ATI PHARMACOLOGY EXAM 1 REVIEW AND EXAM PASSING

1. FDA process for drug approval (legal)
    a. Phases I, II, II, clinical trials
    b. Which Trial does the drug have to pass to be deemed safe and effective?

       Phase III
    c. Phase IV (post Marketing) Prescribers to report any issues with medication
2. Patient rights and drug testing (ethics)
    a. Participation in Clinical Trials
    b. Informed consent
    c. Safety of human subjects
    d. Ethical Principles Define: Autonomy, Beneficence, Non-maleficence, Veracity,

       Justice
    e. Regulations: HIPPA (confidentiality of identifiable patient personal information)
    f. What are the elements of a complete medication order?

       Drug type, dose, frequency and time, route, patient, and document
3. Scheduled drugs I-V (controlled substances) and nurse's role – (legal)
    a. What do the schedules mean in terms of addictive properties? It means how likely a drug is to be misused or addictive

4. Pharmacogenetics and pharmacogenomics
    a. What are genetic polymorphisms?

The gene is the primary organ of hereditary and genetic polymorphism is the one-nucleotide difference in 1% of population that can cause discrepancies in how effective a drug is to the body.

b. How do these impact responses to drugs?

c. Why is considering a patient's racial or ethnic heritage important in what medications and doses they are given?

SO we don't overdose them or give them medications that will hurt them.

5. Medication safety and national patient safety goals related to medications

Need to have 2 forms of ID: state your name and DOB, check the arm band, check the medication against the order and then against label and the label to the ID band before given

a. Medication reconciliation - why is this process important?

Crucial for patient safety and established repor with patients and you don't want to be sending a discontinued medication to the pharmacy.

b. What are LASA drugs (look alike sound alike drugs)? (Give an example)

Example: Paxil can look like Plavix

c. What are High alert drugs? (Give an example)

Drugs given and can cause lots of problems- diabetic drugs, anticoags, and antiplatelet drugs

d. What are the 9 rights of medication administration? (6 main rights and 3 other rights)

Way to prevent medication error. Helps establish repor with patients. You want them to ask questions.

Know the 9 rights- focuses on the 6. Rights: drug, patient, dose, route, time, right documentation.

e. What are methods of preventing medication errors?

Med reconciliation, checking dose 3 times, checking med 3 times before you give it, check right patient, check the 9 rights and speak up and ask questions

f. What are factors affecting patient adherence to drug therapy?
g. What is polypharmacy? Person taking many medications, herbals, or OTC.

Older population experiences this more than any other people

h. Who is most likely to experience poly pharmacy?

6. Differences between prescribed, OTC, medications, and dietary supplements

OTC and prescribes med are approved by FDA, Dietary supplements and herbal are not. Some vitamins have USP classification, but they are not FDA approved. They can cause drug interactions- let provider know.

a. What are the patient teaching issues for each?
b. What is the importance of the patient and nurse being aware of the number and type of active ingredients? Know the number of ingredients in medication because it can cause interaction.
c. Should medication reconciliation include prescription, OTC, Herbal and nutritional supplements, alcohol, and nicotine use? YES, ALL of those are drugs

7. Difference between generic and trade names

SKIP- will use both in exam

a. Which is safest to use? Generic
b. Can a generic drug have more than one trade name? YES

8. **Pharmacokinetics – what body does to the drug**

How do the following pharmacokinetic processes affect drug therapy?

a. Absorption

i. Importance of water on absorption-We tell patients to take with full glass of water unless told other wise ii. Effect of food on absorption- Sometimes it will delay absorption, its dependent on bioavailability.

iii. How do different formulations of drugs (solution, suspension, capsule, tablet, solid) affect absorption? Solution more rapidly than solid. Suspension, you need to shake if given orally. If suspension injection, reconstitute with saline and role with hands between administered.

1. Factors affecting absorption

What are the effects of the following on drug absorption?

i. Stomach pH – will affect if the drug is in an ionized or ionized state.- look at the animation in blackboard.

Ex: If a patient has to take an antibiotic and if they are on an antacid, they need to take antibiotic and hour before or 2 hours after antacid, so no change in gastric ph.

What about if patient is sick and hypotensive? GI blood supply good? No so we will give meds IV route.

ii. Ionization versus non-ionization of the drug to be absorbediii. Other drugs (For example: Think about antacids taken at the same time as an antibiotic)

Ex: If a patient has to take an antibiotic and if they are on an antacid, they need to take antibiotic and hour before or 2 hours after antacid, so no change in gastric ph.

iv Gastric Blood flow and patient's health status

2. What are the nursing considerations when administering and educatintion patients about Extended-release and Enteric coated medications? They cannot be crushed

or chewed so we need to tell patients that. If a pill has groove means "scored" so it can be cut in half. We also cant out these drug forms down a feeding tube because we would need to crush it and mix with water, suck in syringe and flush with water. Cant block the tube- but regardless don't use them this way!

3. Bioavailability- Means the extent to which the drug is absorbed (Poor bioavailability (needs taken on empty stomach) like thyroid medication or osteoporosis medication means patient needs to take the drug on an empty stomach)
4. Which routes of administration produce local versus systemic effects?

Local effects are by nasal inhalation or topical route

Systemic effects by injection or GI route

   i. Which routes are more likely to cause side effects?
   ii. Can topical medications ever cause systemic effects? Yes, rectal can and so

can topical is high does or heat can cause.

   iii. Do nasal steroid and inhaled steroid produce local or systemic effects?

Inhaled steroids are local effect but inhaled anesthesia is

syatemic. b. **Distribution** To: Blood then tissue Drug needs

to bind to albumin (protein).

Bound drug- is inert and doesn't function, no pharm effect. When molecules come off its free drug and we want to see same amount coming in as going out. We reach steady state and equilibrium.

Free drug has pharm effect. Look at animations!!!!

1 Protein binding (plasma protein binding = PPB)

2. Low albumin < 3.0 (hypo-albuminemia) and highly PPB drugs- toxicity –why?

i. What patient factors contributing to low albumin? Poor diet, genetics ii. What are the effects of bound versus free drug? (See Blackboard animation) iii. What is Equilibrium? How does it relate to steady state? The amount of drugs coming in as are leaving to the cellular site of action.

iv. What is competitive protein binding? What may result from this competition? Drug B comes along and if Albumin has a larger specificity and affinity for that Drug B, then it will knock Drug A right off. So we have to be careful so we don't have toxic level of drug A.

Ex: Coumadin binds to Albumin but if you also take aspirin will knock Coumadin off the receptor site and there will be freer Coumadin (and more of it) in the blood leading to toxicity.

3. Protective processes:

i. Brain-Blood brain barrier- protects the brain by preventing harmful substancesfrom entering brain tissues and CSF

ii. P-glycoprotein – a chemical in cell membranes that pumps toxic substances out back out. For example: P-glycoprotein protects the fetus by pumping drugs back into the maternal circulation from the placenta

c. **Metabolism**

What is the purpose of metabolism? Change the drug to water-soluble compound that can be eliminated by kidneys.

If by GI route- it will go to GI tract and will be absorbed, by portal vein to the liver- liver will metabolize- blood will go to right side of heart and

lungs and left side and circulate again and go back to liver to hepatic artery.

All drugs will be metabolized by liver.

GI tract- has fist pass effect since its metabolized first

i. What is the First pass effect? What route of administration is involved in first pass metabolism?
ii. Do drugs that do not go through a first pass effect get metabolized in the liver? Think about the blood supply to the liver.
iii. What are CYP450 inducers?

Ex: Drug A needs enzymes to metabolize it so it's considered a substrate drug

Drug B (a CYP450 inducer) is introduced and it is telling liver to make more metabolizing enzymes (CYP 450) so Drug A is metabolized faster and more completely so lower therapeutic effect of that drug

iv. What are CYP 450 inhibitors?

Ex: If we have Drug A and requires enzymes (CYP450) for metabolism and Drug B (CYP450 inhibitor) is introduced, its going to tell liver to make fewer metabolic enzymes, so free level of drug will be accumulating in body and patient will get toxicity.

-May not know right away-

Some drugs, wont do anything until liver metabolizes them to active metabolite- this is called a **pro drug**

Once metabolism is complete, we have metabolite. These usually are **nontoxic** and **non active**- we urinate them off.

A **pro drug** is active metabolite and some are toxic. Tylenol is produced into a toxic metabolite. Some people take excess and can cause liver failure. And is a cause for liver transplant

v. What are the drug interactions, and consequences of drug interactions between CYP450 substrate drugs and an inhibitor or inducer drugs?

Remember enzymes work on substrates. (See Blackboard animations) d. **Excretion**:

i. What is the main route by which drugs are excreted from the body? Kidney

ii. What does serum creatinine tell us about kidney function and drug clearance? Creatine is the byproduct of protein met and serves as marker for telling us how well kidney

is functioning and how it can clear (rid of) drug. Serum Creatinin 0.8-1.4. Anything above 1.4 is bad

Ex: If patient came in and CREAT 1.4 and next day is 2.4, we would know that something drastic has happened and that the patient is unable to clear drug as well.

iii. What is the half-life of a drug? What does that mean? Tine it takes to get half the dose of drug out of the body. Some are short some are long How many half life's does it take to get a steady state.

Ex: If a drug is 24 hour half life, 2 mg- and we agree that 4 half life's is what it takes to almost reach at steady state. At 5 it is steady state. Between 4 and 5. Look at animation.

Ex: 48 hour half-life= so that's 2 days. 4 half-life's to reach steady state. 2x4= 8, that would mean 8 days to reach steady state.

Ex: 7 days half-life. Taking it everyday. 7x4- 28 days for drug to reach steady state. iv. How many half -lives does it take to reach steady

state? 4 v. How does this affect the dosing interval for a drug? vi. Steady state- what is this? (look at the image in Blackboard) vii. How does a loading dose affect steady state?

What happens if we have to get to stead state faster? We give a dose that's higher than the normal daily does given. Also known as loading dose or bolus.

9. **Pharmacodynamics- drug on body**

a. How drugs work-

i. Mechanism of action (MOA) (receptors, enzymes, or others)- How a drug works with a receptor or enzyme ii. What is the difference between drug actions versus drug effect? Is it binding or inhibiting enzyme iii. What is the difference between the therapeutic drug effects versus an adverse drug response?

TDR- Why you gave the drug in the first place and or the expected outcome.

ADE/ADR- Anything that shouldn't happen. Allergy, side effect.

iv. Define Specificity and affinity

Look in notes in first lecture

v. Define Agonist versus antagonist

Agonist- makes something happen

Antagonist- prevents something from happening. vi. Define Efficacy versus potency

Ex: 10 mg morphine is potent 75 mg of Demerol vii. What is a drug's therapeutic index (margin of safety)? Why the caution with narrow therapeutic index drugs?

Difference between dose of drug that produces effect and a drug that produces toxicity. Narrow margin of safety- we need to draw blood levels. Many drugs with narrow TI

viii. Many drugs with narrow therapeutic indices require drug level monitoring ix. Define therapeutic outcome / effect relative to a drugs action

b. Adverse drug events:

ADRs (Adverse drug reactions)

1. Allergy

a. Requires the involvement of the immune system IgE

i. May be mild ii. May be severe-

anaphylaxis- / angioedema

2. Toxic effects – Often related to the class of drug, high doses, or organ system susceptibility

i. Class Effect- toxicity known to all members of a drug class ii. Organ specific – Cardio toxicity; Hepatotoxicity; Nephrotoxicity; Ototoxicity iii. Teratogenic iv. Mutagenic

3. Pharmacogenetics-related reactions (idiosyncratic)-(**unpredictable**)

4. Side effects are adverse drug reactions (ADRs) but is not usually life threatening (eg.

Rash, nausea, diarrhea)

i. Bothersome may be mild or severe-

predictable ii. Not the same as allergy iii.

Nursing - know early signs and symptoms iv.

Awareness of drug-drug interactions

v. Awareness of synergism (potentiation)

10. **<u>The big 3 (Modified Nursing Process)</u>** The Pharmacology Equivalent of the Nursing