

Pacific University

CommonKnowledge

College of Optometry

Theses, Dissertations and Capstone Projects

4-2004

NBEO pharmacology review guide

Karen K. Chow
Pacific University

Robyn N. Clausen
Pacific University

Recommended Citation

Chow, Karen K. and Clausen, Robyn N., "NBEO pharmacology review guide" (2004). *College of Optometry*. 1471.

<https://commons.pacificu.edu/opt/1471>

This Thesis is brought to you for free and open access by the Theses, Dissertations and Capstone Projects at CommonKnowledge. It has been accepted for inclusion in College of Optometry by an authorized administrator of CommonKnowledge. For more information, please contact CommonKnowledge@pacificu.edu.

NBEO pharmacology review guide

Abstract

This review guide is intended to help optometry students in preparation for the pharmacology section of the National Board Exam (Part I). We have compiled information from various sources into a format that serves as a summary of the key aspects of different drugs. The information provided follows the order of the outline provided by the National Board of Examiners of Optometry, but the specific details are our interpretation of the outline and are not guaranteed to be entirely representative of what will be asked on the examination. We hope having pharmacology information summarized in this format will be helpful to students.

Degree Type

Thesis

Degree Name

Master of Science in Vision Science

Committee Chair

Dennis L. Smith

Subject Categories

Optometry

Copyright and terms of use

If you have downloaded this document directly from the web or from CommonKnowledge, see the "Rights" section on the previous page for the terms of use.

If you have received this document through an interlibrary loan/document delivery service, the following terms of use apply:

Copyright in this work is held by the author(s). You may download or print any portion of this document for personal use only, or for any use that is allowed by fair use (Title 17, §107 U.S.C.). Except for personal or fair use, you or your borrowing library may not reproduce, remix, republish, post, transmit, or distribute this document, or any portion thereof, without the permission of the copyright owner. [Note: If this document is licensed under a Creative Commons license (see "Rights" on the previous page) which allows broader usage rights, your use is governed by the terms of that license.]

Inquiries regarding further use of these materials should be addressed to: CommonKnowledge Rights, Pacific University Library, 2043 College Way, Forest Grove, OR 97116, (503) 352-7209. Email inquiries may be directed to: copyright@pacificu.edu

NBEO PHARMACOLOGY REVIEW GUIDE

By:
Karen K. Chow, B.S.

Robyn N. Clausen, B.S.

**A thesis submitted to the faculty of the
College of Optometry
Pacific University
Forest Grove, Oregon
For the degree of
Doctor of Optometry
April, 2004**

Faculty Advisor: Dennis L. Smith, O.D., M.S., F.A.A.O.

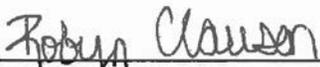
**PACIFIC UNIVERSITY LIBRARY
FOREST GROVE, OREGON**

3 5369 00275 6250

Authors:

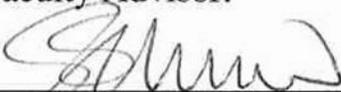


Karen **Chow**



Robyn Clausen

Faculty Advisor:



Dennis **Smith, O.D., M.S., F.A.A.O.**

Biographies:

Karen K. Chow is currently working towards her doctorate of optometry degree at Pacific University College of Optometry and anticipates graduating from the program in May, 2005. Prior to attending Pacific University, Karen earned a Bachelor of Science degree in Molecular Cell Biology at University of Arizona, Tucson.

Robyn N. Clausen is currently **working** towards her doctorate of optometry degree at Pacific University College of Optometry and anticipates graduating from the program in May, 2005. Prior to **attending** Pacific University, Robyn attended South Dakota State University, where she earned a Bachelor of Science degree in Biology.

Abstract:

This review guide is intended to help optometry students in preparation for the pharmacology section of the National Board Exam (Part I). We have compiled information from various sources into a format that serves as a summary of the key aspects of different drugs. The information provided follows the order of the outline provided by the National Board of Examiners of Optometry, but the specific details are our interpretation of the outline and are not guaranteed to be entirely representative of what will be asked on the examination. We hope having pharmacology information summarized in this format will be helpful to students.

Acknowledgements:

We would like to thank Dr. Dennis Smith for his willingness to advise this thesis project. His guidance and expertise have been very valuable.

In addition, we would like to thank Dr. Robert Rosenow, Dr. Nada Lingel, and Dr. Roger Reynolds. We utilized various information presented by them at Pacific University College of Optometry.

CONTENTS

Autonomic and/or neuromuscular junction drugs	1
Autacoid agonists and antagonists	6
Drugs affecting the respiratory system	8
Gastrointestinal agents	10
Chemotherapeutic agents	13
Immunopharmacological agents	25
Anti-inflammatory agents	26
Major drugs acting on the central nervous system	28
General, and local anesthetics	36
Major drugs acting on the endocrine system	39
Major cardiovascular drugs	44
Major drugs acting on the kidneys	50
Resources	52

ADRENERGIC AGONISTS

Mechanism: Increase level of sympathetic activity by directly and/or indirectly stimulating adrenergic receptors.

Direct-acting: Agents that act directly on the postsynaptic adrenergic receptors to produce the same effect as natural stimulation of the receptors

Indirect-acting: Agents that cause an increase in norepinephrine release from presynaptic neurons, and therefore indirectly increase the level of sympathetic activity

Mixed action: Agents that cause an increase in norepinephrine release from presynaptic neurons as well as activating adrenergic receptors of postsynaptic neurons

Specific classes include catecholamines and non-catecholamines →

Catecholamine characteristics:

- Contain a 3,4-dihydroxybenzene group
- Greater potential in alpha and beta receptor activation than non-catecholamines
- Not effective when given orally because of rapid inactivation by catechol-O-methyl transferase (COMT) and monoamine oxidase (MAO)
- Poor CNS penetration due to polar nature

Non-catecholamine characteristics:

- Do not contain a 3,4-dihydroxybenzene group
- Not inactivated by COMT, and less inactivation by MAO than catecholamines, resulting in longer duration of action
- Effective in oral administration

Drugs	Mechanism	Receptors Affected	Indications
Albuterol	Direct-acting	β_2	Bronchospasm
Clonidine	Direct-acting	α_2	Hypertension
Dobutamine*	Direct-acting	β_1	Congestive heart failure
Dopamine*	Direct-acting	β_1	Shock, congestive heart failure
Epinephrine*	Direct-acting	$\alpha_1, \alpha_2, \beta_1, \beta_2$	Acute asthma, open-angle glaucoma, anaphylactic shock
Isoproterenol*	Direct-acting	β_1, β_2	Asthma, cardiac stimulant
Metaproterenol	Direct-acting	β_1, β_2	Bronchospasm
Methoxamine	Direct-acting	α_1	Supraventricular tachycardia
Norepinephrine*	Direct-acting	$\alpha_1, \alpha_2, \beta_1$	Shock
Phenylephrine	Direct-acting	α_1	Nasal decongestant, supraventricular tachycardia
Ritodrine	Direct-acting	β_2	Bronchospasm, premature labor
Terbutaline	Direct-acting	β_2	Bronchospasm, premature labor
Amphetamine	Indirect-acting	$\alpha, \beta, \text{CNS}$	Attention deficit disorder (CNS stimulation)
Ephedrine	Mixed action	$\alpha, \beta, \text{CNS}$	Asthma, nasal decongestant
Metaraminol	Mixed action	$\alpha, \beta, \text{CNS}$	Shock, acute hypotension

*catecholamine

ADRENERGIC ANTAGONISTS

Mechanism: Decrease the amount of sympathetic activity by either binding to and blocking adrenergic receptors or affecting release (and/or re-uptake) of neurotransmitter from the presynaptic neuron

Drugs	Receptors Affected	Indication
Doxazosin	α_1	Hypertension
Phenoxybenzamine	α_1, α_2	Pheochromocytoma, Raynaud's disease, autonomic hyperreflexia
Phentolamine	α_1, α_2	Diagnosis of pheochromocytoma, can trigger arrhythmias (decreased coronary perfusion is a contraindication)
Prazosin	α_1	Hypertension
Terazosin	α_1	Hypertension
Acebutolol	β_1	Hypertension
Atenolol	β_1	Hypertension
Labetalol	$\alpha_1, \beta_1, \beta_2$	Hypertension
Metoprolol	β_1	Hypertension
Pindolol	β_1, β_2	Hypertension
Propranolol	β_1, β_2	Hypertension, glaucoma, migraine, hyperthyroidism, angina pectoris, myocardial infarction
Timolol	β_1, β_2	Glaucoma, hypertension
Guanethidine	Blocks norepinephrine release	Hypertension (rarely used because is commonly causes orthostatic hypotension) *Supersensitivity to norepinephrine caused by its depletion can cause a hypertensive crisis in pheochromocytoma
Reserpine	Prevents the neurotransmitters of presynaptic neuron from entering storage vesicles	Hypertension

CHOLINERGIC AGONISTS

Mechanism: Increase level of parasympathetic activity by directly or indirectly activating cholinergic receptors

Direct-acting: Agents that act directly on the postsynaptic cholinergic receptors to produce the same effect as natural stimulation of the receptors (M = muscarinic receptors, N = nicotinic receptors)

Indirect-acting: Indirectly cause stimulation of cholinergic neurons by inhibiting acetylcholinesterase and thereby prolonging the effectiveness of acetylcholine in the synaptic cleft (cholinesterase inhibitors)

Drugs	Mechanism	Receptors Affected	Indications
Acetylcholine	Direct-acting	M and N	Not used therapeutically because of wide range of action and rapid inactivation by acetylcholinesterase
Bethanechol	Direct-acting	M	Stimulation of atonic bladder in postpartum or postoperative nonobstructive urinary retention
Carbachol	Direct-acting	N	Miosis, lowering intraocular pressure (rarely used)
Pilocarpine	Direct-acting	M	Emergency lowering of intraocular pressure
Edrophonium	Reversible indirect-acting		Diagnosis of myasthenia gravis
Neostigmine	Reversible indirect-acting		Symptomatic treatment of myasthenia gravis, stimulates bladder and GI tract, antidote for tubocurarine
Physostigmine	Reversible indirect-acting		Intestinal and bladder immotility, miosis, intraocular pressure reduction
Pyridostigmine	Reversible indirect-acting		Symptomatic treatment of myasthenia gravis
Isoflurophate	Irreversible indirect-acting		Open-angle glaucoma

CHOLINERGIC ANTAGONISTS/GANGLIONIC BLOCKERS

Cholinergic antagonists are also called cholinergic blockers or anticholinergic drugs

- Mechanism:**
- Antimuscarinic agents compete with acetylcholine for muscarinic receptors. As a result, these agents decrease parasympathetic activity.
 - Ganglionic agents act on nicotinic receptors to block the entire output of the autonomic nervous system. The application of these agents are limited to experimental uses.

Drugs	Mechanism		
Antimuscarinic agents			
Atropine	-Blocks central and peripheral muscarinic receptors	Mydriasis, cycloplegia, reduction of gastrointestinal activity, enuresis, organophosphate poisoning, and generalized reduction of bodily secretions	<ul style="list-style-type: none"> -High dosages: tachycardia -Low dosages: bradycardia -CNS effects: restlessness, confusion, hallucinations, delirium -Others: dry mouth, blurred vision, constipation
Scopolamine	-More potent and longer acting on the CNS than atropine	Motion sickness (Amnesia and sedation with concurrent use of morphine)	-Similar to atropine
Ipratropium	-Similar to atropine but slower onset	Chronic obstructive pulmonary disease	No known side effects
Ganglionic blockers			
Nicotine	<ul style="list-style-type: none"> -Low dosages: ganglionic stimulation (parasympathetic-like effects) -High dosages: ganglionic inhibition (sympathetic-like effects) 		-Many including CNS disturbances
Trimethaphan	-Competitive, short-acting ganglionic agent	Emergency use for lowering blood pressure (in pulmonary edema or dissecting aortic aneurysm)	
Mecamylamine	-Competitive ganglionic agent		

VEUROMUSCULAR TRANSMISSION AGONISTS AND ANTAGONISTS

Category	Drugs	Mechanism	Indications	Precautions/Side Effects
Neuromuscular transmission agonists	-Succinylcholine	-Causes depolarization at neuromuscular junction (nicotinic receptors) -Initially, there is a transient twitching of the muscles as the sodium channels open. This continuous depolarization slows and even prevents repolarization resulting in flaccid paralysis.	Rapid endotracheal intubation at the start of anesthesia administration	-Hyperthermia -Apnea
Neuromuscular transmission antagonists	-Mivacurium -Atracurium -Tubocurarine -Metocurine -Doxacurium -Rocuronium -Vecuronium -Pipercuronium -Pancuronium -Succinylcholine	-Low doses: combine with nicotinic receptor to prevent ACh from binding (reversible) → prevent depolarization of the muscle cell membrane and inhibit muscular contraction -High doses: block ion channels of motor end-plates on muscle cells	Skeletal muscle relaxing during surgery	-Muscle pain following surgery -Increased intraocular pressure -Hyperkalemia -Malignant hyperthermia

AUTACOID AGONISTS

Autacoids are chemicals formed by a tissue that act on that same tissue (function as local hormones).

Naturally occurring autacoids include prostaglandins, histamine, and serotonin

Examples of prostaglandins used therapeutically:

Drugs	Indication
Dinoprost	Abortion
Carboprost	Abortion
Dionoprostone	Abortion
Misoprostol (in combination with Methotrexate)	Abortion
Misoprostol	Inhibiting secretion of HCl in peptic ulcers
Alprostadil	Male impotence

AUTACOID ANTAGONISTS

Category	Drugs	Mechanism	Indications	Precautions/Side Effects
H1-Histamine receptor blockers	First generation agents -Alkylamines -Ethanolamines, -Ethylenediamines, -Phenothiazines, -Piperazines, -Piperidines Second generation agents -Fexofenadine -Astemazole -Loratadine -Cetirizine	Block histamine release at receptor site	-Drugs of choice for allergic rhinitis and urticaria -Motion sickness -Insomnia	-Acute poisoning in young children common in high doses -Sedation/fatigue -Tinnitus -Dizziness -Blurred vision, -Dry mouth -Frequent urination -Possible cardiac arrhythmias with concurrent use of macrolides -Drug interaction with alcohol, monoamine oxidase inhibitors
H2-Histamine receptor blockers	-Cimetidine -Ranitidine -Famotidine -Nizatidine	Prevent stimulation of H ₂ receptors in stomach → decrease gastric acid secretion	-Peptic ulcers -Zollinger-Ellison syndrome -Acute stress ulcers -Gastroesophageal reflux disease	-CNS and GI disturbances -Inhibition of P450 system

BRONCHODILATORS

Goal: To achieve bronchodilation by sympathetic activation or parasympathetic inhibition

Drugs	Mechanism	Location of Response	Precautions/Side Effects	Indications
Epinephrine	β_2 stimulation	Bronchial smooth muscle	CNS disturbances, hemorrhage, cardiac arrhythmias, pulmonary edema	-Allergic or histamine induced bronchoconstriction -Anaphylaxis
Isoproterenol	β_2 stimulation	Bronchial smooth muscle	Similar to epinephrine	-Acute asthma attacks
Metaproterenol	β_2 -selective	Bronchial smooth muscle	Similar to epinephrine	-Asthma -Rebound bronchospasm
Terbutaline	β_2 -selective (short acting)	Bronchial smooth muscle	Similar to epinephrine	-Bronchodilator
Albuterol	β_2 -selective	Bronchial smooth muscle	Similar to epinephrine	-Bronchospasm
Ipratropium bromide	Competitively inhibits action of ACh at the muscarinic receptors in airway	Blocks vagus input to lungs	Cough, drying of oropharynx, headache, dizziness, upper respiratory infections, bronchitis, blurred vision, hypersensitivity	-Use for patients with intolerance to adrenergic agonists
Theophylline	Unknown	Bronchial smooth muscle	CNS, GI, and GU disturbances, hyperglycemia, hypokalemia	-Long-term control asthma treatment

MAST CELL STABILIZERS AND MUCOLYTIC AGENTS

Mast cell stabilizers are used to prevent degranulation of mast cells in order to prevent inflammation of the respiratory airways. Because of their high safety profile, mast cell stabilizers are often considered a safe drug choice for children and pregnant women.

Mucolytic agents are used to liquefy or reduce viscosity of respiratory secretions.

Drugs	Mechanism	Indications	Precautions/Side Effects
Mast cell stabilizers			
Cromolyn, Nedocromil	-Block early phase pulmonary inflammation through inhibition of chloride channels of mast cells -Block late phase pulmonary inflammation through inhibition of chloride channels of eosinophils	Asthma	-Nausea -Headache -Unpleasant taste -Cough* -Wheeze* -Tightness of chest* *Side effects can be prevented if patient uses a β_2 agonist
Mucolytic agents			
Dornase Alfa (DNase) (Pulmozyme)	-Cleaves DNA strands in the respiratory secretions (the viscosity of the secretions is due to DNA from lysed inflammatory cells)	Cystic fibrosis	-Voice changes -Sore throat -Skin rash -Conjunctivitis
N-Acetylcysteine (Mucomyst)	-Cleaves protein complexes in mucus	Chronic bronchopulmonary disease	-Wheeze -Difficulty breathing -Clammy skin -Skin rash -Fever

GASTROINTESTINAL AGENTS – TREATING PEPTIC ULCER DISEASE

Category	Drugs	Mechanism	Indications	Precautions/Side Effects
Antimicrobial agents	-Amoxicillin -Bismuth compounds -Clarithromycin -Metronidazole -Tetracycline	Control <i>Helicobacter pylori</i> growth	-Ulcers caused by <i>Helicobacter pylori</i>	-Depend on specific antimicrobial agent
H ₂ – Histamine receptor blockers	-Cimetidine -Ranitidine -Famotidine -Nizatidine	Prevent stimulation of H ₂ receptors in stomach → decrease gastric acid secretion	-Peptic ulcers -Zollinger-Ellison syndrome -Acute stress ulcers -Gastroesophageal reflux disease	-CNS and GI disturbances -Inhibition of P450 system
Prostaglandins	-Misoprostol	Inhibit HCl secretion and promote mucous and bicarbonate secretion (cytoprotective effect)	-Prevention of NSAID induced gastric ulcers	-Not for use during pregnancy (stimulate uterine contractions)
Proton pump inhibitors	-Omeprazole -Lansoprazole	Irreversibly inhibit enzymes that stimulate HCl secretion	-Peptic and gastric ulcers -Zollinger-Ellison syndrome -Erosive esophagitis	-CNS disturbances -Potentially fatal toxic epidermal necrolysis with omeprazole use (but rare) -Reduced acid levels in stomach (achlorhydria)
Antimuscarinics	-Hyoscyamine -Mepenzolate -Pirenzepine	Prevent parasympathetic stimulation of acid production in stomach	-Peptic ulcers -Zollinger-Ellison syndrome	-Effects of suppressed parasympathetic activity
Antacids	-Aluminum hydroxide -Calcium carbonate -Magnesium hydroxide -Sodium bicarbonate	Neutralize excess acid in stomach	-Peptic ulcers	-GI disturbances -Caution in patients with congestive heart failure or hypertension (affects sodium levels)
Mucosal Protective Agents	-Sucralfate -Colloidal bismuth	Promote mucous secretion in stomach	-Gastric ulcers -Reducing mucosal inflammation and preventing injury	-Very well tolerated due to poor absorption

GASTROINTESTINAL AGENTS – ANTIEMETICS

These agents are used to control nausea and vomiting resulting from chemotherapy

Class	Drugs	Mechanism	Precautions/Side Effects
Phenothiazines	-Prochlorperazine	Block dopamine receptors	-Hypotension -Restlessness -Extrapyramidal effects -Sedation
Substituted benzamides	-Metoclopramide	Prevent vomiting by unknown mechanism	-Sedation -Diarrhea -Extrapyramidal effects
Butyrophenones	-Domperidone -Droperidol -Haloperidol	Block dopamine receptors	-Similar to phenothiazines
Benzodiazepines	-Alprazolam -Lorazepam	Enhance GABA in CNS	-Sedation -Anxiolytic effects -Amnesia
Corticosteroids	-Dexamethasone -Methylprednisolone	Prevent vomiting by unknown mechanism, possibly related to prostaglandin inhibition	-Not for use in immunocompromised -Cushing's syndrome -Increased intraocular pressure
Cannabinoids	-Dronabinol -Nabilone	Prevent vomiting by unknown mechanism	-CNS disturbances
5-HT ₃ serotonin receptor blockers	-Granisetron -Ondansetron	Selectively inhibit activation of 5-HT ₃ receptors	-Headaches

GASTROINTESTINAL AGENTS – ANTIDIARRHEALS AND LAXATIVES

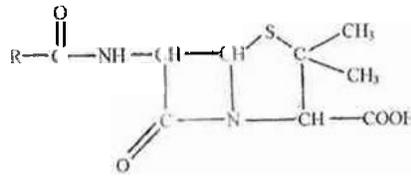
Category	Drugs	Mechanism	Precautions/Side Effects
Antidiarrheals			
Antimotility agents	-Diphenoxylate -Loperamide	-Inhibit acetylcholine release → decrease peristalsis	-CNS and GI disturbances -Toxic megacolon -Not for use in young children -Not for use in patients with severe colitis
Adsorbents	-Kaolin -Pectin -Methylcellulose -Activated attapulgate -Magnesium aluminum silicate	-Adsorb intestinal toxins or intestinal microorganisms	-Interfere with absorption of other drugs
Fluid and electrolyte transport modifiers	-Aspirin -Indomethacin -Bismuth subsalicylate	-Inhibit prostaglandin synthesis -Reduce fluid secretions in bowel	-Effects of reduced prostaglandin levels
Laxatives			
Irritants and stimulants	-Castor oil -Cascara (contains emodin) -Senna (contains emodin) -Aloe (contains emodin) -Pheolphthalein -Bisacodyl	-Increase gut motility	-Abdominal cramps -Atonic colon with prolonged use -Emodin can enter breast milk
Bulking agents		-Increase motility when indigestible material interacts with water and prevents water absorption	-GI disturbances

ANTIBIOTICS: PENICILLINS

Category: Bactericidal beta-lactam antibiotic

Mechanism: prevents bacterial cell wall synthesis by inhibiting peptide bond formation in peptidoglycan

Penicillins all have the same general structure containing a beta-lactam ring (shown in red), but have varying side chains (shown in blue).



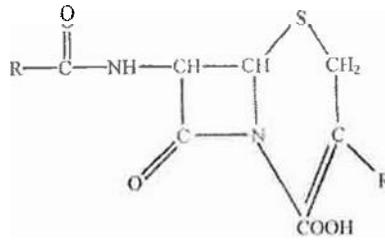
Category	Drugs	Spectrum of Activity	β -Lactamase Resistance	Precautions/Side Effects
Natural Penicillins				
Penicillin G	Penicillin G	-Gram positive (like Staph. and <i>Strep.</i>) -Anaerobic bacterial (above the diaphragm) -Some Gram negative (like N. gonorrhoeae, N. meningitides, and <i>Shigella</i>)	No	-Not acid stable (must be injected) -Cross hypersensitivity with cephalosporins -Jarisch-Herxheimer reaction -Hypernatremia -Hyperkalemia -Interactions with probenecid, parenteral aminoglycosides, anticoagulants, and tetracyclines
Penicillin V	Penicillin V	-Similar to penicillin G	No	-Similar to penicillin G (but is acid stable) -Pseudomembranous colitis -Interstitial nephritis -Interacts with oral contraceptives, probenecid, and tetracyclines
Semi-Synthetic Penicillins				
Aminopenicillins	-Amoxicillin -Ampicillin -Bacampicillin	-Gram positive -Some Gram negative (better than penicillin G) -Limited anaerobes	If combined with clavulanate or sulbactam (to tie up enzyme)	-Similar to penicillin G (but is acid stable)
Extended Spectrum Penicillins	-Carbenicillin -Mezlocillin -Piperacillin -Ticarcillin	-Similar to aminopenicillins	If combined with tazobactam or clavulanate (to tie up enzyme)	-Similar to penicillin G (but is acid stable)
Penicillinase-Resistant Penicillins	-Cloxacillin -Dicloxacillin, -Nafcillin, -Methicillin, -Oxacillin	-Gram positive only	Yes	-Similar to penicillin G (but is acid stable)

ANTIBIOTICS: CEPHALOSPORINS

bactericidal beta-lactam antibiotics

Mechanism: Prevent bacterial cell wall synthesis by inhibiting peptide bond formation in peptidoglycan

Cephalosporins all have the same general structure containing a beta-lactam ring (shown in red), but have varying side chains (shown in blue).



Generation	Drugs	Spectrum of Activity	β -Lactamase Resistance	CNS Distribution	Precautions/Side Effects
1 st	-Cephalexin -Cefadroxil -Cephadrine -Cephalothin	Gram positive > Gram negative	No	Poor	-Nephrotoxicity -Cross hypersensitivity with penicillin -Possible alteration of intestinal flora -Interacts with probenecid, aminoglycoside, ethanol (disulfiram-like reaction), and anticoagulants
2 nd	-Cefaclor -Cefamandole -Cefonicid -Cefoxitin -Cefmetazole -Cefprozil	Slightly more Gram negative activity than 1 st generation, at the expense of Gram positive activity	More than 1 st generation	Moderate	
3 rd	-Cefixime -Cefpodoxime -Ceftibuten -Cefepime	Gram negative > Gram positive	Yes	Good	

ANTIBIOTICS: MACROLIDES

Bacteriostatic antibiotics

Mechanism: Prevent bacterial protein synthesis by binding to 50s subunit of ribosome

Drugs	Spectrum of Activity	Indications	Precautions/Side Effects
Erythromycin	-Gram positive -Some Gram negative	-Upper respiratory infections -Pneumonia -Cellulitis -Prophylactic against bacterial endocarditis - <i>Chlamydia</i> - <i>Legionella</i> - <i>Diphtheria</i> - <i>Mycoplasma</i>	-Hypersensitivity -Pregnancy -Caution with liver problems -GI disturbances -Liver toxicity -Ototoxicity -Inhibits cytochrome P450 enzymes -Interacts with carbamazepine, theophylline, and warfarin
Azithromycin	-Less active against Gram positive -More active against Gram negative (compared to erythromycin)	- <i>Moraxella</i> - <i>Haemophilus</i> - <i>Legionella</i> - <i>Neisseria</i> - <i>Chlamydia trachomatis</i> , -most effective macrolide against <i>Toxoplasma gondii</i>	-Caution with liver problems -Not for use with renal dysfunction -Mild GI disturbances -Headache and dizziness -Rare cholestatic jaundice (no effect on P450 enzymes)
Clarithromycin	-Even more effective than erythromycin against Gram positive	- <i>Helicobacter pylori</i> -Gram positive bacteria in general	-Pseudomembranous colitis -Not approved for children < 6 months -Not for use during pregnancy or nursing -Impairment to liver and kidneys -Mild GI disturbances -Headaches -Rash (less inhibition of P450 than erythromycin)

ANTIBIOTICS: SULFONAMIDES

Bacteriostatic antibiotics

Mechanism: Halt bacterial growth by blocking folic acid synthesis (structurally similar to PABA – para-aminobenzoic acid)

Spectrum of Activity: Broad

Drugs	Indications	Additional Comments	Precautions/Side Effects
Sulfamethoxazole and Sulisoxazole	-Urinary tract infections -Otitis media - <i>Chlamydia</i> - <i>Pneumocystis carinii</i> -Methicillin resistant <i>S. aureus</i> - <i>Shigella</i> - <i>Salmonella</i>	Occasionally used in combination with Phenazopyridine or Trimethoprim	-Cross-sensitivity with carbonic anhydrase inhibitors, thiazide diuretics, and sulfonylureas -Fever -Skin rashes -Photosensitivity -Gastrointestinal disturbances -Stevens-Johnson syndrome
Sulfadiazine	- <i>Toxoplasma gondii</i>	Occasionally combined with Pyrimethamine	-Crystalluria -Hematuria
Sulfadoxine	-Malaria	Occasionally combined with Pyrimethamine	-Hemolytic anemia -Aplastic anemia
Sulfasalazine	-Ulcerative colitis	Not absorbable	
Sodium sulfacetamide		Used topically in ophthalmic preparations; associated with lots of resistance	

ANTIBIOTICS: AMINOGLYCOSIDES, CHLORAMPHENICOL, GLYCOPEPTIDES, AND LINCOSAMINES

Category	Drugs	Mechanism	Spectrum of Activity	Precautions/Side Effects
Aminoglycosides	-Streptomycin -Neomycin -Kanamycin -Amikacin -Gentamycin -Tobramycin -Sisomicin -Netilmicin	-Bacteriocidal -Bind to 30s ribosomal subunit of bacteria	-Primarily Gram negative enteric bacteria *Spectrum enhancement → combined with penicillins to enhance spectrum; the penicillin weakens the wall, making the aminoglycoside more effective	-Nephrotoxicity -Ototoxicity -Narrow therapeutic index -Not for use during pregnancy -Myasthenia gravis -Hypocalcemia -Interact with neuromuscular blocking agents (respiratory paralysis)
Chloramphenicol	-Chloramphenicol	-Bacteriostatic -Binds to 50s ribosomal subunit of bacteria	-Broad spectrum	-Aplastic anemia -Hypoplastic anemia -Thrombocytopenia -Liver and kidney impairment -GI and CNS disturbances -Not for use in infants (gray baby syndrome) -Not for use during pregnancy or nursing
Glycopeptides	-Vancomycin	-Bacteriocidal -Bind to peptide side chain of N-acetylmuramic acid (precursors to bacterial cell walls)	-Gram positive bacteria	-Hypersensitivity -Adjust dosage for kidney and liver patients -Flushing, hypotension and tachycardia with IV administration -Nephrotoxicity -Ototoxicity
Lincosamines	-Clindamycin -Lincomycin	-Bacteriostatic -Bind to 50s ribosomal subunit of bacteria	-Gram positive bacteria -Most anaerobes - <i>Enterococci</i> - <i>Clostridium</i>	-Not for use during pregnancy -GI disturbances -Pseudomembranous colitis -Stevens-Johnson syndrome

ANTIBIOTICS: POLYMXINS, QUINOLONES, TETRACYCLINES, AND TRIMETHOPRIM

Category	Drugs	Mechanism	Spectrum of Activity	Precautions/Side Effects
Polymixins	-Polymixin B -Polymixin E	-Bactericidal -Disrupt cell membranes	-Gram negative bacteria	-Nephrotoxicity -Neurotoxicity
Quinolones	-Ciprofloxacin -Enoxacin -Lomefloxacin -Norfloxacin -Ofloxacin -Cinoxacin -Sparfloxacin -Grepafloxacin	-Bactericidal -Block DNA replication by interfering with DNA gyrase	-Broad spectrum	-Fatal anaphylactic reactions -GI and CNS disturbances -Increased intracranial pressure -Photosensitivity -Cataracts -Tendon and joint ruptures
Tetracyclines	Low-lipid-solubility -Oxytetracycline Intermediate-lipid-solubility -Tetracycline -Demeclocycline High-lipid-solubility -Doxycycline -Minocycline	-Bacteriostatic -Prevent bacterial protein synthesis by binding to 30s ribosomal subunit	-Broad spectrum	-Renal function impairment -Phototoxicity -Not for use in children under 8 years -Not for use during pregnancy -Pseudotumor cerebri -Sulfite sensitivity -Toxicity occurs in outdated products -Interact with antacids, dairy products, iron preparations, anticoagulants, cimetidine, digoxin, insulin, penicillin, and oral contraceptives
Trimethoprim	-Trimethoprim	-Bacteriostatic -Inhibits dihydrofolic acid reductase, which inhibits production of purines	-Broad spectrum	-Glucose-6-phosphate dehydrogenase (G-6-PD) deficiency -Impaired liver function -Impaired kidney function -Megaloblastic anemia -Leukopenia and granulocytopenia -Not for use during pregnancy -Not for use in infants -Interacts with methotrexate, sulfonyleureas, warfarin, mercaptopurine

ANTIFUNGALS

Drugs	Mechanism	Spectrum of Activity/Indications	Resistance	Precautions/Side Effects
Amphotericin B (Polyene)	Disrupts membrane function by binding to ergosterol	- Fungistatic/-cidal - <i>Candida</i> - <i>Histoplasma</i> - <i>Cryptococcus</i> - <i>Coccidioides</i> - <i>Aspergillus</i> - <i>Blastomyces</i>	-Infrequently -Associated with decreased ergosterol content	-Fever -Chills -Renal impairment -Hypotension -Anemia -Neurological effects -Thrombophlebitis
Flucytosine	Disrupts DNA synthesis via cytosine-specific permease	-Fungistatic -Chromblastomycosis -Candidiasis -Cryptococcosis	Results with: -Decreased levels of enzymes needed for conversion of 5-flucytosine to 5-fluorouracil -Increased cytosine synthesis	-Hematologic toxicity -Hepatic dysfunction -GI disturbances
Ketoconazole (Imidazole)	Blocks demethylation of lanosterol (cytochrome P450 enzyme)	-Fungistatic/-cidal -Similar to amphotericin B -Histoplasmosis -Nonmeningeal coccidiomycosis and blastomycosis -Various dermatophytic infections (especially Griseofulvin-resistant strains)	-None documented	-GI disturbances -Endocrine effects -Hepatic dysfunctions
Fluconazole (Imidazole)	Similar to ketoconazole	- <i>Cryptococcus neoformans</i> -Candidemia -Coccidioidomycosis -Blastomycosis -Candidiasis -Histoplasmosis	-HIV-positive patients	-GI disturbances -Rash -Hepatitis (rare) -Not for use in pregnancy (teratogenic)
Griseofulvin	Inhibits fungal mitosis (energy-dependent process)	-Fungistatic -Dermatophytes only (<i>Trichophyton</i> , <i>Microsporum</i> , <i>Epidermophyton</i>)	-Due to lack of energy-dependent uptake system	-Hypersensitivity -Hepatotoxicity
Nystatin (Polyene antibody)	Similar to amphotericin B	- <i>Candida</i>		-Rare (topical use only)

ANTIPARASITIC DRUGS

- Mechanism:**
- The mechanism of many of these agents is not completely understood.
 - Most of the protozoal agents are presumed to interfere with biosynthetic pathways of the parasite.
 - Most of the helminthic agents are presumed to interfere with the neuromuscular function of the worms.

Indications	Drugs
-Intestinal protozoa	-Iodoquinol -Nitroimidazoles (metronidazole) -Quinacrine
-Toxoplasmosis	-Pyrimethamine ("classic triple therapy" containing pyrimethamine, sulfadiazine, and corticosteroids)
-Malaria	-Folate antagonists (pyrimethamine, sulfanamide) -Quinolones (chloroquine, mefloquine, primaquine, quinine)
-Trypanosomes -Leishmaniasis	-Eflornithine -Heavy metals (melarsoprol, sodium stibogluconate, meglumine antimonite) -Nitrofurimox
-Intestinal -Tissue helminths	-Avermectins (ivermectin) -Benzimidazoles (mebendazole, thiabendazole, albendazole) -Phenols (niclosamide) -Piperazines (peperazine, diethylcarbamazine) -Pyrazinoisoquinolines (praziquantel) -Tetrahydropyrimidines (pyrantel pamoate, oxantel)

ANTIVIRAL AGENTS

Category	Drugs	Mechanism	Precautions/Side Effects
Drugs against respiratory viral infections	-Amantadine -Ribavirin -Rimantadine	Mechanism unclear (possibly prevent viral entry into cells)	-CNS disturbances -Use with caution during pregnancy and nursing -Transient anemia with ribavirin
Drugs against herpes and cytomegalovirus infections	-Acyclovir -Cidofovir -Famciclovir -Foscarnet -Ganciclovir -Penciclovir -Trifluridine -Vidarabine	Inhibit viral DNA synthesis	-Headaches -GI disturbances -Use with caution during pregnancy and nursing -Renal precipitation in dehydrated patients
Drugs against retroviral infections (human immunodeficiencyvirus)			
Reverse transcriptase inhibitors (nucleoside analogs)	-Zidovudine -Didanosine -Lamivudine -Stavudine -Zalcitabine	Halt DNA replication by incorporating into growing viral DNA chain	-Anemia -Leukopenia -Headaches -Seizures -Pancreatitis -Peripheral neuropathy -GI & CNS disturbances
Protease inhibitors	-Indinavir -Ritonavir -Saquinavir	Inhibit HIV-1 protease → produce nonfunctional virus particles	-P450 inhibition -Hyperglycemia -Hyperbilirubinemia -Kidney stones

ANTINEOPLASTIC AGENTS

Category	Drugs	Indications	Precautions/Side Effects
Antimetabolites These agents interfere with availability of nucleotides → inhibit DNA/RNA synthesis	-Methotrexate	-Acute lymphocytic leukemia -Choriocarcinoma -Burkitt's lymphoma -Breast cancer -Head/neck carcinomas	-Hypersensitivity reactions -GI disturbances -Kidney and liver dysfunction -Pulmonary toxicity -Neurologic toxicity -Not for use during pregnancy
	-6-Mercaptopurine	-Maintenance of remission in acute lymphoblastic leukemia	-Bone marrow suppression -Liver dysfunction
	-6-Thioguanine	-Acute nonlymphocytic leukemia	-Bone marrow suppression -Liver damage
	-5-Huorouracil	-Slowly growing, solid tumors	-GI disturbances -Alopecia -Ulceration of mucous membranes -Bone marrow suppression
	-Cytarabine	-Acute myelogenous leukemia	-GI disturbances -Granulocytopenia -Liver dysfunction -CNS disturbances with high doses
	-Fludarabine	-Chronic lymphocytic leukemia -Hairy cell leukemia	-GI disturbances -Fever -Edema -Myelosuppression
Alkylating agents These agents alkylate (covalently bond to) DNA	-Mechlorethamine	-Hodgkin's disease	-GI disturbances -Bone marrow suppression
	-Cyclophosphamide -Ifosfamide	-Burkitt's lymphoma -Breast cancer -Nephrotic syndrome -Rheumatoid arthritis	-GI disturbances -Alopecia -Bone marrow suppression -Neurotoxicity -Fibrosis of bladder -Amenorrhea -Testicular atrophy and sterility
	-Nitrosureas	-Brain tumors (able to enter CNS)	-Aplastic anemia -Renal toxicity -Pulmonary fibrosis

ANTINEOPLASTIC AGENTS continued

Category	Drugs	Indications	Precautions/Side Effects
Antibiotics These agents interact with and disrupt the function of DNA	-Dactinomycin	-Wilm's tumor -Gestational choriocarcinoma -Soft-tissue carcinomas	-GI disturbances -Bone marrow suppression -Stomatitis -Alopecia
	-Doxorubicin	-Sarcomas -Carcinomas -Lymphocytic leukemia -Lymphomas	-Similar to dactinomycin -Irreversible cardiotoxicity
	-Daunorubicin	-Acute lymphocytic leukemia -Acute myelocytic leukemia	-Similar to doxorubicin
	-Bleomycin	-Testicular tumors -Squamous cell carcinomas -Lymphomas	-Pulmonary toxicity -Hyperpigmentation of skin -Fever and chills -Mucocutaneous reactions -Alopecia
Microtubule inhibitors These agents bind to tubulin → block metaphase of mitosis	-Vincristine	-Acute lymphoblastic leukemia in children -Wilm's tumor -Ewing's soft-tissue sarcoma -Hodgkin's lymphoma -Non-Hodgkin's lymphoma	-GI disturbances -Alopecia -Peripheral neuropathy
	-Vinblastine	-Metastatic testicular carcinoma -Hodgkin's lymphoma -Non-Hodgkin's lymphoma	-GI disturbances -Alopecia -Bone marrow suppression
	-Paclitaxel	-Advanced ovarian cancer -Metastatic breast cancer -Small-cell lung cancer -Squamous cell carcinoma	-Alopecia -Hypersensitivity -Neutropenia -Peripheral neuropathy -Bradycardia

ANTINEOPLASTIC AGENTS continued

Category	Drugs	Indications	Precautions/Side Effects
Steroid hormone analogs These agents act as hormone agonists to treat hormone-responsive tumors	-Prednisone	-Acute lymphocytic leukemia -Hodgkin's lymphoma -Non-Hodgkin's lymphoma	-Increased intraocular pressure -Osteoporosis -Immunosuppression -Increased appetite -Hypertension -Edema -Peptic ulcers
	-Ethinyl estradiol (estrogen)	-Prostate cancer	-Thromboemboli -Myocardial infarction -Stroke -Hypercalcemia -Dysmenorrhea -Gynecomastia
	-Leuprolide (gonadotropin-releasing hormone analog)	-Prostate cancer	-Impotence -Hot flashes -Tumor flare
Steroid hormone antagonists These agents cause tumor regression in hormone-dependent tumors	-Toremoxifen (estrogen antagonist)	-Estrogen-dependent breast cancer	-Hot flashes -GI disturbances -Rash -Vaginal bleeding -Hypercalcemia -Endometrial cancer
	-Flutamide (androgen antagonist)	-Prostate cancer	-Gynecomastia -GI disturbances
	-Aminoglutethimide (estrogen antagonist)	-Metastatic breast cancer	-CNS depression -Rash

IMMUNOSUPPRESSANTS

Category	Drugs	Mechanism	Indications	Precautions/Side Effects
Antibodies	-Antilymphocyte -Antithymocyte globulin -Muromonab-CD3 (OKT3)	As monoclonal antibodies to CD3 antigens, they block the action of cytotoxic T cells	-Acute phase of allograft rejection	-Rash -Fever and chills -Thrombocytopenia -Leukopenia -Infections -CNS disturbances
Non-selective immunosuppressants	-Azathioprine -Mycophenolate mofetil	Interfere with nucleic acid metabolism, which is required for lymphocyte proliferation	-Rheumatoid arthritis -Systemic lupus erythematosus	-Bone marrow suppression -Infections -Liver toxicity -Lymphoma
Selective immunosuppressants	-Cyclosporine -Tacrolimus (a macrolide antibiotic)	Inhibit production of factors needed for T cell growth	-Uveitis -Type 1 diabetes mellitus -Asthma -Grave's disease -Rheumatoid arthritis -Psoriasis -Corneal transplants -Transplants	-Nephrotoxicity -Liver toxicity -Alteration in lipid levels -Hyperglycemia -Hirsutism -Lymphoma

STEROIDAL ANTI-INFLAMMATORY AGENTS

Corticosteroids prevent the formation of arachidonic acid metabolites. Both the lipoxygenase pathway (which produces leukotrienes) and the cyclooxygenase pathway (which produces thromboxane A_2 and prostaglandins) are blocked.

Drugs	Mechanism	Indications	Precautions/Side effects
Short/medium-acting -Cortisol -Cortisone -Prednisone -Prednisolone -Methylprednisolone	Promotes gene transcription after binding to intracellular receptors	-Addison's disease	-Decreased bone mass -Interference with wound healing -Hypertension -Edema -Peptic ulcers -CNS disturbances -Acute adrenal insufficiency syndrome with abrupt drug removal
Intermediate-acting -Triamcinolone -Fluprednisolone		-Adrenocorticotrophic hormone (ACTH) insufficiency -Corticotropin-releasing factor (CRF) insufficiency -Hypothalamic-pituitary adrenal (HPA) insufficiency -Determination of Cushing's syndrome -Congenital adrenal hyperplasia (CAH)	
Long-acting -Bethamethasone -Dexamethasone -Paramethasone		-Inflammation -Allergies -Appetite stimulation in patients who have undergone chemotherapy	

NON-STEROIDAL ANTI-INFLAMMATORY AGENTS (NSAIDs)

NSAIDs are a group of drugs that have anti-inflammatory, antipyretic, and analgesic effects.

By inhibiting the cyclooxygenase pathway they prevent the formation of prostaglandins and thromboxane A₂.

Category	Drugs	Mechanism	Precautions/Side Effects
Salicylic acids	-Aspirin -Diflunisal -Olsalazine -Sodium Salicylate -Choline magnesium salicylate -Magnesium salicylate	Irreversibly acetylate (and inactivate) cyclooxygenase	-Increased alveolar ventilation at therapeutic doses -Hyperventilation and respiratory alkalosis with higher doses -Respiratory paralysis and respiratory acidosis at toxic doses -Gastric ulcers -GI disturbances -Hemorrhage/prolonged bleeding time -Sodium and water retention -Hyperkalemia -Reye's syndrome with aspirin
Indoleacetic acids	-Indomethacin -Sulindac -Tometin -Diclofenac -Etorolac -Nabumetone -Ketorolac	Reversibly inhibit cyclooxygenase	-GI and CNS disturbances -Acute pancreatitis -Neutropenia -Thrombocytopenia -Aplastic anemia -Hypersensitivity
Propionic acids	-Ibuprofen -Naproxen -Fenoprofen -Ketoprofen -Flubiprofen -Oxaprozin	Reversibly inhibit cyclooxygenase	-GI and CNS disturbances
Oxicam derivatives	-Piroxicam	Mechanism not completely understood	-GI disturbances

NEUROTRANSMITTERS OF THE CENTRAL NERVOUS SYSTEM

Name	Neurotransmitter Class	Location of Production	Function
Acetylcholine (ACh)	Acetylcholine	Widespread	-Widespread -Stimulates release of GABA
Norepinephrine (NE)	Biogenic amine (catecholamine subgroup)	Locus ceruleus (a pigmented area in the pons)	-Both excitatory and inhibitory functions -May facilitate wakefulness/attention -May facilitate active (REM) sleep -Decreased levels associated with depression
Glutamate	Amino acid	Afferent neurons	-The major excitatory neurotransmitter in the brain
Dopamine	Biogenic amine (catecholamine subgroup)	Midbrain substantia nigra cells	-Inhibits release of GABA
Gamma-amino butyric acid (GABA)	Amino acid	Widespread	-Inhibitory effects
Glycine	Amino acid	Spinal cord	-Spinal cord inhibitory interneurons
Serotonin	Biogenic amine	Cells of the median raphe of brainstem	-Decreased levels associated with depression

OPIOID AND NON-OPIOID ANALGESICS

Mechanism:

Opioids

Act on μ , κ , and σ receptors (mainly on μ receptors)

Inhibit nerve firing and presynaptic neurotransmitter release

Decrease pain by inhibiting substance P with its attachment to κ receptors

Nonopioids

-Bind to opioid receptors

-Inhibit re-uptake of serotonin and norepinephrine centrally

Drugs	Receptors Affected	Indications	Precautions/Side Effects	Additional Comments
Opioids				
Morphine	- μ , κ , and σ (full agonist)	-Severe pain (including myocardial infarction)	-Dysphoria -Allergy-induced hypotensive effects -Elevated intracranial pressure -Dependence -Sedation -Severe respiratory depression -GI and GU disturbances	-Relieves pain by raising the pain threshold at spinal cord level -Alters brain's perception of pain
Codeine	- μ , κ , and σ (weak agonist)	-Mild to moderate pain	-Similar to morphine -Low risk of abuse	-A prodrug (10% converts to morphine) -Antitussive
Hydrocodeine/ Oxycodone	- μ , κ , and σ (full agonist)	-Moderate to severe pain	-Similar to morphine	
Propoxyphene	- μ , κ , and σ (weak agonist)	-Moderate pain	-Dizziness -Sedation -Convulsions, CNS depression, coma, and death with high doses -Low risk of abuse -GI disturbances	-Weak analgesic (less potent than aspirin)
Pentazocine	- κ agonist -weak μ and σ antagonist - σ agonist	-Moderate pain via receptor activation in spinal cord -Preoperative medicine	-Dysphoria -Similar to morphine -No respiratory depression -Low risk of abuse	
Non-opioids				
Tramadol	- μ , κ , and σ	-Moderate to severe pain		

SEDATIVE-HYPNOTICS/ANXIOLYTICS

Drugs	Mechanism	Indications	Precautions/Side Effects
Benzodiazepines (-am)	-Enhance GABA receptor binding in the CNS which opens chloride channels, leading to hyperpolarization and decreased neuronal firing	-Anxiety disorders -Muscular disorders -Seizures -Sleep disorders -Acute alcohol withdrawal syndrome -Sedation and amnesia before medical or surgical procedures	-Drowsiness -Confusion
Zolpidem	-Acts on a subset of benzodiazepine receptors	-Little clinical experience with this drug	-Nightmares, agitation, headaches, gastrointestinal upset, dizziness, and drowsiness -No withdrawal effects and little or no tolerance with prolonged use
Buspirone	Targets the following specific receptors: -5-HT _{1A} serotonin receptors -DA ₂ serotonin receptors -5-HT ₂ serotonin receptors	-Generalized anxiety disorders	-Headaches, dizziness, nervousness, lightheadedness -Low risk of abuse -Minimum sedation
Hydroxyzine	-Antihistamine with antiemetic activity	-Anxiety with a history of abuse -Sedation prior to dental procedures or surgery	-Low risk of abuse
Barbiturates	-Interfere with Na ⁺ and K ⁺ transport resulting in the inhibition of reticular activating system -Enhance GABA action	-Anesthesia -Anticonvulsant -Anxiety	-CNS disturbances -Drug hangover -Decreases effectivity of other drugs (enzyme induction) -Addiction -Poisoning (leading cause of death) -Respiratory depression

ANTIPSYCHOTICS

Category	Drugs	Mechanism	Precautions/Side Effects
Phenothiazines	-Chlorpromazine -Fluphenazine -Mesoridazine -Perphenazine -Prochlorperizine -Promazine -Thioridazine -Trifluoperazine -Triflupromazine	Complex and not entirely understood, but the goal is generally to block dopamine receptors (Theories suggest that excess dopamine leads to many psychoses)	-Acute dystonia -Parkinsonism -Malignant syndrome -Akathisia -Tardive dyskinesia
Thioxanthenes	-Thiothixene		
Others	-Clozapine -Haloperidol -Loxapine -Molindone -Olanzapine -Pimozide -Quetiapine -Risperidone		

ANTIPARKINSONIANS

Drugs	Mechanism	Precautions/Side Effects
Levodopa (L-dopa)	<p>Crosses the blood brain barrier as a pro-drug, and then transforms into dopamine</p> <p>*Effective for reducing rigidity, tremors, and other symptoms</p>	<ul style="list-style-type: none"> -Conversion of L-dopa to dopamine in the periphery → nausea, vomiting, cardiac arrhythmias, hypotension -Basal ganglionic effects → hallucinations and dyskinesia -Vitamin B₆ enhances L-dopa breakdown -Monoamine oxidase (MAO) inhibitors promote catecholamine production → hypertensive crisis -Glaucoma → increased IOP -Psychosis -Antipsychotics → Parkinson-like symptoms -Pre-existing heart problems
Carbidopa (dopa decarboxylase inhibitor)	<p>Co-administered with L-dopa to increase the availability of L-dopa in the CNS by slowing its breakdown in the periphery</p>	
Bromocriptine (ergotamine)	<p>Dopamine receptor agonist co-administered with L-dopa (Effective only if patient is L-dopa responsive)</p> <p>*Dyskinesia is lessened</p>	<ul style="list-style-type: none"> -Hallucinations -Confusion -Nausea -Orthostatic hypotension -Worsens symptoms of patients with a previous history of psychosis, vasospasms, peptic ulcers, and myocardial infarctions
Amantadine (antiviral)	<p>Enhances dopamine availability (Dependent upon the integrity of pre-existing dopamine-producing cells)</p> <p>*More effective on rigidity and bradykinesia</p>	<ul style="list-style-type: none"> -Acute toxic psychosis with high dosage -Less CNS and peripheral side effects than levodopa
Deprenyl	<p>Inhibits dopamine metabolism by selectively blocking MAO A action</p>	<ul style="list-style-type: none"> -Decreases hypertensive crisis at recommended dosage (MAO A more selective than MAO inhibitors)
Benzotropine trihexyphenidyl biperiden (antimuscarinic agents)	<p>Co-administered with L-dopa</p>	<ul style="list-style-type: none"> -Mood changes -Xerostomia -Visual problems -GI disturbances (primarily affecting peristalsis) -Glaucoma -Prostatic hypertrophy -Pyloric stenosis

ANTIDEPRESSANTS

All drugs used to treat depression (called thymoleptics) affect levels and activities of norepinephrine, dopamine, and/or serotonin.

Category	Drugs	Mechanism	Precautions/Side Effects
Tricyclic antidepressants *first line of therapy	-Amitriptyline (Elavil) -Amoxapine (Asendin) -Desipramine (Norpramin) -Doxepin (Sinequan) -Imipramine (Tofranil) -Maprotiline -Nortriptyline (Pamelor, Aventyl) -Protriptyline (Vivactil) -Trimipramine (Surmontil)	-Block reuptake of monoamine transmitters (norepinephrine and serotonin) -Also block alpha-adrenergic, histamine, and muscarinic receptors	-React with MAO inhibitors, direct-acting adrenergic drugs, indirect-acting adrenergic drugs, ethanol, and other CNS depressants -Narrow therapeutic index -Use caution in manic-depressive patients (unmask manic behavior)
Serotonin-reuptake inhibitors	-Fluoxetine (Prozac) -Fluvoxamine (Luvox) -Nefazodone (Serzone) -Paroxetine (Paxil) -Sertraline (Zoloft) -Trazodone (Desyrel) -Venlafaxine (Effexor)	-Specifically inhibit reuptake of serotonin	-React with MAO inhibitors
Monoamine oxidase inhibitors (MAOI)	-Isocarboxazid -Phenelzine (Nardil) -Tranylcypromine (Parnate)	-Inactivate the MAO enzymes that degrade norepinephrine, serotonin, and dopamine	-Avoid tyramine-containing foods -Orthostatic hypotension -Avoid co-administration with serotonin-reuptake inhibitors

ANTICONVULSANTS

Drugs	Mechanism	Indications	Precautions/Side Effects
Phenytoin	-Decreases Na ⁺ flux in neurons at resting state or during depolarization → stabilizes neuronal membranes to depolarization and suppresses repetitively firing of neurons	-Suppression of tonic-clonic and partial seizures -Initial epileptic treatment -Status epilepticus (caused by recurrent tonic-clonic)	-CNS depression -GI disturbances -Reversible gingival hyperplasia in children -Megaloblastic anemia (interferes with B ₁₂ metabolism) -Fetal hydantoin syndrome
Carbamazepine	-Inhibits Na ⁺ channels → decreases propagation of abnormal impulses in the brain → inhibits repetitive action potentials (epileptic focus)	-Partial seizures -Tonic-clonic seizures -Trigeminal neuralgia -Manic-depressive episodes	-Stupor -Coma -Respiratory depression -Drowsiness -Vertigo -Ataxia -Blurry vision -GI disturbances -Blood disorders -Neurotoxicity
Phenobarbital	-Activates GABA-mediated neurons → decreases spread of seizure discharges in brain and increases seizure threshold	-Simple partial seizures (not effective for complex partial seizures) -Recurrent seizures in children (including febrile seizures) -Recurrent tonic-clonic seizures	-Nausea and morbilliform rash -Sedation, ataxia, nystagmus, vertigo, and acute psychotic reactions with chronic use -Agitation and confusion with high doses -Rebound seizures with discontinuance
Primidone	-Prodrug (converts to Phenobarbital)	-Adjunctive therapy (not primary treatment for any seizure types)	-Similar to phenobarbital
Valproic acid	-Makes electrical activity in the brain more regular -Enhances GABA	-Myoclonic seizures -Second choice for absence seizures	-Hepatotoxicity -Nausea -Sedation -Ataxia -Tremor -Rash -Alopecia -Blood disorders
Ethanosuximide	-Makes electrical activity in the brain more regular	-First choice for absence seizures	-GI disturbances -Stevens-Johnson syndrome -Blood disorders
Clonazepam (benzodiazepine)	-Prevents seizure spread from epileptogenic focus	-Chronic treatment	-Sedation -Ataxia -Dizziness -Behavior changes

SKELLETAL MUSCLE RELAXANTS

Purpose: To relieve muscle spasticity or muscle spasm, reduce pain, and increase range of motion

Variations: Centrally-acting: Involve stretch reflex arc modification
 Peripherally-acting: Involve disruption of excitation-contraction coupling (direct-acting)

Category	Drugs	Mechanism	Precautions/Side Effects
Centrally-acting	Cyclobenzaprine (Flexeril)	Acts at level of the brain stem to reduce excitatory motor impulses	-Drowsiness -Atropine-like effects (dry mouth, inability to accommodate, gastrointestinal disturbances, confusion)
	Orphenadine (Norflex)	Lessens spinal cord output in response to stretch reflex	-Cholinergic effects -Hallucinations
	Baclofen (Lioresal)	Believed to enhance GABA and inhibit release of excitatory neurotransmitters	-Weakness -Gastrointestinal disturbances -Urinary urgency -Confusion
Peripherally-acting	Dantrolene Sodium (Dantrium)	Disrupts sarcoplasmic reticulum associated muscle contraction	-Dizziness -Insomnia -Gastrointestinal disturbances
Benzodiazepines	Diazepam (Valium)	Stimulate GABA receptors in CNS and increase pre-synaptic inhibition of motor neurons	-Drowsiness -Uncoordinated muscles
Neurotoxin	Botulinum toxin	Binds to the receptor sites of motor nerve terminals and inhibits acetylcholine release	

GENERAL ANESTHETICS

The mechanism of general anesthetics is unclear. These agents do not directly affect receptors. They may work by altering lipids in cell membranes.

Category	Drugs	Mechanism	Indications	Precautions/Side Effects
Benzodiazepines	-Diazepam -Lorazepam -Midazolam	Decrease neural activity	-Sedation	-No analgesic effects -Undesired skeletal muscle activity
Opioids	-Fentanyl -Morphine	Influence opioid receptors → decrease neural activity	-Analgesia prior to cardiac surgery	-Hypotension -Decreased respiratory activity -Muscle rigidity -CNS disturbances
Neuroleptic agent	-Droperidol + fentanyl	Influences adrenergic receptors → decreases neural activity	-Sedation -Antiemetic -Anticonvulsant	-Extrapyramidal effects -Not for use in patients with Parkinson's disease
Dissociative agent	-Ketamine	Decreases neural activity	Sedation in children and young adults (does not cause circulatory depression)	-Increased sympathetic activity -Muscle paralysis -CNS disturbances

GENERAL ANESTHETICS continued

Drugs	Mechanism	Indications	Precautions/Side Effects
Propofol	Decreases neural activity	-Sedation -High intracranial pressure	-Poor analgesic -CNS disturbances
Halothane	Decreases neural activity	-Potent inhaled anesthetic *Drug of choice for use in children	-Poor analgesic -Bradycardia -Cardiac arrhythmias -Hypotension -Toxic effects (fever, anorexia, nausea & vomiting) -Sensitization of heart to catecholamines -Decreased cardiac output -Hypotension -Inhibition of respiratory reflexes -Hepatotoxicity
Enflurane	Decreases neural activity	-Inhaled anesthetic -Potent muscle relaxer	-Hepatotoxicity -CNS disturbances -Slight sensitization of heart to catecholamines -Initial decrease in cardiac output -Initial hypotension -Inhibition of respiratory reflexes
Isoflurane	Decreases neural activity	-Inhaled anesthetic	-Decreased cardiac output -Hypotension -Initially stimulation respiratory reflexes
Methoxyflurane	Decreases neural activity	-Most potent inhaled anesthetic -Does not cause relaxation of uterus	-Not for use in patients with renal failure
Nitrous oxide	Decreases neural activity	-Potent inhaled analgesic -Dental procedures *Safest inhaled anesthetic	-Poor anesthetic -Diffusion hypoxia
Sevoflurane	Decreases neural activity	-Inhaled anesthetic for children	-No known precautions/side effects at this time

LOCAL ANESTHETICS

Category	Mechanism	Indications	Precautions/Side Effects
Amides			
Immediate-acting -Lidocaine -Mepivacaine -Prilocaine	Block voltage-gated sodium channels → inhibit nerve conduction	-Sedation -Nerve block -Arrhythmias (lidocaine) -Intracranial hypertension (lidocaine) -Epidural anesthesia -Dental procedure (mepivacaine, prilocaine, etidocaine) -Lumbar peridural (etidocaine) -Intraabdominal surgery (etidocaine) -Post-operation pain (ropivacaine) -Obstetric surgery (ropivacaine)	-CNS and GI disturbances -Hypotension -Arrhythmias -Decreased heart conduction -Methemoglobinemia with prilocaine -Cardiotoxicity with low doses of bupivacaine and ropivacaine -Respiratory depression
Long-acting -Etidocaine -Bupivacaine -Ropivacaine			
Esters			
Short-acting -Cocaine -Procaine -Chloroprocaine	Block voltage-gated sodium channels → inhibit nerve conduction	-Nerve block -Eye, ear, nose, and throat surgery (cocaine) -Spinal anesthesia (procaine, tetracaine) -Epidural anesthesia	-CNS and GI disturbances -Arrhythmias -Damage of nasal structures with vasoconstriction (cocaine) -Irreversible nerve damage (chloroprocaine) -Hypersensitivity -Respiratory depression
Immediate-acting -Tetracaine			

ADENOHYPHYSAL HORMONES

Hormones released by the anterior pituitary gland (adenohypophysis)

Hormone	Target	Function
Thyroid-stimulating hormone (TSH)	Thyroid gland	Stimulates release of thyroid hormones
Adrenocorticotropic hormone (ACTH)	Adrenal medulla	Stimulates release of adrenal hormones
Follicle-stimulating hormone (FSH)	Ovaries and testes	Stimulates ovarian follicle growth → estradiol production Stimulates sperm production in Sertoli cells
Luteinizing hormone (LH)	Ovaries and testes	Stimulates ovulation and maturation of follicle to corpus luteum → estrogen and progesterone production Stimulates testosterone synthesis in Leydig cells
Prolactin (PRL)	Mammary glands	Stimulates milk production
Growth hormone (GH, somatotropin)	Bones and cartilage	Stimulates increased protein synthesis, lipolysis, and glycogenesis → increased growth of bone and cartilage
Melanocyte-stimulating hormone (MSH)	Melanocytes	Increases rate of melanin production in skin

THYROID AND ANTITHYROID DRUGS

Hormones produced by the thyroid gland include T_4 (thyroxine, tetraiodothyronine), T_3 (triiodothyronine), and calcitonin. Production of these hormones is regulated by levels of thyroid-stimulating hormone (TSH). T_3 and T_4 stimulate carbohydrate metabolism, lipid catabolism, and protein synthesis. Calcitonin decreases plasma calcium concentrations.

Drugs	Indications	Mechanism	Precautions/Side Effects
Levothyroxine (synthetic T_4)	Hypothyroidism	Replenishes T_4	-Caution in cardiovascular disease patients -Caution in metabolic disease patients (diabetes, adrenal insufficiency)
Levothyronine (synthetic T_3)	Hypothyroidism (when levothyroxine is ineffective)	Replenishes T_3	-Similar to levothyroxine
Liotrix (synthetic T_3 and T_4)	Hypothyroidism	Replenishes T_3 and T_4	-Similar to levothyroxine
Radioactive iodine (^{131}I)	Hyperthyroidism	Selective uptake by thyroid cells results in necrosis	-Possible hypothyroidism
Methimazole	Hyperthyroidism	Inhibits bioconversion of inactive T_4 to active T_3	-Possible hypothyroidism -Agranulocytosis -Rash -Arthralgia
Propylthiouracil (PTU)	Hyperthyroidism	Inhibits bioconversion of inactive T_4 to active T_3	-Similar to methimazole
Potassium iodide solution	Hyperthyroidism (including potentially fatal thyroid crisis)	Inhibits iodination of tyrosines → decreases levels of T_3 and T_4	-Possible ulceration of mouth, throat, and other mucous membranes -Rash -Metallic taste

NSULINS

Insulin is a peptide hormone produced by the β cells of the pancreas.

Insulin is needed for cellular uptake of glucose from the bloodstream to store as glycogen.

Diabetics lack the ability to produce insulin in sufficient amounts, and may require insulin treatment.

The duration of action of an insulin agent can be modified by the level of zinc in the preparation. Higher levels of zinc result in longer-acting agents by binding to the insulin and slowing its absorption.

Drugs	Onset	Indications	Precautions/Side Effects
Regular insulin	Rapid	Emergencies (IV administration)	-May result in hypoglycemia -Lipodystrophy -Hypersensitivity
Lispro insulin	Rapid	Faster-acting than regular insulin (usually used with a longer acting agent for glucose control)	-Similar to regular insulin
Semilente insulin suspension	Intermediate	Not suitable for IV administration	-Similar to regular insulin
Isophane insulin suspension	Intermediate	Not suitable for IV administration Not suitable in patients with diabetic ketoacidosis	-Similar to regular insulin
Lente insulin (combination of semilente and ultralente)	Intermediate	Benefit of long-term control that ultralente provides, but more rapid onset (semilente)	-Similar to regular insulin
Ultralente insulin	Slow	Long-term control of glucose levels	-Similar to regular insulin

ORAL HYPOGLYCEMIC AGENTS

These agents are used to control glucose levels in Type 2 diabetes mellitus.

Category	Drugs	Mechanism	Precautions/Side Effects
Sulfonylureas	-Tolbutamide -Chlorpropamide -Tolazamide -Acetohexamide -Glipizide -Glyburide -Glimepiride	-Stimulate pancreatic β cells to release insulin -Decrease levels of glucagon in serum -Enhance affinity of receptors to insulin	-Not for use during pregnancy -Not for use in patients with hepatic or renal insufficiency -Sulfonamide cross sensitivity -Avoid chlorpropamide in elderly -May result in hypoglycemia
Biguanides	-Metformin	-Similar to sulfonylureas but do not stimulate release of insulin	-Not for use in patients with hepatic or renal insufficiency -Interference with B ₁₂ absorption with long-term use -GI disturbances -Potential fatal lactic acidosis (but rare) -Less hypoglycemia than sulfonylureas
α -Glucosidase inhibitor	-Acarbose	-Slows the digestion of carbohydrates \rightarrow decreases glucose absorption following meals -Used alone or in addition to	-GI disturbances -Does not result in hypoglycemia
Thiazolidinediones	-Troglitazone -Rosiglitazone	-Reduce resistance to insulin, making it more effective in liver and skeletal muscle	-Hepatotoxicity -Upper respiratory infections -Headaches -Anemia -Edema -Weight gain -Interfere with oral contraceptives -Interfere with P450 system

ESTROGENS, PROGESTINS, AND ANDROGENS

Category	Drugs	Mechanism	Indications	Precautions/Side Effects
Estrogens	-Chlorotrianisene -Diethylstilbestrol -Estradiol -Estriol -Estrone -Ethinyl estradiol -Mestranol -Quinestrol	Promote gene transcription after binding to intracellular receptors	-Contraception -Post-menopausal hormone therapy -Osteoporosis -Hypogonadism	-Nausea and vomiting -Edema -Headaches -Hypertension -Breast tenderness
Progestins	-Hydroxyprogesterone -Medroxyprogesterone -Norethindone -Norgestrel	Promote protein synthesis after binding to intranuclear protein receptors	-Contraception -Dysfunctional uterine bleeding -Dysmenorrhea -Suppression of postpartum lactation -Endometriosis -Endometriocarcinoma -Precocious puberty	-Edema -Depression -Pulmonary embolism -Acne -Hirsutism -Weight gain -Jaundice due to lipid disturbances
Androgens	-Danazol -Fluoxymesterone -Nandrolone -Stanozolol -Testosterone cypionate	Bind to receptors in target cells and stimulate synthesis of RNA and protein	-Inadequate androgen secretion -Osteoporosis -Severe burns -Pituitary dwarfism -Endometriosis	-Not for use during pregnancy -Masculinization -Acne -Impotence -Decreased spermatogenesis -Gynecomastia -Growth disturbances in children -Lipid disturbances

ANTI-HYPERTENSIVE AGENTS

Blood pressure is directly related to peripheral vascular resistance and cardiac output. The goal of treatment for hypertension is to either reduce peripheral vascular resistance (through vasodilation) or to decrease cardiac output.

Category	Drugs	Mechanism	Precautions/Side Effects	
β adrenergic antagonists	-Acebutolol* -Atenolol* -Betaxolol* -Metoprolol* * β_1 selective ** non-selective	-Propranolol** -Carvedilol** -Timolol** -Nadolol**	-Reduce heart rate and contractility \rightarrow reduce cardiac output -Decrease renin release from kidney \rightarrow vasodilation	-CNS disturbances -Decreased libido -Disturbances in lipid levels -Bronchoconstriction if not β_1 selective -Peripheral vasoconstriction -Bradycardia -Not for use in patients with second or third degree AV block
α_1 adrenergic antagonists	-Prazosin -Terazosin -Doxazosin	Vasodilation	-CNS disturbances -Dyspnea -Nasal congestion -Peripheral edema	
α_2 adrenergic agonists	-Clonidine -Methyldopa (drug of choice for hypertension during pregnancy)	Act within the CNS to inhibit vasomotor centers resulting in decreased sympathetic output \rightarrow causes vasodilation and reduced cardiac output	-CNS disturbances -Decreased sympathetic output throughout the body	
Calcium channel blockers	-Verapamil -Nifedipine -Isradipine -Nicardipine	-Diltiazem -Felodipine -Amlodipine -Nisoldipine	Relaxation of smooth muscle \rightarrow vasodilation	-Postural hypotension, facial flushing, and peripheral edema -Reflex tachycardia with nifedipine -Constipation with verapamil
ACE inhibitors	-Benazepril -Enalapril -Lisinopril -Quinapril	-Captopril -Fosinopril -Moexipril -Ramipril	Inhibit angiotensin-converting enzyme \rightarrow vasodilation	-Dry cough (potentiate bradykinins) -Hyperkalemia -CNS disturbances
Angiotensin II antagonists	-Losartan -Valsartan	Block angiotensin II receptors on blood vessels \rightarrow vasodilation	-CNS disturbances -Upper respiratory infections	

TREATMENT OF CONGESTIVE HEART FAILURE

Congestive heart failure occurs when the heart is unable to supply the demand of the body. The treatment is aimed at decreasing blood volume, increasing the strength of heart contraction, or reducing the load of the heart by decreasing peripheral resistance.

Category	Drugs	Mechanism	Precautions/Side Effects
Diuretics	- Bumetanide - Furosemide - Hydrochlorothiazide - Metolazone	Promote secretion of sodium and water → decrease blood volume	- Orthostatic hypotension - Electrolyte imbalances - CNS & GI disturbances
Vasodilators			
ACE inhibitors	- Captopril - Enalapril - Fosinopril - Lisinopril - Quinapril	Inhibit angiotensin-converting enzyme	- Dry cough (potentiate bradykinins) - Hyperkalemia - CNS disturbances
Direct smooth muscle relaxants	- Hydralazine - Isosorbide - Minoxidil - Sodium nitroprusside	Relax smooth muscle in arteries and veins	- Orthostatic hypotension
Agents that increase contractility			
Cardiac glycosides	- Digitoxin - Digoxin - Indicated after start of diuretic and vasodilation treatment in severe left ventricular systolic dysfunction	Interfere with sodium-potassium pump → decrease heart rate and increase contractility	- Not for use in diastolic or right-sided heart failure - Digitalis toxicity - GI & CNS disturbances
β-adrenergic agonists	- Dobutamine	Increase contractility and cause vasodilation	- CNS disturbances - Cardiac arrhythmias - Pulmonary edema
Phosphodiesterase inhibitors	- Amrinone - Milrinone	Increase contractility	- GI disturbances - Hepatotoxicity

ANTIARRHYTHMIC DRUGS

Category	Drugs	Mechanism	Indications	Precautions/Side Effects
Class IA	-Quinidine -Procainamide -Disopyramide	Blocks sodium channels → slows conduction and prolongs action potentials	-Atrial arrhythmias -AV junction arrhythmias -Ventricular tachyarrhythmias	-Potentially worsens arrhythmias -SA and AV block -Hyperkalemia -GI & CNS disturbances
Class IB	-Lidocaine -Mexilatine -Tocainide	Blocks sodium channels → shortens repolarization and decreases duration of action potentials	-Ventricular arrhythmias (especially during myocardial infarction)	-CNS disturbances -Potential cardiac arrhythmias
Class IC	-Flecainide -Propafenone	Blocks sodium channels → slows conduction and does not affect duration of action potentials	-Refractory ventricular arrhythmias -Premature ventricular contraction	-Aggrevation of congestive heart failure -CNS disturbances -Potential ventricular tachycardia
Class II	-Esmolol -Metoprolol -Pindolol -Propanolol	Blocks β -receptors → slows heart rate and decreases contractility	-Tachyarrhythmias -Atrial flutter -Atrial fibrillation -AV node reentry tachycardia	-CNS disturbances -Decreased libido -Disturbances in lipid levels -Bronchoconstriction if not β -1selective -Peripheral vasoconstriction -Bradycardia -Not for use in patients with second or third degree AV block
Class III	-Amiodarone -Bretylium -Sotalol	Blocks potassium channels → prolongs duration of action potentials	-Prevents arrhythmia occurrence following myocardial infarction -Ventricular arrhythmias	-Tachyarrhythmias -Postural hypotension
Class IV	-Diltiazem -Verapamil	Blocks calcium channels → decreases heart rate and contractility	-Atrial flutter -Atrial fibrillation -Reentry supraventricular tachycardia	-Bradycardia -Hypotension
Digitalis glycosides	-Digitoxin -Digoxin	Interfere with sodium-potassium pump → decrease heart rate and increase contractility	-Atrial fibrillation -Atrial flutter -Paroxysmal atrial tachycardia	-Not for use in patients with diastolic or right-sided heart failure -Digitalis toxicity -GI & CNS disturbances

ANTIANGINAL AGENTS

Angina occurs when there is an insufficient supply of blood to the heart. The goal in treating angina is to increase blood supply to heart and/or decrease the demand of heart.

Category	Drugs	Mechanism	Indications	Precautions/Side Effects
Organic nitrates	-Isosorbide dinitrate -Nitroglycerin	Cause relaxation of smooth muscle → decrease O ₂ demand of heart	-Stable angina -Unstable angina -Prinzmetal's angina -Variant angina	-Headaches -Postural hypotension, facial flushing, and tachycardia with high doses
β-Blockers	-Propranolol -Atenolol -Metoprolol	Slow cardiac rate and decreases cardiac output → decrease O ₂ demand of heart	-Myocardial infarction	-Not for use in patients with diabetes, chronic obstructive pulmonary disease, or peripheral vascular disease
Calcium channel blockers	-Nifedipine -Verapamil -Diltiazem	Block entry of calcium into smooth muscle cells → decrease demand and increase supply of O ₂ to heart	-Variant angina	-Postural hypotension, facial flushing, and peripheral edema -Reflex tachycardia with nifedipine -Constipation with verapamil

ANTICOAGULANTS AND THROMBOLYTICS

Drugs	Mechanism	Indications	Precautions/Side Effects
Anticoagulants			
Heparin	Binds with antithrombin III → indirectly prevents coagulation	<ul style="list-style-type: none"> -Deep vein thrombosis -Pulmonary embolism -Prophylactic treatment for post-surgical venous thrombosis -Acute myocardial infarction -Preferred over other anticoagulants in pregnancy (does not cross placenta) 	<ul style="list-style-type: none"> -Hemorrhage -Hypersensitivity -Thrombocytopenia -Not for use in alcoholics -Not for use following brain, eye, or spinal cord surgery
Warfarin	Inhibits vitamin K → produces inactive clotting factors	<ul style="list-style-type: none"> -Deep vein thrombosis -Pulmonary embolism -Ischemic heart disease -Rheumatic heart disease -Patients with artificial heart valves 	<ul style="list-style-type: none"> -Hemorrhage -Interactions with other protein-bound drugs -Not for use during pregnancy (teratogenic)
Thrombolytics			
Alteplase (tPA – tissue type plasminogen activator)	Activates plasminogen bound to fibrin → lyses fibrin	<ul style="list-style-type: none"> -Myocardial infarction -Pulmonary embolism -Acute ischemic stroke 	<ul style="list-style-type: none"> -Hemorrhage -Not for use during pregnancy -Not for use in patients with a history of strokes -Not for use in patients with metastatic cancer
Streptokinase	Binds to plasminogen → hydrolyses fibrin plugs and degrades fibrinogen, clotting factor V, and clotting factor VII	<ul style="list-style-type: none"> -Acute pulmonary embolism -Deep vein thrombosis -Arterial thrombosis -Acute myocardial infarction 	<ul style="list-style-type: none"> -Similar to Alteplase -Hypersensitivity
Anistreplase	On-site lysis of blood clot	-Similar to streptokinase	-Similar to streptokinase
Urokinase	Direct enzymatic degradation of fibrin and fibrinogen	-Patients who are sensitive to streptokinase	-Similar to altepase

ANTIHYPERLIPIDEMIC AGENTS

Category	Drugs	Mechanism	Precautions/Side Effects
Bile acid binding resins	-Cholestyramine -Colestipol	Bind to bile acids and bile salts in small intestine → hepatocytes replenish supply by converting cholesterol to bile acids	-GI disturbances -Reduced absorption of fat-soluble vitamins -Interference with absorption of certain drugs
HMG-CoA reductase inhibitors	-Atorvastatin -Cerivastatin -Fluvastatin -Lovastatin -Pravastatin -Simvastatin	Inhibit the enzyme needed to make cholesterol precursors (first line treatment for lowering cholesterol)	-GI & CNS disturbances -Myopathies -Alterations in liver function -Not for use during pregnancy or nursing
Fibrates/fibric acid derivatives	-Gemfibrozil -Clofibrate -Fenofibrate	Stimulate lipoprotein lipase activity and also may inhibit the synthesis of cholesterol	-GI disturbances -Gallstones -Malignancy -Inflammation of involuntary muscle tissue -Not for use in patients with liver or kidney dysfunction
Niacin (nicotinic acid)	-Niacin	Inhibits lipid breakdown in adipose tissue → reduces supply of cholesterol precursors to liver	-Flushing -GI disturbances -Hyperuricemia -Liver dysfunction -Glucose intolerance -Ocular side effects

DIURETICS

These drugs promote urine output by increasing both sodium and water excretion.

Category	Drugs	Mechanism	Precautions/Side Effects	Indications
Carbonic anhydrase inhibitors	-Acetazolamide -Methazolamide -Dichlorphenamide	Inhibit carbonic anhydrase in proximal tubule → increased excretion of HCO_3^- , Na^+ , and H_2O	-Sulfonamide cross sensitivity -Alkaline urine (results in metabolic acidosis) -Hypokalemia -Nephrolithiasis -CNS and GI disturbances -Transient myopia -Aplastic anemia	-Hypertension -Glaucoma -Epilepsy -Mountain sickness
Loop diuretics	-Furosemide -Ethacrynic Acid -Bumetanide -Torsemide	Inhibit reabsorption of Na^+ , K^+ , and Cl^- in ascending loop of Henle → decreased H_2O reabsorption *most effective diuretics	-Hypokalemia -Hyponatremia -Hypocalcemia -Hyperuricemia with furosemide and ethacrynic acid -Orthostatic hypotension -Acute hypovolemia -Ototoxicity -CNS and GI disturbances -Sulfonamide cross sensitivity with furosemide	-Hypertension -Acute pulmonary edema -Hypercalcemia -Nephrotic syndrome

DIURETICS continued

Category	Drugs	Mechanism	Precautions/Side Effect	Indications
Thiazide diuretics	-Hydrochlorothiazide -Chlorthiazide -Chlothaldione -Indapamide -Metolazone	Inhibit reabsorption of Na ⁺ and Cl ⁻ at the distal convoluted tubule → increased H ₂ O excretion *most commonly used diuretics	-Hypokalemia -Hyponatremia -Hypercalcemia -Hyperuricemia -Hypomagnesia -Hyperglycemia -Orthostatic hypotension -CNS and GI disturbances -Sulfonamide cross sensitivity	-Hypertension -Congestive heart failure -Nephrotic syndrome when loop diuretics are not effective -Hypercalciuria -Diabetes insipidus
Potassium-sparing diuretics	-Spironolactone -Amiloride -Triamterene	Inhibit reabsorption of sodium and inhibit potassium secretion at the collecting duct Spironolactone → aldosterone antagonist Amiloride and triamterene → sodium channel blockers	-Hyperkalemia -Hyponatremia -CNS and GI disturbances	-Hypertension -Congestive heart failure -Secondary hyperaldosteronism (often used with loop or thiazide diuretics)
Osmotic diuretics	-Mannitol -Urea	Elevate osmolarity of glomerular filtrate → decrease H ₂ O reabsorption	-Electrolyte imbalances	-Increased intracranial pressure -Acute renal failure

Sources

Martini, F.H., Timmons, M.J., & McKinley, M.P. (2000). Human Anatomy, Third Edition. Upper Saddle River, NJ: Prentice Hall.

Mycek, M.J., Harvey, R.A., & Champe, P.C. (2000). Lippincott's Illustrated Reviews: Pharmacology, Second Edition. Philadelphia, PA: Lippincott Williams & Wilkins.

Nester, E.W., Anderson, D.G., Evans Roberts, C.J., Pearsall, N.N., & Nester, M.T. (2001). Microbiology: A Human Perspective, Third Edition. New York, NY: McGraw-Hill.

Olson, J.M. (2002). Clinical Pharmacology Made Ridiculously Simple, Second Edition. Miami, FL: MedMaster, Inc.

In addition to these sources, much information was provided in notes and lectures given by Dr. Robert Rosenow, Dr. Dennis Smith, Dr. Nada Lingel, and Dr. Roger Reynolds at Pacific University College of Optometry.