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paper pharmacology
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(Question 1)
('Ans)

Define receptor?

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 receptor depends on types of chemical bounds that can be established between drug and receptor.
(Different family of drugs recepter)
Receptors can be subdivided into four main classes:
1:ligand-gated ion channels,
2:tyrosine kinase-coupled,
3:intracellular steroid and
4:G-protein-coupled (GPCR).
(second massenger system)
Second messengers are intracellular signaling molecules released by the cell in response to exposure to extracellular signaling molecules—the first messengers.
Examples of second messenger molecules include cyclic AMP, cyclic GMP, inositol trisphosphate, diacylglycerol, and calcium. First messengers are extracellular factors, often hormones or neurotransmitters, such as epinephrine, growth hormone, and serotonin.
Types of second messenger moleculesEdit

There are three basic types of secondary messenger molecules:

Hydrophobic molecules: water-insoluble molecules such as diacylglycerol, and phosphatidylinositols, which are membrane-associated and diffuse from the plasma membrane into the intermembrane space where they can reach and regulate membrane-associated effector proteins

Hydrophilic molecules: water-soluble molecules, such as cAMP, cGMP, IP3, and Ca2+, that are located within the cytosol

Gases: nitric oxide (NO), carbon monoxide (CO) and hydrogen sulfide (H2S) which can diffuse both through cytosol and across cellular membranes.

These intracellular messengers have some properties in common:

They can be synthesized/released and broken down again in specific reactions by enzymes or ion channels.

Some (such as Ca2+) can be stored in special organelles and quickly released when needed.

Their production/release and destruction can be localized, enabling the cell to limit space and time of signal activity.
Common mechanisms of second messenger
There are several different secondary messenger systems (cAMP system, phosphoinositol system, and arachidonic acid system), but they all are quite similar in overall mechanism, although the substances involved and overall effects can vary.
In most cases, a ligand binds to a membrane-spanning receptor protein molecule. The binding of a ligand to the receptor causes a conformation change in the receptor. This conformation change can affect the activity of the receptor and result in the production of active second messengers.
In the case of G protein-coupled receptors, the conformation change exposes a binding site

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for a G-protein. The G-protein (named for the GDP and GTP molecules that bind to it) is bound to the inner membrane of the cell and consists of three subunits: alpha, beta and gamma. The G-protein is known as the "transducer."
When the G-protein binds with the receptor, it becomes able to exchange a GDP (guanosine diphosphate) molecule on its alpha subunit for a GTP (guanosine triphosphate) molecule. Once this exchange takes place, the alpha subunit of the G-protein transducer breaks free from the beta and gamma subunits, all parts remaining membrane-bound. The alpha subunit, now free to move along the inner membrane, eventually contacts another membrane-bound protein - the "primary effector."
The primary effector then has an action, which creates a signal that can diffuse within the cell. This signal is called the "second (or secondary) messenger." The secondary messenger may then activate a "secondary effector" whose effects depend on the particular secondary messenger system.
Calcium ions are one type of second messengers and are responsible for many important physiological functions including muscle contraction, fertilization, and neurotransmitter release. The ions are normally bound or stored in intracellular components (such as the endoplasmic reticulum(ER)) and can be released during signal transduction. The enzyme phospholipase C produces diacylglycerol and inositol trisphosphate, which increases calcium ion permeability into the membrane. Active G-protein open up calcium channels to let calcium ions enter the plasma membrane. The other product of phospholipase C, diacylglycerol, activates protein kinase C, which assists in the activation of cAMP (another second messenger).

(QUESTION 2)
(ANS)
DEFINE DRUG INTERACTION?
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 causes of drug interactions. For example, one drug may alter the pharmacokinetics of another.
(Types of drugs interaction)
What are 3 types of drug interactions?
Drug interactions can be categorised into 3 groups:
1)Interactions of drugs with other drugs (drug-drug interactions),
2) Drugs with food (drug-food interactions)
3)Drug with disease condition (drug-disease interactions).
(pharmacokinetic drugs interaction)
Pharmacokinetic interactions. Modifications in the effect of a drug are caused by differences in the absorption, transport, distribution, metabolism or excretion of one or both of the drugs compared with the expected behavior of each drug when taken individually.
(pharmacokinetic drug interaction example)
Pharmacokinetic interactions occur at the levels of absorption (e.g., levothyroxine and neutralizing antacids), elimination (e.g., digoxin and macrolides), and metabolism, as in the competition for cytochrome P450 enzymes (e.g., SSRIs and certain beta-blockers).
(Question 3)
Differentiate between local anesthesia and general anesthesia?
(Ans)
Anesthetics are used to numb a specific area of the body
(local and regional anesthesia) or to cause a person to be unconscious and not have pain during a procedure such as surgery.
(general anesthesia).
Local anesthesia numbs just a small area of tissue where a minor procedure is to be done.
General anesthesia is a type of anesthesia that is used in major body system surgeries that are needed to depress the whole body. There is harm in using general anesthesia so a qualified anesthesiologist must be present during the procedure. There are risks for the fact that major organs are also suppressed and depressed during the procedure such as the lungs and diaphragm. Thus, careful monitoring must be implemented during the procedure. Examples of surgeries that are in need of general anesthesia are major surgeries such as a heart transplant, brain surgery, repair of hip fractures, and a lot more. The side effects of general anesthesia are also big risks so nurses and doctors carefully monitor a patient post-operation.

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Local anesthesia
, on the other hand, is a type of anesthesia that is used in suppressing a part of the body only. When used, other senses may not be affected such as consciousness, hearing, sight, smell, and a lot more. It is used mostly in minor surgeries such as dental procedures like tooth extractions. It can also be used in circumcision, in dermatological and facial enhancements, and many other procedures. It is also less risky to use than general anesthesia. An anesthesiologist is not needed at times of induction of local anesthesia because the surgeon can monitor this by him or herself.

An anesthesiologist or a doctor who has studied medicine and trained to anesthetize patients is
responsible for medicating patients during these kinds of procedures. They are responsible for
assessing the patient before the surgery, during the surgery, and after the surgery. With their skills,

operations and surgeries will flow smoothly together with the surgeon and the nurses.
Summary:
1. Local anesthesia suppresses pain in a part of the body only while general anesthesia involves
suppressing pain in the whole body.
2. Local anesthesia can be administered by the doctor without the help of an anesthesiologist while in
general anesthesia an anesthesiologist must be present.

3. In general anesthesia there is greater risk of fatality compared to local anesthesia.
(stages of anesthesia)
There are four stages of general anesthesia, namely: analgesia
- stage 1, delirium - stage 2, surgical anesthesia - stage 3 and respiratory arrest - stage 4. As the patient is increasingly affected by the anesthetic his anesthesia is said to become 'deeper. (There are four stages of anesthesia)
Stages of anaesthesiaEdit

Guedel's classification, introduced by Arthur Ernest Guedel in 1937,[22] describes four stages of anaesthesia. Despite newer anaesthetic agents and delivery techniques, which have led to more rapid onset of—and recovery from—anaesthesia (in some cases bypassing some of the stages entirely), the principles remain.
Stage 1
, also known as induction, is the period between the administration of induction agents and loss of consciousness. During this stage, the patient progresses from analgesia without amnesia to analgesia with amnesia. Patients can carry on a conversation at this time.
Stage 2 also known as the excitement stage, is the period following loss of consciousness and marked by excited and delirious activity. During this stage, the patient's respiration and heart rate may become irregular. In addition, there may be uncontrolled movements, vomiting, suspension of breathing, and pupillary dilation. Because the combination of spastic movements, vomiting, and irregular respiration may compromise the patient's airway, rapidly acting drugs are used to minimize time in this stage and reach Stage 3 as fast as possible.
Stage 3, also known as surgical anaesthesia, the skeletal muscles relax, vomiting stops, respiratory depression occurs, and eye movements slow and then stop. The patient is unconscious and ready for surgery. This stage is divided into four planes:

The eyes roll, then become fixed;

Corneal and laryngeal reflexes are lost;

The pupils dilate and light reflex is lost;

Intercostal paralysis and shallow abdominal respiration occur.

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Stage 4, also known as overdose, occurs when too much anaesthetic medication is given relative to the amount of surgical stimulation and the patient has severe brainstem or medullary depression, resulting in a cessation of respiration and potential cardiovascular collapse. This stage is lethal without cardiovascular and respiratory support.

(Question 4)

(Ans) (Heart failure).

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.

(pathophysiology of heart failure)

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..

(Part B)

(Classes of Drugs Used to Treat Heart Failure)

Diuretics. - thiazide diuretics. - loop diuretics. ...
Vasodilators (dilate arteries and veins) - angiotensin converting enzyme (ACE) inhibitors. ...
Cardiostimulatory or inotropic drugs (stimulate contractility) - digitalis. ...
Cardioinhibitory. - beta-blockers.

(mechanism of heart failure)

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(Drug mechanism)

Beta blockers are considered the primary drugs for the pharmacological management of CHF. These drugs provide their beneficial effect by decreasing the excessive activity of the sympathetic nervous system which is characteristic of CHF. Increased sympathetic nervous system activity is a neurohormonal compensation involving the renin-angiotensin system that occurs in CHF patients in order to increase cardiac output of the failing heart and maintain blood pressure. While initially beneficial, these compensations place an ultimately damaging degree of stress on the already failing heart.

As their name suggests, the clinically useful beta blockers used to treat CHF bind to beta-adrenergic receptors on the myocardium, blocking the effects of norepinephrine and epinephrine. By this mechanism, beta blockers normalize sympathetic activity reducing heart rate, cardiac contraction force, and angina. These beta blockers are specifically known as beta-1 cardioselective blockers. Another class of beta blockers, beta-2 blockers, are bronchoconstrictors, and thus provide no real clinical value.

Two FDA approved beta blockers commonly used to treat CHF are Carvedilol and Metoprolol, although there are many others that may be used with the discretion of the treating physician.

Carvedilol is taken orally, with dosages ranging from 3.125 mg taken twice daily to a maximal dose of 50 mg taken twice daily. The half-life of carvedilol is 7-10 hours and it is metabolized extensively and excreted in feces via bile with less than 2% excreted unchanged in the urine.

Metoprolol is taken orally starting at dosages of 12.5-25 mg once a day, which can be doubled every two weeks up to 200 mg daily. Metoprolol has a half-life of 3-7 hours and is mostly metabolized by the liver.

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(Question 5)
(Ans) different b/t Broad spectrum and narrow spectrum?
Broad spectrum antibiotics act against multiple strains and forms of different bacteria which share common structures and metabolic functions that can be attacked and affected to kill them. Narrow spectrum antibiotics are more specific in their course and act against only certain bacteria as a more targeted approach.
(classification of antibiotics drogs)
Top 10 List of Antibiotic Classes (Types of Antibiotics)

Penicillins

Tetracyclines

Cephalosporins

Quinolones

Lincomycins

Macrolides

Sulfonamides

Glycopeptides

Aminoglycosides

Carbapenems.
(Part B)
(mechanism of action of antiviral drug)
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.
Main Targets for Antiviral Drugs
Specific events in virus replication identified as targets for antiviral agents are viral adsorption, penetration, uncoating, and viral nucleic acid synthesis as well as viral protein synthesis.

PAPER END
THANK YOU

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