

**IQRA NATIONAL UNIVERSITY**

**DEPARTMENT OF ALLIED HEALTH SCIENCES**

**Final-Term Examination (Spring-20)**

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PROG: BS DENTAL TECHNOLOGY 3rd semister***

***COURCE TITLE;Pharmacology
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***DEPT: ALLIED HEALTH SCIENCE***

**Note:**

* **Paper is divided into 5 questions**
* **Each question carry equal marks (10) with grand total of 50 marks**
* **Each question is composed of specific parts, pay attention to each part of question or otherwise it will lead to mark deduction**
* **Avoid copy paste from slides, your answer may got canceled if it found a total copy**
1. Define drug receptors, enumerate different receptor families and explain the receptor that shows its effect through second messenger system.

ANS=1

**Drug receptors:**

 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 drugs do not bind

directly to the effectors, viz.enzymes, channels, transporters, structural

proteins,etc. but act through specific regulatory

macromolecules which control the above

listed effectors.

G protein coupled receptors

These receptors are comprised of a single peptide that has seven membrane-spanning regions, and these receptors are linked to a Gprotein (Gs and others) having three subunits, anα subunit that binds guanosine triphosphate (GTP)and β, γ subunit.Binding of the appropriate ligand to the extracellular region of the receptor activates the G protein so that GTP replaces guanosine diphosphate (GDP) on the α subunit. Dissociation of the G protein occurs, and both the α GTP subunit and the β, γ subunit subsequently interact with other cellular effectors, usually an enzyme orion channel. These effectors then change the

concentrations of second messengers that are responsible for further actions within the cell. Stimulation of these receptors results in responses that last several seconds to minutes.

Types of cell receptors

Cell-surface receptors are involved in most of the signaling in multicellular organisms. There are three general categories of cell-surface receptors: ion channel-linked receptors, G-protein-linked receptors, and enzyme-linked receptors.

Second messengers:-

A common pathway turned on by Gs, and other types of G proteins, is the

activation of adenylyl cyclase by α GTP subunits.

G proteins also activate phospholipase C.

G proteins coupled receptors also activate guanylyl cyclase, which converts

(GTP) to cyclic guanosine monophosphate (cGMP),a fourth second messenger that stimulates cGMP-dependent protein kinase.

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

 ANS=2

Definition

It is defined as "an alternation in the duration or magnitude of

pharmacological effects of one drug produced by another drug. food, or

any other substance".

Types

1 (drug-drug interaction) interactions between drugs come to mind

2 (drug-food interactions) interact 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.

Pharmacokinetics DI

2. Pharmacodynamics DI

Pharmacokinetics

Pharmacokinetics involve the effect of a drug on another drug kinetic that includes absorption ,distribution, metabolism and excretion.

Pharmacokinetic interactions

1) Altered GIT absorption.

Altered p

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 form does.

Ex1, antiacids Decrease the tablet dissolution of Ketoconazole (acidic)

Ex2, H2 antagonists Therefore, these drugs must be separated by at least 2h

in the time of administration of both.

b) Altered intestinal bocterial flora;

 EX, 40% or more of the administered digoxin dose is metabolised by the

 intestinal flora.

Antibiotics kill a large number of the normal flora of the intestine increase digoxin conc. and increase its toxicity

c Complexation or chelation;

EX1, Tetracycline interacts with iron preparations or Milk

(Ca2-)Unabsorpable complex

Ex2, Antacid (aluminum or magnesium) hydrowide Decrease absorption of ciprofloxacin by 85% due to chelation

d) Drug-induced mucosal damage

Antineoplastic agents e.g, cyclophosphamide, vincristine, Procarbazine Inhibit

absorption of several drugs eg, digoxin

e) Altered motility

 Metoclopramide (antiemitic) Increase absorption of cyclosporine due to the

increase of stomach empting time Increase the toxicity of cyclosporine

Displaced protein binding (distribu

It depends on the affinity of the drug to plasma protein. The most likely bound

drugs is capable to displace others. The free drug is increased by displacement

by another drug with higher affinity.

Phenytoin is a highly bound to plasma protein (90%), Tolbutamide (96%), and

warfarin (99%)

Drugs that displace these agents are Aspirin, Sulfonamides, phenylbutazone

g) Altered metabolism

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

The liver is the major site of drug metabolism but other organs can also do

eg, wBc,skin,lung, and GIT

CYPAS0 family is the major metabolizing enzyme in phase I (oxidation

process. Therefore, the effect of drugs on the rate of metabolism

of others can involve the following examples.

Enzeme induction

A drug may induce the enzyme that is responsible for the metabolismof

another drug or even itself e-g., Carbamazepine (antiepileptic drug ) increases

its own Metabolism.

Phenytoin increases hepatic metabolism of theophylline Leading to decrease

its level Reduces its action

Ea. Enzyme inhibitien:

It is the decrease of the rate of metabolism of a drug by

another one. This will lead to the increase of the concentration of the

target drug and leading to the increase of its toxicity. Inhibition of the enzyme

may be due to the competition on its binding sites, so the onset of action is

short may be within 24h.

When an enzyme inducer ( e.g. carbamazepine) is administered with an

inhibitor (werapamil) The effect of the ibitor will be predominant

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1. Differentiate between general and local anesthesia, explain stages of anesthesia in detail

ANS=3

Differentiation between general and local anesthesia.

|  |  |  |
| --- | --- | --- |
| FEATURES | Gen, Anaesthsia | Local Anesthsia |
| Site of action | CNS | Peripheral nerves |
| Area of body involved | Whole Body | Restricted Area |
| Consciousness | Last | Unaltered |
| Core of vital function | Essential | Usually not needed |
| Poor health patient | Risky | Safer |
| Use in non cooperative patient | Possible | Not possible |
| Major surjry | Preffered | Cannot be preferred |
| Minor surjery | Not preffered | Preferred |

Stages of anesthesia:-

 FOUR Stages of anesthesia.

STAGE 1:- Stage of Analgesia:

* Starts from beginning of anaesthetic inhalation and lasts upto the loss of consciousness.
* Pain is progressively abolished During this stage.
* Patient remins conscious, can hear and see and feels a dream like state.
* Reflexes and respiration remain normal.
* It is difficult to meaintain use is limited to short procedures only.

STAGE 2:- Stage of delirium and exciltement:

* 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, Involuntry micturition or defecation may occur.
* Heart rate and BP may Rise and pupils dilate dua to sympathetic stimulation.

STAGE 3:- Stage of surgical anaesthesia:

* Extends from onset of regular respiration to cessation of spontaneous breathing this has been divided into 4 planes.
* Plane 1
* Plane 2
* Plane 3
* Plane 4

STAGE 4:- Medullary/ Respiratory paralysis:

* Cessation breathing Failure of circulation Death
* Pupils: Widely dilated
* Muscles are totally flabby
* Pulse id imperceptible.
* BP is very low.

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1.
2. What does heart failure means, explain the pathophysiology of heart failure
3. Classify the drugs used for the treatment of heart failure, explain along with mechanism.

ANS=4

A)

Heart failure is a chronic, progressive condition in which the heart muscle is unable to pump enough blood to meet the body’s needs for blood and oxygen. Basically, the heart can’t keep up with its workload.

Pathophysiology. In heart failure, the heart may not provide tissues with adequate blood for metabolic needs, and cardiac-related elevation of pulmonary or systemic venous pressures may result in organ congestion. This condition can result from abnormalities of systolic or diastolic function or, commonly, both.

B)

# Medicines for congestive heart failure

Some medicines for heart failure improve how well your heart pumps. Others help remove excess fluid from your body or dilate blood vessels so blood can flow more easily so your heart doesn't have to work as hard. A combination of medicines is often used to manage your condition and help you feel better.

Heart failure is an ongoing health condition. To stay as healthy as possible, you might need to take medicine for the rest of your life. It's important that you take your medicines as your doctor prescribed and not miss any doses. Make sure to have prescriptions for these medicines refilled before you run out.

Don't take any over-the-counter medicines until you talk to your doctor to see if they are safe. Don't use nonsteroidal pain relievers (such as ibuprofen, Advil, Motrin, Aleve, and Nuprin), cold and flu remedies (especially those containing pseudoephedrine), and medicines that contain sodium, such as Alka-Seltzer.

Call your doctor right away if you have problems or side effects from your medicine. Do not stop taking your medicine without talking with your doctor or nurse.

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1.
2. Differentiate between broad spectrum and narrow spectrum antibiotics, classify antibiotic drugs

ANS=a

# : Narrow-spectrum antibiotics target a few types of bacteria. Broad-spectrum antibiotics target many types of bacteria. Both types work well to treat infections. But using broad-spectrum antibiotics when they're not needed can create antibiotic-resistant bacteria that are hard to treat.

ANITIBIOTIC DRUGS: Infectious diseases are the major causes of human sickness and death. To overcome such health care issues, antibiotics proved to be promising agents ever since they were introduced in the 1940s. Antibacterials, which are a subclass of antibiotics, have been classified earlier in several ways; however, to make it more easily understandable, we can classify antibacterial agents into five groups: type of action, source, spectrum of activity, chemical structure, and function.

1. Explain briefly the mechanism of action of antiviral agents

B

: Antivirals are a class of medications that are used to treat viral infections. Most viral infections resolve spontaneously in immunocompetent individuals. The aim of antiviral therapy is to minimize symptoms and [infectivity](https://www.amboss.com/us/knowledge/Epidemiology%22%20%5Cl%20%22xid%3D1j02zf%26anker%3DZb8208e3c2c49e99ba9e784fc5ef92308) as well as to shorten the duration of illness. These drugs act by arresting the [viral replication cycle](https://www.amboss.com/us/knowledge/General_virology%22%20%5Cl%20%22xid%3DPn0Wtg%26anker%3DZ0adf033ecb2bd3ae246c4ae5a2ce1e53) at various stages. Currently, antiviral therapy is available only for a limited number of infections. Most of the antiviral drugs currently available are used to treat infections caused by [HIV](https://www.amboss.com/us/knowledge/Human_immunodeficiency_virus%22%20%5Cl%20%22xid%3Dmf0V52%26anker%3DZ854a352d74556f9617875e1e09eb9d73), herpes viruses, [hepatitis B](https://www.amboss.com/us/knowledge/Hepatitis_B%22%20%5Cl%20%22xid%3DOS0I-2%26anker%3DZ8400c8767de06bd1fa7338aa79959829) and C viruses, and [influenza](https://www.amboss.com/us/knowledge/Influenza%22%20%5Cl%20%22xid%3DBm0z3g%26anker%3DZ7f6a08ed676ff91e0fd1a38ea649ecbd) A and B viruses. Because viruses obligate intracellular parasites, it is difficult to find drug targets that interfere with viral replication without also harming the host cells. Unlike other antimicrobials, antiviral drugs do not deactivate or destroy the microbe (in this case, the virus) but act by inhibiting replication. In this way, they prevent the viral load from increasing to a point where it could cause pathogenesis, allowing the body's innate immune mechanisms to neutralize the virus. This article provides an overview of the most commonly used antiviral agents. For more information on antiretroviral agents used in the treatment of [HIV](https://www.amboss.com/us/knowledge/Human_immunodeficiency_virus%22%20%5Cl%20%22xid%3Dmf0V52%26anker%3DZ854a352d74556f9617875e1e09eb9d73), which is known as [highly active antiretroviral therapy](https://www.amboss.com/us/knowledge/Human_immunodeficiency_virus%22%20%5Cl%20%22xid%3Dmf0V52%26anker%3DZ06c68157ba29a3fc6aa11e7247a4bc80) ([HAART](https://www.amboss.com/us/knowledge/Human_immunodeficiency_virus%22%20%5Cl%20%22xid%3Dmf0V52%26anker%3DZ06c68157ba29a3fc6aa11e7247a4bc80)), see [HIV therapy](https://www.amboss.com/us/knowledge/Human_immunodeficiency_virus%22%20%5Cl%20%22xid%3Dmf0V52%26anker%3DZ06c68157ba29a3fc6aa11e7247a4bc80).

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