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Paper : Physiology

Question. 1

Answer:

Drug Receptor

It is defined as a macromolecule or binding site located on the surface or inside the effector cell that serves to recognize the signal molecule / drug and initiate the response to it, but itself has no other function.

The largest number of drug do not bind directly to the effectors viz. enzymes, transporters, structure proteins. ect but act through specific regulatory macromolecules which control the above listed effectors.

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2 Major Receptor families

These receptors may be divided into four families.

1: Ligand-gated ion channels.
2: G proteins coupled receptors
3: Enzyme linked receptors
4. Intracellular receptors.

Ligand-gated ion channels:

The first receptor family ~~co~~mprises ligand-gated ion channels that are responsible for regulation of the flow of ions across cell membranes.

The activity of these channels is regulated by binding of a ligand to the channel.
Response to these receptor is very rapid having durations of a few milliseconds.

G proteins coupled Receptors:

Thes receptors are

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comprised of a single peptide that has seven membrane-spanning regions and these receptors are linked to a G protein having three subunits an α subunit that binds guansine triphosphate and β, γ subunit.

Binding of the appropriate ligand to the extracellular region of the receptor act the G protein so that GTP replaces guanos diphosphate on subunit.

These effectors usually an enzyme or ion channel.

These effectors the change concentrations of second messengers that are responsible for further actions within the cell.

Second messengers:

1: A common pathway turned on by G_s, and other type of G protein, is the activation of adenylyl cyclase by a GTP subunits.

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2. G proteins also activate phospholipase C.

3- G proteins coupled receptors also activate guanylyl cyclase, which converts (GTP) to cyclic guanosise monophosphate.

4. A fourth second messenger that s lates GMP dependent proothic kinase

Question -2

Answer:

Drug interaction:

Definition: It is defined an alternation in the duration or magnitude of pharmacological effect of one drug produced by another drug

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food or any other substance.

Types

1: (Drug-drug interactions) between drug come to mind.

2: (Drug - food interactions) interact may also exist between drugs and food.

3: (Drug - plant interactions) Drugs and medical plants or herbs. E

4: (Dry - disease interactions) two types of drug interactions.

1: Pharmacokinetic
2: Pharmacodynamic.

Drug - Drug interactions:

Drug - drug interactions occur when a drug interacts or interferes with another drug. This can alter the way one or both of the drugs act in

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the body or cause unexpected side effect.

- Codeine + Paracetamol addition (increased analgesic effect).
- Aspirin + warfain synergism (exessive bleeding).

Drug-food interactions

A drug food interactions happens when the food you eat effects the ingredients in a medicine you are taking so the medicine cannot work the way it should.

Benzodiazepines + grapefruit inhabit enzymes involved in drug metabolism.

Drug - disease interactions

Drug & condition interactions occur when a drug worsens or exacerbates an existing medical condition.

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- Nasal decongestants + hypertension increased blood pressure.
- calcium channel blockers + heart failure negative inotropic activity.

Pharmacokinetic interactions:

1: Altered GIT absorption

- Altered PH
- Altered bacterial flora
- formation of drug chelates or complexes.
- Drug induced mucosal damage.
- Altered GIT motility.

a Altered PH:

The non-ionized form of a drug is more lipid soluble and more readily absorbed from GIT than the ionized from does.

example

Antiacids ~~dise~~ decrease the tablet dissolution of ketoconazole (acidic).

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b: Altered intestinal bacterial flora:

Example: 40% or more of the administered digoxin does is metabolised by the intestinal flora.

Antibiotic kill a large number of normal flora of the intestine increase digoxic conic.

c: Complexation or chelation:

Example: 1

Tetracycline interacts with iron preparations as milk (Ca2+) Unabsorbable complex.

Example: 2

Antacid hydroxide decrease absorption of ciprofloxation by 85% - doe do chelation.

D: Drug induced mucosal damage:

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9 Antineoplastic agents.

Example
cyclophosphamide, vincristine, inhibit absoption of several drugs e.g digoxin.

E: Altered motility:

Metoclopramide increase absoption of cyclosporine due to the increase of stomach empting ~~rate~~ time.

F: Displaced protein binding:

It depend on the affinity of the drug to plasma protein. The most likely bound drug is capable to displace other.
phenytoin is a highly bound to plasma protein (90%) and warfarin (99%)

g: Altered metabolism:

The effect of one drug on the metabolism of other is well documented.

Example :

ABC area and all.

Question - 3

Answer

General anesthesia	Local anesthesia
→ General anesthetics which result in a reversible loss of consciousness.	→ Local anesthetic which cause a reversible loss of sensation in a limited region of the body without necessarily affecting consciousness.
→ CNS → Whole body → Last → Essential	Peripheral nervous Restricted area Unaltered Usually not needed.

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=> Risky => possible => preferred => Not preferred	Safer. Not possible. cannot be preferred. preferred.

Stages of Anesthesia

Stage 1 Analgesia

In stage 1 the patient has decreased awarness of pain, sometimes with amnesia. conciousness may be impaired but it not lost.

Stage 2 Disinhibition / excitement

In stage 2 the patient appears to be delirious and exited. Amnesia occur reflexes are enhanced and respiration is typically irregular and incontinence may occur.

Stage 3 surgical Anesthesia

In stage 3 the patient is unconscious and has

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no pain reflexes respiration is very regular and blood pressure is normal.

Stage: 4 Medullary Depression

In stage 4 the patient develops severes respiratory and cardiovascular depression that requires mechanical and pharmacologic support.

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Question- 4

Answer

A) Heart failure

Heart failure means that the heart is unable to pump blood around the ~~blood~~ body properly. It usually occurs because the heart has become to weak or stiff. Its sometimes called congestive heart failure, although this name is not widely used

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nowadays.

PATHOPHYSIOLOGY OF HEART FAILURE :

The main pathophysiology of heart failure is a reduction in the efficiency of heart muscle, through damage or overloading. As such it can be caused by a wide number of conditions, including myocardial infarction, hypertension and amyloidosis. (in which misfolded proteins are deposited in heart muscle causing it to stiffen). over time these increases in workload will produce changes to the heart itself.

B) DRUG USED FOR TREATMENT OF HEART ALONG WITH MECHANISM :

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Cardic glycoside

Increase Ca^{+} increase cardic contractility.

Beta agonists

Dopamine and dobutamine exert a fairly specific positive Inotropic effect.

Phosphodiestase Inhibitors

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

Vasodilator (Nitroprusside)

Produces vasodilation by blocking alph 1 receptors on vascular smooth muscle.

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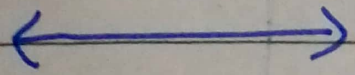
Diuretic

Diuretic work by inhibiting the reabsorption of sodium from the nephron, which turn decrease the amount of water.

Beta Blockers

These drugs therefore normalize sympathetic stimulation of heart rate and myocardial contraction force (negative inotrophic effect).



Question-5

Answer

A)

Board spectrum antibiotics :	Narrow spectrum antibiotics :
effective against both gram + ve and gram - ve	effective against only specific type of

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16 bacteria e.g tetracycline.	bacteria such as isoniazid bacillus bacteria tuberculosis macrolides and penicillins 'G'.
Broad spectrum antibiotics act against multiple strains and forms of different bacteria which share common structure and metabolic functions that can be attached and affected.	Narrow spectrum antibiotics are more specific in their course and act against only certain bacteria as a more targeted approach.

Classification of antibiotic Drug :

- Aminoglycosides - gentamicin, tobramycin amikacic.
- Macrolides - erythromycin, clarithromycin, azithromycin.
- Tetracyclines - Tetracycline,

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Doxycycline.

- chloramphenicol.
- lincomycins - clindamycin.

B)

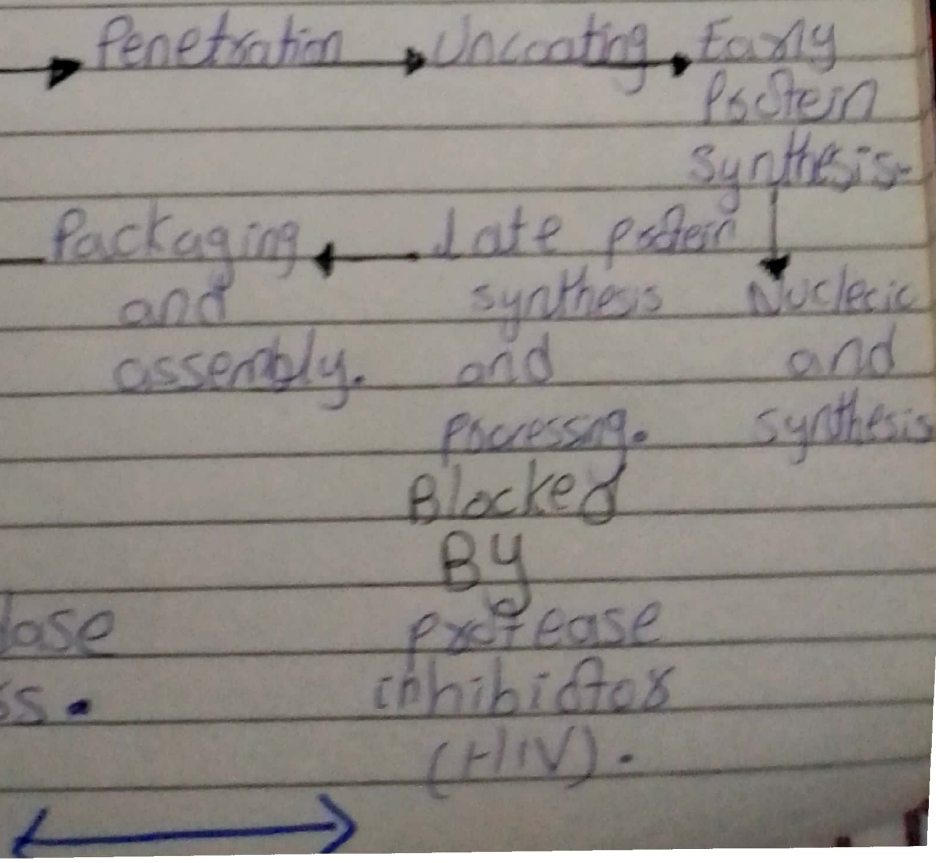
The mechanism of action of antiviral agent:

Blocked By

enfuvirside
maraviroc docosanol
palivizumab.

Blocked By

Amantadine,
rimantadine.



Blocked By

Neuraminidase inhibitors.

Blocked By

Protease inhibitors (HIV).