletal muscle
33. **Captopril** — CHF, ACE inhibitor
34. **Captopril, lisinopril** — HTN, ACE inhibitors
35. **Carbenicillin** — extended spectrum, specific for Pseudomonas infections and indole-positive Proteus species
36. **Cephalosporins 1st gen** — effective against both gram negative and gram positive organisms
37. **Cephalosporins 3rd gen** — increased activity against gram negative, greatly decreased activity against gram positive
38. **Chloramphenicol** — antbiotic, associated with aplastic anemia
39. **Chlorothiazide** — HTN, diuretic, thiazide, when administered with digitalis, will increase penetration of digitalis into myocardium
40. **Chlorpromazine** — prototypic phenothiazine, used in treatment of schizophrenia
41. **Clindamycin** — higher concentration in bone than in serum, mostly affects gram positive organisms
42. **Clindamycin side effects** — GI upset and pseudomonas colitis
43. **Clonidine** — stimulates alpha2 receptors in CNS with resulting decrease in sympathetic outflow
44. **Clonidine** — HTN, stimulates alpha2 receptors in CNS
45. **Cocaine** — norepinephrine reuptake inhibition and release
46. **Codeine** — is the best opioid for suppressing cough reflex
47. **Competitive muscarinic receptor blockers** — atropine, scopolamine, propantheline, are sometimes used to control salivary secretions
48. **Corticosteroids (like Prednisone, Hydrocortisone, Triamcinolone)** — inhibit phospholipase A2, enzymatic step that precedes prostaglandin synthetase
49. **Corticosteroids or glucocorticoids** — suppress immune system in addition to anti-inflammatory activity, so latent infection like TB may go systemic and opportunistic infections like Candidiasis may become more of a problem
50. **Cross-allergenic with penicillin** — cephalosporins and ampicillins are erythromycin is not
51. **Diazepam** — benzodiazepine, Valium, is given most commonly for oral sedation
52. **Dicloxacillin** — penicillin useful against penicillinase-producing bugs (like staphylococcus)
53. **Diflunisal (Dolobid)** — salicylate analgesic, longer half-life than acetaminophen and ibuprofen
54. **Digitalis** — arrhythmia, decreases rate of AV conduction, for atrial fibrillation and paroxysmal tachycardia
55. **Digitalis toxicity** — nausea and vomiting, yellow-green vision, extrasystole, AV conduction block related to coadministration with chlorothiazide
56. **Direct acting cholinergic agonists** — pilocarpine, methacholine, may be used for xerostomia
57. **d-tubocurarine** — non-depolarizing neuromuscular junction blocker
58. **Ephedrine** — causes release of stored norepinephrine and acts at receptor itself
59. **Epinephrine** — rise in BP due to myocardial stimulation that increases ventricular contraction, increase in heart rate, vasoconstriction because of alpha receptor stimulation
60. **Epinephrine reversal** — when epinephrine is administered in the presence of an alpha blocker (Prazosin or Chlorpromaxine), will cause decrease in BP rather than increase because beta-mediated vasodilation predominates
61. **Epinephrine toxicity** — elevated pulse rate in pt's with Grave's disease, will have heightened sympathetic activity and could result in hypertensive crisis
62. **Erythromycin estolate** — associated with allergic cholestatic hepatitis
63. **Ester anesthetics** — procaine, tetracaine, cocaine, metabolized by esterases in the plasma and some in the liver
64. **Ethyacrinic acid** — HTN, loop or high ceiling diuretic, is associated with deafness
65. **Flumazenil** — reverses effect of benzodiazepines
66. **Furosemide** — HTN, diuretic, high-ceiling or loop acting
67. **Ganglionic blockers** — mecamylamine and hexamethonium, produce orthostatic hypotension

68.	**General anesthesia onset and rate of induction**	inversely proportional to solubility of anesthetic in the blood, also influenced by pulmonary ventilation, blood supply to lungs, concentration of anesthetic in inspired mixture
69.	**Glycosides**	CHF, ex: digitalis and digoxin, have positive inotropic effect, increasing force of contraction of myocardium by inhibiting Na+/K+ ATPase and thus increasing Ca2+ influx, reduces compensatory changes associated with CHF like heart size, rate, edema
70.	**Guanethidine**	inhibits release of catecholamines (like norepinephrine)
71.	**Guanethidine**	HTN, neuronal blockers, only for severe hypertension, prevents release and causes depletion of catecholamines taken up into storage vesicles and is released like false transmitter, does not cross blood-brain barrier
72.	**Halothane**	associated with hepatotoxicity, may use atropine before to reduce salivation and bronchial secretions
73.	**Hydralazine**	HTN, directly acts to vasodilate vascular smooth muscle
74.	**Ibuprofen**	much less GI irritation, is anti-inflammatory, will have gastric irritation and bleeding after prolonged use
75.	**Idiosyncratic reactions**	genetically determined abnormal responses to a drug, are most unpredictable because may not be shown until drug is taken for the first time by a pt ex: succinylcholine and atypical plasma cholinesterase
76.	**Irreversible inhibitors of cholinesterase**	organophosphates and insecticides
77.	**Isopreterenol**	beta2 receptor stimulator
78.	**Levodopa with Carbidopa**	levodopa: is a dopamine precursor that can cross the blood brain barrier carbidopa: is a dopa decarboxylase converter blocker used to treat Parkinson's
79.	**Lidocaine**	interacts with propranolol by slowing down heart via beta receptor blockade and keeping lidocaine in the circulation longer and causing toxicity and by competing for the same enzyme in the liver
80.	**Lidocaine (Type 1B drugs)**	arrhythmia, decrease cardiac excitability, for ventricular arrhythmias
81.	**Lithium**	drug of choice for manic phase of manic depression (bipolar disorder)
82.	**Long acting anesthetics**	bupivacaine, tetracaine, etidocaine
83.	**Macrolide interactions**	inhibit metabolism of drugs like seldane, digoxin erythromycin blocks the metabolism of seldane to antihistamine metabolity => will stay unmetabolized and cause cardiac arrhythmias
84.	**MAO inhibitors**	Tranylcypromine or Phenylene
85.	**MAO inhibitors**	blocks enzymatic destruction of norepinephrine
86.	**Methadone**	used in detox of morphine addicts, is full agonist with analgesic properties, when taken orally is not euphoric in addicts, just acts to produce tolerance and physical dependence, withdrawal is less severe because of long half-life
87.	**Methoxamine**	vasoconstrictor that stimulates alpha receptors
88.	**Methyldopa**	HTN, acts centrally to stimulate alpha receptors
89.	**Metoprolol**	HTN, selective beta1 blocker
90.	**Midazolam**	benzodiazepine, water soluble (doesn't cause thrombophlebitis), shorter acting than valium because it doesn't have active metabolites, has more rapid and predictable onset of action when given IM than valium
91.	**Moderate acting anesthetics**	prilocaine, mepivacaine, lidocaine
92.	**Morphine effects**	respiratory depression, euphoria, sedation, dysphoria, analgesia, constipation, urinary retention
93.	**Morphine mechanism of action**	binds mu receptors in CNS causes vomiting by stimulating medullary chemoreceptor trigger zone decrease in ventilation due to loss of sensitivity of medullary resp center to CO2
94.	**Morphine overdose**	coma, respiratory depression, miosis
95.	**Nalbuphine**	mixed agonist-antagonists, has both agonistic and antagonistic activites
96.	**Nalozone**	antagonist to treat overdose of morphine
97.	**Neostigmine**	reversible anticholinesterase, acts peripherally only, has some direct ACh-like activity at NMJ => prolongs activity of endogenous ACh, sometimes for treating xerostomia
98.	**Nitroglycerin**	angina, increases oxygen supply to heart by direct vasodilatory action on smooth muscle in coronary arteries
99.	**Norepinephrine**	leads to decreased heart rate because of baroreceptor reflexes, stimulates alpha and beta1 receptors
100.	**Opioids**	Meperidine, morphine, codeine

101. **Penicillin G**	more sensitive to acid degradation, so is usually injected rather than taken orally, not really used that much anymore
102. **Pentazocine**	mixed agonist-antagonists, has both agonistic and antagonistic activities
103. **Phenobarbital**	long acting barbiturate
104. **Phentolamine**	nonselective alpha blocker, will cause vasodilation
105. **Phenylephrine**	alpha1 receptor agonist
106. **Phenytoin**	arrhythmia, to reverse digitalis induced arrhythmias
107. **Physiological antagonism**	two drugs produce opposite effects but don't act on the same receptor ex: epinephrine and histamine, epinephrine and nitroglycerin
108. **Physostigmine**	reversible anticholinesterase, acts both centrally and peripherally, sometimes for treating xerostomia
109. **Pralidoxime**	enzyme regenerator used in organophosphate toxicity
110. **Prazosin**	HTN, alpha1 blocker, inhibits release of norepinephrine
111. **Prazosin**	alpha blocker, competitive inhibitor of postjunctional adrenergic receptors, associated with epinephrine reversal
112. **Prilocaine**	can cause methemoglobinemia because of toluidine metabolite called orthotoluidine
113. **Probencid**	alters rate of renal clearance of penicillin, is a uricosuric agent that tends to enhance excretion of uric acid by reducing renal tubular transport mechanisms
114. **Prophylaxis for prosthetic joint**	Keflex 2g, take 1hr before tx
115. **Prophylaxis no-no's**	don't use tetracycline because endocarditis is streptococcal infection and some are resistant to tetracyclines
116. **Propranolol**	beta blocker, competitive inhibitor of postjunctional adrenergic receptors
117. **Propranolol**	HTN, nonselective beta blocker
118. **Propranolol**	arrhythmia, for paroxysmal tachycardia
119. **Propranolol**	angina, reduces oxygen demand by preventing chronotropic responses to endogenous epinephrine
120. **Quinidine (Type 1A drugs)**	arrhythmia, increases refractory period of cardiac muscle, for supraventricular tachyarrhythmias and atrial fibrillation
121. **Reserpine**	depletes norepinephrine by inhibiting reuptake, causes depletiong from storage sites
122. **Short acting anesthetics**	procaine
123. **Side effects of 1st generation antipsychotic drugs**	anticholinergic effects and anti-alpha adrenergic side effects, extrapyramidal stimulation resulting in tardive dyskinesia, may have jaundice due to allergic reaction
124. **Side effects of 2nd generation antidepressant drugs**	anticholinergic side effects
125. **Side effects of corticosteroids or glucocorticoids**	gastric ulcers, immunosuppression, acute adrenal insufficiency, osteoporosis, hyperglycemia, redistribution of body fat
126. **Spironolactone**	HTN, diuretic, potassium sparing
127. **Stages of anesthesia**	I: analgesia II: delirium III: surgical anesthesia IV: medullary paralysis (once you start depressing medullary centers, pt will stop breathing and die)
128. **Streptomycin adverse effects**	8th nerve damage, will affect balance and healing
129. **Succinylcholine**	agonist at nicotinic receptors, depolarizing NMJ blocker subject to rapid inactivation by plasma pseudocholinesterase, used to prevent laryngospasm paralyzing dose causes muscle stimulation
130. **Sulfonamides**	compete with PABA in folic acid synthesis so there is folic acid deficiency
131. **TCA antidepressant**	norepinephrine reuptake inhibition
132. **Tetracycline**	higher concentration in gingival fluid than in serum, pretty broad spectrum against gram positive and negative cocci and bacilli
133. **Tetracycline adverse effects**	liver damage or hepatotoxicity, esp in pregnant pts with history of renal disease, superinfection, photosensitivity, discoloration of newly forming teeth, GI symptoms, diarrhea
134. **Tetracycline and penicillin**	cancel each other out because they ahve opposing mechanisms of action
135. **Tetracycline interactions**	will chelate with calcium, reduced by concurrent ingestion of antacids or dairy products
136. **Thiopental**	action is terminated by redistribution of drug out of the chain, will enter and exit the brain rapidly, thus quick onset and short duration of action

137.	**Toxic reactions to anesthetic**	mostly related to excessive blood levels arising from inadvertent intravascular injection CNS stimulation because of inhibition of central inhibitory neurons at higher doses, will inhibit inhibitory and excitatory neurons => generalized state of CNS depression => respiratory depression and death
138.	**Triazolam**	benzodiazepine, Halcion, is ultrashort acting version
139.	**Tricycline antidepressants**	Imipramine or Amitriptyline, are reuptake inhibitors for amine neurotransmitters, were most commonly used in the past, are strong anticholinergics
140.	**Vagal reflex**	injection of pressor dose of norepinephrine may result in decreased heart rate due to activation of baroreceptors that stimulate vagal reflex to reduce heart rate
141.	**Verapamil**	angina, Ca2+ channel blocker, decrease oxygen demand by reducing afterload by reducing peripheral resistance via vasodilation
142.	**Verapamil**	arrhythmia, for supraventricular tachyarrhythmias and paroxysmal tachycardia and atrial fibrillation