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Date = 26/9/20

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PROGRAMME # BS (DENTAL TECHNOLOGY)

PAPER # PHARMACOLOGY

SEMESTER # 3<sup>rd</sup>

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26/9/20

## QUESTION NO: 3

Differentiate between general and local anaesthesia, Explain stages of anaesthesia in detail.

● ANSWER:-

### DIFFERENTIATE BETWEEN GENERAL AND LOCAL ANESTHESIA:-

FEATURES	GENERAL ANESTHESIA	LOCAL ANESTHESIA.
• Site of action	Central Nervous System	Peripheral nerves
• Area of body involved	whole body	Restriated area
• Consciousness	Lost	Unaltered
• Care of vital functions	Essential	Usually not needed
• Poor health Patients	Risky	Safer



	Possible	Not possible
• Use in non cooperative patients		
• Major Surgery	Preferred	Cannot be Preferred
• Minor surgery	Not preferred	Preferred

Explain stages of anesthesia in detail.

ANSWER:-

### STAGES OF ANESTHESIA:-

#### STAGE: 1 ANALGESIA:-

- Analgesia without amnesia

#### STAGE: 2 EXCITEMENTS:-

- Nausea, vomiting, hyperreactivity, irregular respiration

#### STAGE: 3 SURGICAL ANESTHESIA:-

- Sleep, normal respiration and blood pressure.

## • STAGE: 4 MEDULLARY DEPRESSION.

- Depression of vasomotor and respiratory centers coma and death.

## • STAGE: 1 ANALGESIA.

- Starts from beginning of anesthetic inhalation and lasts upto loss of consciousness.
- Pain is progressively abolished during this stage.
- Patients remains conscious, can hear, and see, and feels a dream like state.
- Reflexes and respiration remain normal.
- It is difficult to maintain use is limited to short procedure only.



## STAGE : 2 STAGE OF DELIRIUM AND EXCITEMENT:-

From loss of consciousness, to beginning of regular respiration

- Excitement - patient may shout, struggle and hold his breath.
- Muscle tone increases, jaws are tightly closed
- Breathing is jerky; vomiting, involuntary micturition or defecation may occur.
- Heart rate and BP may rise and pupils dilate due to sympathetic stimulation.
- No stimulus or operative procedure carried out during this stage
- Breathing are commonly seen. Potentially dangerous responses can occur during this stage including vomiting, laryngospasm and uncontrolled movement.
- This stage is not found with modern anaesthesia paraneosthetic medication, rapid induction etc.

## STAGE: 3 STAGE OF SURGICAL ANESTHESIA:-

-> Extends from onset of regular respiration to cessation of spontaneous breathing.

This has been divided into 4 planes: plane

-> 1 Roving eye balls. This plane ends when eyes become fixed.

-> Plane: 2: Loss of corneal and laryngeal reflexes.

-> Plane: 3 Pupil starts dilating and light reflex is lost

-> Plane: 4 Intercostal paralysis, shallow abdominal respiration, dilating pupil.



## STAGE: 4 MEDULLARY / RESPIRATOR. Y PARALYSIS:-

- Cessation of breathing failure of circulation death
- Pupils: widely dilated
- Muscles are totally flabby
- Pulse is imperceptible
- BP is very low.

QNO:5

Part a:-  
Differentiate between broad and narrow spectrum antibiotics, classify antibiotic drugs?

ANSWER:

DIFFERENTIATE BETWEEN BROAD AND NARROW SPECTRUM:-

- **Narrow** spectrum antibiotics target a few types of bacteria.
- **Broad** spectrum antibiotics target many types of bacteria.
- But using broad spectrum antibiotics when they are not needed can create antibiotic resistant bacteria that are hard to treat.



**Broad** spectrum antibiotics effective against both gram +ve and gram -ve bacteria e.g. tetracycline.

**Narrow** spectrum antibiotics effective against only specific type of bacteria such as  
~~iso~~ isoniazid → bacillus bacteria tuberculosis, macrolides and penicillins G<sup>n</sup>.

## CLASSIFICATION OF ANTIBIOTIC DRUGS:-

- Cell wall inhibitors
- Glycopeptides
- Protein synthesis inhibitors.
- Topoisomerase inhibitors
- Anti-metabolites
- Anti-mycobacterials.
- Sulfones.

## CELL WALL INHIBITORS:-

Include

- Penicillins
- Cephalosporins
- Carbapenems
- glycopeptides.

## GLYCOPEPTIDES:-

✓ (all GP and *C. difficile* - the oral form).

- Vancomycin

## PROTEIN SYNTHESIS INHIBITORS:-

• 50S ribosome inhibitors.

- Macrolides
- Lincosamides

• 30S ribosomes inhibitors

- Aminoglycosides
- Tetracyclines.



## TOPOISOMERASE INHIBITORS.

- Fluoroquinolones
- Rifampin
- Metronidazole

## Anti Metabolites.

- Trimethoprim
- Sulfamethoxazole
- Nitrofurantoin

## PLASMA MEMBRANE:-

- Polymyxins
- Polymyxin B
- Colistin
- Lipopeptide
- daptomycin

## DNA SYNTHESIS:-

- Fluoroquinolones
- Ciprofloxacin
- Levofloxacin
- Moxifloxacin

## RNA SYNTHESIS:-

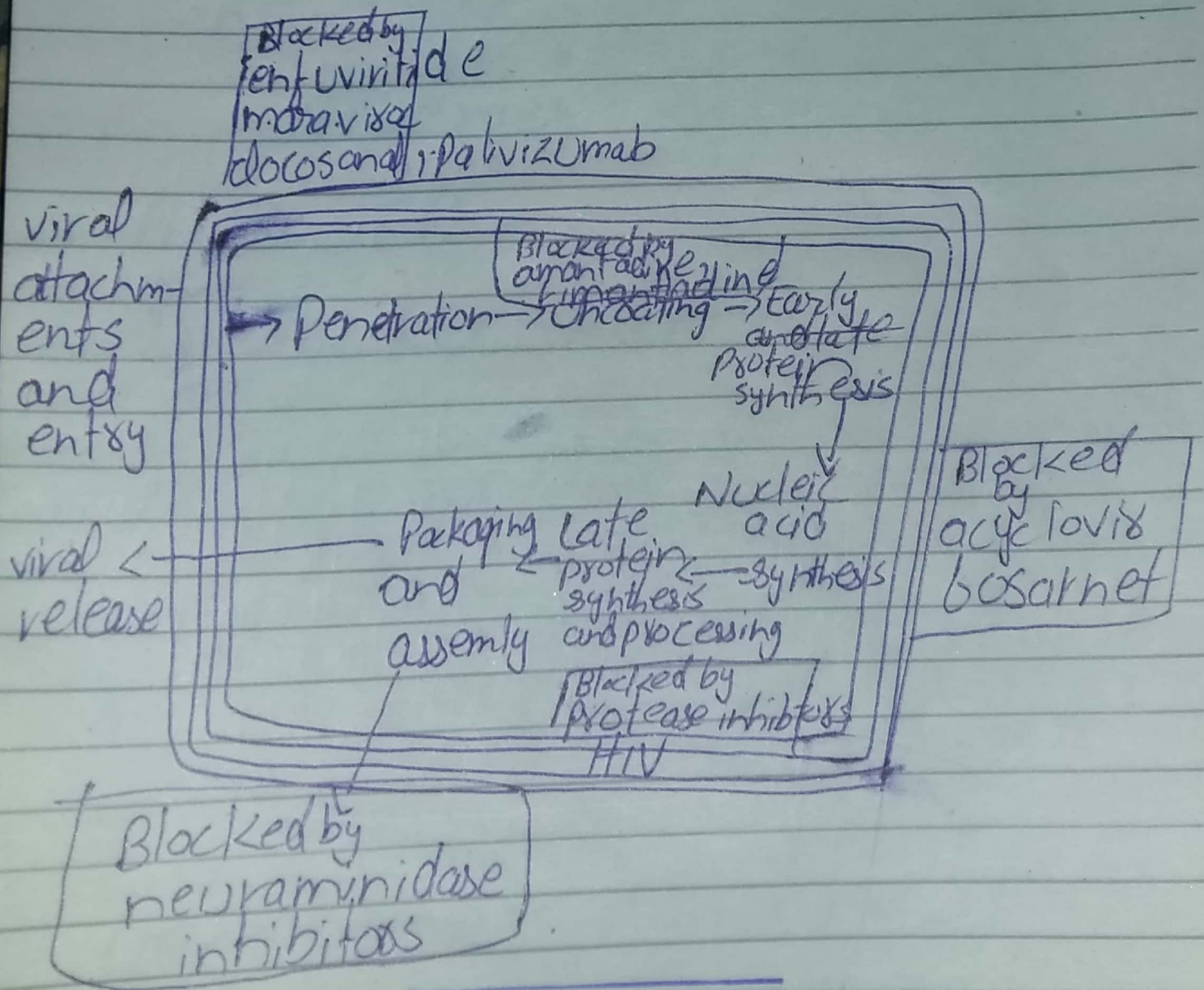
- Rifamycins
- Rifampin

## METABOLIC PATHWAYS:-

- Folic acid synthesis
- Sulfonamides
- Sulfones
- Trimethoprim
- Mycolic acid synthesis
- Isoniazid.



# MOA OF ANTIVIRAL AGENTS:-



QNO: 1

Define drug receptors, enumerate different receptor families and explain the receptors that shows its effect second messenger system.

ANSWER:-

**DRUG RECEPTORS:-**

Receptor is a macromolecule in the membrane or inside the cell that specifically (chemically) bind a ligand (drug). The binding of a drug to receptors depends on types of chemical bonds that can be established b/w drug and receptor.

**RECEPTORS FAMILY:-**

- 1) Ligand-gated ion channels.
- 2) G proteins coupled receptors
- 3) Enzyme linked receptors,
- 4) Intra cellular receptors.



Second messengers are small intracellular molecules that mediate the effects of first messengers, neurotransmitters and hormones. Some of the important second messengers in the nervous system are cAMP, cyclic guanosine monophosphate (cGMP), diacylglycerol (DAG), inositol triphosphate (IP<sub>3</sub>), and Ca<sup>2+</sup> ions.

### Second messengers

- 1- A common pathway turned on by G<sub>s</sub> and other types of G proteins is the activation of adenylyl cyclase by  $\alpha$ GTP subunits
- 2- G proteins also activate phospholipase C.
- 3- G proteins coupled receptors also activate guanylyl cyclase, which converts (ATP) to cyclic guanosin monophosphate (cGMP)
- 4- A fourth second messenger that stimulates cGMP-dependent protein kinase. =====



QNO:2

Define drug interactions, enumerate its various types and explain pharmacokinetic drug interaction and its factors with examples.

ANSWER:-

## DRUG INTERACTIONS:-

A drug interaction is a change in the action or side effects of a drug caused by concomitant administration with a food, beverage, supplement, or another drug. There are many cause of drug interactions.

An alternation in the duration or magnitude of pharmacological effects of one drug produced by another drug, food, food, or any other substance.



## TYPES:-

1) DRUG-DRUG Interactions/interactions between drugs come to mind.

2- DRUG-FOOD Interactions/interactions may also exist between drugs and foods.

3- (Drug-plant Interactions) drugs and medicinal plants or herbs

4- DRUG-DISEASE Interactions

But there are essentially two types of drug interactions:

- 1- Pharmacokinetics D.I.
- 2- Pharmacodynamics D.I.

# PHARMACOKINETIC DRUG INTERACTION:-

These occur when one drug alters the absorption, distribution, metabolism, or excretion of another, thus increasing or decreasing the amount of drug available to produce its pharmacological effects.

Hence such interactions are also called as ADME interactions.

## CLASSIFICATION:-

- Absorption interactions
- Distribution interactions
- Metabolism interactions.
- Excretion interactions.



# DRUG ABSORPTION INTERACTIONS.

- Absorption interactions are those where the absorption of the object drug is altered.
- Since the oral route is the one, most frequently used to administer drugs, interactions influencing absorption are more likely to occur within the gastro. gastrointestinal tract.
- The net effect of such an interaction is:
  - Faster or slower drug absorption.
  - More or, less drug absorption.

## FACTORS:-

- a) changes in gastrointestinal pH
- b) changes induced by chelation
- c) changes in gastrointestinal motility.



# CHANGES IN GASTROINTESTINAL PH:-

- Absorption in the gut is governed by the gut pH, lipid solubility and pKa of the drug.
- While changes in gastric pH induced by H<sub>2</sub> and proton pump blockers and antacids containing AlMg formulations have been shown to significantly reduce drug bioavailability.
- However the alteration in pH has certain clinical implications as it can result in a significantly reduce drug bioavailability.
- However, the alteration in pH has certain clinical implications as it can result in a significant reduction in the absorption of ketoconazole and itraconazole which are insoluble in water and are only ionized at low pH, hence gastric activity plays an important part in this



interactions.

## CHANGES INDUCED BY CHELATION

• The various possible drug interactions that occur due to alterations in drug absorption the most clinically significant interactions occur due to chelation or formation of insoluble complexes.

## CHANGES IN GASTROINTESTINAL MOTILITY:-

- Drug that alter the stomach emptying rate can effect the rate of absorption of drugs as most of them are absorbed in the small intestine.
- Drugs with anticholinergic properties like propantheline or those altering bowel motility, like Diphenoxylate may effect the absorption of other drugs. Eg. Propantheline increases the absorption of slow dissolving Digoxin by 30% as the reduced gut motility pass into solution



making a greater amount available for absorption but this effect is not seen with fast dissolving tablets.

- Metoclopramide on the other hand produces the opposite effect on motility and digoxin absorption.

## EXAMPLES:-

Digoxin, particularly when given intravenously is an example of a drug that is well described by two ~~compartment~~ compartment pharmacokinetics. After an intravenous dose is administered, plasma concentrations rise and then rapidly decline as drug distributes out of plasma and into muscle tissue.



Q4  
What does heart failure mean, explain the pathophysiology of heart failure.

ANSWER:-

### HEART FAILURE

Heart failure is a chronic, progressive condition in which heart muscle is unable to pump enough blood to meet the body's needs for blood and oxygen.

OR  
Congestive heart failure is a chronic condition in which the heart is unable to pump a sufficient quantity of blood to meet the needs of peripheral tissues.

# PATHOPHYSIOLOGY OF HEART FAILURE:-

Cardiac Lesion:

Ischemia  
infarction  
myopathy

hypertension  
valve disease  
other

↓  
Decreased Cardiac Performance  
impaired pumping ability

Myocardial cell changes  
structural damage  
altered calcium transport

Neurohormonal Compensation  
↑ sympathetic activity  
↑ renin-angiotensin II  
↑ Aldosterone

Increased Cardiac workload due to:  
↑ vascular resistance  
↑ fluid volume



Q:4

Part: B

Classify the drugs used for the treatment of heart failure, explain along with mechanism.

## MECHANISM OF HEART FAILURE.

Heart failure begins after an index event produces an initial decline in pumping capacity of the heart. After this initial decline in pumping capacity of the heart, a variety of compensatory mechanisms are activated, including the adrenergic nervous system, the renin-angiotensin system, and the cytokine system.

## TREATMENT OF HEART FAILURE:-

## Drugs used in heart failure

Positive inotropic drugs

Vasodilators

Miscellaneous drugs for chronic failure

Cardiac glycosides (digoxin)

Beta agonists (dobutamine)

PDE inhibitors (milrinone)

Nitroprusside  
Nitrates  
Hydralazine

Loop diuretics  
ACE inhibitors  
Nesiritide

Beta blockers  
Spironolactone

**Cardiac glycoside**  
Increases Ca<sup>+</sup>, increases cardiac contractility

**Beta agonists**  
Dopamine and dobutamine exert a fairly specific positive inotropic effect, presumably through their ability to stimulate beta-1 receptors on the myocardium

### Phosphodiesterase Inhibitors

These agents cause a cAMP-mediated increase in intracellular calcium, which subsequently increases the force of contraction within the myocardial cell

**Vasodilator (Nitroprusside)**  
Produces vasodilation by blocking alpha-1 receptors on vascular smooth muscle

### Diuretics

Diuretics work by inhibiting the reabsorption of sodium from the nephron, which, in turn, decreases the amount of water that is normally reabsorbed with sodium, thus increasing water excretion

### Beta blockers

These drugs therefore normalize sympathetic stimulation of the heart and help reduce heart rate (negative chronotropic effect) and myocardial contraction force (negative inotropic effect)