**Name of subject : Introduction to pharmacology**

**Name of Lecturer: Dr.Tayseer Shaker Mahmood**

**Date :**

**Time of subject : 45 minutes**

**Target group: students of nursing department**

**1+2**

بسم الله الرحمن الرحيم


صدق الله العظيم

## Dedication

**To Al-Hadi University College and its students in nursing department**

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## General objective

**In the end of lecture, the student must be:**

**1. Understand what pharmacology**

**2. Know effect of pharmacology on the drug pathway and its therapeutic effect**

## Specific objective

**In the end of lecture, the student must be:**

1. Define pharmacology
2. Enumerate route of drug administration
3. Classify Drug nomenclature
4. Identified Nature & purposes Of Drugs
5. Enumerate Sources of Drugs

## Introduction

**Pharmacology:** is the branch of biology concerned with the study of drug action.

**Clinical pharmacology:** is the basic science of pharmacology with an added focus on the application of pharmacological principles and methods in the medical clinic and towards patient care and outcomes.

## A drug

is any substance that when inhaled, injected, smoked, consumed, absorbed via a patch on the skin, or dissolved under the tongue causes a temporary physiological (and often psychological) change in the body, effect on the cell, tissue, organ, or organism.

## History of drug:

The first drugs were likely discovered through accident and observation. As early humans tried different plant, animal, and mineral substances, they realized that some substances produced specific effects.

the Chinese documented the use of herbal medicine to cure illness in humans and valuable animals.

## Drug nomenclature

drugs have 3 types of names

1.Chemical names, are the scientific names, based on the molecular structure of the drug. example, "-(isopropylamino)-3-(1-naphthyloxy) propan-2-ol" is a chemical name for propranolol..

.2.Generic or nonproprietary names, constructed out of affixes.  Marketed drug might also have a company code or compound code. ex: ponstan (mefenamic acid.(

3. Trade names, which are brand names. Generic names for drugs are now a days.

**Nature & purposes Of Drugs**

**Drug is a substance which is used for the following purposes:**

**1.Symptomatic :relieve disease symptoms. Aspirin .**

**2.Preventative: to avoid getting a disease. Flu vaccine.**

**3.Diagnostic: help determine disease presence. Radioactive dyes.**

**4.Curative: eliminate the disease. Antibiotics.**

**5.Health Maintenance: help keep the body functioning normally. Insulin.**

**Route of administration**

**Administration of drugs can be administered via a number of routes, and many can be administered by more than one:**

- **1. Orally, as a liquid or solid, that is absorbed through the intestines.**
- **2. Inhaled, (breathed into the lungs), as an aerosol or dry powder. (This includes smoking a substance(**
- **• 3. Injection as solution, suspension or emulsion either: intramuscular, intravenous, intraperitoneal, intrathecal or subcutaneous injection.**
- **Insufflation, or snorted into the nose. 4**
- **5.Rectally as a suppository, that is absorbed by the rectum or colon.**
- **6.Sublingually, diffusing into the blood through tissues under the tongue.**
- **7.Topically, usually as a cream or ointment. A drug administered in this manner may be given to act locally or systemically.**
- **Vaginally to treat vaginal infections.8**

# Sources of Drugs : 3+ 4

**There are 4 sources of drug:**

**I. NATURAL SOURCES: Most primitive and abundant.**

**Drugs are obtained from the following natural sources:**

**A) PLANTS SOURCES**

**B) ANIMAL SOURCES**

**C) MINERAL SOURCES**

**D) MICROORGANISMSA- PLANTS SOURCES**

**A) PLANTS SOURCES**

**Following categories of drugs are derived from roots, leaves or barks of plants:**

**a) Alkaloids**

**They are composed of carbon, hydrogen, nitrogen and oxygen.They are bitter in taste and are often poisonous. These are, therefore, used in small doses. They are insoluble in water. Some examples of alkaloids:**

**-Atropine from atropa belladoma , -Nicotin from tobacco**

**b) Glycosides**

**They are ether-like combination of sugar moiety with non-sugar moiety. They are called glycosides, if the sugar moiety is glucose. Some examples are digitoxin, digoxin and ouabain.**

**c) Oils**

**They are liquids which are insoluble in water. They are of three types and are used for various medicinal purposes.**

**For examples are clove oil, peppermint oil, eucalyptus oil and ginger oil.**

**They may be of vegetable origin e.g. olive oil, castor oil, and peanut oil or of animal origin e.g. cod liver oil, shark liver oil..**

**For examples are clove oil, peppermint oil, eucalyptus oil and ginger oil.**

**They may be of vegetable origin e.g. olive oil, castor oil, and peanut oil or of animal origin e.g. cod liver oil, shark liver oil.**

## B-ANIMAL SOURCES

**Some animal sources continue to be used to procure some modern drugs for example: Insulin, extracted from pork and beef pancreas, is used for the treatment of diabetes mellitus. Thyroid powder for treating hypothyroidism. Heparin is used as an anticoagulant. Hormones and vitamins are used as replacement therapy. Vaccines (cholera, T.B., smallpox .(**

## C-MINERAL SOURCES

**Minerals or their salts are useful pharmacotherapeutic agents.For example: Ferrous sulfate is used in iron deficiency anaemia , Magnesium sulfate is employed as purgative, Aluminium hydroxide and sodium bicarbonate are used as antacids for hyperacidity and peptic ulcer. Kaolin (aluminium silicate) is used as adsorbent in antidiarrheal mixtures. Radioactive isotopes of iodine, phosphorus, gold are employed for the diagnosis/ treatment of diseases particularly malignant conditions.**

## D-MICROBIOLOGICAL SOURCES

**Many life-saving drugs are obtained from fungi, moulds and bacteria e.g. penicillin from Penicillium notatum , chloramphenicol from Streptomyces venezuelae.**

- **SEMISYNTHETIC SOURCES**

**Sometimes semi-synthetic processes are used to prepare drugs when the synthesis of drugs(complex molecules) may be difficult, expensive for examples: human insulin Prepared by chemically modifying substances that are available from natural source to improve its potency, efficacy and also reduce side effects.**

**i) Semi synthetic drugs from plant sources**

**Heroine from Morphine. Bromoscopolamine from scopolamine. Homoatropine from atropine.**

**ii) Semi synthetic drugs from animal sources:**

**Animal insulin changed to be like human insulin 6-aminopenicillanic acid derivative.**

- **SYNTHETIC SOURCES**

**At present majority of drugs used in clinical practice are prepared synthetically, such as aspirin, oral antidiabetics, antihistamines, Most of the synthetic drugs are prepared synthetically i.e. by chemical process ( reaction) with the help of the knowledge of phytochemical investigation.**

- **BIOSYNTHETIC SOURCES (genetically engineered drugs(**

**This is relatively a new field which is being developed by mixing discoveries from molecularbiology, recombinant DNA technology, DNA alteration, gene splicing, immunology and immune-pharmacology. Some of the recent developments are genetically engineered novel vaccines (hepatitis-B vaccine.(**

**TEST:**

**Write a note about the sources of drugs with example.**

**.2Why are synthetic drugs used most widely?**

**.3Write a note about biosynthetic sources of drug along with example**

## PROPERTIES OF IDEAL DRUG 5

**Drug should be**

- **.1Reversible action**
  - **Example: General Anesthetic**
- **.2Predictability**
  - **Know how patient will respond**
- **.3Ease of Administration**
  - **Number of doses should be low and easy to administer**
- **.4Freedom from drug interactions**
- **.5Low Cost**
- **.6Chemical Stability**
  - **No lose of effectiveness with storage**
- **.7Simple generic name**
  - **Easy to remember and pronounce**

**.8 Safe and Effective**

**Factors that determine intensity of drug response**

- **Administration- dosage size and route**
- **Pharmacokinetic processes**
- **Pharmacodynamics**

**Over-the-counter (OTC) drugs:**

**Are medicines sold directly to a consumer without a prescription from a doctor, as opposed to prescription drugs, which may be sold only to consumers possessing a valid prescription .**

**Important cases for using over-the-counter medicines:**

- **ACHES, PAINS, AND HEADACHES**

**Over-the-counter pain medicines can help with headache, arthritis pain, sprains, and other minor joint and muscle problems.**

- **Acetaminophen -- Try this medicine first for your pain .**
- **Nonsteroidal anti-inflammatory drugs (NSAIDs) -- You can buy some NSAIDs, such as ibuprofen and naproxen, without a prescription.**

- **FEVER**

**Acetaminophen (Tylenol) and ibuprofen (Advil, Motrin) help reduce fever in children and adults.**

- **COLD, SORE THROAT, COUGH**

**Cold medicines can treat symptoms to make you feel better.**

**Cough medicines:**

- **Guaifenesin -- Helps break up mucus .**
- **Liquid cough medicines with dextromethorphan -- Decongestants**

- **ALLERGIES**

**Antihistamine pills and liquids work well for treating allergy symptoms.**

- **Antihistamines that may cause sleepiness -- Diphenhydramine chlorpheniramine**
- **Antihistamines that cause little or no sleepiness -- Loratadine**
- **Eye drops Soothe or moisten the eyes**

- **STOMACH UPSET**

- **Antidiarrhea medicines such as loperamide (Imodium – (**

- **SKIN RASHES AND ITCHING**

- **Antihistamines taken by mouth -- May help with itching or if you have allergies**

**Name of subject : Responsibility of nurse for drug administration**

**Name of Lecturer: Dr.Tayseer Shaker Mahmood**

**Date :**

**Time of subject : 45 minutes**

**Target group: students of nursing department**

## General objective

**In the end of lecture, the student must be:**

**1. Understand what Responsibility of nurse for drug administration**

## Specific objective

**In the end of lecture, the student must be:**

**1. Define Responsibility of nurse for drug administration**

**2. Enumerate The responsibility of nurse related to drug**

**3. identified the Nursing Process**

**4.Explain effect of drug interaction**

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# Introduction

**The nurse literally plays the role of a lifeguard in medication administration. She often provides the last opportunity for the health-care team to identify and correct errors in prescribing and distributing medication. Although the physician prescribes the medication and the pharmacist fills the prescription, the nurse usually administers the medication. She is the last link in medication administration**

**Some point should the nursing note through drug administration to patient:**

- **Only administer drug was prepared**
- **Know the purpose and expected outcomes**
- **Do not leave drug at bedside**
- **Check armband before administering**
- **IM injections - no more than 3ml at one site**
- **Wear gloves for parenteral injections**
- **Washing hands before beginning medication pass**
- **Keep medication cart clean**
- **Wear gloves if helping put pills in**

**The responsibility of nurse related to drug :**

**.1 The Physician's Order (card(**

**The medication administration record (MAR) is based on the physician's order and provides the information the nurse needs to administer medication. The MAR contains the name of the patient, the name and dosage of the medication to be administered, frequency/time of administration, and the method of introducing the drug into the patient's body (route of administration (**

## .2 Patient Identification

**Prior to administering medication, the nurse check the patient's identity .**

**To prevent errors, the nurse uses two sources of identification and checks for matching information**

- **She compares the patient's wristband identification with a written document such as a MAR or physician's order .**
- **The nurse may ask the patient to state his name and birthdate and match the information to the patient's wristband.**

## Patient Rights 3 :

**In safely treating the patient, the nurse observes six patient rights, ensuring that she administers the right medication, in the right dosage, to the right patient, at the right time, via the right route, in accordance with the physician's orders. She completes the process with the right documentation. In addition, before a nurse administers medication, she references the action and expected effect of the drug. She monitors the patient and reports any adverse reactions to the medication.**

## .4 Preventing Errors:

**Nursing supervisor suspects that a prescribed dosage of medication is unsafe. The nurse also identifies prescribed medications, OTC , and client allergies that can interfere with the physician's recommended drug therapy. She gathers data specific to the patient's medical history and compares it to the MAR to identify unsuited drug combinations or possible allergic reactions to medication.**

## .5 Nurses' Rights:

**Medical facility guidelines also ensure six rights of the nurse .**

**The nurse has the right to clearly written medication orders that specify the dosage, route, and time for medication administration. The nurse also has the right to receive the correct form of the drug from the pharmacist and to access information about the drug .**

**She has the right to report problems in the medication system, to stop the administration process if she identifies an unsafe condition.**

**she has the right to work in a medical facility that provides guidelines and policies for safe administration of medication.**

**Test :**

**Q\ what is nursing responsibilities ?**

**Q\ what is included MAR?**

# References:


Lippincott Illustrated Reviews: Pharmacology, 7th Edition

**Pharmacology and Nursing Process, by Linda Lane Lilley,ISBN13: 978-0323358286 ,8th Edition**


**Name of subject :** *Nursing Process*

**Name of Lecturer: Dr.Tayseer Shaker Mahmood**

**Date :**

**Time of subject : 45 minutes**

**Target group: students of Nursing department**

## General objective

**In the end of lecture, the student must be:**

**1. Know Nursing Process toward patients and related to drug administration**

## Specific objective

**In the end of lecture, the student must be:**

**1. Define Nursing Process**

**2. Enumerate Nursing Process in order to application in field**

**3. Classify each process and their importance**

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## Overview of the Nursing Process: 7

**The nursing process is a systematic, rational, and continuous method of planning, providing, and evaluating individualized nursing care to optimize the administration of medications. The nursing process involves critical thinking throughout each of its five steps: assessment, nursing diagnosis, planning**

## .1ASSESSMENT

**Assessment This is the first stage of the nursing process. It involves the collection of information from the patient and their family/carers concerning their condition and perceived problems. Also, collecting subjective and objective data. Subjective is information that the patient tells you, how they are feeling, levels and sensation of pain. Objective information is that which can be measured such as blood pressure or weight. Primary information is that which is gained from the patient themselves whereas secondary data is information from other sources, such as family members. from the patient, significant others, medical records (including laboratory and diagnostic tests) and others involved in the patient's care. in the assessment step the nurse gathers data about the drug(s) that he or she is responsible for administering and monitoring.**

## .2NURSING DIAGNOSIS

**The second step of the nursing process involves clustering the data gathered during the assessment, analyzing it for patterns, and making inferences about the patient's potential or actual problems. As well as strengths .Strengths might be self-caring abilities or independence in certain areas. Or prior knowledge or experience of the illness. Actual problems are those that come directly**

## 3.PLANNING

Once the data have been analyzed and nursing diagnoses identified, the planning phase begins. During this phase, **goals** and **outcome** criteria are formulated. Nurses keeping patient comfort and safety .The goals and outcome criteria identify the expected behaviors or results of drug therapy.

SMART goals should be identified which are:

**S**pecific, **M**easurable, **A**chievable, **R**ealistic and **T**imely

**G**oal centered, that the care planned will meet and achieve the goal set

## .4INTERVENTION

The intervention phase or (Implementation) of the nursing process involves carrying out the planned activities, being mindful that ongoing assessment of the patient is needed before every intervention. For example, perhaps a patient has a laxative ordered daily but has been having loose stools all night. The nurse will need to assess this patient's current condition (i.e., complaint of loose stools) and make a decision about how to proceed with the intervention (e.g., withhold the medication and notify the prescriber.(

.5EVALUATION

The most important part of the nursing process after the assessment is done it has the care achieved the desired result and determining progress toward identified goals .

- This should not just occur at the end of a course of treatment or care, but should occur continuously as care is being
- Evaluation at the end of a course of treatment involves reassessment of all the plan of care to determine if the expected outcomes have been achieved.
- States that evaluation is determine that the assessment was accurate and complete, the diagnosis correct, the goals realistic and achievable, and the prescribed actions appropriate.With evaluation the whole process starts again

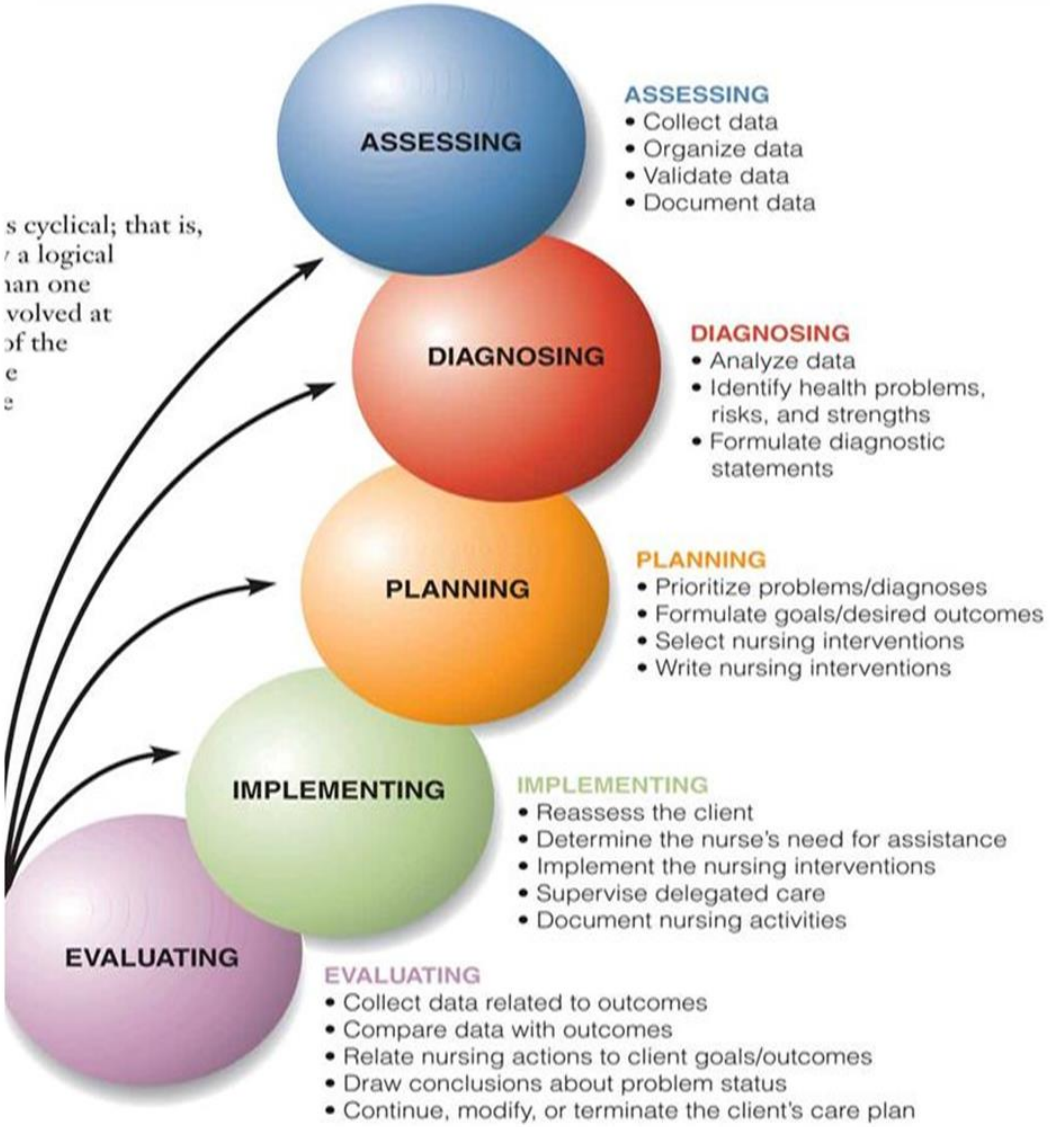
. Evaluation may involve reviewing pertinent laboratory and other diagnostic tests, observing patient performance of a learned procedure, or interviewing patients and significant others about the effects of their medications.

-Documentation is an essential component of all phases of the nursing process

**. The aim of the nursing process is used to:**

- enhance the quality of patient care .
- provide individualized, safe, effective, and thoughtful patient care.
- incorporate safe administration and monitoring of drug therapy into the overall plan of care for each patient.

**Figure 11-1 The nursing process in action.**

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Test

Q\enumerate nursing processes ?

Q\ what is the most important step in nursing processes and why?

References:

Pharmacology and Nursing Process, by Linda Lane Lilley,ISBN13: 978-0323358286 ,8th Edition

**Name of subject : *Pharmacokinetics***

**Name of Lecturer: Dr.Tayseer Shaker Mahmood**

**Date :**

**Time of subject : 45 minutes**

**Target group: students of Nursing department**

**8**

**General objective**

**In the end of lecture, the student must be:**

**1. Know the pharmacokinetic of the drug and their effect in the body**

**Specific objective**

**In the end of lecture, the student must be:**

**1. Define pharmacokinetics**

**2. Enumerate the steps of pharmacokinetics**

**3. identified factors that affect each step.**

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# Pharmacokinetics

**Movement of drugs in the body and the body affect on it.**

- **Four Processes:**
  - **Absorption**
  - **Distribution**
  - **Metabolism**
  - **Excretion**
- **Drug concentration at sites of action influenced by several factors, such as:**
  - **Route of administration**
  - **Dose**
  - **Characteristics of drug molecules (e.g., lipid solubility(**

- **Absorption: First, absorption from the site of administration permits entry of the drug (either directly or indirectly) into plasma.**

- **Distribution: Second, the drug may then reversibly leave the bloodstream and distribute into the interstitial and intracellular fluids.**

- **Metabolism: Third, the drug may be biotransformed by metabolism by the liver or other tissues.**

- **Elimination: Finally, the drug and its metabolites are eliminated from the body in urine, bile, or feces.**

**Drug Absorption:**

- **Factors affect drug absorption:**

**.1 Routes of Drug Administration**

- **Oral (per os, p.o(.**

**, Inhalation vapors, gases, smoke Mucous membrane**

**intranasal (sniffing ,,sublingual**

**rectal suppositories, Injection (parenteral(**

**intravenous (IV (intramuscular (IM(**

**subcutaneous (SC(intraperitoneal (IP; nonhumans only(**

- **Transdermal**

**.2 Lipid solubility**

**.3 pKa = pH at which 50% of drug molecules are ionized (charged(**

- **Only uncharged molecules are lipid soluble.**
- **The pKa of a molecule influences its rate of absorption through tissues into the bloodstream.**
- **pH varies among tissue sites**
  - **e.g., stomach: 3-4,**
  - **intestines: 8-9**

**Drug Distribution:**

**Drug distribute through Cell Membranes and to Capillaries Factors affect drug distribution:**

**.1 Drug affinities for plasma proteins**

- **Bound molecules can't cross capillary walls**

**.2 Blood Brain Barrier 1.Tight junctions in capillaries 2.Less developed in infants**

**.3 Disease: Cerebral trauma can decrease integrity**

**Drug metabolism:**

- **Biotransformation Liver microsomal enzymes in hepatocytes transform drug molecules into less lipid soluble by- products.**
  - **Phase I:Cytochrome P450 enzyme family**
  - **Phase II: Conjugation**
- **Factors Influencing Biotransformation**
  - **Genetic**
  - **Environmental (e.g., diet, nutrition(**
  - **Physiological differences (e.g., age, gender differences in microsomal enzyme systems(**

**Drug elimination:**

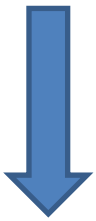
- **Two-stage kidney process (filter, absorption(**
- **Metabolites that are poorly reabsorbed by kidney are excreted in urine.**
- **Some drugs have active (lipid soluble) metabolites that are reabsorbed into circulation (e.g., pro-drugs(**
- **Other routes of elimination: lungs, bile, skin**

- **Kidney functions:**
  - **excretes products of body metabolism**
  - **closely regulates body fluids and electrolytes**
  - **The human adult kidney filters approx. 1 liter of plasma per minute, 99.9% of fluid is reabsorbed.**
  - **Lipid soluble drugs are reabsorbed with the water.**
- **Half-Life Formula**

## Homework

**Q\ how the kinetics affect on the body?**

**next**



## Pharmacodynamic

## 9

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## Pharmacodynamic :

**The study of the effect of drugs on the body**

- **Pharmacodynamics places particular emphasis on dose–response relationships, that is, the relationships between drug concentration and effect.**

**Drug Properties:**

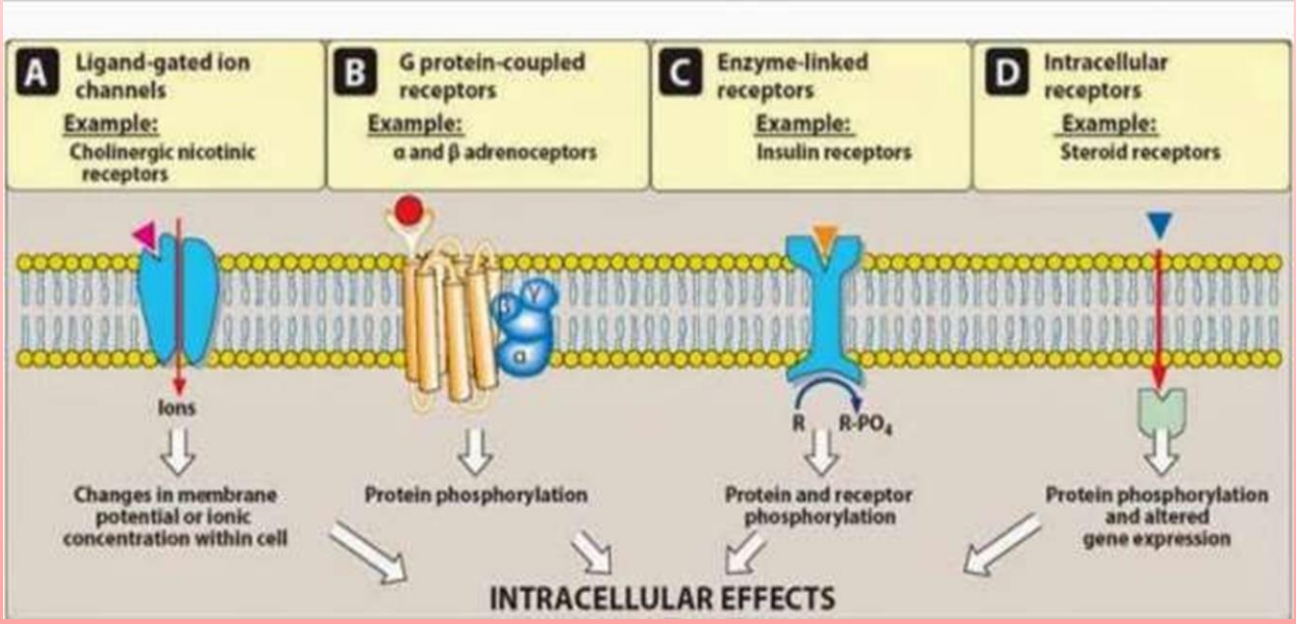
- **Drug binding to a receptor is mediated by the chemical structure of the drug that allows it to interact with complementary surfaces on the receptor.**
- **Agonists activate cellular signaling pathways to alter physiologic activity.**
- **Antagonists bind to the receptor but cannot initiate a change in cellular function. Occupation of the receptor without activation results in blockade of the actions of agonists.**
- **Partial agonists can block the actions of full agonists.**

**Receptor:**

- **Any cellular macromolecule to which a neurotransmitter or drug binds to initiate its effects. The endogenous function of a receptor is to participate in neurotransmission or physiologic regulation.**

## Type of Receptor Structures:

- Ion Channel Receptors
- G Protein-Coupled Receptors
- Enzymes linked receptors
- Intracellular receptors

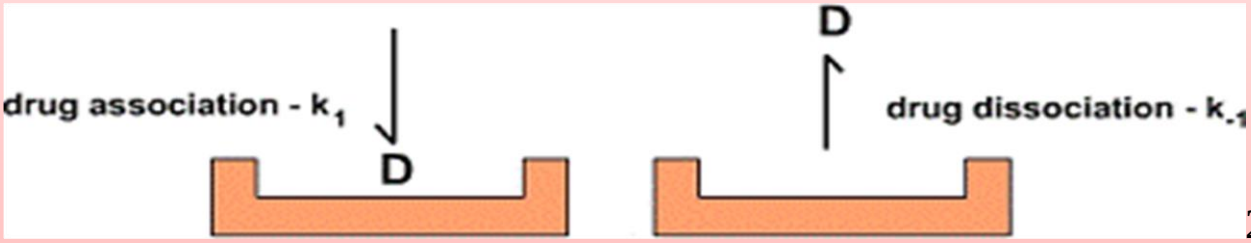



## Factors affecting Drug Action:

- Affinity is a measure of the tightness with which a drug binds to the receptor.
- Intrinsic activity is a measure of the ability of an agonist that is bound to the receptor to generate an activating stimulus and produce a change in cellular activity.

**Affinity:**

1) Affinity describes the strength of binding to receptors.




2) k1 describes the rate at which a drug associates with the receptor whilek-1 describes the ease at which a drug dissociates from its receptor.

**Dose-Response Functions:** **10**

– **Efficacy** (ED50 = median effective dose)

– **Lethality** (LD50 = median lethal dose)

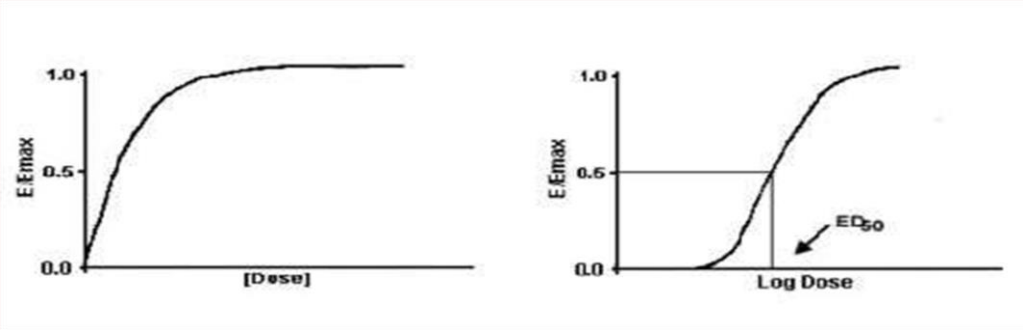
– **Therapeutic Index** = LD 50 /ED 50

• **Dose-response relationship**

As the concentration of the drug increases, more molecules will occupy more receptors, which then produces a greater response

## DOSE-RESPONSE CURVES:

To present the data, the concentration of the drug is plotted on the x-axis and the effect

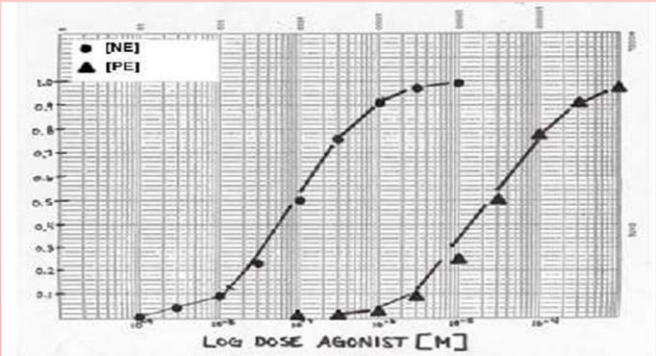



would be presented on the y-axis. A plot of drug concentration ([D]) versus effect (E/Emax

in the graphs).

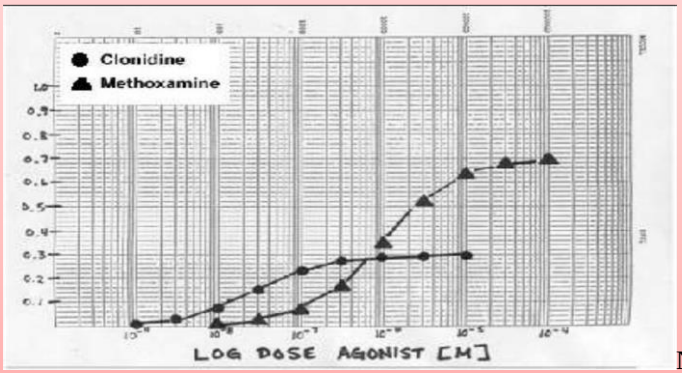
## Potency:

1) Potency refers to the concentration of a drug required to produce a given physiologic effect. Drugs with high receptor affinity will exhibit greater potency than those with lower affinity.



## Efficacy:

1) Efficacy is often used to describe the maximal level of response a drug can produce.

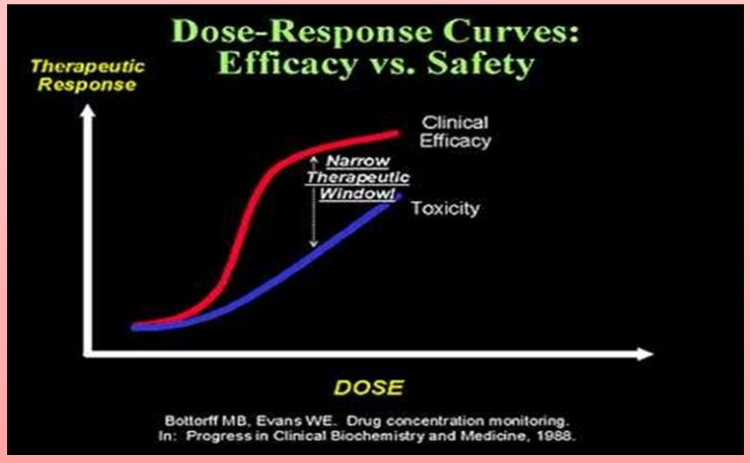



Norepinephrine would have a greater efficacy than methoxamine which in turn would have a greater efficacy than clonidine.

## Therapeutic Index:

- The Therapeutic Index is the ratio of the LD50 of a drug to produce a lethal effect to the ED50 to produce a therapeutic effect.

- **ED50:** The dose required to produce theraputic effect
- **LD50:** The dose required to produce death in 50% of a population



**Drug interaction effect:**

## Addictive Effect

An effect in which two substances or actions used in combination produce a total effect the same as the sum of the individual effects

## Synergistic Effect

The use of two or more drugs that produce a greater effect of one drug used alone

Ex. NSAID added to codeine for pain relief

## Antagonistic Effect

The use of a second drug reduces the effect of another drug. The second drug has an antagonistic effect

A second drug may bind to the same receptor as the first drug, thus preventing the agonist response

**Tolerance:**

– A diminished response to a drug as a result of continued use

– Not all drugs produce tolerance

– When tolerance is developed for one drug in a category, a cross tolerance may developed for another drug in the same category

- 2 major mechanisms that cause pharmacological tolerance

– *Enzyme Induction*

- The liver produces more drug-metabolizing enzymes

– *Receptor Effects*

- Responsiveness of the receptors decreased

## Test:

## Q\ what is the difference between efficacy and potency?

## References:

Lippincott Illustrated Reviews: Pharmacology, 7th Edition

# Aderse drug reaction:

- **Side Effects**

– Expected responses based on the pharmacologic action of the drug

- **Allergic Reactions**

– Exaggerated immune response to a certain drug

- **Organ Cytotoxic Effects**

– Adverse effects on organs

- **Idiosyncratic Reactions**

– Reaction that is particular to an individual or defined group of people

- **Drug-drug Interactions**

– Interaction of 2 or more drugs that result in a disadvantage to a patient

- **Drug-food Interactions**

– Interaction of a drug with food that results in an adverse patient reaction

- **Drug-herb Interactions**

– Interaction of a drug with herbal products that results in an adverse patient reaction

- **Drug Use During Pregnancy**

– Most drugs cross the placenta

– Thus, posing an adverse reaction to the child

## Individual Variation In Drug Responses:

- I. Body Weight and Composition
- II. Age
- III. Gender
- IV. Pathophysiology
  - A. KIDNEY DISEASE
  - B. LIVER DISEASE
- V. Tolerance
- VII. Variability In Absorption
  - BIOAVAILABILITY
- VIII. Failure to Take Medicine as Prescribed

**Name of subject :** ***Drug therapy across Life Span***

**Name of Lecturer: Dr.Tayseer Shaker Mahmood**

**Date :**

**Time of subject : 45 minutes**

**Target group: students of Nursing department**

## General objective

**In the end of lecture, the student must be:**

**1. Know the effect of drug on human along life span**

## Specific objective

**In the end of lecture, the student must be:**

**1. Define life span**

**2. Enumerate the most important features in every level of life span**

**3.Identified kinetics of drug in every level of life span**

**5. Enumerate adverse drugs reaction and individual variation to drug action**

| no | subject | page |
|---|---|---|
| 1 | Drug therapy across Life Span | 44 |
| 2 | Pregnancy | 44 |
| 3 | Breast-feeding | 45 |
| 4 | Neonatal and Pediatric | 46 |
| 5 | The Elderly (geriatrics) | 47 |
| | | |

## Drug therapy across Life Span

- Pregnancy
- Breast-feeding
- Neonatal and Pediatric

## Pregnancy

- **First trimester** is the period of greatest danger for drug-induced developmental defects
- Drugs cross the placenta by diffusion
- During the last trimester the greatest percentage of maternally absorbed drug gets to the fetus
- FDA pregnancy safety categories

| FDA Pharmaceutical Pregnancy Categories | |
|---|---|
| Category A | Adequate and well-controlled human studies demonstrate no risk. |
| Category B | Animal studies demonstrate no risk, but no human studies have been performed.<br>OR<br>Animal studies demonstrate a risk, but human studies have demonstrated no risk. |
| Category C | Animal studies demonstrate a risk, but no human studies have been performed. Potential benefits may outweigh the risks. |
| Category D | Human studies demonstrate a risk. Potential benefits may outweigh the risks. |
| Category X | Animal or human studies demonstrate a risk. The risks outweigh the potential benefits. |

## Breast-feeding

•Breast-fed infants are at risk for exposure to drugs consumed by the mother •Consider risk-to-benefit ratio

**Table 3-2 Classification of Young Patients**

| Age Range | Classification |
|---|---|
| Younger than 38 wk gestation | Premature or preterm infant |
| Younger than 1 mo | Neonate or newborn infant |
| 1 mo to younger than 1 yr | Infant |
| 1 yr to younger than 12 yr | Child |

NOTE: The meaning of the term *pediatric* may vary with the individual drug and clinical situation. Often the maximum age for a pediatric patient may be identified as 16 years of age. Consult manufacturer's guidelines for specific dosing information.

## Neonatal and Pediatric:

**Pharmacokinetics:**

•Absorption

-Gastric pH less acidic
–Gastric emptying is slowed
–Intramuscular absorption faster and irregular

•Distribution

–The younger the person, the greater the % of total body water
–Decreased level of protein binding
–Immature blood-brain barrier—more drugs enter the brain

•Metabolism

–Liver immature, does not produce enough microsomal enzymes
–Older children may have increased metabolism, requiring higher doses than infants
–Other factors

•Excretion

–Kidney immaturity affects glomerular filtration rate and tubular secretion –Decreased perfusion rate of the kidneys may reduce excretion of drugs

Factors Affecting Pediatric Drug Dosages :

•Skin is thin and permeable
•Stomach lacks acid to kill bacteria
•Lungs have weaker mucus barriers
•Body temperatures less well regulated and dehydration occurs \
•Liver and kidneys are immature, impairing drug metabolism and excretion

**Methods of Dosage Calculation for Pediatric Patients :**

**•Body surface area method**
**•Body weight dosage calculations**
–Using mg/kg

## The Elderly (geriatrics)

•Elderly: older than age 65

–*Healthy People 2010:* older than age 55

•Use of OTC medications

•Increased incidence of chronic illnesses

**Physiologic Changes in the Elderly Patient:**

**•Cardiovascular problems**

**•Gastrointestinal problem**

**•Hepatic problem**

**•Renal problem**

**The Elderly problematic Medications:**

•Analgesics, including NSAIDs

•Anticoagulants

•Anticholinergics

•Antidepressants

•Antihypertensives

•Cardiac glycosides (digoxin)

•Sedatives and hypnotics

•Thiazide diuretics

**Test**

**Q\ whats difference between kinetics in younger and in elderly man?**

**Name of subject :** ***Physiology of nervous system***

**Name of Lecturer: Dr.Tayseer Shaker Mahmood**

**Date :**

**Time of subject : 45 minutes**

**Target group: students of Nursing department**

## General objective

**In the end of lecture, the student must be:**

1. **Know the effect of nervous system on drug effect on the body**
2. **Understand different effect of drug according to different types of**

## Specific objective

**In the end of lecture, the student must be:**

**1. Define central and peripheral nervous system**

**2. Enumerate sympathetic and parasympathetic receptors**

**3.Identified cholinergic and nicotinic receptors**

**4. Enumerate agonist and antagonist of**


| no | subject | page |
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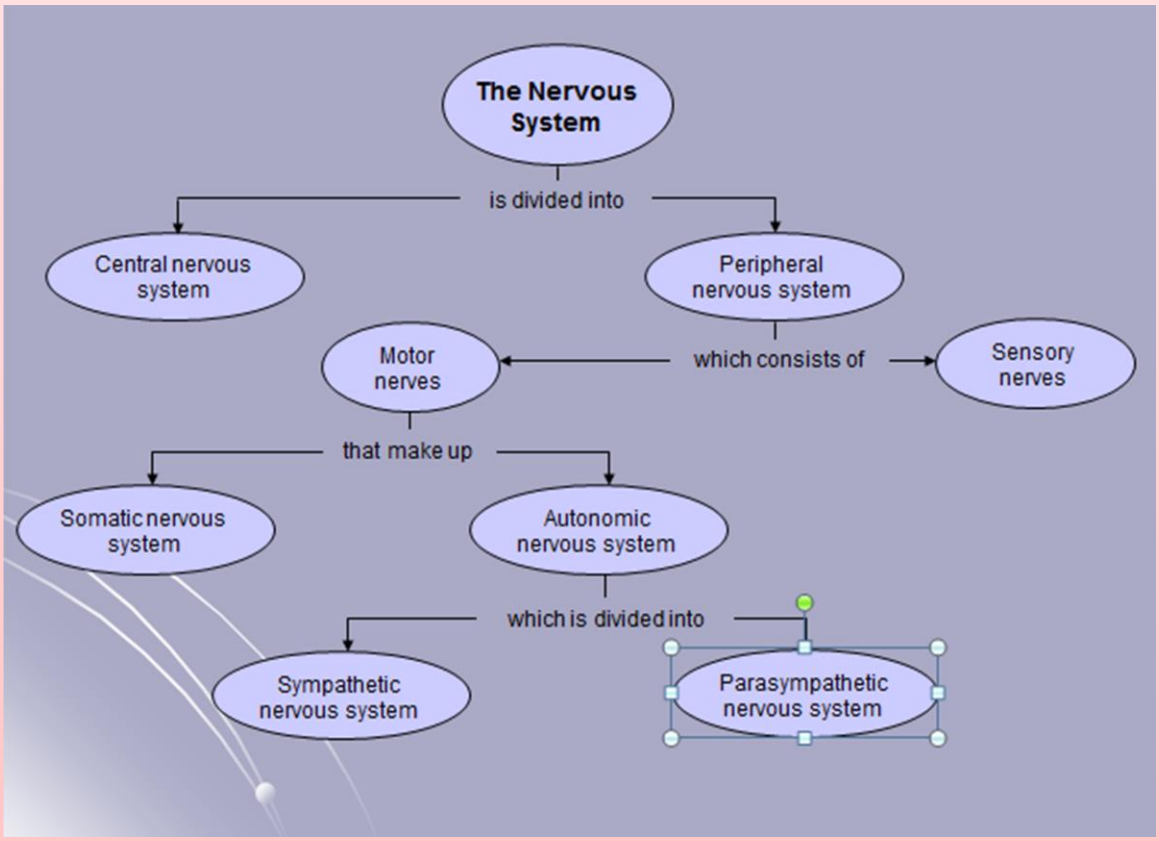
| 1 | Physiology of nervous system | 49 |
| 2 | Peripheral nervous system ( PNS ) | 49 |
| 3 | Autonomic nervous system(ANS) Neurotransmitters: | 52 |
| 4 | Cholinergic neurons | 52 |
| 5 | Cholinergic drugs agonists Direct acting(cholinomimetics) | 53 |
| 6 | Indirect acting cholinomimetics (AchE inhibitors) | 53 |
| 7 | Cholinergic antagonists | 54 |

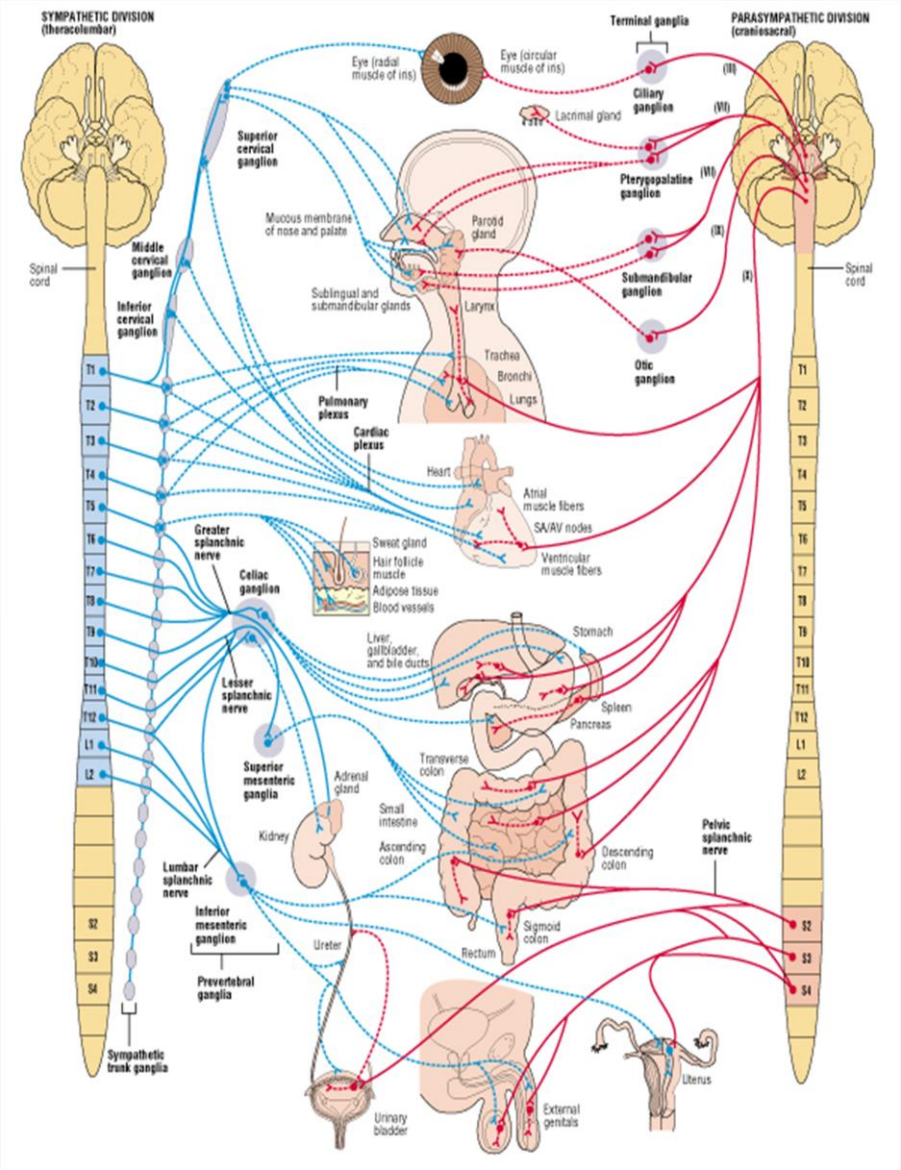
# Physiology of nervous system




## Peripheral nervous system ( PNS )

- NERVES that connect the central nervous system to the rest of the body

1. Motor Division – impulses from CNS to muscles or glands
    - Two Parts:
        - Somatic Nervous System
        - Autonomic Nervous System
    - Subdivided into two system that have opposite effects on the same organs:
        1. **Parasympathetic** – decreases heart rate
            - Controls internal organs during normal activity
        2. **Sympathetic** – increases heart rate
            - Controls internal organs during high stress activity
            -
2. Sensory Division – transmits impulses from sense organs to CNS




1. Sympathetic (thoracolumbar) division
   - preganglionic  cell bodies in thoracic and first 2 lumbar segments of spinal cord
2. Parasympathetic (craniosacral) division
   - preganglionic cell bodies in nuclei of 4 cranial nerves and the sacral spinal cord

- **Autonomic nervous system(ANS) Neurotransmitters:**
- Classified as either cholinergic or adrenergic neurons based upon the neurotransmitter released.
- **Cholinergic** neurons release acetylcholine from preganglionic neurons & from parasympathetic postganglionic neurons
- **Receptor:**
- Nicotinic
- Muscarinic
- **Adrenergic** neurons release norepinephrine (NE) from postganglionic sympathetic neurons only
- **Receptor:**
  - Alpha1 and Beta1 receptors produce excitation
  - Alpha2 and Beta2 receptors cause inhibition
  - Beta3 receptors (brown fat) increase thermogenesis

**Cholinergic neurons**

They are found in:
CNS, adrenal medulla, autonomic ganglia (sympath. And parasympath )

**Cholinergic recepters**

**1-Muscarinic Recepters (M 1-5).**
**2-Nicotinic Recepters (Nn, Nm).**

Location:

Nervous system contain all 5 sub types.
M1: Gastric parietal cell( increase gas. Acid)
M2: Heart ( S A node, Contractility)
M3: Smooth muscle(eye,Gut, bladder wall,sphincters , bronchi and exocrine gland).

**Nicotinic Recepters**

These are :
Nn:Neuronal type ganglionic recepters they found in the CNS,Autonomic gang. And adrenal medulla).
Nm:Muscle type or end plate receptors, found in the skeletal m.(neuromuscular junction.

## Cholinergic drugs agonists(cholinomimetics) 13

1-Direct acting cholinomimetic.
2-Indirect acting cholinomimetic(ACh inhibiters).

### 1-Direct acting cholinomimetic

They act by binding directly to cholinreceptor.

**Acetylcholine:**

Is therapeutically of no importance because its rapid inactivation by Ach E. have both musc. And nicotinic activity.

**Pharmacological action:**

CVS: decrease HR and C.O.
B.V.: vasodilitation
B. P.: hypotention

Respiratory system: Bronchospasm, increase bronchial secretion.
GIT: Increase
Salivary secretion
intestinal secretion and motility
Relaxation of sphinictors
G.U.T.: Expultion of urine.
Eye: miosis(constriction of pupil)
Ex:

- **Carbachol (carbamylCholine) :for glaucoma**
- **Bethanecole :To stimulate atonic bladder in post partum of postop. Non-obst. Urinary retention**
- **Pilocarpine :Sweating and salivation.**

## Indirect acting cholinomimetics (AchE inhibitors)

AChE enz that cleaves Ach to choline and acetate, Inhibition of this enz. Prolong the life time of the endogenous Ach.

These inhibitors are either reversible or irreversible agents:

A- The reversible are:

1- Physostigmine :As antidote of drug with antichlinergic action

2- Neostigmine :Symptomatic treatment of Myasthenia gravis.

3- Pyridostigmine :Used in chronic management of Myasthenia gravis.

## Cholinergic antagonists

1- Muscarinic receptor blockers.

2- Nicotinic receptor blockers (Ganglionic blockers) (Nn).

3- N-M blockers :

a- Competitive (non-depolarizing)

b- Noncompetitive (polarizing)

**Muscarinic receptor blockers**

**1.Atropine**

**2.Homatropine**

**3.Hyosine(scopolamine)**

**1- Atropine**

Its acompetitive antagonist of mus. Receptors in both central and peripheral N.S.

**Action:**

CVS:heart : at low doses (brady cardia)
at high doses (tacky cardia)
B.V.: at high dose cause vasodilitation.
Resp.: bronchial m. relaxation.
U.T. : decrease motility and urinary retention may occure.
G.I.T.: decrease tone and peristalysis.
Eye : mydriatic agent (increase IOP).
Exocrine gland : Decrease:
salivery sec.
sweat gland sec.
lacrimal gland sec.
bronchial sec.

**Therapeutic uses**

1- Ophthalmic: topical atropine(mydriasis)
2-Anti-spasmodic.
3- Anti-secretory agent (to block sec. preop.)
4- For bradycardia following M.I.
5- antidote for cholinomimetic agents.

**Ganglionic Blockers (Nn blockers)**
These specifically act on Nic. Receptors in the autonomic gang.
Rarely used therapeutically.
They often serves in experimental pharmacology.
**These includes:**

**1.Nicotine**
Low doses causes (tachycardia, increase B.P., vasoconstriction).
High doses (decrease B.P. and motor activity of both bowel and urinary bladder)

**2- Trimethaphan**
**3- Mecamylamine**

## Test

**Q\ what happened to the body when the person faced sudden things?**

**Q\ how the indirect agonist cholinergic drug act?**


**References:**

**Lippincott Illustrated Reviews: Pharmacology, 7th Edition**

**Pharmacology and Nursing Process, by Linda Lane Lilley,ISBN13: 978-0323358286 ,8th Edition**

**Name of subject : Adrenergic & anti-Adrenergic Drugs**

**Name of Lecturer: Dr.Tayseer Shaker Mahmood**

**Date :**

**Time of subject : 45 minutes**

**Target group: students of nursing department**

**14**

**General objective**

**In the end of lecture, the student must be:**

1. **Know the effect of adrenergic and anti adrenergic drugs**

## Specific objective

## In the end of lecture, the student must be:

1. Define adrenergic division
2. Enumerate drug act directly on adrenergic receptors
3. Enumerate drug act indirectly on adrenergic receptors
4. Identifiy drug act antagonist and block adrenergic receptors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| 1 | Fight or flight response results in: | 58 |
| 2 | Direct adrenergic drug action | 58 |
| 3 | Indirect adrenergic drug action | 58 |
| 4 | INDICATIONS FOR USE | 58 |
| 5 | ANTI-ADRENERGICS: Sympatholytic | 59 |
| 6 | Inderal | 59 |

# ADRENERGIC AND ANTI-ADRENERGIC DRUGS

## Fight or flight response results in:

1. Increased BP
2. Increased blood flow to brain, heart and skeletal muscles
3. Increased muscle glycogen for energy
4. Increased rate of coagulation
5. Pupil dilation

**Direct adrenergic drug action**

- Affects postsynaptic alpha 1 and beta receptors on target effector organs
- Examples: epinephrine, Isuprel, norepinephrine, phenylephrine

**Indirect adrenergic drug action**

occurs by stimulation of postsynaptic alpha 1, beta 1 and beta 2 receptors.Cause release of norepinephrine into the synapse of nerve endings or prevent reuptake of norepinephrine.

- Examples include cocaine and TCAs**Tricyclic antidepressants**

# INDICATIONS FOR USE

- **Emergency drugs in treatment of acute cardiovascular, respiratory and allergic disorders**
- **In children, epinephrine may be used to treat bronchospasm due to asthma or allergic reactions**
- **Phenylephrine may be used to treat sinus congestion**

## ANTI-ADRENERGICS: Sympatholytic

Block or decrease the effects of sympathetic nerve stimulation, endogenous catecholamines and adrenergic drugs

## ALPHA 1 ANTAGONISTS:

**Minipress (prazosin)—prototype.**

**Hytrin (terazosin) and Cardura (doxazosin)—both are longer acting than Minipress.**

## BETA ADRENERGIC BLOCKING MEDICATIONS

Prevent receptors from responding to sympathetic nerve impulses, catecholamines and beta adrenergic drugs.

## EFFECTS OF BETA BLOCKING DRUGS

**Decreased heart rate**
**Decreased force of contraction**
**Decreased CO( cardiac output)**
**Slow cardiac conduction**
**Decreased automaticity of ectopic pacemakers**
**Decreased renin secretion from kidneys**
**Decreased BP**
**Bronchoconstriction**
**Less effective metabolism of glucose. May result in more pronounced hypoglycemia and early s/s of hypoglycemia may be blocker (tachycardia)**
**Decreased production of aqueous humor in eye**
**May increase VLDL and decrease HDL**
**Diminished portal pressure in clients with cirrhosis**

## Ex:Inderal (propranolol) is prototype

**Useful in treatment of hypertension, dysrhythmias, angina pectoris, MI**
**Useful in pheochromocytoma in conjunction with alpha blockers (counter catecholamine release)**
**migraines**

## Test

**Q\what is therapeutic use of propranolol?**

**Q\ enumerate effect of beta blockers?**

**References:**

**Lippincott Illustrated Reviews: Pharmacology, 7th Edition**

**Name of subject : Drugs affecting the Cardiovascular system**

**(Antihypertensive Drugs, Antianginal, drugs for heart failure, myocardial infarction and antiarrhythmic drugs )**

**Name of Lecturer: Dr.Tayseer Shaker Mahmood**

**Date :**

**Time of subject: 45 minutes**

**Target group: students of nursing department**

## General objective

**In the end of lecture, the student must be:**

1. **Understand the disorders in CVS and different mechanism and drugs to treat it**

## Specific objective

## In the end of lecture, the student must be:

**1.define hypertension,heart failure, angina,arrhythmia and MI**

**2. Enumerate drugs that treat different disorder in CVS and their mechanisms**


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| 1 | Antihypertensive Drugs | 63 |
| 2 | Heart failure | 65 |
| 3 | Myochardial infarction | 65 |
| 4 | angina | 66 |
| 5 | antiarrythmia | 66 |
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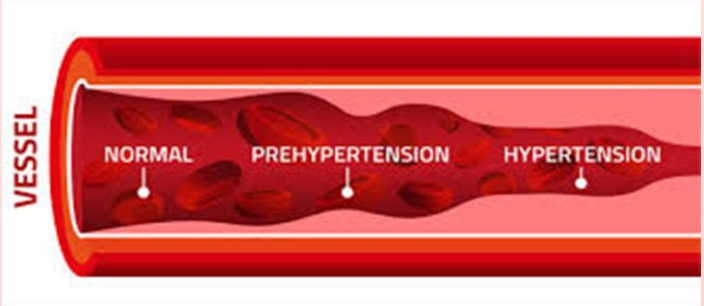
## ■ Antihypertensive Drugs




- **Diuretics:**
  - **Thiazides**: Hydrochlorothiazide, chlorthalidone
  - **Loop diuretics** : Furosemide
  - **K+ sparing**: Spironolactone, triamterene and amiloride

Action: Acts on Kidneys to increase excretion of Na and H2O – decrease in bl volume – decreased BP

- **Angiotensin-converting Enzyme (ACE) inhibitors:**

  Is an enzyme that responsible for convert angiotensin I to angiotensin II (potent vasoconstriction)in kidney.
  - Captopril, lisinopril., enalapril, ramipril and fosinopril

Action: Inhibit synthesis of Angiotensin II – decrease in peripheral resistance a blood volume

- **Angiotensin (AT1) blockers:**
  - Losartan, candesartan, valsartan and telmisartan

Action: Blocks binding of Angiotensin II to its receptors

- **Centrally acting:**
  - Clonidine, methyldopa

Action: Act on central α2A receptors to decrease sympathetic outflow – fall in BP

- **ß-adrenergic blockers:**
  - **Non selective:** Propranolol \ **Cardioselective: Metoprolol** (others: atenolol, esmolol, betaxolol)

Action: Bind to beta adrenergic receptors and blocks the activity

- **ß and α – adrenergic blockers:**
  - Labetolol and carvedilol
- **α – adrenergic blockers:**
  - Prazosin, terazosin, doxazosin, phenoxybenzamine and phentolamine

Action: Blocking of alpha adrenergic receptors in smooth muscles – vasodilatation

- **Calcium Channel Blockers (CCB):**

  Verapamil, diltiazem, nifedipine, felodipine, amlodipine, nimodipine Action: Blocks influx of Ca++ in smooth muscle cells – relaxation of SMCs – decrease BP

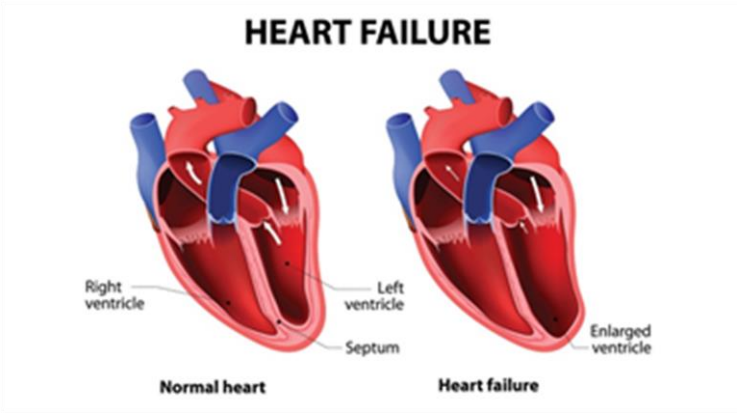
- **K+ Channel activators:**
  - Diazoxide, minoxidil, pinacidil and nicorandil

Action: Leaking of K+ due to opening – hyper polarization of SMCs – relaxation of SMCs

- **Vasodilators:**
  - Arteriolar – Hydralazine (also CCBs and K+ channel activators)
  - Arterio-venular: Sodium Nitroprusside

# Heart failure: 16

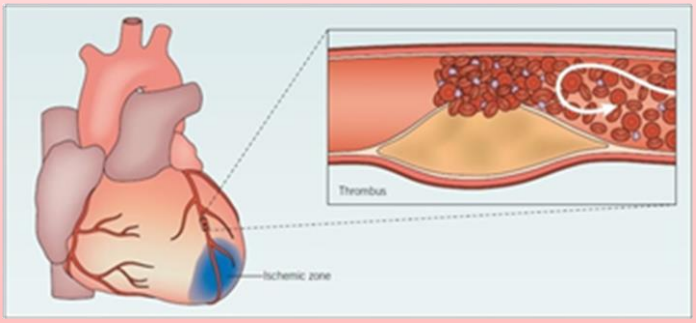
Causes :is when the heart is unable to pump sufficiently to maintain blood flow to meet the body's needs.




**Medication:**

- angiotensin-converting enzyme (ACE) inhibitors (ACE-I) or angiotensin receptor blockers
- Beta-adrenergic blocking agents
- Aldosterone Antagonists
- Diuretics

**Myocardial infarction (MI),** commonly known as a **heart attack**, causes: occurs when blood flow decreases or stops to a part of the heart, causing damage to the heart muscle.

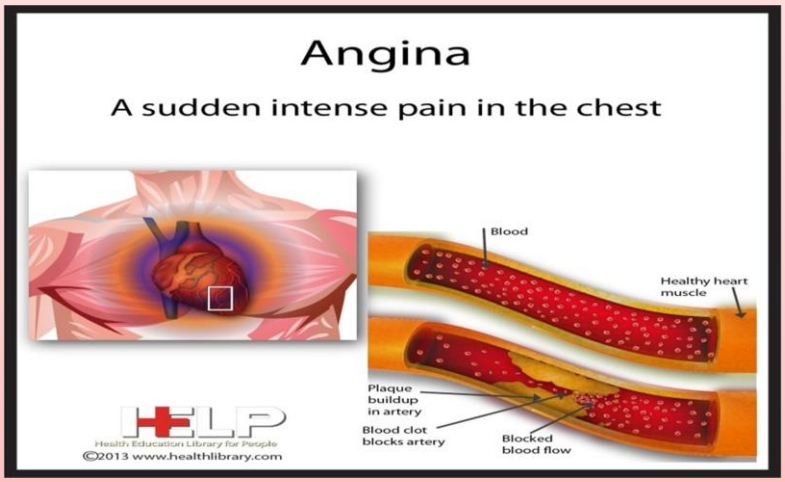



**Treatment in general aims to unblock blood vessels, reduce blot clot enlargement, reduce ischemia, and modify risk factors with the aim of preventing future MIs.**

**Medication**

- **Statins,** drugs that act to lower blood cholesterol, decrease the incidence and mortality rates of myocardial infarctions
- **Aspirin**

**Angina causes:** is usually due to obstruction or spasm of the coronary arteries that provide heart with blood.

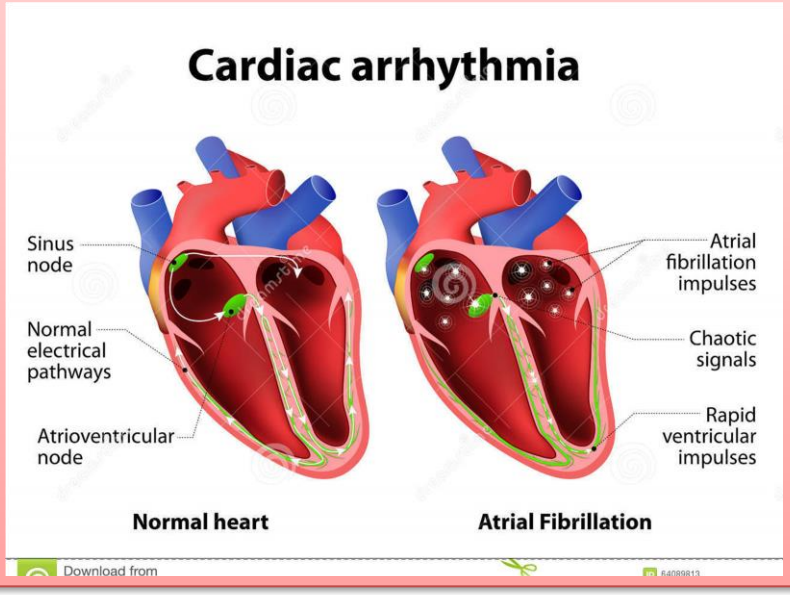



**Medication:**

- **nitroglycerin.** It is a potent **vasodilator** that decreases myocardial oxygen demand by decreasing the heart's workload.
- **Beta blockers** and **calcium channel blockers** act to decrease the heart's workload, and thus its requirement for oxygen.

**Antiarrhythmic agents**, also known as **cardiac dysrhythmia medications**, are a group of pharmaceuticals that are used to suppress abnor rhythms of the heart (cardiac arrhythmias), such as atrial fibrillation, atrial flutter, ventricular tachycardia, and ventricular fibrillation.

Causes: if the electrical impulses that coordinate the heartbeat don't work properly causing heart to beat too fast or too slow or irregularly.



**Test**

**Q\give the mechanism of action of the following drugs:**

**Nitroglycerin, Captopril, diltiazem, minoxidil, statin**

## References:

Lippincott Illustrated Reviews: Pharmacology, 7th Edition

**Name of subject : Drugs for disorders in Endocrine System**

**Name of Lecturer: Dr.Tayseer Shaker Mahmood**

**Date :**

**Time of subject: 45 minutes**

**Target group: students of nursing department**

## General objective

**In the end of lecture, the student must be:**

**1.Understand the disorders in**

## Specific objective

**In the end of lecture, the student must be:**

**1.Define hyperthyroidism, diabetes mellitus**

**2. Enumerate drugs that treat different disorder in thyroid , pancreas, and female &male reproductive glands and their**


| no | subject | page |
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| 2 | Disorder of Thyroid gland | 69 |
| 3 | Disorder of female & Male reproductive system hormones : | 70 |
| 4 | Disorder of Pancreas gland | 72 |
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# *Introduction to the Endocrine System*

Hormones, produced by endocrine glands, are important in regulating and integrating the bodies functions. They have roles in metabolism, fluid balance, growth, development and reproduction. Hormones can be subdivided into classes based on their chemical structure; steroid hormones, amino-acid derived hormones and peptide hormones.

## Disorder of Thyroid gland

**Hyperthroidism**

Hyperthyroidism is elevated circulating thyroid hormone, and when the levels are very high the condition is known as thyrotoxicosis, a medical emergency. Graves' disease, an autoimmune disease causing the excessive secretion of thyroid hormone, accounts for 60-90% cases of thyrotoxicosis.

**Treatment:** for hyperthyroidism is drugs or surgery, or drugs prior to surgery. The drugs used include **propylthiouracil**, **Carbimazole ,methimazole, iodide** and **radioactive iodine.** Adjuvant treatment of hyperthyroidism is to treat the consequences of hyperthyroidism on the heart and includes **β-adrenoceptor antagonist and Ca2+ channel blockers**.

**Mechanism of action:**

**Propiothiouracil** inhibits 2 enzymes involved in the synthesis of the thyroid hormones; thyroid gland peroxidase and peripheral tissues iodothyronine 5/-deiodinase (Figure 15.1). **Carbimazole** only inhibits thyroid gland peroxidase (Figure 15.1). The incidence of side effects is relatively low with these drugs.

**High doses of iodide** inhibit its own transport into thyroid gland, and therefore the synthesis of thyroxine. High dose iodide also inhibits the release of thyroid hormone. and this inhibition last about 2 days.

**Hypothyroidism**

Congenital Hypothyroidism (often called cretinism) is most common preventable cause of mental retardation. Hypothyroidism, which when it is severe is known as myxedema) is the most common disorder of thyroid function and is most often **due to iodine deficiency**, and is also known as primary hypothyroidism).

**Treatment** :of hypothyroidism is to use **iodated table salt** as prevention, which promotes the synthesis of the thyroid hormones. In hypothyroidism, replacement therapy with **thyroxine** is used as treatment.

**Hyperparathyroidism**

**Primary hyperparathyroidism:** (high secretion of the parathyroid hormone from the parathyroid glands) is a common endocrine disorder, especially in postmenopausal women. It is usually caused by parathyroid adenoma (tumours) or spontaneous hyperplasia.

**high levels of parathyroid hormone**, :

increased resorption of calcium from bones, and increased absorption of calcium from the gastrointestinal tract and kidneys. With high plasma levels of calcium and high urine phosphate levels, there can be renal stones, and bone pain leading to lesions and fractures. **Secondary hyperparathyroidism:** is associated with chronic renal insufficiency.

**Treatment** :1.**Surgery** removing part of the parathyroid gland .

2.**Cinacalcet** is the drug used to treat hyperparathyroidism. Cinacalcet increases the sensitivity of calcium-sensing receptors to calcium, which turns off the stimulus for parathyroid hormone secretion. cinacalcet inhibits parathyroid cell proliferation and parathyroid hormone synthesis.

## Disorder of female & Male reproductive system hormones :

**oestrogens and progestogens**

The oestrogens are also used in:
disorders of menstruation (e.g. endometrial hyperplasia),
prostate cancer
hirsutism (excessive androgen-dependent hair growth).
Oestrogens and progestogens are used together as hormone replacement therapy (HRT). HRT is a combination of a low dose of an oestrogen, such as oestradiol, and a progestogen, usually medoxyprogesterone acetate. HRT can be used relatively safely for up to 5 years to relieve moderate-severe menopausal symptoms.
The progestogen is mainly used to prevent endometrial cancer with the oestrogen. In women that have had a hysterectomy (removal of the uterus), oestrogen is safe and effective alone in preventing menopausal symptoms.
**Contraception:**Most oral contraceptives contain an oestrogen-like drug (oestrogen) and a progestogen combined in various doses to mimic the 28-day menstrual cycle. The oestrogen alone will inhibit ovulation, but often leads to 'break-through' bleeding, which can be prevented by adding a progestogen. The progestogen also decreases the production of luteinizing hormone to prevent ovulation and fertilization. **Drugs:** oestrogen is **ethinyl-oestradiol.** Progesterone itself is not used as a contraceptive as it is not active after oral administration. Oral active progestogens commonly used in combined oral contraceptives formulations are low dose **levonorgestrel and norethisterone**. The 'minipill' is a progestogen-only oral contraceptive Without the oestrogen, there is a higher risk of 'break-through' bleeding.

**adverse effects:** Combined oral contraceptives increased risk of thrombosis and myocardial infarction especially in smokers and women aged over 25 years of age. This increased risk is related to the oestrogen rather than progestogen part of the combined oral contraceptive.

**Drugs and the hormones of male reproduction**

The main male sex hormone **testosterone** is used to **treat androgen deficiency**. Androgen deficiency is associated with testicular failure caused by cryptorchidism (failure of one or both testes to descent into scrotum), orchitis (inflammation of the testes), orchidectomy (surgical removal of one or both testes). Androgen deficiency can also be due to insufficiency of the pituitary-hypothalamic axis or in delayed male puberty.

For a longer action, **the enanthate ester of testosterone** is used, and it is administered intramuscularly every 1-4 weeks. **Adverse effects** are mainly related to route of administration, and with intramuscular can be bleeding, infection and extrusion.

**Androgens are anabolic agents** stimulate the formation and maintenance of muscular and skeletal proteins. For these reasons, the anabolic steroids have become drugs of abuse in sport.

**Benign prostatic hyperplasia** (BPH) is excessive growth of the prostate gland, which surround the urethra and obstructs the bladder to make urination difficult. In severe prostatic hyperplasia, surgery to remove part or the entire prostate may be necessary. Prior to this, **a1-adrenoceptor blocker such as prazosin or tamsulosin,** which is the first example of an uroselective blocker shows selectivity for a1A-adrenoceptors (which are found on the prostate)

**finasteride**, which is a 5α-reductase inhibitor. In the prostate gland, testosterone is metabolised to 5α-dihydotestosterone, which is more potent than testosterone as an androgen, and promotes prostrate grown. The enzyme responsible for this metabolism is 5α-reductase, and consequently inhibition of the enzyme with finasteride prevents the growth of the prostate.

# Pancreas gland 18

## Diabetes Mellitus

Diabetes means large volume of urine flowing through. Diabetes mellitus is when the urine is sweet. Diabetes insipidus is when the urine is tasteless. Diabetes insipidus is a rare condition due to impaired renal conservation of water. Diabetes mellitus is the subject of this chapter.

In diabetes the underlying syndromes is **hyperglycemia**, high circulating levels of glucose. The glucose sticks to many sites to interfere with many processes in the body, and this underlies the complications with diabetes. The glucose sticks to haemoglobin, and we use this to monitor the ongoing glucose levels in diabetes.

**There are two types of diabetes mellitus.** **Type I** diabetes mellitus (which was previously known as insulin-dependent diabetes mellitus (IDDM)) only accounts for 10% of diabetes. autoimmune disease of the pancreatic cells that secrete insulin. Once the pancreatic cells are destroyed the individual no longer produces or stores insulin. As insulin is essential to the management of glucose in the body, the resulting high levels of glucose can have major effects, if left untreated.

**Physiology of the Pancreas**

Insulin is secreted from a specialized part of the pancreas known as the Islets of Langerhans. The beta cells of the islets secrete insulin. The secretion of insulin is increased by glucose, amino acids, and fatty acids. For glucose, the mechanism is that the glucose is transported into beta cells, where it is metabolized . This glucose metabolism leads to ATP formation, and ATP closes the K+-ATP-dependent channel. Normally, the K+-ATP channel is open and K+ moves out of the cell through this ion channel, keeping the membrane potential low. When the K+-ATP channel is blocked by ATP from the metabolism of glucose, the amounts of K+ in the cell increases, and this depolarization opens the voltage-dependent Ca2+ channels. Ca2+ entry into the beta cells promotes insulin secretion.

- **Treatment :**
- **Insulin Replacement Therapy**

The logical treatment for low insulin secretion or low sensitivity to insulin is to replace the insulin. The insulin for replacement is made my recombinant DNA techniques. Insulin replacement therapy is indicated for all subjects with Type 1 diabetes. Type 2 diabetes also end up by producing very little insulin and becoming very insensitive to the insulin they produce, and need to use insulin replacement therapy.

- **Metformin**

metformin has been used in type 2 diabetes for many years, Metformin inhibits **gluconeogenesis,** which is the conversion of amino acids to glucose in the liver. Metformin increases the sensitivity to insulin in type 2 diabetes, Metformin is **orally active**, Metformin is **anti-hyperglycemic**, it reduces hyperglycemia to normoglycemia. **Metformin is not hypoglycemic** i.e. it does not reduce normoglycemia to hypoglycemia. **Thus, hypoglycemic adverse effects are rare with metformin.** The most common **unwanted effects** of metformin are gastrointestinal disturbances, such as nausea and diarrhoea. One **advantage** of metformin over some of the anti-diabetic drugs is that there is **no weight gain** with metformin. In contrast, insulin replacement therapy and the sulphonylureas commonly cause weight gain, which tends to make the diabetes worse

- **Acarbose**

Carbohydrates are broken down to glucose in the gut by the enzyme α-glucosidase, and the glucose is absorbed. Acarbose is an inhibitor of α-glucosidase, thereby inhibiting the formation of glucose in the gut. Acarbose is active after oral administration, and is used in the treatment of Type 2 diabetes only. Acarbose has a different mechanism of action to metformin, which means it can be used with metformin to have an additive effect on plasma glucose levels.

- **Sulfonylureas**

The sulfonylureas inhibit the K+ATP channel in the pancreatic beta cells to increase insulin secretion (Figure 14.1). The sulfonylureas are active **after oral administration** and are given orally once or twice daily, which is a major advantage over insulin. Their disadvantage over insulin is that they are **not effective in type 1 diabetes,** as they require the presence of insulin to work. The major **adverse effect** of the sulfonylureas is **hypoglycemia**, the sulfonylureas **cause a weight gain** of a few kilograms, which tends to counter some of the benefit with these agents. The oral sulfonylureas may become less effective with time, **this is due to the progression of beta-cell failure or of the insulin resistance, making less endogenous insulin available or making the available insulin less effective. When this happens, the type 2 diabetic, may have to proceed to insulin replacement therapy.**

- **Glitazones**

An example of a glitazone is **pioglitazone**. The glitazones stimulate the nuclear receptor peroxisome proliferation-activate receptor-γ (PPARγ), to proteins in liver and leads to a reduced glucose output from liver All of these effects combine to decrease plasma glucose. The glitazones are active **after oral administration**, and are used in type 2 diabetes. The major **adverse effects** of the glitazones are **weight gain**, **oedema** and **anaemia.** Hypoglycemia is not usually a problem with the glitazones

- **Glucagon-Like Peptide-1, Exenatide( s\c) and Sitagliptin(oral)**

GLP-1 is produced by the gut, and has a major role in controlling glucose levels. GLP-1 stimulates insulin secretion, and inhibits glucagon secretion, both of which will lead to a decrease in glucose levels.

# Test:

## Q\ what is the difference between hypo and hyper thyrodism?

## Q\what is the function of parathyroid gland?

## Q\how can cured the deficiency of estrogen and progesterone?

# References:

Lippincott Illustrated Reviews: Pharmacology, 7th Edition

**Name of subject|:Drugs of disorders of Respiratory and Digestive System**

**Name of Lecturer: Dr.Tayseer Shaker Mahmood**

**Date :**

**Time of subject : 45 minutes**

**Target group: students of nursing**

## General objective

**In the end of lecture, the student must be:**

**1. Understand the main diseases of respiratory and digestive systems**

**2. Know groups of drugs to treat disorders of Respiratory and Digestive System**

## Specific objective

**In the end of lecture, the student must be:**

**1. Define histamine and its function**

**2 Understand mechanism of asthma**

**3.enumerate different drugs for asthma**

**4.know the drugs that treat rihinitis,cold,**

**5.define the antitussive , mycolytic and expectorant**

**6. define PPI**

**7.explain the different between laxative and drastic and puragative**

**8. explaine therapy of H-pylori**

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# Respiratory and Digestive System Drugs

## Antihistamine drugs:

**histamine :**

**The primary site the mast cell granules (or basophils)**

- **Mast cells are important in that they release histamine in response to potential tissue injury**

The most important mechanism for histamine release is in response to an immunological stimulus.

**Mechanism of Action –**

**Histamine mediates its effects by interacting with receptors.**

**Receptor Types $H_1$, $H_2$,and $H_3$ types.**

**Pharmalogical effects of Histamine:**

1)Histamine promotes( intestinal and Bronchiolar ) smooth muscle contraction which is an H1 receptor mediated effect

2)Histamine significant increase in gastric acid and gastric pepsin secretion which is an H2 receptor mediated effect

3) Vasodilation of arterioles and precapillary sphincters which is an H1and H2 receptor mediated effect

## Receptor antagonists:

**selective blockade of histamine receptors ($H_1$, $H_2$, $H_3$ types)**

## H1 receptor antagonists:

**First-generation** ( diphenhydramine,, promethazine.., chlorpheniramine)

**Second-generation** (orally active, hepatic metabolism ,,,.$H_1$ receptor blockers: competitive antagonism

## Therapeutic uses:

**1） Allergic Reactions: (allergic rhinitis , Atopic dermatitis, hay fever, urticaria(风疹）**

**2） Motion sikness: ( vestibular disturbances,,,,,)**

**3)Sedation and hypnotics. : these agents to be used has sleep-aids, i.e. hypnotics.**

**The newer $H_1$ antagonists, by contrast, cause minimal or no sedation.**

## adverse effect

- 1) Inhibition of CNS
- 2) Some first-generation $H_1$ antagonists have strong antimuscarinic actions (atropine-like effects)
- 3) others: Second-generation overdosage: may induce cardiac arrhythmias

## $H_2$ receptor antangnists:

- **Cimetidine (Tagamet)**
- **Ranitidine (Zantac)**
- **Famotidine (Pepcid)**

## Clinical uses:

- Peptic Ulcer and Duodenal Disease
- Gastric Ulcer: reduce symptoms ( promote healing for benign gastric ulcers )
- Gastroesophageal Reflux Disorder (erosive esophagitis)

**Zollinger-Ellison syndrome:** acid hypersecretion -- caused by gastrin-secreting tumor

Systemic mastocytosis and multiple endocrine adenomas:

## adverse effects:

- Most common adverse effects: diarrhea , dizziness , somnolence , headache , rash, thrombocytopenia , neutropenia , aplastic anemia)

# Asthma

- Reactive airway disease,,,Chronic inflammatory lung disease,,,, Inflammation causes varying degrees of obstruction in the airways ,Asthma is reversible in early stages

**Triggers of Asthma:** 1.Allergens 2.Exercise 3.Respiratory Infections 4.Nose and Sinus problems

5.Drugs and Food Additives 6.GERD 7.Emotional Stress

**Asthma Pathophysiology:**

**Early-Phase Response**

-Peaks 30-60 minutes post exposure, subsides 30-90 minutes later

-Characterized primarily by bronchospasm

-Increased mucous secretion, edema formation, and increased amounts of tenacious sputum

-Patient experiences wheezing, cough, chest tightness, and dyspnea

**Late-Phase Response**

-Characterized primarily by inflammation

-Histamine and other mediators set up a self-sustaining cycle increasing airway reactivity causing hyperresponsiveness to allergens and other stimuli

-Increased airway resistance leads to air trapping in alveoli and hyperinflation of the lungs

-If airway inflammation is not treated or does not resolve, may lead to irreversible lung damage

**Asthma Drug Therapy**

**-Long-term control medications:**-Achieve and maintain control of persistent asthma

**-Quick-relief medications:**-Treat symptoms of exacerbations

- **Bronchodilators**,,,,,β**-adrenergic agonists**,,, **(e.g., albuterol, salbutamol[Ventolin])**

- Acts in minutes, lasts 4 to 8 hours ,Short-term relief of bronchoconstriction ,Treatment of choice in acute exacerbations

**Bronchodilators:** 1.Useful in preventing bronchospasm precipitated by exercise and other stimuli

2.Overuse may cause rebound bronchospasm,,,,3.Too frequent use indicates poor asthma control and may mask severity.**Bronchodilators (longer acting)**: 8 – 12 or 24 hr; useful for nocturnal asthma ,,,Avoid contact with tongue to decrease side effects,,,Can be used in combination therapy with inhaled corticosteroid

- **Anti-inflammatory drugs: Corticosteroids** (e.g., beclomethasone, budesonide):Suppress inflammatory response

Inhaled form is used in long-term control. Systemic form to control exacerbations and manage persistent asthma Inhibit release of mediators from macrophages and eosinophils

**Mast cell stabilizers** (e.g., cromolyn, nedocromil):Inhibit release of histamine Inhibit late-phase response Effective in exercise-induced asthma when used 10 to 20 minutes before exercise **Leukotriene modifiers (e.g. Singulair)**Leukotriene – potent bronchco-constrictors and may cause airway edema and inflammation, Have broncho-dilator and anti-inflammatory effects

## Definition of Rhinitis

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Rhinitis is inflammation of the lining of the nasal cavity. As the lining of the nasal cavity and the para nasal sinuses is continuous, inflammatory process tend to involve both areas to a greater or lesser extent.

**Classification of Rhinitis**

- Allergy: -Seasonal ,Perennial, food related ,drug induced
- Infectious :-Acute ,-Chronic

**Allergic rhinitis**

- Its an IgE mediated hypersensitivity response to allergen lead to rhinitis ,associated allergic conjunctivitis and asthma may occur.
- The disease is common ,prevalence depend on age ,gender, geographical distribution

**Allergic rhinitis classification:**

• 1. Intermittent (Seasonal) : Tree pollen,Grass pollen,Weed pollen,Ragweed .pollen,Out door mold spores

2. Persistent (Perennial): House Dust mites,Pet dander,In door mold spores, Cockroach

3. Mild

4. Moderate - severe

**Clinical features:**

- Sneezing , may be in paroxysm.
- Rhinorrhea
- nasal obstruction and loss of smell
- Itchiness of nose ,eye,palate
- Tearing ,itching ,redness of eyes
- Burning sensation in the throat.
- symptom related to asthma (cough,shortness of breath, wheeze)

**treatment:**

- Avoidance of allergins .
- Drugs . ***Oral or local non-sedative $H_1$ blocker, Intra-nasal steroid, Local cromolyn***

***Intra-nasal decongestant (<10 days) or oral decongestant***

- ***Nasal Corticosteroids***

• *Beclomethasone dipropionate: Beconase 42mcg/spray- 2 sprays each nostril daily*

• *Budesonide:Rhinocort ,,Ciclesonide: Omnaris ,,Flunisolide : Nasalide, Fluticasone : Veramyst: ,,Mometasone furoate,,Triamcinolone acetonide:*

- .Immunotherapy (desensitization) .
- Surgery .

# Antitussives:

## Actions and Uses

- Some antitussives depress cough center located in medulla and are called centrally acting drugs
- Some antitussives are peripherally acting drugs, which act by anesthetizing stretch receptors in the respiratory passages, thereby decreasing coughing
- Antitussives are used to relieve nonproductive cough

**Antitussives: Adverse Reactions:**

- Central nervous system reactions:Sedation; dizziness; lightheadedness
- Gastrointestinal reactions: Nausea; vomiting; constipation
- The only monograph topical antitussives are **camphor & menthol**

**Drugs:**

**Codein** For treatment and management of pain (systemic). It is also used as an antidiarrheal and as a cough suppressant.

**Dextromethorphan** For treatment and relief of dry cough.

**Hydrocodone** For relief of moderate to moderately severe pain. Also used for the symptomatic relief of nonproductive cough, alone or in combination with other antitussives or expectorants.

**Methadone** For the treatment of dry cough, drug withdrawal syndrome, opioid type drug dependence, and pain.

**Butorphanol** For the relief of moderate to severe pain.

**Benzonatate** For the symptomatic relief of cough. Has also been applied locally in the oral cavity in adults by releasing the drug from the liquid-filled capsules to provide oropharyngeal anesthesia for conscious intubation.

**Ethylmorphine** Ethylmorphine is an analgesic used for pain relief.

**Pipazethate** For the treatment of cough.

# Mucolytics and Expectorants: Actions :

Drug with mucolytic activity appears to reduce viscosity of respiratory secretions by direct action on mucus

Expectorants increase production of respiratory secretions, which in turn appears to decrease viscosity of mucus, helps to raise secretions from respiratory passage

**Uses:**

- Acute bronchopulmonary disease
- Pulmonary complications of cystic fibrosis
- Pulmonary complications associated with surgery
- Post-traumatic chest conditions
- Atelectasis due to mucus obstruction
- Acetaminophen overdosage

**Expectorant drugs**

Many mucokinetic drugs are available, including Sodium citrate or Potassium citrate, Potassium iodide, Guaifenesin, Tolu balsam, Vasaka, Ammonium chloride

**Mucolytics**

Many mucolytic drugs are available, including acetylcysteine, ambroxol, bromhexine, carbocisteine, erdosteine, mecysteine, and dornase alfa

# Common cold

- It is a self-limiting viral infection of the upper respiratory tract
- Common cold cannot be prevented or cured
- **Antibiotics: ineffective**
- **The main and common causative agents:**
- **- rhinoviruses** → 50% of cases
- **- coronaviruses**, **respiratory syncytial virus** (RSV), **influenza virus** (types A,B,C); **echovirus; coxackie virus, adenovirus, parainfluenza virus**

Pathophysiology:

- Rhinoviruses bind to intercellular adhesion molecule on epithelial cells in the nose and nasopharynx
- The virus replicates and infection spreads
- Infected cells release chemokine "distress signal" and cytokines that activate inflammatory mediators & neurogenic reflexes

**Signs & Symptoms:**

**Symptoms appear in sequence 1-3 days after infection:**

- The 1st symptom to appear: **sore throat**
- 2nd : nasal symptoms (stuffiness (congestion), rhinorrhea, postnasal drip)
- Watering eyes, sneezing
- Then, cough (infrequent) by day 4-5

**Less common:** headache, chills, pyrexia (<37.8), sinus pain & myalgia

**Management of common cold:**

- The effective means of prevention: frequent hand cleansing with soap or soap substitutes
- Cold symptoms are usually self-limiting and resolve within 1-2 weeks whether treated or not
- General therapeutic measures: rest & maintain fluid intake

# Test:

# Q\mention the effect of histamine?

# Q\what is the phases of asthma and how treated?

## DRUGS USED IN THE TREATMENT OF PEPTIC ULCER 21

Acid peptic diseases include gastro esophageal reflux, peptic ulcer (gastric & duodenal) and stress related mucosal injury.

99% of peptic ulcer is caused by the infection with Helicobacter pylori or by non-steroidal anti inflammatory drugs.

**Drugs is divided in to two groups.**

Agents that reduce intragastric acidity.

Agents that promote mucosal defense

**1.** Antacids: MECHANISM OF ACTION :
Antacids are the weak bases react with gastric hydrochloric acid to form salt and water and reduce intragastric acidity. It also promotes mucosal defense mechanism of stimulation of mucosal PGS production.

- **Sodium bicarbonate (baking soda) :**
- Formulations containing magnesium hydroxide or aluminium hydroxide

**2.** H2 RECEPTOR ANTAGONISTS:Cimetidine,,Ranitidine
**3.** PROTON PUMP INHIBITORSPPI•
Irreversibly inhibits H+/K+ ATPase. **Omeprazole** :Nonenteric coated powder.

- **H.PYLORI TREATMENT REGIMENS:**

2 antibiotics + PPI for 10 –14 days
Triple therapy
PPI –20mg BD
Clarithromycin 500 mg –bd +Amoxicillin1g –bd
If the Patient is allergic to penicillin, metronidazole 500mg bd. After completion of triple therapy, PPI should be continued for 4-6 weeks to ensure complete ulcer healing.
For patients with resistant infections quadruple therapy is recommended.
PPI + Amoxicillin + Clarithromycin + Bismuth sub citrate -2 tablets 4 times daily.

# Laxatives

## Definition :

Laxatives : Latin : laxare : to loosen

- Drugs which loose the bowels (intestines) or
- Drugs which producing, increasing and hastening intestinal evacuation

**Uses :**

- In constipation
- In evacuation of bowls , prior to diagnostic procedure or surgery.

**Classification :**

Depending upon the intensities of drug effect they are classified as :

- **Laxatives :** Eliminate the soft formed stools also known as Mild Purgatives.
  e.g.
  Karaya gum ,, Senna , Carboxy methyl cellulose , Isapgol,, Liquid-paraffin

- **Purgatives** : fluid evacuation
  e.g. Castor oil , Aloe , Cascara sagrada ,, Rhubarb

- **Drastics** : Act intensely by irritating the mucous membrane of the intestine.
  e.g. Jalap , Colocynth ,Podophyllum

## Emesis (vomiting)

- **Act of forceful expulsion of gastric contents through the mouth**

Emetic Drugs - Uses

- Acute cases of poisoning (except in corrosives substances poisoning or if pt is not fully conscious)
- Alcoholic intoxication
- Removal of foreign bodies from the oesophagus

## Emetic Drugs

- **Centrally acting**: Apomorphine
- directly stimulate the CTZ or VC
- **Reflexly acting**: Ipecacuanha
- stimulate the VC by irritating gastric & duodenal mucosa which stimulate afferent fibres of vagus nerve
- **Locally acting:** Alum, Sodium Chloride (Conc Solution)
- **Other Drugs as Adverse Effect:** Morphine, Digitalis, Emetine, Aspirin, Quinine & Anticancer drugs

## Antiemetics(Classification)

- **Anticholinergics**: Hyoscine, Dicyclomine
- **H1 antihistaminics**:
  – Promethazine,
  – Diphenhydramine,
  – **Doxylamine,**
  – Dimenhydrinate, Cyclizine, Meclizine, Cinnarizine
- **Neuroleptics:** Chlorpromazine, Prochlorperazine, Haloperidol
- **Prokinetic agents: Metoclopramide, Domperidone**, Cisapride, Mosapride,Renzapride
- **5-HT3 antagonists**: **Ondansetron, Granisetron**.Dolasetron. Ramosetron

**Test:**

**Q\mention the effect of ppi?**

**Q\what is triple and quaderaple therapy of H-pylori??**

**References**:
Lippincott Illustrated Reviews: Pharmacology, 7th Edition

**Pharmacology and Nursing Process, by Linda Lane Lilley,ISBN13: 978-0323358286 ,8th**

**Name of subject:Chemotherapeutic Drugs \ Principles of Antimicrobial Therapy**

**Name of Lecturer: Dr.Tayseer Shaker Mahmood**

**Date :**

**Time of subject : 45 minutes**

**Target group: students of nursing department**

**22**

## General objective

**In the end of lecture, the student must be:**

1. understand mechanism of antibiotics therapy
2. Know groups of antibiotics to treat different infection and their

## Specific objective

**In the end of lecture, the student must be:**

1. Define antibiotics
2. enumerate principles of selection of antibiotic
3. enumerate spectrum of antibiotic
4. know the advantage of combination therapy with antibiotics
5. explain the complication of abuse of antibiotics
6. know the differences mechanisms of action of antibiotics
8. define B-lactam antibiotics

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## Chemotherapeutic Drugs

### Principles of Antimicrobial Therapy

Antimicrobial drugs are effective in the treatment of infections because of their **selective toxicity**; that is, they can injure or kill an invading microorganism without harming the cells of the host.

**SELECTION OF ANTIMICROBIAL AGENTS**

Selection of the most appropriate antimicrobial agent requires knowing:

1) the organism's identity, 2) the organism's susceptibility to a particular agent, 3) the site of the infection, 4) patient factors, 5) the safety of the agent, and 6) the cost of therapy.

**Bacteriostatic versus bactericidal drugs:**

- • **Bacteriostatic drugs** arrest the growth and replication of bacteria, thus limiting the spread of infection until the immune system attacks, immobilizes, and eliminates the pathogen. If the drug is removed before the immune system has scavenged the organisms, enough viable organisms may remain to begin a second cycle of infection.

- • **Bactericidal drugs** kill bacteria at drug serum levels achievable in the patient. Because of their more aggressive antimicrobial action, bactericidal agents are often the drugs of choice in seriously ill and immunocompromised patients.

**Minimum inhibitory concentration** (MIC) is the lowest antimicrobial concentration that prevents visible growth of an organism after 24 hours of incubation.

**Minimum bactericidal concentration** (MBC): is the lowest concentration of antimicrobial agent that results in a 99.9% decline in colony count after overnight broth dilution incubations.

**DETERMINANTS OF RATIONAL DOSING**

Rational dosing of antimicrobial agents is based on their pharmacodynamics (the relationship of drug concentrations to antimicrobial effects) and pharmacokinetic properties (the absorption, distribution, metabolism, and elimination of the drug). Three important properties that have a significant influence on the frequency of dosing are concentration dependent killing, time-dependent killing, and post-antibiotic effect (PAE).

**A. Concentration-dependent killing** :Certain antimicrobial agents, including aminoglycosides and *daptomycin*, show a significant increase in the rate of bacterial killing as the concentration of antibiotic increases. So, giving drugs that exhibit this concentration-dependent killing by a once-a-day bolus infusion achieves high peak levels, favoring rapid killing of the infecting pathogen.

**B. Time-dependent (concentration-independent) killing:**In contrast, β-lactams, glycopeptides, macrolides, *clindamycin*, and *linezolid* do not exhibit concentration-dependent killing. The clinical efficacy of these antimicrobials is best predicted by the percentage of time that blood concentrations of a drug remain above the MIC. This effect is sometimes called concentration-independent or time-dependent killing.

**C. Postantibiotic effect** :The PAE is a persistent suppression of microbial growth that occurs after levels of antibiotic have fallen below the MIC. Antimicrobial drugs exhibiting a long PAE (for example, aminoglycosides and fluoroquinolones) often require only one dose per day, particularly against gram-negative bacteria.

**CHEMOTHERAPEUTIC SPECTRA**

**A. Narrow-spectrum antibiotics**

Chemotherapeutic agents acting only on a single or a limited group of microorganisms are said to have a narrow spectrum. For example, *isoniazid* is active only against Mycobacterium tuberculosis.

**B. Extended-spectrum antibiotics**

Extended spectrum is the term applied to antibiotics that are modified to be effective against gram-positive organisms and also against a significant number of gram-negative bacteria. For example, *ampicillin* is considered to have an extended spectrum because it acts against gram-positive and some gram-negative bacteria.

**C. Broad-spectrum antibiotics**

Drugs such as *tetracycline*, fluoroquinolones and carbapenems affect a wide variety of microbial species and are referred to as broad-spectrum antibiotics. Administration of broad-spectrum antibiotics can drastically alter the nature of the normal bacterial flora and precipitate a superinfection due to organisms such as Clostridium difficile. .

**xCOMBINATIONS OF ANTIMICROBIAL DRUGS**

This strategy reduces the possibility of superinfections, decreases the emergence of resistant organisms, and minimizes toxicity.

**A. Advantages of drug combinations**

Certain combinations of antibiotics, such as β-lactams and aminoglycosides, show synergism; that is, the combination is more effective than either of the drugs used separately.

**B. Disadvantages of drug combinations**

coadministration of an agent that causes bacteriostasis plus a second agent that is bactericidal may result in the first drug interfering with the action of the second. For example, bacteriostatic tetracycline drugs may interfere with the bactericidal effects of penicillins and cephalosporins. Another concern is the risk of selection pressure and the development of antibiotic resistance by giving unnecessary combination therapy.

**DRUG RESISTANCE**

- **1- Genetic alterations leading to drug resistance:** Acquired antibiotic resistance requires the temporary or permanent alteration of bacterial genetic information. Resistance develops due to the ability of DNA to undergo spontaneous mutation or to move from one organism to another.

- **2- Altered expression of proteins in drug-resistant organisms**: can be represented by different mechanisms, which are:*1) Modification of target sites 2) Decreased accumulation and 3) Enzymatic inactivation.*

**PROPHYLACTIC USE OF ANTIBIOTICS**

Certain clinical situations, such as dental procedures and surgeries, require the use of antibiotics for the prevention rather than for the treatment of infections. In dental practice, a prophylactic treatment can be prescribed for patients with heart problems such as implanted prosthetic heart valves and rheumatic heart disease.

**COMPLICATIONS OF ANTIBIOTIC THERAPY**

**A. Hypersensitivity**

**B. Direct toxicity**

**C. Superinfections**

**SITES OF ANTIMICROBIAL ACTIONS**

Antimicrobial drugs can be classified in a number of ways:

1) by their chemical structure (for example, β-lactams or aminoglycosides)

2) by their mechanism of action (for example, cell wall synthesis inhibitors

3) by their activity against particular types of organisms (for example, bacteria, fungi, or viruses).

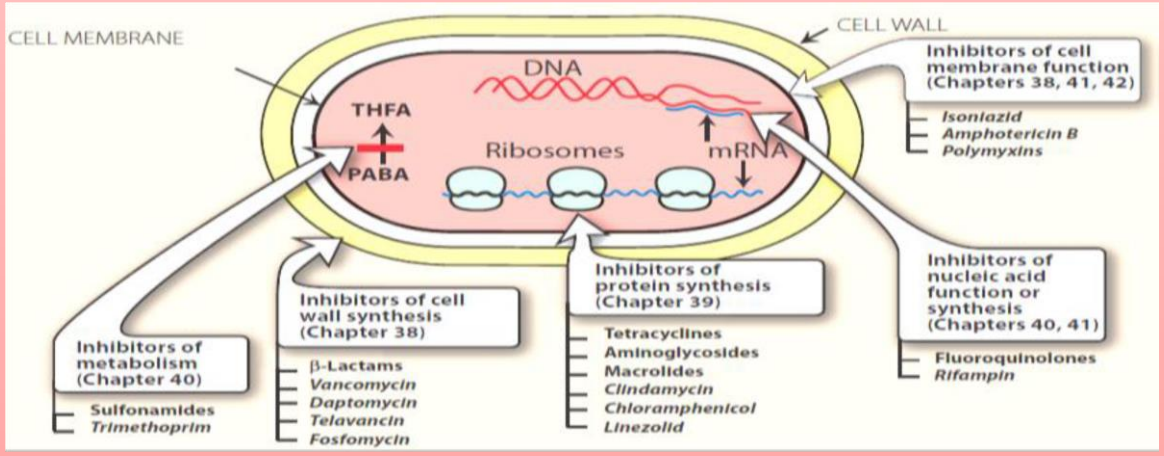



**Figure 1: Classification of some antimicrobial agents by their sites of action. (THFA = tetrahydrofolic acid; PABA = *p*-aminobenzoic acid.)**

**Cell Wall Inhibitors** (part 1) 23

Some antimicrobial drugs selectively interfere with synthesis of the bacterial cell wall—a structure that mammalian cells do not possess. The cell wall is composed of a polymer called peptidoglycan that consists of glycan units joined to each other by peptide cross-links.

The most important members of this group of drugs are the β-lactam antibiotics (named after the β-lactam ring that is essential to their activity), vancomycin, and daptomycin.

**PENICILLINS**

The penicillins share features of chemistry, mechanism of action, pharmacology, and immunologic characteristics with cephalosporins, monobactams, carbapenems, and β- lactamase inhibitors. All are β-lactam compounds, so named because of their four membered lactam ring .

**Mechanism of Action**

The penicillins interfere with the last step of bacterial cell wall synthesis (transpeptidation or cross-linkage), resulting in exposure of the osmotically less stable membrane. Cell lysis can then occur, either through osmotic pressure or through the activation of autolysins. These drugs are bactericidal and work in a time-dependent fashion. **Penicillins are only effective against rapidly growing organisms that synthesize a peptidoglycan cell wall. Consequently, they are inactive against organisms devoid of this structure, such as mycobacteria, protozoa, fungi, and viruses.**

**Resistance**

Resistance to penicillins and other β-lactams is due to one of four general mechanisms: (1) inactivation of antibiotic by β-lactamase, (2) modification of target PBPs (penicillin-binding protein), (3) impaired penetration of drug to target PBPs, and (4) antibiotic efflux.

**Clinical Uses**

- • Except for amoxicillin, oral penicillins should be given 1–2 hours before or after a meal; they should not be given with food to minimize binding to food proteins and acid inactivation.
- • Amoxicillin may be given without regard to meals.
- • Blood levels of all penicillins can be raised by simultaneous administration of probenecid, 0.5 g (10 mg/kg in children) every 6 hours orally, which impairs renal tubular secretion of weak acids such as β-lactam compounds.
- • Penicillins, like all antibacterial antibiotics, should never be used for viral infections and should be prescribed only when there is reasonable suspicion of, or documented infection with, susceptible organisms.

**B. Extended-Spectrum Penicillins (Amoxicillin, Aminopenicillins, Carboxypenicillins, and Ureidopenicillins)**

• These drugs have greater activity than penicillin against Gram-negative bacteria because of their enhanced ability to penetrate the Gram-negative outer membrane.

• Ampicillin and amoxicillin are available in combination with one of several β-lactamase inhibitors: **clavulanic acid** and **sulbactam**. The addition of a β-lactamase inhibitor extends the activity of these penicillins to include β-lactamase-producing strains of *S aureus* as well as some β -lactamase-producing Gram-negative bacteria.

**Cell wall inhibitors (part 2)**

**Cephalosporins (CPs)**

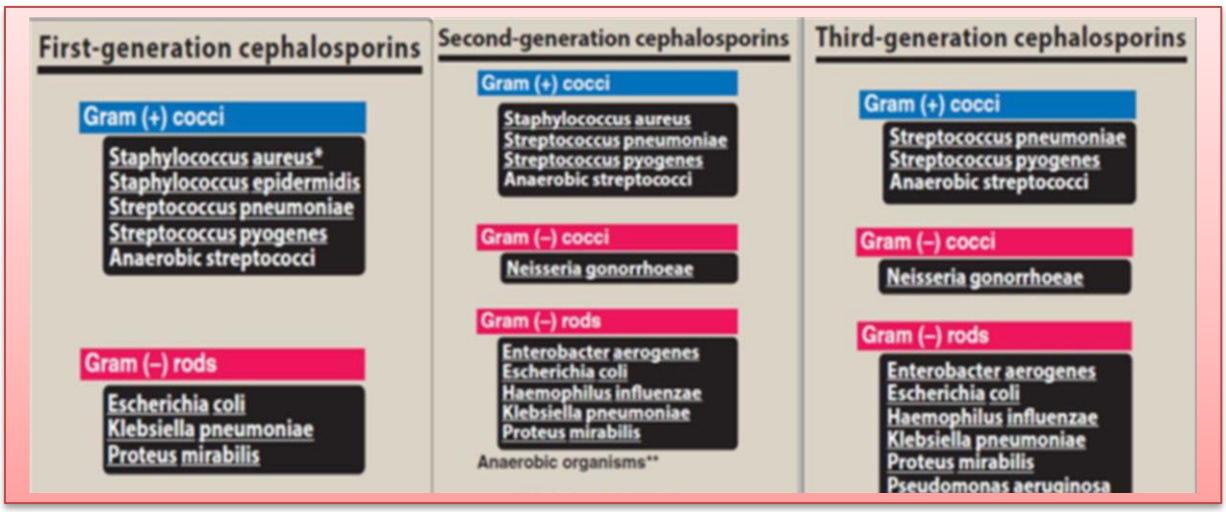
The CPs are β-lactam antibiotics that are closely related both structurally and functionally to the penicillins. CPs have the same mode of action as penicillins, and they are affected by the same resistance mechanisms. However, they tend to be more resistant than the penicillins to certain β-lactamases.

**Antibacterial spectrum**

CPs have been classified as first, second, third, fourth, and advanced generation, based largely on their bacterial susceptibility patterns and resistance to β-lactamases.

## Fourth-generation cephalosporins

Antibacterial coverage comparable to that of the third-generation class; however, demonstrate greater stability against lactamases.




**OTHER β-LACTAM ANTIBIOTICS**

**A. Carbapenems**

- • Carbapenems are synthetic β-lactam antibiotics that differ in structure from the penicillins. Imipenem, meropenem, doripenem, and ertapenem are the drugs of this group currently available.

**Antibacterial spectrum:**

- • Imipenem resists hydrolysis by most β-lactamases, but not the metallo-β-lactamases.
- • This drug plays a role in empiric therapy because it is active against β-lactamase–producing gram-positive and gram-negative organisms, anaerobes, and P. aeruginosa (although other pseudomonal strains are resistant and resistant strains of P. aeruginosa have been reported to arise during therapy).

## β-LACTAMASE INHIBITORS

Hydrolysis of the β-lactam ring, either by enzymatic cleavage with a β-lactamase or by acid, destroys the antimicrobial activity of a β-lactam antibiotic. β-Lactamase inhibitors, such as *clavulanic acid, sulbactam*, and *tazobactam*, contain a β-lactam ring but, by themselves, do not have significant antibacterial activity or cause any significant adverse effects. Instead, they bind to and inactivate β-lactamases, thereby protecting the antibiotics that are normally substrates for these enzymes. The β-lactamase inhibitors are therefore formulated in combination with β- lactamase–sensitive antibiotics.

## PROTEIN SYNTHESIS INHIBITORS 24

## TETRACYCLINES (Tets)

### Mechanism of action

The drugs bind reversibly to the 30S subunit of the bacterial ribosome. This action prevents binding of tRNA to the mRNA–ribosome complex, thereby inhibiting bacterial protein synthesis.

### Antibacterial spectrum

The Tets are bacteriostatic antibiotics effective against a wide variety of organisms, including gram-positive and gram-negative bacteria, protozoa, spirochetes, mycobacteria, and atypical species. They are commonly used in the treatment of acne and Chlamydia infections (doxycycline).

### Resistance

The most commonly encountered naturally occurring resistance to Tets is **an efflux pump** that expels drug out of the cell, thus preventing intracellular accumulation. Other mechanisms of bacterial resistance to Tets include **enzymatic inactivation** of the drug and production of bacterial proteins that prevent Tets from binding to the ribosome. Resistance to one Tet does not confer universal resistance to all Tets.

### Adverse effects

- • **Gastric discomfort:** Epigastric distress commonly results from irritation of the gastric mucosa and is often responsible for noncompliance with Tets. Esophagitis may be minimized through coadministration with food (other than dairy products) or [Note: Tet should be taken on an empty stomach.]
- • **Effects on calcified tissues:** Deposition in the bone and primary dentition occurs during the calcification process in growing children. This may cause discoloration and hypoplasia of teeth and a temporary stunting of growth. The use of Tets is limited in pediatrics.
- • **Phototoxicity:** Severe sunburn may occur in patients receiving a Tet who are exposed to sun or ultraviolet rays.
- more frequently with Tet. Patients should be advised to wear adequate sun protection.
- • Dizziness, vertigo, and tinnitus may occur particularly with minocycline, which concentrates in the endolymph of the ear and affects function.
- • **Contraindications:** The Tets should not be used in pregnant or breast-feeding women or in children less than 8 years of age.
- **Note:** Tigecycline is a protein synthesis inhibitor, which is indicated for the treatment of complicated skin and soft tissue infections, as well as complicated intra-abdominal infections. Its adverse effects are similar to those of the Tets and include photosensitivity, discoloration of permanent teeth when used during tooth development, and fetal harm when administered in pregnancy. Acute pancreatitis, including fatality, has been reported with therapy.

**Aminoglycosides (AGs)**

AGs are used for the treatment of serious infections due to aerobic gram-negative bacilli. However, their clinical utility is limited by serious toxicities. AGs are derived from either Streptomyces sp. (have -mycin suffixes) or Micromonospora sp. (end in -micin).

**Mechanism of action**

AGs diffuse through porin channels in the outer membrane of susceptible organisms. These organisms also have an oxygen-dependent system that transports the drug across the cytoplasmic membrane resulting in protein synthesis disruption.

Antibiotics that disrupt protein synthesis are generally bacteriostatic; however, AGs are unique in that they are bactericidal. The bactericidal effect of AGs is **concentration dependent**; that is, efficacy is dependent on the maximum concentration (Cmax) of drug above the minimum inhibitory concentration (MIC) of the organism.

## Adverse effects

Therapeutic drug monitoring of **gentamicin,** t**obramycin**, and **amikacin** plasma levels The elderly are particularly susceptible to **nephrotoxicity and ototoxicity**

**MACROLIDES (MCDs)**

- • **Erythromycin** was the first of these drugs to find clinical application, both as a drug of first choice and as an alternative to penicillin in individuals with an allergy to β-lactam antibiotics.
- • **Clarithromycin** (a methylated form of erythromycin) and azithromycin have some features in common with, and others that improve upon, erythromycin.
- • **Telithromycin**, a semisynthetic derivative of erythromycin

**Mechanism of action**

The macrolides bind **irreversibly** to a site on the 50S subunit of the bacterial ribosome, thus inhibiting translocation steps of protein synthesis. They may also interfere with other steps, such as transpeptidation. Generally considered to be **bacteriostatic,** they **may be bactericidal at higher doses.** Their binding site is either identical to or in close proximity to that for clindamycin and chloramphenicol.

**Contraindications:** Patients with **hepatic dysfunction** should be treated cautiously with *erythromycin, telithromycin,* or *azithromycin,* because these **drugs accumulate in the liver**

**Drug interactions:** *Erythromycin, telithromycin,* and *clarithromycin* inhibit the hepatic metabolism of a number of drugs, which can lead to toxic accumulation of these compounds. An interaction with *digoxin* may occur. In this case, the antibiotic eliminates a species of intestinal flora that ordinarily inactivates *digoxin*, thus leading to greater reabsorption of the drug from the enterohepatic circulation.

**CHLORAMPHENICOL (CHL.)**

***The use of* CHL*, a broad-spectrum antibiotic, is restricted to life-threatening infections for which no alternatives exist.***

**Mechanism of action :** CHL binds reversibly to the bacterial 50S ribosomal subunit and inhibits protein synthesis at the peptidyl transferase reaction.

**Antibacterial spectrum**

*CHL* is active against many types of microorganisms including chlamydiae, rickettsiae, spirochetes, and anaerobes. The drug is primarily bacteriostatic, but depending on the dose and organism, it may be bactericidal.

**CLINDAMYCIN**

✓ Clindamycin has a mechanism of action that is the same as that of erythromycin.

✓ Clindamycin is used primarily in the treatment of infections caused by gram-positive organisms, including MRSA and streptococcus, and anaerobic bacteria.

✓ Resistance mechanisms are the same as those for erythromycin, and cross-resistance has been described.

✓ Clostridium difficile is always resistant to clindamycin, and the utility of clindamycin for G -ve anaerobes is decreasing due to increasing resistance.

✓ Clindamycin is available in both IV and oral formulations but use of the oral form is limited by gastrointestinal intolerance. It distributes well into all body fluids including bone but exhibits poor entry into the CSF.

✓ Clindamycin undergoes extensive oxidative metabolism to inactive products and is primarily excreted into the bile. Low urinary elimination limits its clinical utility for urinary tract infections. Accumulation has been reported in patients with either severe renal impairment or hepatic failure.

✓ In addition to skin rashes, the most common adverse effect is diarrhea, which may represent a serious pseudomembranous colitis caused by overgrowth of C. difficile. Oral administration of either metronidazole or vancomycin is usually effective in the treatment of C. difficile.

## Test

**Q\enumerate site of action of antibiotics?**

**Q\what is the side effect of tetracycline?**

**Q\give the most important side effect of gentamycin?**

**Q\what is the difference between extended and broad spectrum of antibiotics?**


**References:**

Lippincott Illustrated Reviews: Pharmacology, 7th Edition

**Pharmacology and Nursing Process, by Linda Lane Lilley,ISBN13: 978-0323358286 ,8th Edition**

**Name of subject : Narcotic and analgesic**

**Name of Lecturer: Dr.Tayseer Shaker Mahmood**

**Date :**

**Time of subject : 45 minutes**

**Target group: students of nursing department**

**25**

**General objective**

**In the end of lecture, the student must be:**

**1. understand uses of Narcotic and analgesic**

**2. Know difference of narcotic and non narcotic drugs**

# Specific objective

## In the end of lecture, the student must be:

## 1. Define narcotic drug

## 2. Classify groups of drugs for narcotic and analgesic

## 4.Explain difference between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# Introduction \ Narcotic and analgesic

**Sources of Pain**

- Nociceptive—free nerve endings that receive painful stimuli
- Neuropathic –damaged nerves

- **Neurotransmitters**—chemicals that exert inhibitory or excitatory activity at post-synaptic nerve cell membranes. Examples include: acetylcholine, norepinehprine, epinephrine, dopamin, and serotonin.
- **Neuromodulators**—endogenous opiates. Hormones in brain. Alpha endorphins, beta endorphins and enkephalins. Help to relieve pain.

## Opioid receptors—

binding sites not only for endogenous opiates but also for opioid analgesics to relieve pain. Several types of receptors: Mu, Kappa, Delta, Epsilon and Sigma.

Mu Receptors

- Location: CNS incl. brainstem, limbic system, dorsal horn of spinal cord
- Morphine sulfate and morphine sulfate agonists bind to Mu receptors

## Narcotic Analgesics

- Relieve moderate to severe pain by inhibiting release of Substance P in central and peripheral nerves; reducing the perception of pain sensation in brain, producting sedation and decreasing emotional upsets associated with pain

Can be given orally, IM, sub q, IV or even transdermally

## Narcotics—Mechanisms of Action:

- Bind to opioid receptors in brain and SC and even in periphery

**Indications for UseBefore and during surgery**

- Before and during invasive diagnostic procedures
- During labor and delivery
- Tx acute pulmonary edema
- Treating severe, nonproductive cough

**Dosage:**

☐ Dosages of narcotic analgesics should be reduced for clients receiving other CNS depressants such as other sedative-type drugs, antihistamines or sedating antianxiety medications

**Agonists**

☐ Alfenta (alfentanil)—short duration

☐ Codeine

☐ Sublimaze or Duragesic (Fentanyl)—short duration

☐ Dilaudid (hydromorphone)

☐ Demerol (meperidine)—preferred in urinary and biliary colic, less resp. depression newborns

☐ Morphine

☐ OxyContin

Darvon (propoxyphene)

Ultram (tramadol)

Methadone

**Agonists/Antagonists:**

Have lower abuse potential than pure agonists

☐ Buprenex (buprenorphine)

☐ Nubain (nalbuphine)

☐ Talwin (pentazocine)

☐ Stadol (butohanol)—also in nasal spray

**Antagonists:**

Revex (nalmefene)—longer duration of action than Narcan

Narcan (naloxone)

ReVia (naltrexone)-used in maintenance of opiate-free states in opiate addicts

# Analgesic,Antipyretic, and Anti-inflammatory Drugs:26

- **Mechanism of action**—inactivate cyclo-oxygenases, enzymes required for the production of prostaglandins
- **ASA and traditional NSAIDs inhibit both COX 1 and COX 2**
- **COX 1** is present in all tissues esp. GI, kidneys, endothelial cells and in platelets
- Found in brain, bone, kidneys, GI tract, and the female reproductive system
- Overall, prostaglandins produced by COX 2 are associated with pain and inflammation
- **Prostaglandins important in:**

Protection of kidneys and stomach

Regulate vascular tone and platelets in CV system

**COX2:**

- Found in brain, bone, kidneys, GI tract, and the female reproductive system

Overall, prostaglandins produced by COX 2 are associated with pain and inflammation

**Indications for Use:**

- Treat mild to moderate pain or inflammation
- Musculoskeletal disorders; HA; dysmenorrhea, minor trauma and surgery
- Low dose ASA for risk of MI or stroke
- Celebrex is indicated for familial polyposis

**Aspirin (ASA):**

- Home remedy for headaches, colds, influenza and other respiratory infections
- For fever
- For inflammation
- ASA and COX 2 are ok, COX 2 have little effect on platelet function
- Poisoning can occur with large doses. Saturate the metabolic pathway, slow elimination and cause drug accumulation

# NSAIDS

- **Propionic acid** derivatives such as ibuprofen, ketoprofen (Orudis), naproxen and fenoprofen (Nalfon)
- **Acetic acid** derivatives include indomethacin (Indocin), sulindac (Clinoril) and tolmetin (Tolectin)---these drugs have more severe adverse reactions than the proprionic acid derivatives
- IV **indomethacin** is approved for the tx of patent ductus arteriosus in premature infants.
- Remember: patent ductus is a communication between the pulmonary artery and the aorta
- Toradol (ketoralac) is used only for pain. Is the only NSAID that can be given by injection. Use limited to 5 days as can cause bleeding.
- **Oxicam drugs** include Mobic (meloxacam) and Feldene (piroxicam)
- Celebrex (celecoxib)
- Affect bleeding only while drug is still in the system

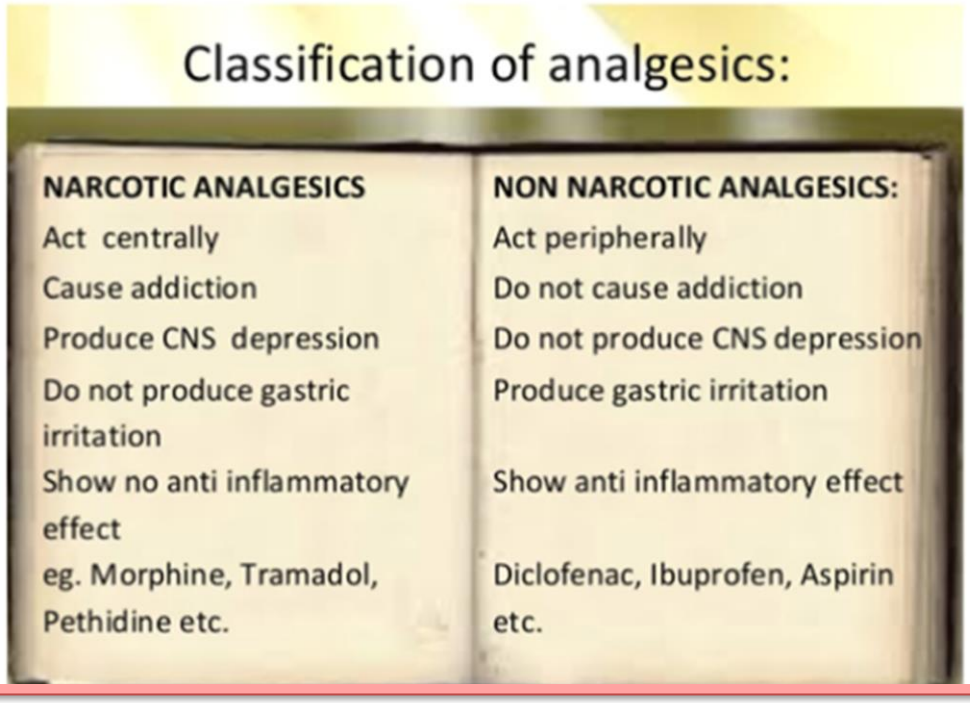
**Effects of Nonsteroidals on Other Drugs:**

- Decrease effects of ACEI, beta blockers and diuretics
- Affect sodium and water retention
- Inhibit renal prostaglandin synthesis

**Acetaminophen:**

- Equal in effectiveness to ASA in analgesic and antipyretic effects
- Lacks anti-inflammatory actions
- Ethanol induces drug-metabolizing enzymes in liver. Resulting rapid metabolism of acetaminophen produces enough toxic metabolite to exceed glutathione. Need glutathione to inactivate toxic metabolites.
- Toxicity occurs with 20g or more.
- Treatment—gastric lavage, charcoal, antidote is Mucomyst (acetylcysteine). Provides cysteine, a precursor to glutethione.

## Classification of analgesics:



| NARCOTIC ANALGESICS | NON NARCOTIC ANALGESICS: |
|---|---|
| Act centrally | Act peripherally |
| Cause addiction | Do not cause addiction |
| Produce CNS depression | Do not produce CNS depression |
| Do not produce gastric irritation | Produce gastric irritation |
| Show no anti inflammatory effect | Show anti inflammatory effect |
| eg. Morphine, Tramadol, Pethidine etc. | Diclofenac, Ibuprofen, Aspirin etc. |

## Test:

**Q\what is the mechanism of action of narcotic drugs?**

**Q\ is acetoaminophen have sedation effect? And why?**

## References


Lippincott Illustrated Reviews: Pharmacology, 7th Edition

**Pharmacology and Nursing Process, by Linda Lane Lilley,ISBN13: 978-0323358286 ,8th Edition**

**Name of subject :anti-viral, antiparasite,antihelminth drugs**

**Name of Lecturer: Dr.Tayseer Shaker Mahmood**

**Date :**

**Time of subject : 45 minutes**

**Target group: students of nursin department**

**27**

**General objective**

**In the end of lecture, the student must be:**

**1.know the drugs that treat viral,parasite,worm infections**

# Specific objective

## In the end of lecture, the student must be:

## 1.define anti-viral, antiparasite,antihelminth drugs

## 2. enumerate drugs to treat each infection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## Antiviral drug:مضادات الفايروسات

- drug used to treat the DNA virus,( herpesvirus اسم الفايروس ) : is acyclovir.

**Aciclovir** دواء لعلاج الاصابات الفايروسية is very good against (herpes simplexاسم الفايروس )

(which causes تسبب cold sores القروح الباردة , conjunctivitis التهاب ملتحمة العين ,

mouth ulcersتقرحات الفم , genital infections التهابات تناسلية , encephalitis التهاب الدماغ).

- Drugs used to Treat the Flu ادوية علاج الانفلونزا: **Zanamivir** (Relenza) aerosoland **oseltamivir** orally.فمويا
- Drugs used to Treat Human immunodeficiency virus infection and acquired immune deficiency syndrome (HIV/AID الادوية التي تستعمل لعلاج ) الفايروسات المسببة نقص المناعة

The first drug developed for the treatment of HIV was **AZT**, which is also known as **azidothymidine** and **zidovudine**

## Antifungal drug:مضادات الفطريات

The fungal cell الخلية الفطرية membrane غشاء is made up of متكونة منergosterol , which differs يختلف from the phospholipid plasma membrane of mammalian cellsعن غشاء خلايا الانسان او الحيوان. Thus, drugs الدواء that interfere يتداخل with ergosterol will affect the fungal cell membrane , but not have effects in humans e.g. adverse effects.

**Amphotericin** اسم الدواء is a mixture مزيج of anti-fungal substances derived from the bacteria, *Streptomyces.* مضاد فطريات مشتق من بكتريا

- Amphotericin binds يرتبط مع to ergosterol to create pores يكون فتحات in the fungi cell membrane في الغشاء and this leads to the death of the fungus قتل الفطر
- i.e. is fungicidal.سام للفطريات

## Anthelmintics مضادات الديدان

Drugs used to treat parasitic worm ديدان infections: helminthic infections

Unlike protozoa البدائيات لا تشبه , helminths are large كبيرة and have complex cellular structures تركيبات خلوية معقدة

Drugs: الادوية المستعملة

albendazole (Albenza) ivermectin (Stromectol), mebendazole (Vermox)

praziquantel (Biltricide).,pyrantel (Antiminth),, thiabendazole (Mintezol)

# Test:

## Q\ what is the mechanism of action of acyclovir?

## Q\ why the antibiotics don't used to treat fungal infection?

# References


Lippincott Illustrated Reviews: Pharmacology, 7th Edition

Pharmacology and Nursing Process, by Linda Lane Lilley,ISBN13: 978-0323358286 ,8th Edition

**Name of subject : drugs for CNS disorders**

**Name of Lecturer: Dr.Tayseer Shaker Mahmood**

**Date :**

**Time of subject : 45 minutes**

**Target group: students of nursing**

**General objective**

**In the end of lecture, the student must be:**

**1. understand different disorders in CNS**

**2. Know difference drugs used to treat mental or sleep disorders**

**Specific objective**

**In the end of lecture, the student must be:**

**1.define Parkinson's disease**

**2.enumerate causes of Parkinson's disease**

**3.explain mechanism of action of drugs for Parkinson's disease**

**4. Define Alzheimer's disease**

**5. enumerate causes of Alzheimer's disease**

**6.Explain mechanism of action of drugs for Alzheimer's disease**

**7. define epilepsy**

**8. explain causes and drugs to treat epilepsy**

**9.enumerate drugs used to treat sleep disorders**

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# Drugs for CNS disorders

## Drugs for Parkinson's Disease:

- Dopamine is a neurotransmitter that helps send signals in the brain.
- Neurotransmitters are chemicals made by nerve cells called neurons.
- They're used to communicate messages across different parts of the brain and between the brain and the rest of the body.

Dopamine is involved mainly in controlling movement. Dopamine plays a role in the brain's reward system, helping to reinforce certain behaviors that result in reward. Dopamine also helps to aid the flow of information to the brain regions responsible for thought and emotion. Dopamine creates feelings of pleasure.

### What Is Parkinson's Disease?

Parkinson's disease mostly affects older people but can also occur in younger adults. The **symptoms** are the result of the gradual degeneration of nerve cells in the part of the brain that controls body movement. People with Parkinson's don't have enough dopamine in their brains to control their movements.

### What Causes Parkinson's?

It's not clear what causes the abnormal nerve function linked to Parkinson's disease. **Heredity**, **toxins** in the environment, and **oxidative stress** may play some role.

**Drugs:**

1. **Levodopa** (L-dopa): is a drug used to treat Parkinson's disease. Symptoms of Parkinson's disease start to show up when dopamine-producing cells in the brain die. Levodopa, a precursor chemical to dopamine, helps to boost dopamine levels in the brain. Once **levodopa** reaches the brain, it transforms into **dopamine**.

2. **Dopamine Agonists** :are a class of drugs that bind and activate dopamine receptors in the brain. They mimic the action of naturally-occurring dopamine in the brain, causing the neurons to react as they would to dopamine.

- Dopamine agonists are used to **treat low dopamine conditions**, including **Parkinson's disease**.
- Dopamine agonists also are sometimes used to treat depression .

Common dopamine agonist drugs include:

- ✓ Mirapex (ramipexole)
- ✓ Neupro (rotigotine)
- ✓ Requip (ropinirole)

-----**Side effects** associated with dopamine agonists include low blood pressure, dizziness when standing up, hallucinations, and impulse control disorders, such as pathological gambling, compulsive eating, and hypersexuality.

3. **Sinemet** is a mix of **levodopa and another drug called carbidopa. Carbidopa** makes the levodopa work better, so you can take less of it. That prevents many common side effects of levodopa, such as nausea, vomiting, and irregular heartrhythms.

4. **Safinamide (Xadago)** is an add-on medicine that may be prescribed when individuals taking levdopoa and carbidopa have a breakthrough of Parkinson's symptoms that were previously under control. The most common **side effects** are trouble falling or staying asleep.

5. **Dopamine Supplements**:

Dopamine is found in many types of food, but dopamine itself can't cross into the brain from the bloodstream, so eating foods that contain dopamine won't raise dopamine levels in the brain. But dopamine's precursor molecule**, tyrosine**, can cross the blood-brain barrier, according to a review published in November 2015 in the Journal of Psychiatric Research.

**Tyrosine is an amino acid** found naturally in protein-rich foods, such as cheese, nuts, and meat. In fact, the Parkinson's disease drug(Levodopa ) was originally synthesized from one form of tyrosine.

**Epilepsy** :is a common condition that affects the brain and causes frequent **seizures.**

- ✓ Seizures are bursts of electrical activity in the brain that temporarily affect its works. They can cause a wide range of symptoms.
- Epilepsy can start at any age, but usually starts either in childhood or in people over 60. It's often lifelong, but can sometimes get slowly better over time.

**Anti-epileptic drugs (AEDs)**

AEDs are the most commonly used treatment for epilepsy. They help control seizures in about 70% of people.

- AEDs **work by** changing the levels of chemicals in brain. They don't cure epilepsy, but can stop seizures happening.
- AEDs are available in a number of different forms, including tablets, capsules, liquids and syrups. You usually need to take the medicine every day.

**Types of AEDs:**

sodium valproate, carbamazepine,lamotrigine,levetiracetam.

**Alzheimer's** is a type of dementia that causes problems with memory, thinking and behavior. Symptoms usually develop slowly and get worse over time, becoming severe enough to interfere with daily tasks.

- As Alzheimer's progresses, **brain cells die and connections among cells are lost, causing cognitive symptoms to worsen.** While current medications cannot stop the damage in brain cells, they may help stabilize symptoms for a limited time by affecting certain chemicals involved in carrying messages among the brain's nerve cells. Doctors sometimes prescribe both types of medications together.

**Types of Drugs**

two types of medications —

1. **cholinesterase inhibitors (**Aricept, Exelon, Razadyne): Prevent the breakdown of acetylcholine, a chemical messenger important for learning and memory.
2. **memantine** (Namenda) combination of memantine and donepezil (Namzaric) — to treat the cognitive symptoms (memory loss, confusion, and problems with thinking and reasoning) of Alzheimer's disease.

- Can cause **side effects**, including headache, constipation, confusion and dizziness.

# Antidepressants 29

are a class of drugs that **reduce symptoms of depressive disorders** by **correcting chemical imbalances of neurotransmitters in the brain**. Chemical imbalances may be responsible for changes in mood and behavior.

1. **Selective serotonin reuptake inhibitors (SSRIs)**

SSRIs affect a chemical in your brain called serotonin.

- paroxetine
- sertraline.

2. **Serotonin and norepinephrine reuptake inhibitors (SNRIs)**

- duloxetine
- venlafaxine
- desvenlafaxine.

3. **Tricyclic antidepressants**

Tricyclic antidepressants affect three brain chemicals. They are serotonin, norepinephrine, and dop

**drugs:**

- amitriptyline
- clomipramine
- desipramine

4. **Monamine oxidase inhibitors (MAOIs)**

MAOIs affect an enzyme in your brain called monamine.

Types of MAOIs are:

- isocarboxazid
- phenelzine
- selegiline

**Anxiety** causes sleeping problems, and new research suggests sleep deprivation can cause an anxiety disorder.

-----drugs used in the treatment of **insomnia** include:

**1.nonbenzodiazepine receptor agonists,**

**2.benzodiazepine receptor agonists**,

**3. selective melatonin receptor agonist ramelteon**

**4.sedating antidepressants.**

**Benzodiazepines: \ action:**enhance GABA receptor function to promote sleep.

Can be grouped, according to their elimination half-life, into:

- Short/intermediate half-life agents include alprazolam (intermediate), lorazepam (short), oxazepam (short), temazepam (intermediate) and triazolam (ultra-short);
- long half-life agents include diazepam, chlordiazepoxide, flurazepam and nitrazepam.
- short elimination half-life are preferred to minimise daytime sedation, but they can cause rebound symptomatology more often than agents with longer elimination half-life.

**Test**

**Q\ what is the best group of drugs to treat insomnia?**

**Q\ is the epilepsy can cured?**

## References


Lippincott Illustrated Reviews: Pharmacology, 7th Edition

**Pharmacology and Nursing Process, by Linda Lane Lilley,ISBN13: 978-0323358286 ,8th Edition**

**Name of subject : Toxicity**

**Name of Lecturer: Dr.Tayseer Shaker Mahmood**

**Date :**

**Time of subject : 45 minutes**

**Target group: students of nursing department**

**30**

**Specific objective**

**In the end of lecture, the student must be:**

**1. Define Toxicity**

**2. enumerate cases of toxicity and their treatment**

**3.give the most antidotes used for**

| no | subject | page |
|---|---|---|
| 1 | Introduction to toxicity | 120 |
| 2 | treatment | 121 |
| 3 | Poison and antidote | 121 |


# Toxicity

refers to how poisonous or harmful a substance can be. In the context of pharmacology, drug toxicity occurs when a person has accumulated too much of a drug in his bloodstream, leading to adverse effects on the body. Drug toxicity may occur when the dose given is too high or the liver or kidneys are unable to remove the drug from the bloodstream, allowing it to accumulate in the body.

# Treatment

There are several ways in which drug toxicity may be treated. If the toxicity is the result of an acute overdose, then a person may undergo stomach pumping to remove drugs that have not yet been absorbed. Activated charcoal may be given to bind the drugs and prevent them from being absorbed into the blood (instead, it is eliminated from the body through stool). Other medication may also be given as an antidote.

| POISON | ANTIDOTE |
|---|---|
| Paracetamol | N-acetylcysteine - **see guideline** |
| Opioids | Naloxone |
| Benzodiazepines | Flumazenil |
| Sodium channel blockers | $NaHCO_3$ |
| Iron | Desferroxamine |
| Digoxin | Digoxin fab-fragments (Digi-bind) |
| Organophosphates | Pralidoxime, atropine |
| Beta blockers, $Ca^{2+}$ channel blockers | Insulin/dextrose euglycaemic therapy |

Test:

Q\define toxicity

Q\ in what case we use anti emetic drugs as route to save patients from toxicity?

# References:


Lippincott Illustrated Reviews: Pharmacology, 7th Edition

**Pharmacology and Nursing Process, by Linda Lane Lilley,ISBN13: 978-0323358286 ,8th Edition**

**Casarett & Doull's Toxicology: The Basic Science of Poisons, 9th edition.,Curtis D.**


Thank you