



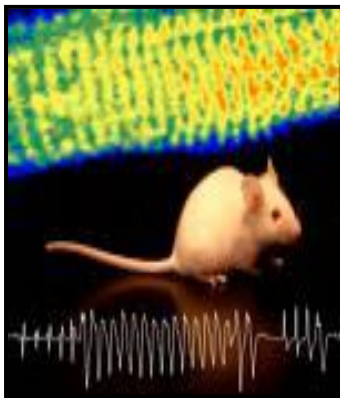
**KING ABDULAZIZ UNIVERSITY**  
**Faculty of Medicine**



## ***MEDICAL PHARMACOLOGY CORE COURSE***

***(PHARM311)***

***Study Guide***



***Phase II, MBBS***  
***1431/1432 H/(2010/2011 G)***

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ابنائى وبناتى الطلاب السلام عليكم وبعد

مرحبا بكم فى قسم علم الادويه

عن أبى سعيد الخدرى رضى الله عنه أن رسول الله صلى الله عليه وسلم قال :

**" ما خلق الله من داءٍ إلا وجعل له شفاءً، علمه من علمه، وجهله من جهله، إلا السام "**

رواه ابن ماجه والسام الموت وفى قوله صلى الله عليه وسلم : " علمه من

علمه ، وجهله من جهله " حث للأطباء المسلمين على البحث

والاستقصاء لاكتشاف أدوية لأمراض لم يعرفها بعد دواء . وقد ربط

النبي صلى الله عليه وسلم الشفاء بموافقة الدواء للداء ، فلكل

دواء مقدار معين يعمل به ، وينبغي ألا يزيد ولا ينقص.

لذلك انتم تدرسون علم الدواء والأمل معقود عليكم لتحقيق

الاستخدام الأمثل للدوية الحالية والمضي قدما فى المستقبل

لاكتشاف وتطوير أدوية جديدة -

إن علم الادويه علم متعدد الروافد ومتعدد العطاء للعلوم

الأخرى وفهم علم الادويه سوف يؤهلكم بقاعدة قوية لفة م

علم العلاج وتحقى قلاستخدام الأمثل للدواء وهذا م

ينتظره المجتمع السعودى منكم وإنها أمانه وانتم بإذن الله

أهلا لحمل الامانه

أنصحكم باستيعاب أهداف كل محاضره وتركيز على العناصر الأساسية

ثم الاستعداد بالنظر فى المراجع المذكورة والانتباه لما يقدمه

المحاضر والحرص على حضور المحاضرات هو مفتاح النجاح

والتفوق- لا تتردوا فى سؤال أعضاء هيئة التدريس عما أشكل

عليكم فهمه

اسأل الله لكم التوفيق وعليكم ببذل الجهد والمثابرة فى مدارسة

المادة وسوف تجدون أعضاء القسم مستعدون لتذليل كل  
الصعاب التي تواجهكم لفهم واستيعاب المنهج الدراسي  
وتوجيهكم للطرق المثلى للمذاكرة والفهم

**د عمر إبراهيم محمد سعادة**

رئيس قسم علم الادويه

## **COURSE DESCRIPTION AND ORGANIZATION**

*The aim of pharmacology core course is to introduce you to the basic principles of pharmacokinetics & pharmacodynamics ; to recognize adverse drug effects and drug-drug interaction and to understand the rational basis of selection optimal drugs & dosing regimens in view of patients profile*

*The course includes and covers the following topics : clinically important drugs affecting autonomic, cardiovascular systems ; chemotherapy; .clinically important drugs for treatment of hyper-lipidemia, coagulation disorders, , cancer chemotherapy, bacterial, fungal and viral infections. It also covers important classes of drugs affecting CNS : Non-Steroidal Anti-inflammatory Drugs, Central Nervous System Stimulants; Antidepressant , Antiparkinsonian Drugs and Local anaesthetics will be also discussed. Practical sessions illustrate prescription writing, pharmacokinetic calculations, drug forms and routes of drug administration, and demonstrate the effect of certain drugs on isolated organs e.g. effect of autonomic drugs on isolated rabbit intestine . Self directed learning will also be implemented to make the students aware of Drug-drug , drug –food or drug-herbal interactions.*

*The course consists of lectures, practical classes and tutorials. (SDL )*

Core Course	Code/No	Course Units			Credit Hours
		Lectures	Practical	Tutorial (SDL)	
Pharmacology	311	25	4	1	

## MAJOR COURSE OBJECTIVES

The course offered to 3<sup>rd</sup> year medical students in pharmacology consists of scheduled lectures, practical and SDL which ensure smooth flow of the scientific material, in a controlled manner, through several pathways to achieve our objectives. There is some suggestion for optimal utilization of these classes by the students.

A. Lectures: The aim of the lecture is not to give all information but to highlight the clinically relevant topics and to explain difficult points: students are advised to

- 1) Understand the course objectives of each lecture and read the topic from the recommended textbook.
- 2) Pay attention during the lecture; write down your notes and, questions.
- 3) Make a summary, utilize self-testing in order to assess your grasping of the subject if is possible to study the lecture in the same day which is highly recommended.

B. Practical class: For optimal utilization of the practical class time it is advisable to:

1. Read your practical worksheet so as to have view of what is expected from you to perform, observe and draw conclusions on your practical work.
2. Use a record of the practical session according to instructions.
3. Use the practical time for discussing difficult theoretical or practical points with the instructor.

C. Tutorials: For optimal benefit of the tutorial, the tutorial will be reserved for open discussion about the subjects listed in the tutorial schedule. The students will be assigned these topics and will be asked to present them and be ready to change the most recent knowledge about these topics and how to defend their thoughts on scientific bases

**STUDY STRATEGIES AND CLASS PARTICIPATION EXPECTATIONS**

**Instructional Methods**

*The main instructional material includes lectures and practical to streamline the applied and clinical aspects of the lectures, and tutorials session to stimulate the students to participate in the teaching/learning activities.*

**Instructional Materials And Resources**

**1. Required Text(s)**

**1) Lippincott's Illustrated Reviews: Pharmacology, 4th Edition**

by [Richard A Harvey](#) ;[Pamela C Champe](#) ;[Richard Finkel](#) ;[Luigi Cubeddu](#) &, [Michelle A Clarke](#) (Editors) . Lippincott Williams & Wilkins **2009**

**2 ) Katzung & Trevor's Pharmacology Examination and Board Review: Eighth Edition** by *Anthony Trevor, Bertram Katzung, and Susan Masters* . MCGraw Hill, **2008**

**2. Essential References**

**1-Rang & Dale's Pharmacology:** by Humphrey P. Rang ; James M. Ritter ; Rod Flower Churchill Livingstone; 6 edition **2007**

**3- Recommended Books and Reference Material (Journals, Reports, etc)**  
(

**1- Integrated Pharmacology:** by [Clive Page](#) ;[Brian Hoffman](#) ;[Michael Curtis](#) ; [Michael Walker](#) (Authors) ; 3rd edition , Mosby & Elsevier; **2006**

**2- Goodman & Gilman's The Pharmacological Basis of Therapeutics (by [Laurence Brunton](#)), [John Lazo](#) (), [Keith Parker](#) ; McGraw-Hill Professional; 11 edition, **2007****

**4. Electronic Materials, Web Sites etc**

<http://www.Cochrane.org>. Mechanism of drug action Chemotherapy

<http://www.cdc.gov/ncidod/srp/drugs/drug-service.html>

<http://www.who.int/tdrbcancer> chemotherapy

<http://www.cancer.gov/> cancer topics

<http://www.nlm.nih.gov/medlineplus/cancer.html> ANS & CNS

<http://www.merck.com>

<http://www.psychiatryonline.com/>

**5- Other learning material such as computer-based programs/CD, professional standards/regulations**

*Core*

*Software for experimental pharmacology & clinical pharmacokinetics*



## ASSESSMENT

### 1. Formative:

*This form of assessment is designed to give you feedback to help you to identify areas for improvement. It includes a mixture of MCQs, short answer-questions (SAQs), and independent learning activities in all subjects. These will be given during tutorial and practical sessions. The Answers are presented and discussed immediately with you after the assessment. The results will be made available to you.*

### 2. Summative

*This type of assessment is used for judgment or decisions to be made about your performance. It serves as:*

- a. Verification of achievement for the student satisfying requirement*
- b. Motivation of the student to maintain or improve performance*
- c. Certification of performance*
- d. Grades*

*In this Course your performance will be assessed according to the following:*

<b>6.. Schedule of Assessment Tasks for Students During the Semester</b>			
<b>DATES</b>			
<i>Asses men t</i>	<i>Assessment task (eg. essay, test, group project, examination etc.)</i>	<i>Week due</i>	<i>Proportion of Final Assessment</i>
<b>1.</b>	<b>Quiz Exam</b>		<b>10 %</b>
<b>2.</b>	<b>Mid-Block exam</b>		<b>30 %</b>
<b>3.</b>	<b>Assignments</b>		<b>10</b>
<b>4.</b>	<b>OSPE</b>		<b>10</b>
<b>5.</b>	<b>Final written exam</b>		<b>40%</b>
	<b>Total</b>		<b>= 100 Marks</b>

*Grades*

<i>85 - 100</i>	<i>A</i>	<i>Excellent</i>
<i>75 - 84</i>	<i>B</i>	<i>Very good</i>
<i>65 - 74</i>	<i>C</i>	<i>Good</i>
<i>60 - 64</i>	<i>D</i>	<i>Pass</i>
<i>Less than 60</i>	<i>F</i>	<i>Fail</i>

*All grades will be assigned as follows:*

*Exams: Exams will include short answer and multiple choice questions (MCQs). They will cover material presented in lecture, readings, and discussion. All exams must be taken on the date scheduled. Assignment paper: The purpose of the work is to provide you with the opportunity to explore an area of basic medical sciences or medical education in depth. The paper is to be a 10-15 page literature review of the topic will constitute 20% of your final grade. Policy: Topics must be approved in writing by the coordinator. Directions for topic submission will be discussed during the first week of class. Topics that have not been approved will not be accepted.*

*All papers must reference a minimum of eight references from refereed journals. All papers must be typed, double-spaced, have 1 inch margins.*

## ***Students support***

All teaching staff are available daily for individual student consultations and academic advice from the start time of the module throughout the whole module period. Office hours would be announced as a schedule at the start of the module showing periods per week each faculty member are available in his office to be contact with students to answer their quires) . The following is a list of the faculty members and staff of the Department of Pharmacology. Students are welcome to contact any of the members of the department to answer any of their inquiries.

### **Male Section: Men Medical Complex**

***Pharmacology dept., Bld No.( 7 )***

<b><i>Name/Status</i></b>	<b><i>Room No</i></b>	<b><i>Phone No</i></b>	<b><i>E-Mail Address</i></b>	<b><i>Office Hours</i></b>
<b><i>Dr. Omar Ibrahim Mohammad Saadah ( chairman )</i></b>	<b><i>763/G</i></b>	<b><i>20106</i></b>	<b><i><a href="mailto:saadaho@hotmail.com">saadaho@hotmail.com</a></i></b>	<b><i>10 am-1.00 pm</i></b>  <b><i>EXCEPT</i></b> <b><i>Sunday and</i></b> <b><i>Monday</i></b>
<b><i>Prof. Osman Hassan Osman Omer</i></b>	<b><i>766G</i></b>	<b><i>20108</i></b>	<b><i><a href="mailto:osmanomer39@hotmail.com">osmanomer39@hotmail.com</a></i></b>	<b><i>10 am-1.00 pm</i></b>
<b><i>Prof. Mansour Ibrahim Suliman</i></b>		<b><i>20106</i></b>	<b><i><a href="mailto:misuliman@hotmail.com">misuliman@hotmail.com</a></i></b>	<b><i>1-3 PM at</i></b> <b><i>KFRC</i></b>
<b><i>Prof. Abdel-Moneim Mahmoud Osman</i></b>	<b><i>754A</i></b>	<b><i>20218</i></b>	<b><i><a href="mailto:moneimosman@hotmail.com">moneimosman@hotmail.com</a></i></b>	<b><i>10AM-01 PM</i></b>
<b><i>Dr. Ahmed Shaker Ali</i></b>	<b><i>755G</i></b>	<b><i>22330</i></b>	<b><i><a href="mailto:Ahmedshaker21@yahoo.com">Ahmedshaker21@yahoo.com</a></i></b>	<b><i>9.00AM- 10.00</i></b> <b><i>daily</i></b>
<b><i>Dr. Lateef Mohiuddin Khan</i></b>	<b><i>740G</i></b>	<b><i>??</i></b>	<b><i><a href="mailto:Lmkhan@hotmail.com">Lmkhan@hotmail.com</a></i></b>	

### **Female Section: Women Medical Complex**

***Pharmacology dept., Bld No.( 6 )***

<b><i>Name/Status</i></b>	<b><i>Room No</i></b>	<b><i>Phone No</i></b>	<b><i>E-Mail Address</i></b>	<b><i>Office Hours</i></b>
<b><i>Prof. Magda Mohamed Hagrass</i></b>	<b><i>673 G,</i></b>	<b><i>23100</i></b>	<b><i><a href="mailto:magyhagrass@hotmail.com">magyhagrass@hotmail.com</a></i></b>	<b><i>9.00AM- 11.00</i></b> <b><i>AM except</i></b> <b><i>Wednesday</i></b> <b><i>11.00 AM-</i></b> <b><i>12.00AM</i></b>
<b><i>Prof. Mai Abdul Alim Abdul Sattar Ahmad</i></b>	<b><i>672 G,</i></b>	<b><i>23102</i></b>	<b><i><a href="mailto:drm_aalim2000@yahoo.com">drm_aalim2000@yahoo.com</a></i></b>	<b><i>9.00 AM To</i></b> <b><i>11.00 AM</i></b>

*Core*

**Department Web Site**

**[www.kau.edu.sa/Pharmacology](http://www.kau.edu.sa/Pharmacology)**

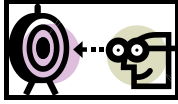
**( CURRENTLY NOT ACTIVATED )**

Time Allocations

<i>List of topics</i>	<i>Contact hours</i>		
	<i>Lecture</i>	<i>Practical</i>	<i>SDL</i>
1. <i>Pharmacodynamics</i>	<i>1</i>		
2. <i>Pharmacokinetics of absorption and distribution</i>	<i>1</i>		
3. <i>pharmacokinetics of metabolism and elimination</i>	<i>1</i>		
4. <i>Factors affecting drug response</i>	<i>1</i>		
5. <i>Unwanted effects of drugs:</i>	<i>1</i>		
6. <i>Directly-acting cholinergic drugs:</i>	<i>1</i>		
7. <i>Anti-cholinergic drugs</i>	<i>1</i>		
8. <i>Adrenergic agonists : Direct acting drugs</i>	<i>1</i>		
9. <i>Adrenergic agonists : indirect &amp; mixed acting drugs</i>	<i>1</i>		
10. <i>Adrenergic antagonists</i>	<i>1</i>		
11. <i>Hypolipidemic drugs</i>	<i>1</i>		
12. <i>Drugs used in Coagulation disorders</i>	<i>1</i>		
13. <i>Antimicrobials : Cell- wall inhibitors</i>	<i>1</i>		
14. <i>Antimicrobials : protein synthesis inhibitors</i>	<i>1</i>		
15. <i>Antimicrobials : Quinolones &amp; folate antagonists</i>	<i>1</i>		
16. <i>Antimicrobials : miscellaneous</i>	<i>1</i>		
17. <i>Antiviral drugs:</i>	<i>1</i>		
18. <i>Antifungal drugs:</i>	<i>1</i>		
19. <i>Anticancer drugs : Antimetabolites; antibiotics;</i>	<i>1</i>		
20. <i>Anticancer drugs ; Alkylating agents:microtubule inhibitors; steroids</i>	<i>1</i>		
21. <i>Non-steroidal anti-inflammatory drugs:</i>	<i>1</i>		
22. <i>CNS stimulants</i>	<i>1</i>		
23. <i>Local anesthetics</i>	<i>1</i>		
24. <i>Antiparkinsonian drugs:</i>	<i>1</i>		
25. <i>Antidepressant Drugs:</i>	<i>1</i>		
<b><i>Practical/ tutorial/ SDL.</i></b>			
1. <i>Drug forms and routes of drug administration</i>		<i>1</i>	
2. <i>Practical pharmacokinetics</i>		<i>1</i>	
3. <i>Effect of autonomic drugs on rabbit eye</i>		<i>1</i>	
4. <i>Drug-drug interaction</i>			<i>1</i>
5. <i>Prescription writing</i>		<i>1</i>	
<b>subtotal</b>	<b>25</b>	<b>4</b>	<b>1</b>
<b>Total contact</b>	<b>30 h</b>		

## **Icons (standards)**

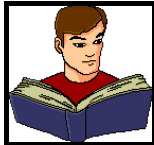
The following icons have been used to help you identify the various experiences you will be exposed to.



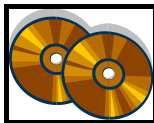
*Learning objectives*



*Content of the lecture*



*Independent learning from textbooks*



*Independent learning from the CD-ROM.*

*The computer cluster is in the 2<sup>nd</sup> floor of the medical library, building*

*No. 7.*



*Independent learning from the Internet*



*Problem-Based Learning*



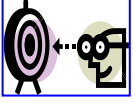

*Self- Assessment (the answer to self-assessment exercises will be discussed in tutorial sessions)*



*The main concepts*

# *Topic Outlines*

*25 : Lectures ,  
4: practical class  
1: SDL*

<b>Lecture : 1. PHARMACODYNAMICS</b>		<b>1 Lecture</b>
Department : Pharmacology		Students notes
Lecturer: <i>prof. Osman (M.sec) Prof. Magda (F. sec )</i>		
TEACHING LOCATION :		
	<p><b>Objectives</b></p> <p>At the end of the lecture you should be able to</p> <ol style="list-style-type: none"> <li>1. <i>Specify whether an antagonist is competitive or irreversible based on its effect on the dose-response curve of an agonist</i></li> <li>2. <i>Give examples of competitive and irreversible pharmacologic antagonists, and physiologic and chemical antagonists</i></li> <li>3. <i>Name the main 4 receptor subfamilies</i></li> <li>4. <i>Describe the term desensitization phenomenon and its underlying mechanism</i></li> </ol>	
	<p><b>Topics</b></p> <ol style="list-style-type: none"> <li>a. <i>Competitive and irreversible pharmacologic antagonists</i></li> <li>b. <i>Physiologic antagonists, chemical antagonists</i></li> <li>c. <i>Signaling mechanisms: intracellular receptors, G-protein receptors, tyrosine-kinase receptors, and ion channel receptors</i></li> <li>d. <i>Desensitization or tachyphylaxis of receptors</i></li> </ol>	<p><i>(Insert here handouts and additional pages for notes if needed)</i></p>



**Continued**



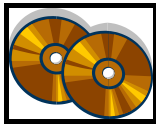
**Remember**

- *Types of receptors; definition of agonist, partial agonist; antagonist; types of antagonists. potency , efficacy, understand the curves ; therapeutic index, therapeutic range Spare receptors is identified by finding that  $EC_{50} < K_d$  of the agonist.*
- *Quantal dose- response curve can be used for determination of therapeutic index & variation in sensitivity to a drug within the population studied.*
- *Steroid hormones receptors are intracellular receptors .*
- *Maximal efficacy is the largest response a drug can produce regardless of dose*
- *Drug A is 100 times more potent than drug B : If drug 1 mg of drug A produce the same magnitude of effect produced by 100 mg of drug B.*



**Text book**

*Basic and clinical Pharmacology, 8<sup>th</sup> edition, { Examination & Board Review } A. J. Trevor & B.G. Katzung Mcgraw Hill ; CHAPTER 2*



*CD ; will be provided by the dept. to demonstrate types of receptors*



**Independent learning from the Internet**



**Self- Assessment** *(the answer to self-assessment exercises will be discussed in tutorial sessions)*

*Which of the following factors will determine the number of drug-receptor complexes formed?*

- A. Efficacy of the drug.*
- B: Receptor affinity for the drug.*
- C. Therapeutic index of the drug.*
- D. Half-life of the drug.*
- E. Rate of renal secretion.*

**Which of the following provide information about the variation in sensitivity to a drug within the population studied?**

- A- Maximal efficacy**
- B- Therapeutic index**
- C- Drug potency**
- D- Graded dose-response curve**
- E- Quantal dose-response curve**

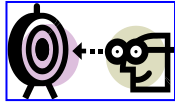
## Lecture: 2. Pharmacokinetics of absorption and distribution 1Lecture

Department : Pharmacology

Lecturer: *Dr Ahmed Shaker (M.sec) Prof Magda (F. sec)*

TEACHING LOCATION :

Student Notes:



### Objectives

At the end of the lecture you should be able to

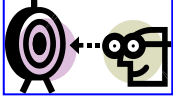

1. **Review** of important PK process & terms
2. **Discuss** details of drug metabolism.
3. **Describe** Renal elimination of drugs
4. **Summarize** Pharmacokinetic after single IV dose



### Topics

1. **Introduction** : Review of some terms Bioavailability, volume of distribution, , protein binding , first pass effect, enzyme induction and enzyme
2. **Details of phase I & phase II metabolic pathways**
3. **Examples of clinically significant enzyme induction & enzyme inhibition**
4. **Renal elimination , Ion trapping & Assessment of renal function**
5. **1<sup>st</sup> order elimination , half life , elimination rate constant & non-linear kinetics**
6. **Estimation of volume of distribution**
7. **Clinical application of volume of distribution to estimate the dose required to attain certain peak level**
8. **Estimation of peak & trough levels**

(Insert here handouts and additional pages for notes if needed)

<b>Lecture: 3. Pharmacokinetics of metabolism and elimination 1 Lecture</b>	
Department : Pharmacology	Student Notes:
Lecturer: <b>Dr Shaker (M.sec) Prof Magda Hagrass (F. sec )</b>	
EACHING LOCATION :	
 <b>Objectives</b> At the end of the lecture you should be able to <ol style="list-style-type: none"> <li>1. <b>Summarize</b> Pharmacokinetics after IV infusion</li> <li>2. <b>Summarize</b> Pharmacokinetics after repeated IV injection</li> <li>3. <b>Summarize</b> Pharmacokinetics after single and repeated oral administration</li> </ol>	
 <b>Topics</b> <ol style="list-style-type: none"> <li>1. Concept of the clearance &amp; estimation of drug clearance</li> <li>2. Estimation of steady state level after IV infusion &amp; the need of a Loading dose</li> <li>3. Concept of accumulation , time to attain steady state level</li> <li>4. Estimation of steady state level after repeated IV injection</li> <li>5. Estimation of steady sate level after repeated oral administration</li> </ol>	(Insert here handouts and additional pages for notes if needed)

**Continued****Remember**

*Factors affecting drug bioavailability , what is the meaning of 1<sup>st</sup> pass effect ,( pre-systemic metabolism ) ; what is the difference between phase I & phase II metabolism*

*Volume of distribution and estimation of loading dose*

*Half- life , time to attain steady state & dosing interval*

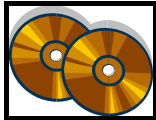
*Clinical Importance of altered protein binding for drugs strongly bound to plasma protein*

*Clinical Importance of Enzyme induction & inhibition*

*Assessment of renal function & dose adjustment.*

**Text book**

1. *Lippincott's illiterate review , 3<sup>rd</sup> Edition, R.D. Howland M.J. Mycek. Lippincott's Williams & Wilkinsp 1-22*
2. *Handout & solved problems provided by the lecturers*

**CD**

*CP – Pham & an educational program for PK analysis will be provided.*



*Independent learning from the Internet valuable web [www.icp.org.nz](http://www.icp.org.nz) (pK animation)*

*<http://www.pjonline.com/pdf/cpd>*

*/pj\_20040619\_pharmacokinetics01.pdf { basic Pk }\*\*\**

*/pj\_20040626\_pharmacokinetics02.pdf ( variability in dose requirement ) /pj\_20040807\_pharmacokinetics04.pdf { TDM }*

*/pj\_20040731\_pharmacokinetics03.pdf ( dose adjustment )*



**Self- Assessment** *(the answer to self-assessment exercises will be discussed in tutorial sessions or with staff during office hours )*

**Which one of the following is a phase I drug metabolism reaction?**

- A. Acetylation.
- B. Conjugation with glucronic acid
- C. Conjugation with Glycine
- D. Conjugation with sulphate
- E. N-Oxidation. and N- Hydroxylation.

**All of the following factors affect drug absorption EXCEPT**

- A- Transport of drugs through membranes.
- B- Increased blood flow to the site of administration
- C- Increased surface area of the site of absorption.
- D- Binding of a drug to its receptor.
- E- Increased lipid solubility

**The following factors which influence drug bioavailability are true EXCEPT**

- A- The formulation and dosage form.
- B- interaction with food or other drugs
- C- .Drug stability in the GIT
- D- The drug lipid solubility and molecular weight
- E- The degree of protein binding

**Factors which can increase the fraction of unbound drug include the following EXCEPT**

- A- Renal impairment ( uraemia )
- B- Low plasma albumin levels
- C- Late pregnancy
- D- Displacement from binding site by other drugs,
- E- Drugs with high molecular weight
- F-

**The following statements concerning distribution of drugs are true EXCEPT:**

- A. The rate of delivery of drugs to tissues such as muscles is usually slow.
- B. Blood - brain barrier prevents many polar drugs from entering into the brain.
- C. Protein bound drugs can't distribute into tissues.
- D. Drugs strongly bound to plasma proteins usually show low volume of distribution
- E. Volume of distribution is used to calculate the dosing interval.

**Drug metabolism usually results in a product that is:**

- A. More likely to distribute intracellularly.
- B. Less lipid-soluble than the original drug.
- C. More likely to be reabsorbed by kidney tubules.
- D. More lipid-soluble than the original drug.
- E. More likely to produce adverse effects.

**Core**

**Which one of the following statements is CORRECT?**

**A. Weak bases are absorbed efficiently across the epithelial cells of the stomach.**

**B. Coadministration of atropine speeds the absorption of a second drug.**



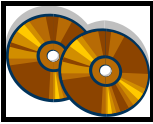


**C. Drugs showing a large  $V_d$  can be efficiently removed by dialysis of the plasma.**

**D. Stressful emotions can lead to a slowing of drug absorption.**

**E. If the  $V_d$  for a drug is small, most of the drug is in the extraplasmic space..**





<i>Continued</i>	
 <p><i>Remember</i></p>  <p><i>Text book</i></p> <ol style="list-style-type: none"> <li>1. <i>Integrated Pharmacology. 3<sup>rd</sup> edition. Page, Curtis, Sutter, Walker, and Hoffman. Mosby</i></li> <li>2. <i>Clinical Pharmacology, 9<sup>th</sup> edition P.N. Bennett and M.J. Brown. Churchill Livingstone</i></li> </ol>  <p><i>CD</i></p>  <p><i>Independent learning from the Internet</i></p>  <p><i>Self- Assessment (the answer to self-assessment exercises will be discussed in tutorial sessions)</i></p> <p><i>- Substances strongly suspected to know to be capable of harming the fetus when consumed by a pregnant woman include all of the following <u>EXCEPT</u>:-</i></p> <ol style="list-style-type: none"> <li><i>A. Sex hormones.</i></li> <li><i>B. Warfarin.</i></li> <li><i>C. Azithromycin.</i></li> <li><i>D. Anticancer drugs.</i></li> <li><i>E. Alcohol.</i></li> </ol> <p><i>- Where a drug causes an allergic (Immunological) illness, all of the following statements are true <u>EXCEPT</u>:-</i></p> <ol style="list-style-type: none"> <li><i>A. There is a linear relationship of dose to effect.</i></li> <li><i>B. Safe desensitization is possible.</i></li> <li><i>C. Re-exposure to a small dose is enough to cause illness.</i></li> <li><i>D. It is unsafe to shift to another member of the same chemical class.</i></li> <li><i>E. There has been a preceding</i></li> </ol>	

*A pregnant woman was hospitalized and catheterized with a Foley catheter. She developed a urinary tract infection caused by Pseudomonas aeruginosa, and was treated with gentamicin. Which of the following adverse effects was a risk to the fetus when the woman was on gentamicin?*

- A. Skeletal deformity.*
- B. Hearing loss.*
- C. Teratogenesis.*
- D. Blindness.*
- E. Mental retardation*

*Pharmacokinetic drug interactions can result from all of the following EXCEPT:*

- A. Impaired absorption.*
- B. Induction of the drug microsomal enzyme metabolizing system.*
- C. Inhibition of the drug microsomal enzyme metabolizing system .*
- D. Inhibition of renal excretion.*
- E. The combination of a bacteriostatic antibiotic and a bactericidal antibiotic.*

*All of the following agents would be capable of inducing the cytochrome P-450*

*metabolizing system EXCEPT:*

- A. Barbiturates.*
- B. Rifampin.*
- C. Phenytoin.*
- D. Carbamazepine.*
- E. Ketoconazole.*

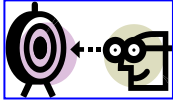

*- Which of the following agents interferes with most of the cytochrome P450*

*enzymes and, thus leads to many drug-drug interactions?*

- A. Famotidine.*
- B. Nizatidine.*
- C. Ranitidine.*
- D. Cimetidine.*
- E. Omeprazole.*

<b>Lecture : 6. Directly Acting Cholinergic Drugs</b>	<b>1 Lecture</b>
Department : Pharmacology	Students notes
Lecturer: <b>Prof Osman (M.sec) Prof Magda (F. sec )</b>	
TEACHING LOCATION :	
<div data-bbox="236 376 408 479" data-label="Image"> </div> <p data-bbox="432 421 596 461"><b>Objectives</b></p> <p data-bbox="285 488 858 519">At the end of the lecture you should be able to</p> <ol data-bbox="285 542 1150 1124" style="list-style-type: none"> <li>1. <i>List the locations and types of acetylcholine receptors in the major organs systems</i></li> <li>2. <i>Describe the effects of acetylcholine on the major organs</i></li> <li>3. <i>Relate the different pharmacokinetic properties of the various choline esters and cholinomimetic alkaloids to their chemical structures</i></li> <li>4. <i>List the major clinical uses of cholinomimetic agonists</i></li> <li>5. <i>Describe the pharmacodynamic differences between direct-acting and indirect-acting cholinomimetic agents</i></li> <li>6. <i>List the major signs and symptoms organophosphate insecticide poisoning and acute nicotine poisoning</i></li> </ol> <div data-bbox="236 1173 367 1326" data-label="Image"> </div> <p data-bbox="391 1223 491 1263"><b>Topics</b></p> <p data-bbox="285 1379 847 1411"><u>Cholinomimetic drugs (cholinergic drugs)</u></p> <ol data-bbox="272 1451 1121 1760" style="list-style-type: none"> <li>1. <i>Direct-acting: Muscarinic choline esters, eg. Ach and others, pilocarpine</i></li> <li>2. <i>Direct-acting: Nicotine</i></li> <li>3. <i>Indirect-acting cholinergic drugs: Carbamates, eg. Physostigmine, edrophonium, neostigmine and the organophosphates</i></li> </ol>	(Insert here handouts and additional pages for notes if needed)

<b>Lecture: 7. Anticholinergic ( Antimuscarinic) drugs</b>	<b>1 Lecture</b>
Department : Pharmacology	Student Notes:
Lecturer: <b>Prof Osman (M.sec) Prof Magda (F. sec )</b>	
TEACHING LOCATION :	
<div data-bbox="236 376 408 479" data-label="Image"> </div> <p data-bbox="432 421 596 461"><b>Objectives</b></p> <p data-bbox="285 490 858 521">At the end of the lecture you should be able to</p> <ol data-bbox="285 544 1177 965" style="list-style-type: none"> <li>1. Describe the effects of atropine on the major organ systems</li> <li>2. List the signs and symptoms of atropine poisoning</li> <li>3. List the major clinical indications and contraindications for the use of muscarinic antagonists</li> <li>4. Describe the effects of ganglion-blocking nicotinic antagonists</li> <li>5. List one antimuscarinic agent promoted for each of the following uses: to produce mydriasis and cycloplegia; to treat parkinsonism; peptic ulcer, and asthma</li> </ol> <div data-bbox="236 1061 367 1169" data-label="Image"> </div> <p data-bbox="389 1111 493 1151"><b>Topics</b></p> <ol data-bbox="285 1200 1149 1285" style="list-style-type: none"> <li>a. Antimuscarinic agents: atropine, scopolamine, and ipratropium</li> <li>b. Ganglionic blocking agents: Nicotine, mecamylamine, trimethophan</li> </ol>	<p data-bbox="1203 1258 1497 1402"><i>(Insert here handouts and additional pages for notes if needed)</i></p>

<p><b>Lecture: 8. Adrenergic Agonists</b>  <b>9. Adrenergic Agonists</b></p>	<p><b>1 Lecture</b>  <b>1 Lecture</b></p>
<p>Department : Pharmacology</p>	<p>Student Notes:</p>
<p>Lecturer: Prof Osman (M.sec) Prof Magda (F. sec )</p>	
<p>TEACHING LOCATION :</p>	
<p> <b>Objectives</b></p> <p>At the end of the lecture you should be able to</p> <ol style="list-style-type: none"> <li>1. List tissues that have significant numbers of alpha receptors</li> <li>2. List tissues that have significantly numbers of beta receptors</li> <li>3. Describe the major organ system effects of a pure alpha agonist, a pure beta agonist, and a mixed alpha and beta agonist. Give examples of each type of drug</li> <li>4. Describe a clinical situation in which the effect of an indirect sympathomimetic would differ from those of a direct agonist</li> </ol> <p>List the major clinical applications of the adrenoceptor agonists</p> <p> <b>Topics</b></p> <ol style="list-style-type: none"> <li>a. Direct -acting agonists: alpha agonists, alpha-one selective, alpha 2 selective and non-selective</li> <li>b. Beta agonists: beta-one selective, beta-2 selective and non-selective</li> <li>c. Indirect-acting: amphetamine, tyramine, mixed-acting: ephedrine</li> </ol>	
<p>(Insert here handouts and additional pages for notes if needed)</p>	

<b>Lecture: 10. adrenergic blockers</b>	<b>1 Lecture</b>
Department : Pharmacology	Student Notes:
Lecturer <b>Prof Osman Hassan (M.sec) Prof. Magda (F. sec )</b>	
EACHING LOCATION :	
<div data-bbox="236 376 408 479" data-label="Image"> </div> <p data-bbox="432 421 596 461"><b>Objectives</b></p> <p data-bbox="284 488 858 519">At the end of the lecture you should be able to</p> <ol data-bbox="284 542 1177 1182" style="list-style-type: none"> <li>1. Describe the effects of an alpha blocker on the hemodynamic responses to epinephrine</li> <li>2. Describe the effects of an alpha-blocker on the hemodynamic responses to norepinephrine</li> <li>3. Compare the effects of propranolol, labetalol, metoprolol, and pindolol</li> <li>4. Compare the pharmacokinetics of propranolol, atenolol, esmolol, and nadolol</li> <li>5. Describe the clinical indications, and toxicities of typical alpha- and beta-blockers</li> <li>6. Define the drugs which affect adrenergic neuronal transmitter uptake or release such as cocaine, guanethidine and reserpine</li> </ol> <div data-bbox="236 1227 367 1330" data-label="Image"> </div> <p data-bbox="391 1272 494 1312"><b>Topics</b></p> <ol data-bbox="284 1361 1177 1998" style="list-style-type: none"> <li>a. Alpha-adrenergic blockers: non-selective blockers, such as phenoxybenzamine, phentolamine,</li> <li>b. Selective-alpha-one blockers such as prazosin, terazosin, tamsulosin</li> <li>c. Beta-adrenergic blockers: non-selective such as propranolol. Selective-beta-one blockers such as atenolol, metoprolol, and esmolol. Beta-antagonists with partial agonist activity such as pindolol and acebutalol.</li> <li>d. Antagonists of both alpha and beta adrenoceptors such as labetalol and carvedilol</li> <li>e. Drugs affecting neurotransmitter release or uptake: reserpine, guanethidine and cocaine</li> </ol>	<p data-bbox="1203 1258 1500 1402"><i>(Insert here handouts and additional pages for notes if needed)</i></p>

*Continued*

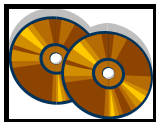


*Remember : write down important key points provided during the lectures*



*Text book*

*Lippincott's Pharmacology 3<sup>rd</sup> edition. R.D. Howland, M.J. Mycek.  
Lippincott's Williams & Wilkins Chapters 3- 7 pp 35-81*



*CD*



*Independent learning from the Internet*

*Many valuable web for example*

[www.frca.co.uk](http://www.frca.co.uk)

*Google : search for AnesthesiaUK – Home > Recourses*

*>Pharmacology : explore the articles*

*\* Autonomic nervous system & \* Autonomic nervous system II*



*Self- Assessment (the answer to self-assessment exercises will be discussed in tutorial sessions)*

*- Botulinum toxin causes paralysis by:*

- A. Inhibiting choline acetyltransferase.*
- B. Blocking transport of choline into neurons .*
- C. Blocking release of acetylcholine from storage vesicles.*
- D. Inhibiting acetylcholinesterase.*
- E. Blocking the synapse at ganglia.*

- Which of the following medications is used to prevent premature labor?

- A. Neostigmine.
- B. Tamsulosin.
- C. Atracurium.
- D. Clonidine.
- E. Terbutaline.

- Which of the following drugs is used to diagnose myasthenia gravis?

- A. Atropine.
- B. Neostigmine.
- C. Bethanechol.
- D. Edrophonium.
- E. Pralidoxime.

From the list below, choose the depolarizing neuromuscular blocker most likely to be used in "rapid sequence intubation," a procedure that is done when the stomach contents have a high risk of refluxing and causing aspiration.

- A. Baclofen.
- B. Succinylcholine.
- C. Neostigmine.
- D. Homatropine.
- E. Pralidoxime.

- The drug of choice for treating decreased salivation accompanying head and neck irradiation is:

- A. Physostigmine.
- B. Pilocarpine.
- C. Scopolamine.
- D. Carbachol.
- E. Acetylcholine.



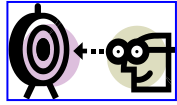
**Lecture: 11. hypolipidemic Drugs****1 Lecture**

Department : Pharmacology

Student Notes:

Lecturer: **Prof.Moneim (M.sec)** **Prof. Mai Abdul Sattar (F. sec)**

TEACHING LOCATION :

**Objectives**

At the end of the lecture you should be able to

- a. Describe types and the proposed role of lipoproteins in the formation of atherosclerotic plaques
- b. Describe the five main classes of drugs used to treat hyperlipidemia, and describe their mechanisms of action, effects upon serum lipid concentrations and adverse effects
- c. Based on a set baseline serum lipid values, propose a rational drug treatment regimen
- d. Surge the merits of combined drug therapy for some diseases and list 3 rational drug combinations

**Topics**

- a. Hyperlipoproteinemia: treatment strategies, diet, and drugs
- b. HMG-CoA reductase inhibitors e.g. atorvastatin, lovastatin; mechanism of action, indications and adverse effects.
- c. Fibrates e.g. fenofibrate, gemfibrozil; mechanism of action, indications and adverse effects.
- d. Niacin (nicotinic acid); mechanism of action, indications and adverse effects.
- e. Bile acid sequestrants e.g. cholestyramine ; mechanism of action, indications and adverse effects.
- f. Cholesterol absorption inhibitors: ezetimibe ; mechanism of action, indications and adverse effects.

(Insert here handouts and additional pages for notes if needed)



**Remember**

**-Avoid combinations of HMG- CO A reductase inhibitors (statins) and fibrates for fear of developing of sever myopathy.**

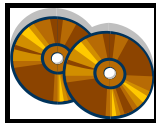


**Text book**

**1-Lippincott's Illustrated Reviews: Pharmacology, 4th Edition**

by [Richard A Harvey](#) ;[Pamela C Champe](#) ;[Richard Finkel](#) ;[Luigi Cubeddu](#) &, [Michelle A Clarke](#) (Editors) . Lippincott Williams & Wilkins **2009**

**2 ) Katzung & Trevor's Pharmacology Examination and Board Review: Eighth Edition by Anthony Trevor, Bertram Katzung, and Susan Masters . MCGraw Hill, **2008****



**CD**



**Independent learning from the Internet**

**Self- Assessment** (the answer to self-assessment exercises will be discussed in tutorial sessions)

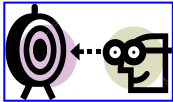

**We administer a drug with the intent of lowering a patient's elevated LDL and total cholesterol levels, and raising HDL levels. The drug we choose inhibits cholesterol synthesis by inhibiting 3-hydroxy-3-methylglutaryl-coenzyme A (better known as [HMG CoA] reductase). Which of the following drugs best fits this description and works by the stated mechanism of action?**

- A. Cholestyramine.**
- B. Gemfibrozil.**
- C. Lovastatin.**
- D. Nicotinic acid (niacin).**
- E. Ezetimibe**

**Select the hypolipidemic drug that enhances lipoprotein**

**Core**

<p><i>synthesis, fatty acid oxidation and LDL receptor expression in liver through peroxisome proliferator activated receptor <math>\alpha</math>:</i></p> <ul style="list-style-type: none"><li><i>A- Lovastatin</i></li><li><i>B- Atorvastatin</i></li><li><i>C- Fenofibrate</i></li><li><i>D- Nicotinic acid</i></li><li><i>E- Ezetimibe</i></li></ul>		

<b>Lecture: 12 Drugs used in Coagulation Disorders</b>	
Department : Pharmacology	
Lecturer: <i>Prof.Moneim (M.sec) Prof. Mai Abdul Sattar(F. sec )</i>	
EACHING LOCATION :	
 <p><b>Objectives</b></p> <p>At the end of the lecture you should be able to</p> <ol style="list-style-type: none"> <li>1. <i>List 4 types of anticoagulants and describe their mechanisms of action</i></li> <li>2. <i>Compare the oral anticoagulants, standard heparin, and LMW heparins in terms of their pharmacokinetics, mechanisms of action and toxicities</i></li> <li>3. <i>Compare the pharmacokinetics, clinical uses, and toxicity of the major antiplatelet drugs</i></li> <li>4. <i>List the main thrombolytic drugs, explain how they act, their adverse effects and clinical uses</i></li> </ol>	<p><i>(Insert here handouts and additional pages for notes if needed)</i></p>
 <p><b>Topics</b></p> <ol style="list-style-type: none"> <li>1. <i>Oral anticoagulants: warfarin</i></li> <li>2. <i>Injectable anticoagulants: heparin, LMW heparins, lepirudin, danaparoid</i></li> <li>3. <i>Platelet aggregation inhibitors: aspirin, ticlopidine and clopidogrel, Abciximab, Eptifibitide and tirofiban, dipyridamole</i></li> <li>4. <i>Thrombolytic drugs: alteplase, streptokinase, urokinase</i></li> <li>5. <i>Drugs used to treat bleeding: aminocaproic acid, tranexamic acid, protamine sulfate, vitamin K, Aprotinin</i></li> </ol>	

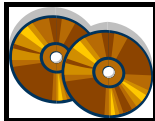
**Continued****Remember**

- Warfarin is contraindicated for a pregnant woman.
- Drug interactions are very important with oral anticoagulants
- Continuous monitoring with the use of anticoagulants is mandatory

**Text book****1-Lippincott's Illustrated Reviews: Pharmacology, 4th Edition**

by [Richard A Harvey](#) ;[Pamela C Champe](#) ;[Richard Finkel](#) ;[Luigi Cubeddu](#) &, [Michelle A Clarke](#) (Editors) . Lippincott Williams & Wilkins **2009**

**2 ) Katzung & Trevor's Pharmacology Examination and Board Review: Eighth Edition** by Anthony Trevor, Bertram Katzung, and Susan Masters . **MCGraw Hill, 2008**

**CD****Independent learning from the Internet**

**Self- Assessment** (the answer to self-assessment exercises will be discussed in tutorial sessions)

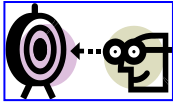

Which one of the following agents is **LEAST** likely to enhance the anticoagulant effects of warfarin?

- A. Aspirin.
- B. Cholestyramine.
- C. Cimetidine.
- D. Quinidine.
- E. Thyroxine.



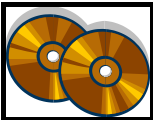
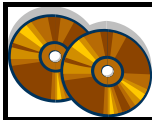


A 30-year-old pregnant woman requires heparin for prophylaxis of thromboembolism. Which of the following best summarizes heparin's main mechanism of action?

- A. Activates plasminogen .
- B. Increases the plasma level of Factor IX.
- C. Inhibits platelet aggregation in vitro.
- D. Inhibits synthesis of prothrombin and coagulation Factors VII, IX, and X.
- E. Inhibits thrombin and early coagulation steps.

<b><i>Continued</i></b>	
<p><b><i>A 42-year-old man with an acute MI is treated with alteplase. Which of the following most accurately describes how this drug exerts its intended effect?</i></b></p> <ul style="list-style-type: none"><li><b><i>A. Blocks platelet ADP receptors.</i></b></li><li><b><i>B. Inhibits platelet thromboxane production.</i></b></li><li><b><i>C. Inhibits synthesis of vitamin K–dependent coagulation factors.</i></b></li><li><b><i>D. Prevents aggregation of adjacent platelets by blocking Glycoprotein IIB/IIIa receptors.</i></b></li><li><b><i>E. Promotes conversion of plasminogen to plasmin.</i></b></li></ul>	

<p><b>Lecture : 13- 16: Antimicrobial therapy and antibiotics</b>  <b>4lectures</b></p>	
<p><i>Department : Pharmacology</i></p>	<p><i>Students notes</i></p>
<p><b>Lecturer: <i>Proof. Osman (M.sec) Prof. Mai Abdul Sattar(F. sec )</i></b></p>	
<p><b>TEACHING LOCATION :</b></p>	
<div style="display: flex; align-items: flex-start;"> <div style="margin-right: 20px;">  </div> <div> <p><b>Objectives</b></p> <p>At the end of the lecture you should be able to</p> <ol style="list-style-type: none"> <li>1. Describe the mechanism of action of penicillins, cephalosporins and other beta-lactam antibiotics. Discuss their pharmacokinetics, clinical uses and adverse effects</li> <li>2. <i>Define the actions and uses of vancomycin</i></li> <li>3. <i>Describe the pharmacodynamic and pharmacokinetic properties of the protein synthesis inhibitors: tetracyclines, aminoglycosides, macrolides/ketolides, chloramphenicol, clindamycin and the quinupristin/dalfopristin and the linezolid</i></li> <li>4. <i>Discuss the pharmacodynamic and pharmacokinetic effects, clinical uses and adverse effects of the fluoroquinolones and folic acid antagonists</i></li> </ol> </div> </div>	
<div style="display: flex; align-items: flex-start;"> <div style="margin-right: 20px;">  </div> <div> <p><b>Topics</b></p> <p>∴</p> <ol style="list-style-type: none"> <li>1. <i>Cell -wall inhibitors:Penicillins and cephalosporins, carbapenems, monobactams. Beta-lactamase inhibitors, vancomycin</i></li> <li>2. <i>Protein synthesis inhibitors: Tetracyclines, aminoglycosides, chloramphenicol, macrolides, clindamycin quinupristin/dalfopristin and linezolid</i></li> <li>3. <i>Fluoroquinolones and folic acid antagonists</i></li> </ol> </div> </div>	

*(Insert here handouts and additional pages for notes if needed)*

<b>Continued</b>		
	<b>Remember</b>	
<p>*Cross allergy is present- to some extent- between penicillins and cephalosporins. *Quinolones are contraindicated for patients less than 18 year old. *Sulphonamides are contraindicated for patients with G-6- p Deficiency. *Antibiotics should be used for proper duration to obtain complete cure.</p>		
	<b>Text book</b>	
<p><b>1-Lippincott's Illustrated Reviews: Pharmacology, 4th Edition</b> by <a href="#">Richard A Harvey</a> ;<a href="#">Pamela C Champe</a> ;<a href="#">Richard Finkel</a> ;<a href="#">Luigi Cubeddu</a> &amp; <a href="#">Michelle A Clarke</a> (Editors) . Lippincott Williams &amp; Wilkins <b>2009</b></p> <p><b>2 ) <u>Katzung &amp; Trevor's Pharmacology Examination and Board Review: Eighth Edition</u> by Anthony Trevor, Bertram Katzung, and Susan Masters . MCGraw Hill, <b>2008</b></b></p>		
		<b>CD</b>
	<b>Independent learning from the Internet</b>	
	<b>Self- Assessment</b> <i>(the answer to self-assessment exercises will be discussed in tutorial sessions)</i>	
<p><b>In which one of the following infections is ciprofloxacin ineffective?</b></p> <ul style="list-style-type: none"><li><b>A. Urinary tract infections due to a <math>\beta</math>-lactamase-producing strain of klebsiella.</b></li><li><b>B. Pneumonia due to <i>Streptococcus pneumoniae</i>.</b></li><li><b>C. Exacerbation of chronic bronchitis due to <i>Moraxella catarrhalis</i>.</b></li><li><b>D. Urinary tract infection due to <i>Escherichia coli</i>.</b></li><li><b>E. Urinary tract infections due to <i>Pseudomonas aeruginosa</i>.</b></li></ul>		



*A patient with degenerative joint disease is to undergo insertion of a hip prosthesis. To avoid complications due to postoperative infection, the surgeon will pretreat this patient with an antibiotic. This hospital has a significant problem with methicillin-resistant Staphylococcus aureus. Which of the following antibiotics should the surgeon select?*

- A. Ampicillin.*
- B. Imipenem/cilastatin.*
- C. Gentamicin/piperacillin.*
- D. Vancomycin.*
- E. Cefazolin.*

*A patient with a gunshot wound to the abdomen, which has resulted in spillage of intestinal contents, is brought to the emergency room. Which antibiotic would you select to effectively treat an infection due to Bacteroides fragilis?*

- A. Aztreonam.*
- B. Clindamycin.*
- C. Gentamicin.*
- D. Azithromycin.*
- E. Doxycycline.*

*A pregnant woman was hospitalized and catheterized with a Foley catheter. She developed a urinary tract infection caused by Pseudomonas aeruginosa, and was treated with gentamicin. Which of the following adverse effects was a risk to the fetus when the woman was on gentamicin?*

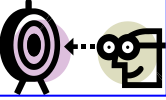

- A. Skeletal deformity.*
- B. Hearing loss.*
- C. Teratogenesis.*
- D. Blindness.*
- E. Mental retardation*

*A thirty-five-year-old male, formerly a heroin abuser, has been on methadone maintenance for the last thirteen months. Two weeks ago, he had a positive PPD test, and a chest radiograph showed evidence of right upper lobe infection. He was started on standard antimycobacterial therapy. He has come to the emergency department complaining of "withdrawal symptoms." Which of the following antimycobacterial drugs is likely to have caused this patient's acute withdrawal reaction?*

- A. Ethambutol.*
- B. Isoniazid.*
- C. Pyrazinamide.*
- D. Rifampin.*
- E. Streptomycin.*

*The combination of sulfadiazine and pyrimethamine is effective against which one of the following opportunistic infections in the AIDS patient?*

- A. Disseminated herpes simplex.*
- B. Cryptococcal meningitis.*
- C. Toxoplasmosis.*
- D. Oral candidiasis.*
- E. Tuberculosis*

<b>Lecture : 17. Antiviral Drugs</b>	<b>1 Lecture</b>
<i>Department : Pharmacology</i>	<i>Students notes</i>
<b>Lecturer : <i>Proof. Osman (M.sec) Prof. Mai (F. sec)</i></b> <b>TEACHING LOCATION :</b>	
 <b>Objectives</b> <p>At the end of the lecture you should be able to</p> <ol style="list-style-type: none"> <li>1. <i>Identify the main steps in viral replication</i></li> <li>2. <i>Describe the mechanisms of action and of resistance of the major antiherpes drugs</i></li> <li>3. <i>Describe the pharmacokinetic properties, the clinical uses, and the toxic effects of the antiherpes drugs</i></li> <li>4. <i>Describe the mechanisms of action and of resistance of the major antiretroviral drugs</i></li> <li>5. <i>Describe the pharmacokinetic properties, the clinical uses, and the toxic effects of the antiretroviral drugs</i></li> <li>6. <i>Identify the significant antiviral properties of amantadine, neuraminidase inhibitors, interferons, fusion inhibitor and ribavarine</i></li> </ol>	
 <b>Topics</b> <ol style="list-style-type: none"> <li>1. <i>Antiherpes drugs: acyclovir, foscarnet, ganciclovir, cidofovir, vidarabine, idouridine, trifuridine</i></li> <li>2. <i>Anti-HIV agents: (NRTIs) zidovudine, didanosine, zalcitabine, lamivudine, stavudine, abacavir</i></li> <li>3. <i>Anti-HIV agents: non-nucleoside reverse transcriptase inhibitors (NNRTIs), nevirapine, delavirdine, efavirenz</i></li> <li>4. <i>Anti-HIV agents: protease inhibitors, indinavir, ritonavir, saquinavir</i></li> <li>5. <i>Miscellaneous antiviral drugs: amantadine, oseltamivir and zanamivir, inteferons, ribavarin</i></li> </ol>	<p><i>(Insert here handouts and additional pages for notes if needed)</i></p>



*Remember*

- \* **Acyclovir is the drug of choice for herpetic infections.**
- \* **Crystalluria is an adverse effect of IV administration of acyclovir.**
- \* **Myelosuppression is increased when gancyclovir is combined with zidovudine**

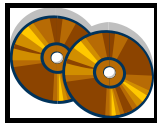
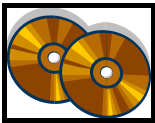


*Text book*

**1-Lippincott's Illustrated Reviews: Pharmacology, 4th Edition**

by [Richard A Harvey](#) ;[Pamela C Champe](#) ;[Richard Finkel](#) ;[Luigi Cubeddu](#) &, [Michelle A Clarke](#) (Editors) . Lippincott Williams & Wilkins **2009**

**2 ) Katzung & Trevor's Pharmacology Examination and Board Review: Eighth Edition by Anthony Trevor, Bertram Katzung, and Susan Masters . MCGraw Hill, 2008**



*CD*



*Independent learning from the Internet*



*Self- Assessment (the answer to self-assessment exercises will be discussed in tutorial sessions)*

**Which one of the following antiviral drugs share activity against hepatitis B and HIV viruses?**

- A. Lamuvidine.**
- B. Interferon.**
- C. Ribavirin.**
- D. Zidovudine.**
- E. Amantadine.**

**2. Amantadine used prophylactically against influenza A is thought to act by :**

- A. Preventing production of viral protein.**
- B. Preventing viron release.**
- C. Preventing viral penetration into the host cell.**
- D. Preventing uncoating of viral DNA.**
- E. Causing lysis of infected host cell by release of**

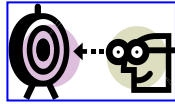
*intracellular lysosomal enzymes.*

**3. Which of the following anti –CMV drugs is likely to cause additive myelosuppression with zidovudine ?**

- A. Acyclovir.**
- B. Ganciclovir.**
- C. Amantadine**
- D. Foscarnet.**
- E. Ribavirin.**

**4-Gancyclovir is preferred over acyclovir in the following condition:**

- A. Herpes simplex keratitis**
- B. Herpes zoster**
- C. Chickenpox**
- D. Cytomegalovirus retinitis in AIDS patients**
- E. Monilia infection**

**Lecture: 18. Antifungal Drugs****1 Lecture***Department : Pharmacology**Lecturer : Prof. Osman (M.sec) Prof. Mai (F. sec )**TEACHING LOCATION :***Objectives**

At the end of the lecture you should be able to

1. Describe the mechanisms of action of the major drugs used for fungal infections
2. Describe the clinical uses and pharmacokinetics of amphotericin B, flucytosine, fluconazole, itraconazole, ketoconazole, voriconazole, caspofungin, clotrimazole, fluconazole, griseofulvin, terbinafine, and nystatin
3. Identify the toxic effects of the major antifungal drugs
4. Identify the main topical antifungal agents

**Topics**

- a. Antifungal drugs for systemic mycoses: amphotericin B, fluconazole, itraconazole, ketoconazole, voriconazole, caspofungin, flucytosine
- b. Antifungal drugs for cutaneous mycoses: nystatin, miconazole, clotrimazole, griseofulvin, terbinafine.

*Student Notes:*

*(Insert here handouts and additional pages for notes if needed)*



*Remember*

**\*Amphotericin B** The drug of choice in life threatening systemic mycoses, used in combination with flucytosin in cryptococcal meningitis .

**\*Ketoconazole and amphotericin B** should not be given together because the decrease in ergosterol of fungal membrane reduces the fungicidal action of amphotericin B.

**\*Ketocinazole** should be avoided in pregnancy.

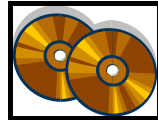
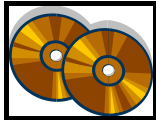


*Text book*

**1-Lippincott's Illustrated Reviews: Pharmacology, 4th Edition**

by [Richard A Harvey](#) ;[Pamela C Champe](#) ;[Richard Finkel](#) ;[Luigi Cubeddu](#) &, [Michelle A Clarke](#) (Editors) . Lippincott Williams & Wilkins **2009**

**2 ) Katzung & Trevor's Pharmacology Examination and Board Review: Eighth Edition** by *Anthony Trevor, Bertram Katzung, and Susan Masters* . **MCGraw Hill, 2008**



*CD*



*Independent learning from the Internet*



**Self- Assessment** (the answer to self-assessment exercises will be discussed in tutorial sessions)

**1. The dose –limiting toxicity of amphotericin B is:**

- A. Myelosuppression.**
- B. Infusion –related adverse effects.**
- C. Renal toxicity.**
- D. Hypotension.**
- E. Hepatitis**

**2. Which one of the following antifungal drugs is not used for systemic fungal Infection?**

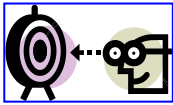

- A. Flucytosine.**
- B. Griseofulvin.**
- C. Fluconazole.**
- D. Ketoconazole.**
- E. Amphotericin B**

**3. Which antifungal is effective in both dermatophytosis as well as systemic mycosis?**

- A. Amphotericin B**
- B. Griseofulvin**
- C. Clotrimazole**
- D. Ketoconazole**
- E. Terbinafine**

**Adverse effects of ketoconazole includes all of the following EXCEPT**

- A. Gynaecomastia**
- B. Oligospermia**
- C. Kidney damage**
- D. Liver toxicity**
- E. Menstual irregularities**

<b>Lecture: 19, 20 Anticancer drugs :</b>	<b>2 Lectures</b>
Department : Pharmacology	<i>Student Notes:</i>
Lecturer: <b>Prof. Moneim (M.sec) Prof Moneim (F. sec )</b>	
EACHING LOCATION :	
<div data-bbox="236 456 408 560">  </div> <p data-bbox="427 501 596 539"><b>Objectives</b></p> <p data-bbox="284 568 858 602">At the end of the lecture you should be able to</p> <ol data-bbox="284 622 1134 987" style="list-style-type: none"> <li>1. <i>the principle of cancer chemotherapy</i></li> <li>2. <i>2- the goal of treatment</i></li> <li>3. <i>3-The treatment regimens and scheduling</i></li> <li>4. <i>4-The problem associated with chemotherapy</i></li> <li>5. <i>5-The different classes of anticancer drugs</i></li> <li>6. <i>6-The mechanism of action of each anticancer drug</i></li> <li>7. <i>7-The interaction between anticancer drugs and other drugs</i></li> </ol> <div data-bbox="240 1032 371 1140">  </div> <p data-bbox="395 1081 499 1120"><b>Topics</b></p> <ol data-bbox="284 1167 1134 2089" style="list-style-type: none"> <li>1. <i>Cell cycle specificity of anticancer drugs</i></li> <li>2. <i>.Different anticancer protocols MOPP, CHOP, FAD, COPP</i></li> <li>3. <i>.Side effects of anticancer drugs ( Bone marrow depression, alopecia, GI Bleeding ---)</i></li> <li>4. <i>.Classes of anticancer drugs ( their mechanism of action, toxicity and interaction)</i> <ul data-bbox="284 1496 1171 2089" style="list-style-type: none"> <li>▪ <i>Antimetabolite ( Methotrexate, 5-FU, 6-MP, Arabinoside cytosine, Fludarabine, cladribine, capcitapine, gemcitabine</i></li> <li>▪ <i>Alkylating agents ( Nitrogen mustard, cyclophosphamide, chlorambucil, Nitrosourea, melphalan, dacarbazine)</i></li> <li>▪ <i>3-Antibiotics (Doxorubicin, epirubicin, daunorubicin, bleomycin)</i></li> <li>▪ <i>Microtubule inhibitors (Vincristine, vinblastine, Paclitaxel, docetaxel)</i></li> <li>▪ <i>Steroid hormone and their antagonists ( Prednisolone, Tamoxifen , Aromatase inhibitors progestins ,Leuprolide and goserline</i></li> <li>▪ <i>6-Monoclonal antibodies (Trastuzumab, Retuximasb, bevacizumab, cetuximab)</i></li> <li>▪ <i>7-Platinum compouns ( cisplatin , Carboplatin)</i></li> <li>▪ <i>8-Podoiophyllotoxin (Etoposide)</i></li> <li>▪ <i>9-tyrosine Kinase inhibitors (Imatinib)</i></li> </ul> </li> </ol>	
	<i>(Insert here handouts and additional pages for notes if needed)</i>



*Core*

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<i>Continued</i>	

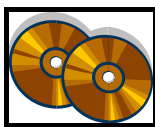


**Remember**



**Text book**

*Lippincott's illiterate review , 3<sup>rd</sup> Edition, R.D. Howland M.J. Mycek. Lippincott's Williams & Wilkinsp*



**CD**



**Independent learning from the Internet**



**Self- Assessment (the answer to self-assessment exercises will be discussed in tutorial sessions)**

*An HIV-positive woman is diagnosed with CMV retinitis. She has been on a HAART regimen containing zidovudine. Which of the following anti-CMV drugs is likely to cause additive myelosuppression with zidovudine?*

- A. Ganciclovir.*
- B. Acyclovir.*
- C. Amantadine.*
- D. Foscarnet.*
- E. Ribavirin.*

*A 25 year-old male AIDS patient has a fever of 102°F and complains of severe headaches during the past week. Staining of his CSF with India ink reveals Cryptococcus neoformans. The patient is admitted to the hospital and is treated with:*

- A. Intravenous amphotericin B plus flucytosine.*
- B. Oral ketoconazole.*
- C. Intrathecal amphotericin B.*
- D. Oral fluconazole.*
- E. Intravenous amphotericin B plus ketoconazole.*

**MESNA is antidote for:**

- A-Methotrexate toxicity.**
- B-5-FU toxicity.**
- C-6-MP toxicity.**
- D-Cisplatin toxicity.**
- E-Cyclophosphamide –induced hemorrhagic cystitis.**

**Which of the following is the main mechanism by which vincristine is exerting its effects?**

- A-Alkylating DNA, causing cross-linking between DNA strands.**
- B-Blocking microtubular assembly and mitosis during M-phase.**
- C-Intercalating DNA strands.**
- D-Stabilizing assembled microtubule array, thereby preventing mitosis.**
- E-Inhibiting topoisomerase. Preventing repair of DNA strand breaks.**

**Which of the following anticancer drugs can be used for the treatment of brain tumors?**

- A-Busulfan.**
- B-5-FU.**
- C-Adriamycin.**
- D-Methotrexate.**
- E-Lomustine.**

**Choose from the following- for questions 1,2, 3**

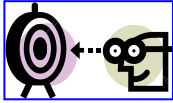

- A. Sulbactam**
- B. Aztreonam.**
- C. Ethambutol.**
- D. Voriconazole.**
- E. Isoniazid**

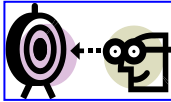

**1- Monobactam, has antimicrobial activity against Enterobacteriaceae and P.aeruginosa.**

**Can be safely used in penicillin allergic patients. [B]**

**2- Antimycobacterial drug. Can cause optic neuritis which results in decreased visual acuity and loss of ability to discriminate between red and green. [C]**

**3- Broad-spectrum antifungal agent. Orally active and can penetrate CNS. [D]**

<b>Department : Pharmacology</b>	<i>Students notes</i>
<b>Lecturer: <i>Proof. Osman (M.sec) Prof Magda (F. sec)</i></b>	
<b>TEACHING LOCATION :</b>	
<div style="display: flex; align-items: flex-start;"> <div style="margin-right: 10px;">  </div> <div> <p><b>Objectives</b></p> <p>At the end of the lecture you should be able to</p> <ol style="list-style-type: none"> <li>1. <i>Contrast the functions of COX-1 and COX-2</i></li> <li>2. <i>Describe the effects of aspirin on prostaglandin synthesis</i></li> <li>3. <i>Contrast the actions and toxicity of aspirin, the older nonselective NSAIDs and the COX-2-selective drugs</i></li> <li>4. <i>List the toxic effects of aspirin and salicylates</i></li> <li>5. <i>Describe the actions of paracetamol (acetaminophen), its pharmacokinetic effects, its clinical uses and adverse effects</i></li> </ol> </div> </div> <div style="display: flex; align-items: flex-start;"> <div style="margin-right: 10px;">  </div> <div> <p><b>Topics</b></p> <ol style="list-style-type: none"> <li>1. <i>Aspirin and salicylates, diflunisal, ibuprofen</i></li> <li>2. <i>Indomethacin, piroxicam, mefenamic acid, diclofenac,</i></li> <li>3. <i>COX-2-selective NSAID's: celecoxib,</i></li> <li>4. <i>Acetaminophen</i></li> </ol> </div> </div>	<p><i>(Insert here handouts and additional pages for notes if needed)</i></p>

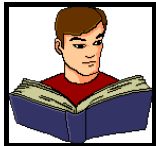
<b>Lecture: 22. ( CNS ) Central Nervous System Stimulants    1 Lecture</b>		
<i>Department : Pharmacology</i>	<i>Student Notes:</i>	
<i>Lecturer: <b>Prof. Osman (M.sec) Prof Magda (F. sec)</b></i>		
<b>TEACHING LOCATION :</b>		
<div style="display: flex; align-items: center; margin-bottom: 10px;">  <div> <p><b>Objectives</b></p> <p>At the end of the lecture you should be able to</p> <ol style="list-style-type: none"> <li>1. <i>Identify the major classes of CNS stimulants</i></li> <li>2. <i>Describe the effects of psychomotor stimulant drugs</i></li> <li>3. <i>Identify their pharmacodynamic and pharmacokinetic properties, their medical use, their adverse effects and their addiction potentials</i></li> <li>4. <i>Describe the effects of hallucinogenic drugs on the CNS and their adverse effects</i></li> </ol> </div> </div> <div style="display: flex; align-items: center; margin-bottom: 10px;">  <div> <p><b>Topics</b></p> <ol style="list-style-type: none"> <li>1. <i>Methylxanthine derivatives: Caffeine, theobromine and theophylline</i></li> <li>2. <i>Nicotine, cocaine, amphetamine</i></li> <li>3. <i>Hallucinogens: lysergic acid diethylamide(LSD), tetrahydrocannabinol, and phencyclidine (PCP)</i></li> </ol> </div> </div>		
		<i>(Insert here handouts and additional pages for notes if needed)</i>

<b>Lecture: 23. Local Anaesthetic Drugs</b>	<b>1 Lecture</b>
Department : Pharmacology	Student Notes:
Lecturer: <b>Prof. Osman (M.sec)</b> <b>Prof Magda (F. sec)</b>	
TEACHING LOCATION :	
<div data-bbox="236 376 408 479" data-label="Image"> </div> <p data-bbox="432 421 596 461"><b>Objectives</b></p> <p data-bbox="285 488 858 519">At the end of the lecture you should be able to</p> <ol data-bbox="285 542 1177 1012" style="list-style-type: none"> <li>1. Describe the mechanism of blockade of the nerve impulses by local anesthetics</li> <li>2. Discuss the relation between pH, pKa, and the speed of onset of local anesthesia</li> <li>3. List the factors that determine the susceptibility of nerve fibers to blockade</li> <li>4. Describe the pharmacokinetics, clinical uses and adverse effects of the major ester group and the amide group of local anesthetics</li> </ol> <div data-bbox="236 1115 367 1223" data-label="Image"> </div> <p data-bbox="391 1167 491 1207"><b>Topics</b></p> <ol data-bbox="285 1249 1177 1617" style="list-style-type: none"> <li>1. Chemical structure of ester type and amide type local anesthetics</li> <li>2. Mechanisms of action, and effect of local anesthetics on different organ systems</li> <li>3. Susceptibility of the different types of nerves to blockade by local anesthetics</li> <li>4. Ester L.A.: procaine, cocaine, tetracaine, benzocaine</li> <li>5. Amide L.A.: lidocaine, mepivacaine, bupivacaine, ropivacaine</li> </ol>	(Insert here handouts and additional pages for notes if needed)

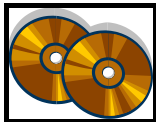
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**Remember**



**Text book**



**CD**



**Independent learning from the Internet**



**Self- Assessment** (the answer to self-assessment exercises will be discussed in tutorial sessions)

All of the following statements regarding aspirin and diflunisal are correct **EXCEPT**:

- A. Aspirin is unique among the NSAIDs in that it irreversibly acetylates and inactivates cyclooxygenase.
- B. Diflunisal is metabolized to salicylate and therefore, can cause salicylate intoxication.
- C. Diflunisal is three-to-four fold more potent than aspirin as analgesic.
- D. Diflunisal does not enter the CNS and therefore cannot relieve fever.
- E. Aspirin is a non-selective COX-2 inhibitor.

*An 8-year-old girl has a fever and muscle aches from a presumptive viral infection. Which one of the following drugs would be most appropriate to treat her symptoms?*

- A. Acetaminophen.*
- B. Aspirin.*
- C. Celecoxib.*
- D. Codeine.*
- E. Indomethacin –*

*The pka of lidocaine is 7.9. In infected tissue at pH 6.9, the fraction of ionized form will be:*

- A. 1%.*
- B. 10%.*
- C. 50%.*
- D. 90%.*
- E. 99%.*

*The neuroleptic drug chlorpromazine can produce all of the following effects EXCEPT:*

- A. Constipation.*
- B. Sexual dysfunction.*
- C. Nausea and vomiting.*
- D. Postural hypotension.*
- E. Extrapyramidal symptoms.*

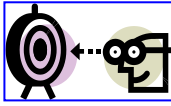

*- A local anesthetic drug that is only used topically (e.g. to mucous membranes) but cannot be given parenterally because of its physicochemical properties is:*

- A. Benzocaine.*
- B. Tetracaine.*
- C. Lidocaine.*
- D. Procaine.*
- E. Bupivacaine*

*An antiparkinsonian drug that is a selective inhibitor of monoamine oxidase type B (MAO-B) is:*

- A. Bromocriptine.*
- B. Carbidopa.*
- C. Tolcapone.*
- D. Pramipexole.*
- E. Selegiline.*

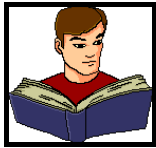


<b>Lecture: 24. Antiparkinson Drugs</b>	<b>1 Lecture</b>
Department : Pharmacology	Student Notes:
Lecturer : <b>Dr Osman (M.sec) Prof Magda (F.sec)</b>	
TEACHING LOCATION	
 <b>Objectives</b> <p>At the end of the lecture you should be able to:</p> <ol style="list-style-type: none"> <li><i>1. Describe the neurochemical imbalance underlying the symptoms of Parkinson's disease</i></li> <li><i>2. Identify the mechanisms by which levodopa, dopamine receptor agonists, MAOI, and muscarinic blocking drugs alleviate parkinsonism.</i></li> <li><i>3. Describe the therapeutic and toxic effects of the major antiparkinsonian drugs</i></li> <li><i>4. Identify the compounds that inhibit dopa decarboxylase and COMT and describe their use in parkinsonism.</i></li> <li><i>5. Identify the chemical agents and drugs that cause parkinsonism symptoms</i></li> </ol>	
 <b>Topics</b> <ol style="list-style-type: none"> <li><i>1. Levodopa, carbidopa combination</i></li> <li><i>2. Bromocriptine, and other dopamine agonists</i></li> <li><i>3. Pramapixole and ropinirole</i></li> <li><i>4. Seligiline, entacapone, tolcapone</i></li> <li><i>5. Trihexphenidyl</i></li> </ol>	<p><i>(Insert here handouts and additional pages for notes if needed)</i></p>

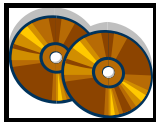
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**Remember**



**Text book**



**CD**



**Independent learning from the Internet**



**Self- Assessment** (the answer to self-assessment exercises will be discussed in tutorial sessions)

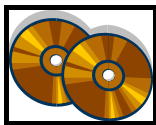
**An antiparkinsonian drug that is a selective inhibitor of monoamine oxidase type B (MAO-B) is:**

- A. Bromocriptine.**
- B. Carbidopa.**
- C. Tolcapone.**
- D. Pramipexole.**
- E. Selegiline.**

<b>Lecture: 25. Antidepressant Drugs</b>	<b>1 Lecture</b>
Department : Pharmacology	Student Notes:
Lecturer <b>Dr Osman (M.sec) Prof Magda (F. sec)</b>	
TEACHING LOCATION :	
<div data-bbox="236 376 408 479" data-label="Image"> </div> <p data-bbox="432 421 596 461"><b>Objectives</b></p> <p data-bbox="285 490 858 521">At the end of the lecture you should be able to</p> <ol data-bbox="285 600 1171 1137" style="list-style-type: none"> <li>1. <i>Describe the probable mechanisms and the major properties of tricyclic antidepressants</i></li> <li>2. <i>List the toxic effects that occur during chronic therapy and with an acute overdose of TCA</i></li> <li>3. <i>Identify the second- and third generation heterocyclic antidepressants and their distinctive properties</i></li> <li>4. <i>Identify the selective serotonin reuptake inhibitors and list their major characteristics</i></li> <li>5. <i>Describe the therapeutic use and toxic effects of MAO inhibitors</i></li> <li>6. <i>Identify the major drug interactions associated with antidepressant drugs</i></li> </ol> <div data-bbox="236 1189 367 1294" data-label="Image"> </div> <p data-bbox="391 1234 480 1265"><b>Topics</b></p> <ol data-bbox="285 1346 1171 1816" style="list-style-type: none"> <li>a. <i>Amine hypothesis of mood; TCA: pharmacodynamic and pharmacokinetic characteristics, clinical uses and adverse effects</i></li> <li>b. <i>Heterocyclic antidepressants: second and third – generation drugs: mechanism of action pharmacokinetics, clinical uses and drug interactions</i></li> <li>c. <i>Selective serotonin reuptake inhibitors (SSRI): mechanism of actions, pharmacokinetics, uses and adverse effects</i></li> <li>d. <i>MAO inhibitors : uses and drug interactions</i></li> </ol>	<p data-bbox="1203 1256 1497 1400"><i>(Insert here handouts and additional pages for notes if needed)</i></p>

**Continued****Remember****Text book**

1. *Lippincott's Pharmacology, 3<sup>rd</sup> Edition, & Basic and Clinical*
2. *Pharmacology, 10<sup>th</sup> Edition, B.G. Katzung*

**CD****Independent learning from the Internet****Self- Assessment** (the answer to self-assessment exercises will be discussed in tutorial sessions)

A 55-year-old male patient was diagnosed with mental depression, for which fluoxetine was prescribed. The patient's condition is improved, however he complained of sexual dysfunction. Which of the following drugs might be useful in this patient?

- A. Amitriptyline (TCA).
- B. Lithium.
- C. Fluvoxamine.
- D. Citalopram.
- E. Mirtazapine.

*When mental depression is accompanied with neuropathic pain,*

**Core**

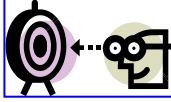

*the patient would use:*

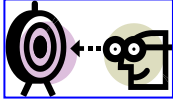

- A. Fluoxetine.**
- B. Sertraline.**
- C. Duloxetine.**
- D. Mirtazapine.**
- E. Phenelzine.**

*Which of the following antidepressants is used to decrease the craving for*

*nicotine in tobacco abusers?*

- A. Mirtazapine.**
- B. Bupropion.**
- C. Nefazodone.**
- D. Trazodone.**
- E. Fluoxetine.**

<b>Practical 1 : Drug forms and routes of drug administration</b>	
<b>Department : Pharmacology</b>	<b>Student Notes:</b>
<b>Lecturer : Dr Saad Mahrous &amp; Dr. Ibrahim</b>	
<b>TEACHING LOCATION :</b> برجاء استكمال أسماء أعضاء هيئة التدريس للطلاب والطالبات	
 <b>Objectives</b> <p>At the end of the lecture you should be able to</p> <p><b><u>Objectives:</u></b></p> <ol style="list-style-type: none"> <li>1- Know different dosage forms, advantage and disadvantages of each form.</li> <li>2- Know different routes of drug administration, advantage and disadvantages of each form.</li> </ol>	
 <b>Topics</b> <p><b><u>contents:</u></b></p> <ol style="list-style-type: none"> <li>1. Different forms of tablets and factors affecting drug absorption</li> <li>2. Syrup, Suspension and Emulsion.</li> <li>3. Difference between ampoule and vial.</li> <li>4. Difference between cream and ointment</li> <li>5. Inhalers</li> <li>6. Skin patches</li> <li>7. Sachets and their solubility</li> <li>8. Advantages &amp; disadvantages of Enteral route (oral, buccal, rectal and sublingual)</li> <li>9. Advantages &amp; disadvantages of Parenteral route (IV, IM, ID, SC.....etc)</li> </ol>	(Insert here handouts and additional pages for notes if needed)

Practical 2 : pharmacokinetics.	
<b>Department : Pharmacology</b>	<b>Student Notes:</b>
<b>Lecturer : <i>Dr . Shaker , Dr. Saad Mahrous &amp; Dr. Ibrahim</i></b>	
<b>TEACHING LOCATION :</b> برجاء استكمال أسماء أعضاء هيئة التدريس : للطلاب والطالبات	
 <b>Objectives</b> At the end of the lecture you should be able to <u><b>Objectives:</b></u> <ol style="list-style-type: none"> <li><b><i>To demonstrate Clinical application of pharmacokinetic principles in design of optimal dosage regimen for drugs with narrow therapeutic range</i></b></li> </ol>	
 <b>Topics</b> <u><b>contents</b></u> <ol style="list-style-type: none"> <li><b><i>Review of basic pharmacokinetic paramters, volume of distribution, clerance, half life, steady state , peak &amp; trough levels.</i></b></li> <li><b><i>Utilize simulation Pharmacokinetic software to demonstrate the following :</i></b> <ul style="list-style-type: none"> <li><b><i>Estimation of steady state peak and trough levels after repeated IV injection ( using genatmicin as model )</i></b></li> <li><b><i>Estimation of steady state level after IV infusion with and without loading dose ( using theophylline as model )</i></b></li> </ul> </li> </ol>	<i>(Insert here handouts and additional pages for notes if needed)</i>

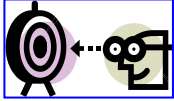
Practical 3 : Effect of autonomic drugs on Rabbit eye.

Department : Pharmacology

Lecturer : **Dr Saad Mahrous & Dr. Ibrahim**

TEACHING LOCATION : برجاء استكمال أسماء أعضاء هيئة التدريس :  
للطلاب والطالبات

Student Notes:



### Objectives

At the end of the lecture you should be able to

#### Objectives:

1. To know the mechanism of different autonomic drugs on the Rabbit eye.
2. To recognize the different changes that can be produced by drops acting on the eye.



### Topics

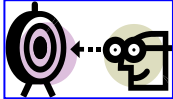

#### Contents:

To demonstrate the following:

1. Effect of myotic drugs:
  - a. Direct (e.g. Pilocarpine).
  - b. Indirect (e.g. Physostigmine).
2. Effect of mydriatic drugs:
  - a. Direct (e.g. Atropine).
  - b. Indirect (e.g. Cocaine).

(Insert here handouts and additional pages for notes if needed)



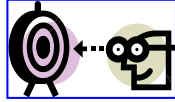
<b>Practical 4 : Prescription writing</b>	
<b>Department : Pharmacology</b>	<b>Student Notes:</b>
<b>Lecturer : <i>Dr Saad Mahrous &amp; Dr. Ibrahim</i></b>	
<b>TEACHING LOCATION :</b> <span style="background-color: yellow;">برجاء استكمال أسماء أعضاء هيئة التدريس للطلاب والطالبات</span>	
 <b>Objectives</b> At the end of the lecture you should be able to <u><b>Objectives:</b></u> <ol style="list-style-type: none"> <li><i>1. - how to prescribe proper medicine and to know the rational steps in writing a prescription and the common prescribing errors</i></li> <li><i>2- Get the knowledge to consider the pathophysiology of the diagnosis selected, select the specific therapeutic objective, and determine the proper dosing regimen</i></li> </ol>	
 <b>Topics</b> <u><b>Contents</b></u> <ol style="list-style-type: none"> <li><i>1. Ordinary prescription</i></li> <li><i>2. Narcotic prescription</i></li> </ol>	<i>(Insert here handouts and additional pages for notes if needed)</i>

**SDL 1 : D-Drug-Drug interactions.**

Department : Pharmacology

Lecturer : **Dr Saad Mahrous & Dr. Ibrahim**TEACHING LOCATION : **برجاء استكمال أسماء أعضاء هيئة التدريس للطلاب والطالبات**

Student Notes:

**Objectives**

At the end of the lecture you should be able to

**Objectives:**

3. *Describe the primary pharmacokinetic mechanisms that underlie drug interactions*
4. *Describe how the pharmacodynamic characteristics of different drugs administered concomitantly may lead to additive, synergistic, or antagonistic effects*
5. *Identify specific drug interactions that occur commonly in clinical practice*

**Topics****contents:**

1. *Pharmacokinetic interactions: Interactions based on absorption, distribution and binding, interactions based on metabolic clearance, interactions based on renal functions.*
2. *Pharmacodynamic interactions: interactions based on opposing actions, interactions based on additive effects*

*(Insert here handouts and additional pages for notes if needed)*