

CHAPTER 2

DRUGS ACTING ON THE AUTONOMIC NERVOUS SYSTEM

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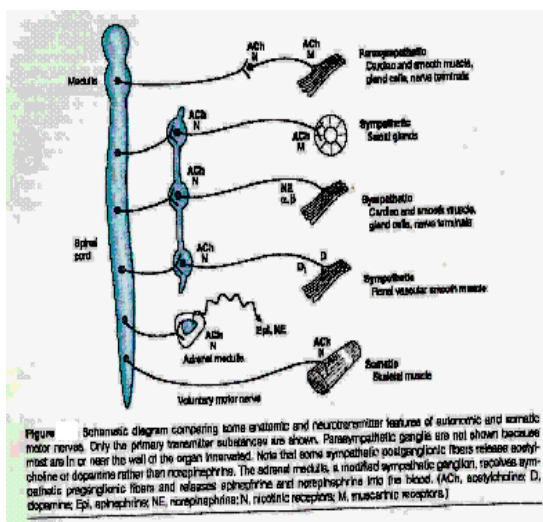
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INTRODUCTION

All of the body's major functions are controlled by the nervous system. The central and peripheral nervous systems are separated. The somatic and autonomic nervous systems, which control voluntary and involuntary functions, are both part of the peripheral nervous system. The ANS is in charge of the body's vegetative functions. Circulation, respiration, digestion, and body temperature regulation are examples of these functions.

The autonomic nervous system is divided into two major subdivisions based on anatomic and physiologic criteria; the two subdivisions are sympathetic (thoracolumbar) and parasympathetic (craniosacral). Based on their anatomical location relative to the ganglia, autonomic nerves are divided into two neuron systems: preganglionic and postganglionic. The cell body of a preganglionic neuron is found in the spinal cord or brain.



The parasympathetic nervous system is primarily concerned with the conservation and restoration of function. The sympathetic nervous system, on the other hand, is concerned with energy expenditure, i.e., it performs nearly opposite functions to parasympathetic nerve stimulation, and it is typically associated with arousal or in emergency situations, i.e., it prepares the body for fight-or-flight responses.

To understand autonomic nervous system pharmacology, you must first understand how the system works and the mechanisms that underpin its functions, such as nerve transmission. The autonomic nervous system contains two important

neurotransmitters. Acetylcholine and noradrenaline are the two (norepinephrine) Acetylcholine is a neurotransmitter that is released after the parasympathetic nervous system is stimulated to act on effector organs (cells) to elicit their response, but it also acts as a neurotransmitter: It is found in the sympathetic and parasympathetic nervous systems' ganglia. Between some neurons in the CNS, and at preganglionic nerve endings to the adrenal medulla. The passage of an impulse across a synapse is the process of neurotransmission.

The enzyme choline acetyltransferase catalyzes the conversion of acetyl coenzyme A and choline into acetylcholine in the cytoplasm of nerve fibers. When an action potential reaches the terminal and the latter is stimulated, acetylcholine is released into the synaptic cleft from the cytoplasm into vesicles to be stored. The molecule binds to and activates an acetylcholine receptor (cholinergic receptor) on the effector cell after it is released from the presynaptic terminal. Finally, the acetyl cholinesterase enzyme hydrolyzes it into choline and acetate, terminating the transmitter's action. Muscarinic and nicotinic cholinergic receptors are the two types of cholinergic receptors.

Most autonomic effector cells in peripheral visceral organs have muscarinic responses, whereas parasympathetic and sympathetic ganglia, as well as skeletal muscle, have nicotinic responses. The effect of parasympathetic nervous system activity in an organ can be achieved by stimulating parasympathetic nerve fibres that supply the organ or by administering acetylcholine or other parasympathomimetics to the effector cells. Cholinergic activity is the term for this.

The neurotransmitter noradrenaline is released by postganglionic sympathetic nerves to have an effect on effector cells. Noradrenergic or adrenergic sympathetic fibres are found post-ganglionic. Except in sweat glands and blood vessels to skeletal muscles, where acetylcholine is released as a neurotransmitter, sympathetic nerve activity can be demonstrated by sympathetic nerve stimulation or application of noradrenaline, adrenaline, or other sympathomimetics, i.e. 'adrenergic activity.'

When a nerve is stimulated, adrenergic neuron terminals synthesise noradrenaline, store it in vesicles, and release it to effector cells. Several processes, including drug action sites, are used to synthesise the transmitter from the precursor tyrosine (amino acid). Noradrenaline produces its effects after it is released into receptor sites. Reuptake into the nerve terminal (reuptake1), diffusion away from the synaptic cleft and subsequent reuptake into the perisynaptic glia or smooth muscle (reuptake2), or enzyme degradation all result in the termination of noradrenergic transmission. The most important mechanism for noradrenaline effects termination is reuptake into the

nerve terminal. Adrenergic receptors are receptors that respond to adrenergic nerve transmitters. On the basis of agonist and antagonist selectivity, these receptors are divided into alpha and beta adrenoceptor types. Subclasses of receptors exist based on drug selectivity. They are alpha 1 and 2, as well as beta 1 and 2.

Table 2.1: Distribution of adrenoceptor subtypes and their actions

Type	Tissue	Actions
Alpha₁	Most vascular smooth muscles	Contraction
	Pupillary dilator muscle	Mydriasis
	Heart	Increase force of contraction
Alpha₂	Adrenergic nerve terminals	Inhibition of transmitter release
	Platelets	Aggregation
Beta₁	Heart	Increased rate and force of contraction
Beta₂	Respiratory, uterine, and vascular smooth muscle	Relaxation
	Human liver	Glycogenolysis
Beta₃	Fat cells	Lipolysis

There are five key aspects of neurotransmitter function that could be pharmacologically targeted. Synthesis, storage, release, receptor activation, and transmitter action termination are the steps involved.

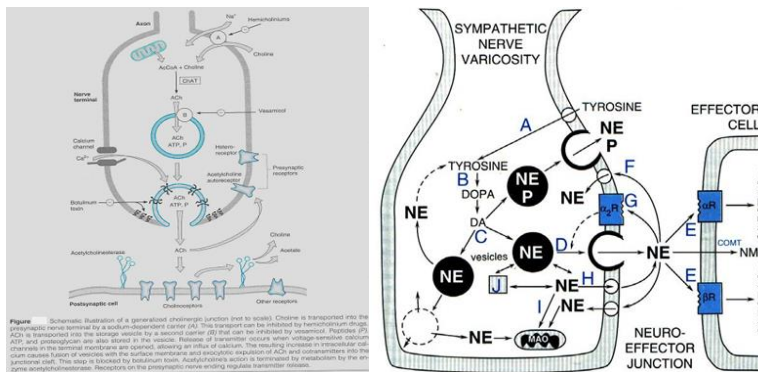


Fig 2.1: Proposed site of action of drugs on the synthesis, action, and fate of norepinephrine at sympathetic neuroeffector junctions

AUTONOMIC DRUGS

Several drugs that affect the autonomic nervous system have been classified into groups to help people understand them better.

1. Medications that affect the sympathetic nervous system
 - a) Adrenergic drugs, also known as sympatholytics, are drugs that mimic the effects of sympathetic nerve stimulation.
 - b) Sympatholytics are drugs that block the sympathetic nerve's or sympathomimetics' activity.
2. Medications that affect the parasympathetic nervous system
 - a) Drugs that mimic acetylcholine or the effects of parasympathetic nerve stimulation are known as parasympathomimetics or cholinergic drugs.
 - b) Parasympatholytics: These are drugs that block the activity of the parasympathetic nervous system or cholinergic drugs.

CHOLINERGIC DRUGS

Cholinergic drugs are also called parasympathomimetics because their effect mimics the effect of parasympathetic nerve stimulation. Administration of these drugs will result in an increase in the parasympathetic activities in the systems innervated by cholinergic nerves.

There are two groups of cholinergic drugs:

1. bind to and activate muscarinic or nicotinic receptors (mostly both), and include the subgroups listed below:
 - a. **Esters of choline:** methacholine, carbachol, betanechol
 - b. **Cholinergic alkaloids:** pilocarpine, muscarine, arecoline, nicotine
2. Indirect-action: inhibits acetylcholinesterase enzyme action
 - a. **Reversible:** neostigmine, physostigmine, edrophonium
 - b. **Irreversible:** Organophosphate compounds; echothiophate

The actions of acetylcholine may be divided into two main groups:

1. **Nicotinic actions:** those produced by stimulation of all autonomic ganglia and the neuromuscular junction
2. **Muscarinic actions:** those produced at postganglionic cholinergic nerve endings

ESTERS OF CHOLINE

The prototypical cholinergic agent is ACETYLCHOLINE. It functions as a neurotransmitter at all cholinergic sites in the body; it has never been used in medical therapeutics due to its unique pharmacokinetic properties; the discussion that follows is for academic purposes only.

Pharmacokinetics

Because acetylcholine is poorly absorbed from the gastric mucosa, it is ineffective when administered orally. Parenteral administration is the preferred method. It is rapidly hydrolyzed into acetic acid and choline in the blood by the enzyme cholinesterase, making its duration of action very short and unreliable for therapeutic purposes.

Pharmacodynamics

It has two types of actions, as previously stated: nicotinic and muscarinic; the muscarinic actions are of primary interest and are discussed below.

- Cardiovascular system

Heart→ slow heart rate

Blood vessels→ vasodilator

Blood pressure→ falls because of the effect on the heart and blood vessels

- Gastrointestinal tract** It stimulates the tone and motility of the GI tract but the sphincters will be relaxed
- Urinary tract** It stimulates the detrusor muscle and relaxes the internal urethral sphincter resulting in evacuation of bladder
- Bronchioles** It increases bronchial secretion and brings about bronchoconstriction
- Eye-** Stimulation of the constrictor pupillae and ciliary muscles causes miosis and accommodation for near objects, respectively.
- Salivary, gastric, bronchial, lachrymal, and sweat gland secretions are stimulated by exocrine glands.

Synthetic Choline Esters. Metacholine, carbachol, and betanecol are examples of synthetic choline derivatives. Compared to acetylcholine, these drugs have the following advantages:

- They have longer duration of action,

- They are effective orally as well as parenterally, and
- They are relatively more selective in their actions.

CARBACHOL

Pharmacokinetics

It is completely absorbed from the gastro intestinal tract and is stable towards hydrolysis by cholinesterase enzyme; therefore it can be given both orally and parenterally with almost similar dosage.

Pharmacodynamics

It has similar actions to those of acetylcholine with pronounced effects on the gastro intestinal tract and the urinary bladder

Indications

- Glaucoma
- Retention of urine (postoperative)
- Paralytic ileus

BETANECHOL

This drug is similar to carbachol in every way, including pharmacokinetics, pharmacodynamics, and clinical indications; however, it has a better advantage over carbachol because it has fewer side effects due to the lack of nicotinic effects.

1. Contra indications to the use of choline esters
2. They may cause bronchial constriction and increase bronchial secretions in people with bronchial asthma.
3. Hyperthyroidism is dangerous because it can cause atrial fibrillation.
4. Gastric ulcer disease as a result of increased gastric acid secretion
5. Coronary insufficiency, as the resulting hypertension compromises coronary blood flow.
6. Obstruction of the mechanical intestinal and urinary outlets

CHOLINERGIC ALKALOIDS

1. Nicotine, lobeline, and other compounds with nicotinic actions.
2. Muscarine, pilocarpine, and other muscarinic agents are examples.

PILOCARPINE

Pharmacokinetics

This drug is easily absorbed from the gastrointestinal tract and is not hydrolyzed by the enzyme cholinesterase. In the urine, it is partly destroyed and partly undamaged.

Pharmacodynamics

The drug directly stimulates muscarinic receptors, causing all of acetylcholine's muscarinic effects.

Indications

- Glaucoma

ANTICHOLINESTERASE DRUGS

Cholinesterase Inhibitors are classified into three Chemical Groups:

1. Quaternary amine-containing simple alcohols, such as edrophonium
2. Neostigmine, physostigmine, and other carbamate and related quaternary or tertiary amines
3. Organic phosphate derivatives, such as isofluorophate and echothiophate

PHYSOSTIGMINE

Pharmacokinetics

This drug is completely absorbed from the gastrointestinal and is highly distributed throughout the body; it can cross the blood brain barrier.

Pharmacodynamics

Because it inhibits cholinesterase, it increases and prolongs the effect of endogenous acetylcholine at various sites. It has no effect on cholinergic receptors directly.

Indications

- Glaucoma
- Atropine over dosage

NEOSTIGMINE

Pharmacokinetics

This drug is poorly absorbed from the gastro intestinal tract and is poorly distributed throughout the body; it cannot cross the blood brain barrier.

Pharmacodynamics

It inhibits the cholinesterase enzyme similarly to physostigmine, but unlike physostigmine, it has a direct nicotinic effect on skeletal muscles.

Indications

- Myasthenia gravis
- Paralytic Ileus
- Reversal of effect of muscle relaxants, e.g. tubocurarine
- Post operative urine retention

Organophosphates Because compounds like echothiophate, isofluorophate, and others combine irreversibly with cholinesterase, hydrolysis is very slow.

They could be used to treat glaucoma. Insecticides such as parathion and malathion are also organophosphates. Organophosphate poisoning is a leading cause of morbidity and mortality around the world. It is usually the result of:

- Occupational exposure as in persons engaged in spraying insecticides,
- Accidental exposure, and
- Ingestion of any of these compounds with suicidal intent.

ANTICHOLINERGICS

Anticholinergics stop acetylcholine and other cholinergic drugs from acting on effector cell cholinergic receptors. There are two types of anticholinergic drugs:

1. Antinicotinic which include ganglion blockers such as hexamethonium, trimethaphan, etc., and neuromuscular blockers such as gallamine, tubocurarine, pancuronium, etc.
2. Antimuscarinics include tertiary amines like atropine, scopolamine, tropicamide, and propantheline, ipratropium, and benztropine, as well as quaternary amines like propantheline, ipratropium, and benztropine.

ATROPINE

The plant *Atropa belladonna* contains atropine, which is the prototype of muscarinic antagonists.

Pharmacokinetics

Atropine is fully absorbed from all routes of administration, with the exception of the skin wall, where it is only partially absorbed; it has a good distribution. About 60% of the drug is excreted unchanged in the urine.

Pharmacodynamics

Atropine counteracts the effects of acetylcholine by competing for muscarinic receptors in the peripheral and central nervous systems; thus, atropine has the opposite effect as acetylcholine.

Organ-system Effects:

CNS: - lower doses produce sedation

- higher doses produce excitation, agitation and hallucination

Eyes: - relaxation of constrictor pupillae (mydriasis)

- relaxation or weakening of ciliary muscle (cycloplegia-loss of the ability to accommodate)

CVS: - blocks vagal parasympathetic stimulation (tachycardia)

- vasoconstriction

Respiratory: - bronchodilatation and reduction of secretion

GIT: - decreased motility and secretions

GUS: - Relaxes smooth muscle of ureter and bladder wall; voiding is slowed.

Sweat Glands: - suppresses sweating

Clinical Indications

Pre-anesthetic medication is used to reduce secretion and prevent vagal tone from becoming too strong due to anaesthesia. In cases of intestinal, biliary, and renal colic, as an antispasmodic a heart blockage Hyperhidrosis Poisoning by organophosphates

Side Effects

- Dryness of the mouth, tachycardia and blurred vision
- Retention of urine

Contraindications

- Glaucoma
- Bladder outlet obstruction.

HYOSCINE (SCOPOLAMINE)

This drug has the same effect as atropine, with the following exceptions: -It has a shorter duration of action -It is more CNS depressant

All other characteristics are similar to those of atropine. It has some benefits over atropine. Among them are: Better for preanesthetic medication because of strong antisecretory and antiemetic action and also brings about amnesia Can be used for short- travel motion sickness

SYNTHETIC ATROPINE DERIVATIVES

In the treatment of various conditions, the Synthetic Atropine Derivatives are used however, their actions are same as Atropine's except they have few side effects. These drug classes comprise:

1. Mydriatic atropine substitutes, which have a relatively short duration of action than atropine and are used locally in the eye; examples include Homatropine, Eucatropine, and others.
2. Antisecretory antispasmodic atropine substitutes: Effective more localized to the GI. Drugs include: propantheline and hyoscine
3. Antiparkinsonian atropine substitute: - drugs like Benztropine, Trihexyphenidyl
4. oxybutynin and other atropine substitutes that reduce urinary bladder activity
5. Atropine substitutes used in bronchial asthma drugs like ipratropium

ADRENERGIC DRUGS

These drugs have effects that are similar to sympathetic nerve stimulation, as their name suggests; they can be divided into two groups based on their chemical structure.

1. **Catecholamines:-** these are compounds which have the catechol nucleus. Catecholamines interact with receptor sites on the cell membrane to have a direct effect on sympathetic effector cells. Adrenaline, noradrenaline, dopamine, isoprenaline, and dobutamine are all members of this group-Noncatecholamines: - lack the catechol nucleus.

They may directly act on the receptors or may indirectly release the physiologic catecholamines-

For example: Ephedrine, Phenylephrine, and Amphetamine

Adrenergic drugs, like cholinergic drugs, are classified according to their mode of action and the receptors they affect.

- a. Direct mode of action: directly interact with and activate adrenoreceptors, e.g., adrenaline and noradrenaline
- b. Indirect mode of action: their actions are dependent on the release of endogenous catecholamines. This may be
 - i. Storage catecholamines, such as amphetamine and tyramine, are displaced from adrenergic nerve endings.
 - ii. Cocaine, tricyclic antidepressants, and other drugs that block catecholamine reuptake

Direct and indirect sympathomimetics both cause adrenoreceptor activation, which results in some or all of the catecholamines' typical effects.

Organ-system: Effects of Activation of the Adrenergic System

1. CVS:

- a. Heart: increased contraction rate and force, cardiac output, myocardial demand, and AV conduction
- b. Blood vessels and blood pressure: skin and mucous membrane blood vessels constrict.
 - Dilatation of skeletal muscle vessels
 - At low doses, adrenaline increases systolic and decreases diastolic blood pressure, but at higher doses, it increases both.
 - Both systolic and diastolic blood pressure are raised by noradrenaline.

2. Smooth Muscle:

- a. Bronchi: relaxation.
 - b. Uterus: relaxation of the pregnant uterus
 - c. GIT: relaxation of wall muscles and contraction of sphincters
 - d. Bladder: relaxation of detrusor muscle; contraction of sphincter and trigone muscle
3. **Eye:** mydriasis; reduction of intraocular pressure in normal and glaucomatous eyes
 4. **Respiration:** Bronchodilatation; relief of congestion; mild stimulation of respiration
 5. **Metabolic:** Increased hepatic glycogenolysis; decreased peripheral glucose intake; increased free fatty acids in the blood (lipolysis)
 6. **CNS:** excitement, vomiting, restlessness
 7. **Skeletal muscle:** facilitation of neuromuscular transmission and vasodilatation

Drugs Acting on the Adrenergic Receptor Subtypes

	α_1	α_2	β_1	β_2
Agonist	Phenylephrine Methoxamine	Clonidine Oxymetazoline	Dobutamine Isoproterenol Terbutaline	Salbutamol Terbutaline Isoetharine
Antagonist	Proposing Phentolamine Phenoxybenzamine	Yohimbine Phentolamine Phenoxybenzamine	Propranolol Pindolol Atenolol Metoprolol Tombolo	Propranolol Pindolol Butoxamine Tombolo

Adrenaline stimulates all the four receptor subtypes.

Noradrenaline stimulates both alpha and beta receptors, but has a low affinity for beta2. All beta receptors, as well as some alpha receptors, are blocked by labetalol.

ADRENALINE

The Adrenal medulla cells and chromaffin tissues in the body produce this, which is the prototype of adrenergic drugs.

Pharmacokinetics

In the gastrointestinal tract, adrenaline is rapidly destroyed, conjugated, and oxidised. Because it is ineffective when taken orally, it should be administered intramuscularly or subcutaneously. Intravenous injections are extremely dangerous and can cause ventricular fibrillation.

When its relaxing effect on the bronchi is desired, the drug can be administered via nebulizer or applied topically to mucus membranes to cause vasoconstriction. Because of the drug's extensive metabolism in the liver, only a small amount of it is excreted unchanged in the urine.

Pharmacodynamics

Adrenaline stimulates all adrenergic receptors directly, as well as causing sympathetic nerve stimulation. Depending on the type of receptor stimulated, its action can be divided into two categories.

Vasoconstriction in the skin and viscera, mydriasis, platelet aggregation, and a slight increase in blood glucose are among the side effects. The β effects include increased contractility and heart rate with a shorter refractory period (β_1), muscle and coronary vessel vasodilation (β_2), bronchial and uterine relaxation (β_2), hyperglycemia, lactic acidemia, and an increase in circulating free fatty acids (β_2).

Indications

1. Bronchial asthma (acute)
2. Anaphylaxis
3. Local haemostatic to stop epistaxis bleeding
4. To prolong the action, use local anaesthesia.
5. Atrial fibrillation

Adverse reactions

1. Anxiety, restlessness, tremor in the head
2. Anginal pain
3. Arrhythmias and palpitations in the heart
4. A rapid increase in blood pressure
5. Extreme vasoconstriction with gangrene of the extremities
6. Conjunctival hyperemia, tears

Contra Indications

- i. Coronary Diseases
- ii. Hypertension
- iii. Injection Around End Arteries
- iv. Digitalis Therapy
- v. Hyperthyroidism

NOR ADRENALINE

The neurochemical mediator released by nerve impulses and various drugs from postganglionic adrenergic nerves is not adrenaline. It also accounts for 20% of the catecholamine output of the adrenal medulla.

Pharmacokinetics

Because noradrenaline, like adrenaline, is ineffective when taken orally, it must be administered intravenously with caution. Because of its strong vasoconstrictor effect, which causes necrosis and sloughing, it is not given subcutaneously or intramuscularly. Only a small amount is excreted unchanged in urine, similar to adrenaline.

Pharmacodynamics

When compared to adrenaline, nor adrenaline is primarily a receptor agonist with less agonist action. Indication In hypotensive states, adrenaline is not used as a hypertensive agent. For example, during spinal anaesthesia or following sympathectomy.

Adverse effects include

- Common side effects include anxiety, headaches, and bradycardia.
- Severe hypertension in people who are hypersensitive
- The drug's extravasation causes necrosis and sloughing.

Isoprenaline Dopamine, Dobutamine

These are catecholamines that have properties similar to adrenaline and noradrenaline. Dopamine is a precursor to noradrenaline and is found in nature. Isoprenaline and dobutamine, the other two, are synthetic. These drugs have an advantage over the others because their action is more selective, resulting in fewer side effects than adrenaline and no adrenaline. Dopamine and dobutamine are two drugs that can be used to treat shock.

NON- CATECHOLAMINES

The majority of non-catecholamines work by releasing physiologic catecholamines from postganglionic nerve endings.

EPHEDRINE

Pharmacokinetics

Ephedrine is absorbed from all parenteral sites as well as the gastrointestinal tract. It has a wide distribution throughout the body and is resistant to liver enzyme hydrolysis. The majority of the drug is excreted in the urine unchanged. It has a longer duration of action than catecholamines due to its metabolic stability.

Pharmacodynamics

Both α and β receptors are stimulated by ephedrine. The drug's effect on various organs and systems is similar to that of adrenaline, partly through direct action on receptors and partly through indirect action on tissue stores of noradrenaline. It also has a mild CNS stimulant effect.

Indications:

1. Mydriasis
 2. Heart block
 3. Nocturnal enuresis
 4. Bronchial asthma: - usually as a prophylactic for prevention of attacks
 5. Nasal decongestion
- Side effects

The side effects are similar to those of adrenaline, but it can also cause insomnia and urine retention.

Contraindications

They are the same as Adrenaline.

The rest of the catecholamines are classified based on their selectivity for specific receptors, but it is extremely difficult to exhaust all of them. More specifically, where they are clinically indicated, their effect and pharmacology are discussed.

ADRENERGIC BLOCKERS

Adrenergic receptor blockers may be considered in two groups:

1. α adrenergic receptor blocker
2. Adrenergic receptor blocker

These drugs block the effects of adrenaline, noradrenaline, and other sympathomimetic amines, whether they are released naturally or injected. Catecholamines in the blood are more easily antagonised than the effects of sympathetic nerve stimulation. The drugs work by competing for or receptors on the effector organs with catechoamines. They have no effect on the substances' production or release.

Adrenergic Blockers: Antibodies to the alpha adrenergic receptor can be reversible or irreversible. Reversible antagonists, such as phentolamine, tolazoline, prazosin, yohimbine, and others, dissociate from receptors. Irreversible antagonists, such as phenoxybenzamine, bind to the receptor so tightly that their effects may last long after the drug has been cleared from the plasma.

Pharmacologic Effects: Anti-alpha receptor drugs lower blood pressure and peripheral vascular resistance. As a result, postural hypotension and reflex tachycardia are common side effects of these medications. Other minor effects include miosis, nasal stuffiness, etc.

Prazosin: This medication is effective in the treatment of hypertension. It has a strong affinity for the α_1 receptor and a weak affinity for the α_2 . Because α_1 receptors are blocked by prazosin, both arterial and venous smooth muscles relax. As a result, it reduces blood pressure, venous return, and cardiac output. It also lowers the tone of the urinary bladder's internal sphincter.

Indications

- Essential hypertension
- Benign prostatic hyperplasia
- Raynaud's syndrome

ADRENERGIC BLOCKING DRUGS

The selectivity for receptors in different tissues of the β - adrenergic receptor blocking drugs in use can be classified.

1. Non-selective beta blockers, such as propranolol, pinadolol, and timolol, block all of adrenaline's receptor effects.
2. Drugs that block only the β_1 effects (those on the heart) while having little effect on the bronchi and blood vessels (β_1 -selective blockers), such as atenolol, practalol, acebutalol, and so on.

PROPRANOLOL

Propranolol is a non-selective β adrenergic blocker; it has also other actions like membrane stabilization.

Pharmacokinetics: Following oral administration, propranolol is almost completely absorbed. However, the liver metabolises the majority of the administered dose, leaving only 1/3 of it to reach the systemic circulations. It binds to plasma to the tune of 90-95 percent. It is passed through the urine.

Pharmacodynamics

The drug has the following main actions.

1. Cardiovascular system
 - Bradycardia
 - Reduces force of contraction
 - Reduces blood pressure
2. Central nervous system
 - Anti-anxiety action
3. Respiratory system
 - Bronchoconstriction
4. Kidneys:
 - Decrease renin secretion
5. Metabolic system
 - Hypoglycemia
6. Eye
 - Decrease the rate of Aqueous humor production

Indications

- Cardiac arrhythmias
- Myocardial infarction

- Hypertension
- Prophylaxis against angina
- Prophylaxis against migraine attacks
- Anxiety states (suppression of the physical manifestations of situational anxiety)
- Thyrotoxicosis
- Glaucoma

Adverse reactions

- Heart failure
- Bronchospasm
- Heart block
- GI disturbances like nausea, vomiting
- Hypotension and severe bradycardia
- Allergic reaction
- Withdrawal symptoms in case of abrupt discontinuation
- Vivid dreams night mare and hallucinations
- Masking of hypoglycemia in diabetic patients
- Cold hands

Contraindications and Precautions:

- Bronchial asthma
- Diabetes mellitus
- Heart failure
- Peripheral vascular disease

Questions

- i. What is the autonomic nervous system?
- ii. How are drugs affecting the Autonomic nervous system classified?
- iii. Discuss the effects of Acetylcholine.
- iv. Discuss the effects and clinical uses of atropine.
- v. Discuss the effects of Adrenaline.
- vi. Discuss the effects and contraindications of propranolol.