

A REVIEW OF ANTI HYPERTENSIVE DRUGS

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ABSTRACT

Hypertensive drugs play a crucial role in managing hypertension, reducing blood pressure and preventing cardiovascular complications. The review highlights innovative approaches in hypertension management. The traditional treatment like Diuretics, beta blockers, ACE inhibitors, calcium channel blockers and the novel mechanisms of drugs include RNA based therapy, natriuretic peptide based therapies, endothelin receptor antagonist, NAD⁺-boosting compounds, gut microbiome targeted therapies helps in advanced way to improve blood pressure control and offer cardio renal benefits while reducing adverse effects including hyperkalemia. This article provides an overview on traditional drugs and how novel strategies can overcome in treatment of hypertension.

KEYWORDS: *Haemodynamic, Electrophysiology of heart, Traditional Antihypertensive drugs, Novel mechanism in treating hypertension.*

INTRODUCTION

HEAMODYNAMIC

Hemodynamics is the study of blood flow, blood pressure, and the circulatory system's resistance to blood flow. It involves analyzing the forces that drive blood through blood vessels and the heart's pumping action. Understanding hemodynamics is crucial for diagnosing and managing cardiovascular diseases.

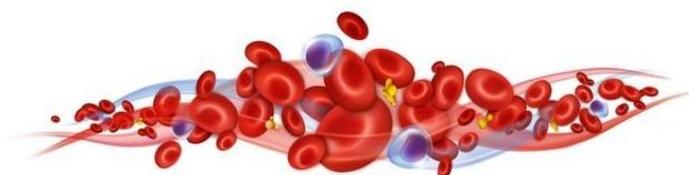


Figure No. 1: Blood Circulation.

Components of Hemodynamics

1. **Blood Pressure (BP):** The pressure exerted by blood on the walls of blood vessels, measured in millimeters of mercury (mmHg).
2. **Cardiac Output (CO):** The volume of blood pumped by the heart per minute, measured in liters per minute (L/min).
3. **Systemic Vascular Resistance (SVR):** The resistance to blood flow in the systemic circulation, measured in dynes/second/cm⁵.
4. **Blood Viscosity:** The thickness and stickiness of blood, affecting its flow through blood vessels.
5. **Vascular Compliance:** The ability of blood vessels to expand and accommodate changes in blood pressure.

Hemodynamic Parameters

1. **Mean Arterial Pressure (MAP):** The average pressure in the arteries during a cardiac cycle, calculated as $(2 \times \text{diastolic BP} + \text{systolic BP})/3$.
2. **Central Venous Pressure (CVP):** The pressure in the superior and inferior vena cava, reflecting right atrial pressure.
3. **Pulmonary Artery Pressure (PAP):** The pressure in the pulmonary artery, reflecting right ventricular pressure.
4. **Pulmonary Capillary Wedge Pressure (PCWP):** The pressure in the pulmonary capillaries, reflecting left atrial pressure.
5. **Cardiac Index (CI):** The cardiac output adjusted for body surface area, measured in L/min/m².

Hemodynamic States

1. **Normotension:** Normal blood pressure, typically defined as systolic BP <120 mmHg and diastolic BP <80 mmHg.
2. **Hypertension:** Elevated blood pressure, typically defined as systolic BP \geq 140 mmHg and diastolic BP \geq 90 mmHg.
3. **Hypotension:** Low blood pressure, typically defined as systolic BP <90 mmHg.
4. **Shock:** A life-threatening condition characterized by inadequate tissue perfusion, often accompanied by hypotension.

Factors Affecting Hemodynamics

1. **Heart Rate:** An increase in heart rate can increase cardiac output, while a decrease can decrease cardiac output.
2. **Contractility:** An increase in contractility can increase cardiac output, while a decrease can decrease cardiac output.
3. **Preload:** An increase in preload can increase cardiac output, while a decrease can decrease cardiac output.
4. **Afterload:** An increase in afterload can decrease cardiac output, while a decrease can increase cardiac output.
5. **Blood Volume:** An increase in blood volume can increase cardiac output, while a decrease can decrease cardiac output.

Interventions to manage cardiovascular disorders.

The Conduction System of the Heart

The conduction system of the heart is a complex network of specialized cells and fibers that generate and conduct electrical impulses to regulate the heartbeat. The conduction system consists of the following components:

1. **SA Node (Sinoatrial Node):** Located in the right atrium, the SA node is the natural pacemaker. It generates electrical impulses at a rate of 60-100 beats per minute (bpm).
2. **AV Node (Atrioventricular Node):** Located between the atria and ventricles, the AV node relays the electrical impulse from the SA node to the ventricles. It also acts as a delay, allowing the atria to fully contract before the ventricles contract.
3. **Bundle of His:** A bundle of specialized fibers that carries the electrical impulse from the AV node to the ventricles.
4. **Bundle Branches:** The Bundle of His divides into left and right bundle branches, which carry the electrical impulse to the left and right ventricles, respectively.
5. **Purkinje Fibers:** A network of fibers that spread the electrical impulse throughout the ventricles, causing them to contract.

The Conduction Pathway

1. The SA node generates an electrical impulse, which spreads through the atria, causing them to contract.
2. The impulse reaches the AV node, which relays it to the Bundle of His.
3. The Bundle of His carries the impulse to the bundle branches, which divide into left and right branches.
4. The impulse travels through the bundle branches to the Purkinje fibers, which spread it throughout the ventricles.
5. The ventricles contract, pumping blood out of the heart.

Electrical Activity of the Heart

The electrical activity of the heart can be recorded using an electrocardiogram (ECG). The ECG tracing shows the following waves and intervals:

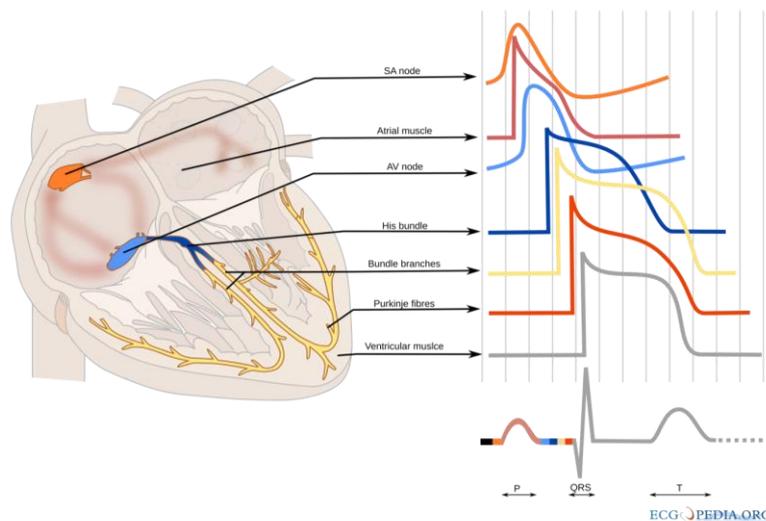


Figure No. 2: ECG waves arising from various parts of the Heart

1. **P Wave:** Represents *atrial depolarization* (contraction).
2. **QRS Complex:** Represents *ventricular depolarization* (contraction).
3. **T Wave:** Represents *ventricular repolarization* (relaxation).
4. **PR Interval:** Measures the time *between atrial and ventricular depolarization*.
5. **QT Interval:** Measures the time *between ventricular depolarization and repolarization*.

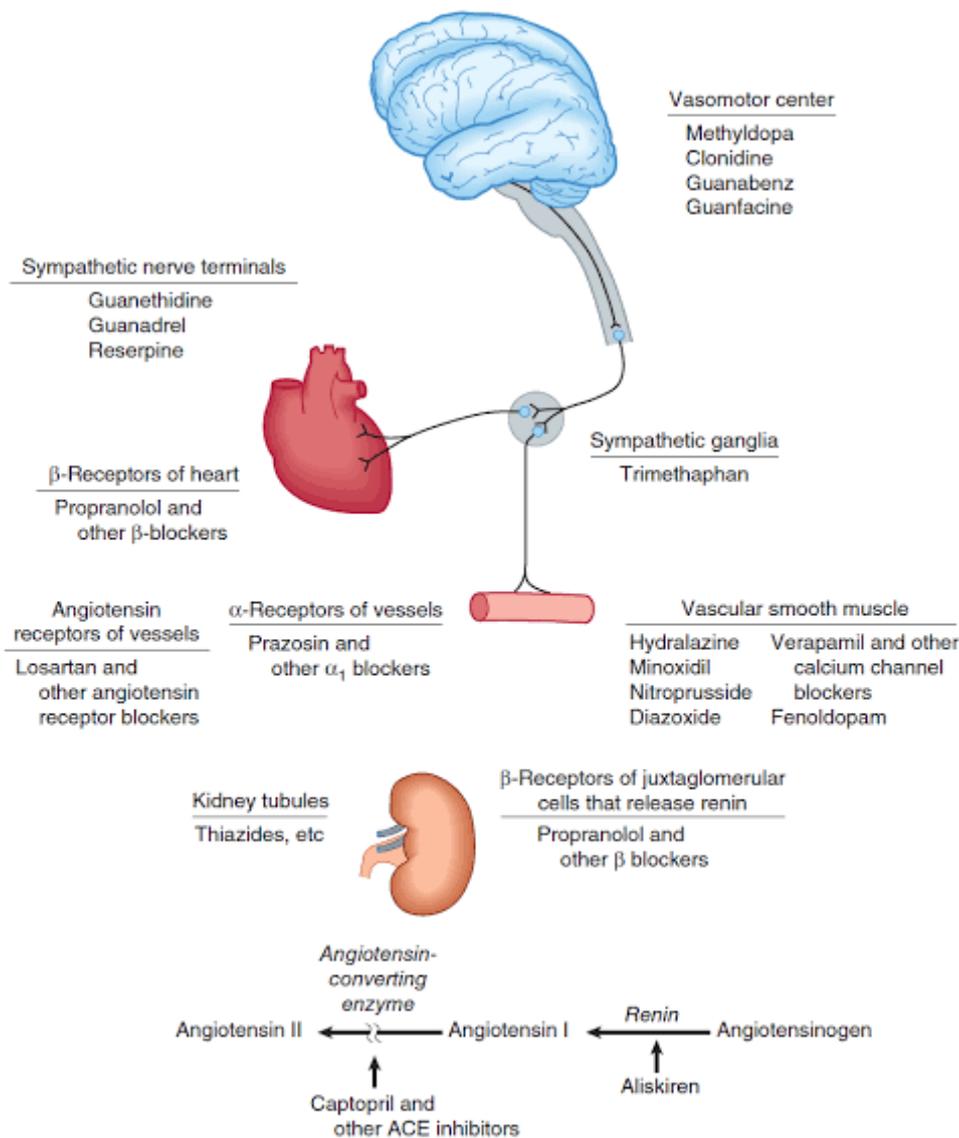
Abnormalities in Conduction

Abnormalities in conduction can lead to arrhythmias, which are irregular heart rhythms. Some common arrhythmias include:

1. **Atrial Fibrillation:** Rapid, irregular atrial contractions.
2. **Ventricular Tachycardia:** Rapid, abnormal ventricular contractions.
3. **Heart Block:** Delayed or blocked conduction through the AV node or bundle branches.
4. **Bundle Branch Block:** Delayed or blocked conduction through one of the bundle branches.
5. Let's dive into drugs used to treat *CVS disorder*

Antihypertensive Drugs

Antihypertensive drugs decrease elevated blood pressure by interfering with normal BP regulation mechanisms.



Sites of action of the major classes of antihypertensive drugs.
 Source : Basic & Clinical Pharmacology 14th Edition

Figure No. 3: Showing mechanism of action for traditional drugs.

The Traditional drugs used in the management of hypertension

Let us see how traditional drugs work in the treatment of hypertension.

Major classes include

1. Diuretics

Diuretics have been the standard antihypertensive drugs over the past nearly 5 decades, though they do not lower BP in normotensives.

- **Mechanism:** Increase urine production, reducing fluid volume and blood pressure.
- **Examples:** *Thiazides (e.g., hydrochlorothiazide), Loop diuretics (e.g., furosemide), Potassium-sparing diuretics (e.g., spironolactone)*
- **Action:** Diuretics work by inhibiting sodium reabsorption in the kidneys, leading to increased urine production and reduced fluid volume. This decreases blood pressure by reducing cardiac output and peripheral resistance.

2. Beta Blockers

They are mild antihypertensive and do not significantly lower BP in normotensives. Used alone they suffice in 30-40% patients mostly stage I cases. Additional BP lowering may be obtained when combined with other drugs.

- **Mechanism:** Reduce heart rate and contractility, decreasing cardiac output and blood pressure.
- **Examples:** *Non-selective beta blockers (e.g., propranolol), Beta-1 selective blockers (e.g., metoprolol), Alpha-beta blockers (e.g., carvedilol)*
- **Action:** Beta blockers work by blocking beta-1 receptors in the heart, reducing heart rate and contractility. This decreases cardiac output and blood pressure. Beta blockers also reduce sympathetic tone, decreasing peripheral resistance.

3. Angiotensin-Converting Enzyme (ACE) Inhibitors

The ACE inhibitors are one of the first choice drugs in all grades of essential as well as renovascular hypertension (except those with bilateral renal artery stenosis).

- **Mechanism:** inhibit conversion of angiotensin I to angiotensin II, reducing vasoconstriction and blood pressure.
- **Examples:** Lisinopril, Enalapril, Captopril
- **Action:** ACE inhibitors work by blocking the conversion of angiotensin I to angiotensin II, a potent vasoconstrictor. This reduces peripheral resistance and blood pressure. ACE inhibitors also increase bradykinin levels, leading to vasodilation.

4. Calcium Channel Blockers

Calcium channel blockers are another class of first line antihypertensive drugs.

- **Mechanism:** Inhibit calcium influx into smooth muscle cells, reducing vasoconstriction and blood pressure.
- **Examples:** *Dihydropyridines (e.g., amlodipine), Non-dihydropyridines (e.g., verapamil, diltiazem)*
- **Action:** Calcium channel blockers work by inhibiting calcium influx into smooth muscle cells, reducing vasoconstriction and peripheral resistance. This decreases blood pressure and reduces cardiovascular risk.

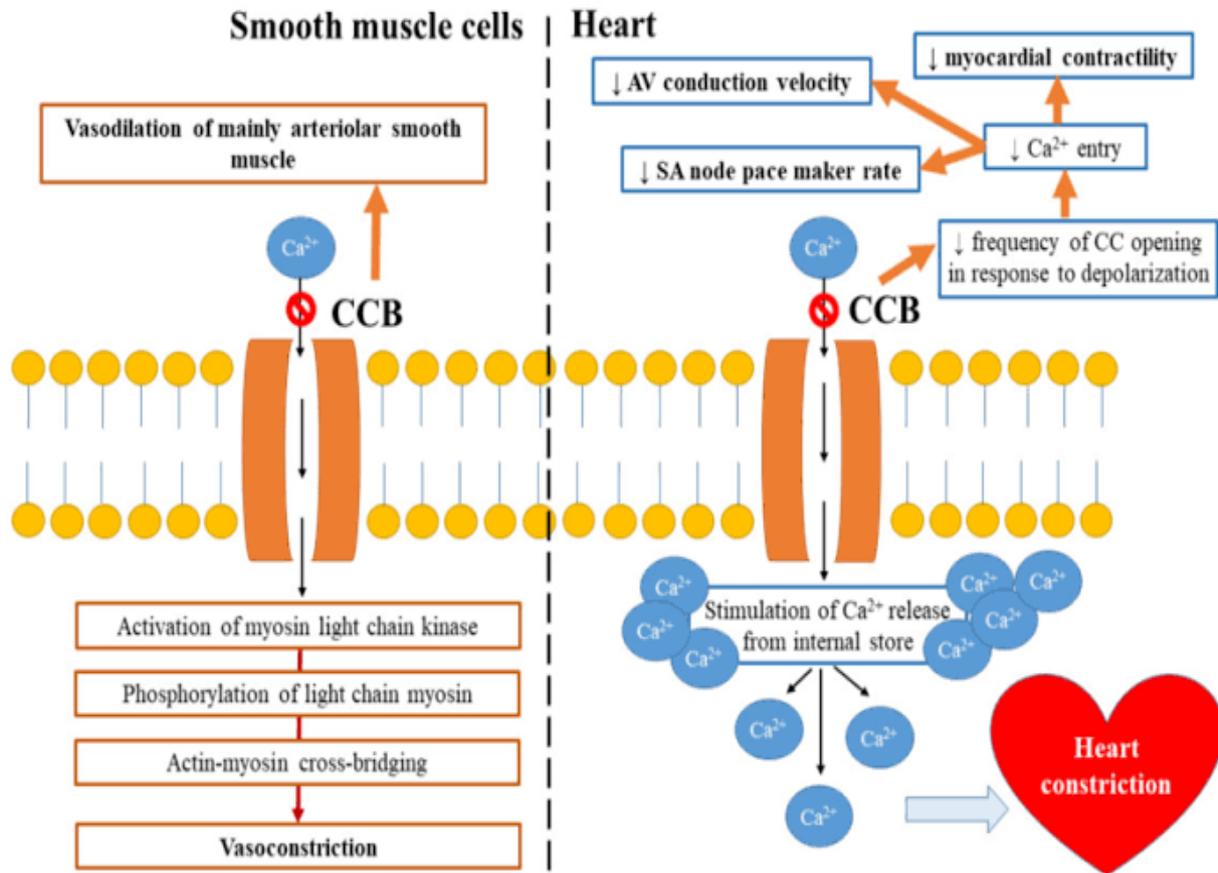


Figure No. 4: showing representation of calcium channel blockers.

Table 1: showing traditional drugs used for the treatment of hypertension and cardiovascular related disorders.

DRUG CLASS	MECHANISM OF ACTION	EXAMPLES	SIDE EFFECTS
Diuretics	Thiazide and loop diuretics increase renal excretion of sodium and water (Na&H ₂ O), lowering blood volume.k ⁺ sparing diuretics reduce sodium reabsorption while retaining potassium.	THIAZIDE: hydrochlorothiazide, chlorothalidone. LOOP: furosemide, bumetanide etc.... K ⁺ SPARING: spironolactone, eplerenone	Hypokalaemia, dehydration, gynecomastia.
Beta blockers	Blocks beta 1 adrenoreceptors reduce heart rate and cardiac output.Some beta blockers also have alpha blocking effects.	Metoprolol,atenolol,carvedilol, bisoprolol	Bradycardia, fatigue, potential, reduced diabetes.
Ca ⁺ channel blockers	Inhibit calcium influx into vascular smooth muscle and cardiac cells promoting vasodilation and decreasing cardiac.	Dihydropyridines: Amlodipine, felodipine	Peripheral oedema, headache.

NOVEL MECHANISMS AND TARGETS

This novel drugs include Advancing Antihypertensive Drug Development Emerging Therapeutic Strategies and Novel Targets.

1. RNA-based Therapy: Antisense Oligonucleotides (ASOs)

RNA-based therapies are a new class of treatments that target the genetic roots of disease. Antisense oligonucleotides (ASOs) are a type of RNA-based therapy that can specifically target and modulate the expression of genes involved in disease.

Mechanism of Action (MoA): ASOs are synthetic single-stranded nucleotides designed to specifically bind to target messenger RNA (mRNA) molecules. By binding to mRNA, ASOs can modulate gene expression by either degrading the mRNA or blocking its translation into protein. In the context of hypertension, ASOs can target genes involved in blood pressure regulation, such as angiotensinogen (AGT) mRNA.

Example: IONS-AGT-LRx is an ASO that targets AGT mRNA, which codes for angiotensinogen, a precursor to angiotensin II, a potent vasoconstrictor. By reducing AGT expression, IONS-AGT-LRx can lower blood pressure.

2. Natriuretic Peptide-Based Therapies

Natriuretic peptides (NPs) are a family of hormones that play a crucial role in regulating blood pressure and cardiovascular function. NP-based therapies aim to enhance the protective effects of NPs, promoting vasodilation, natriuresis, and diuresis.

Mechanism of Action (MoA): NP-based therapies enhance the protective effects of NPs by either increasing their levels or mimicking their actions. This leads to increased cyclic guanosine monophosphate (cGMP) production, promoting smooth muscle relaxation, vasodilation, and reduced blood pressure.

Example: Sacubitril/valsartan is a combination therapy that includes a neprilysin inhibitor (sacubitril) and an angiotensin receptor blocker (valsartan). Neprilysin is an enzyme that breaks down NPs, so inhibiting it increases NP levels, promoting vasodilation and reducing blood pressure.

3. Endothelin Receptor Antagonists (ERAs)

Endothelin-1 (ET-1) is a potent vasoconstrictor that plays a significant role in blood pressure regulation. ERAs are a class of medications that target the endothelin system, blocking the effects of ET-1 and promoting vasodilation.

Mechanism of Action (MoA): ERAs block the effects of ET-1 by antagonizing its receptors, leading to vasodilation and reduced blood pressure. ET-1 is a potent vasoconstrictor that also promotes fibrosis, inflammation, and vascular remodeling.

Example: Bosentan is a dual endothelin receptor antagonist that blocks both ETA and ETB receptors. By blocking ET-1's vasoconstrictive effects, bosentan can reduce blood pressure and improve cardiovascular outcomes.

4. NAD⁺ Boosting Compounds

Nicotinamide adenine dinucleotide (NAD⁺) is a coenzyme involved in various cellular processes, including energy metabolism and DNA repair. NAD⁺ levels decline with age and boosting NAD⁺ levels has been shown to improve vascular function and reduce blood pressure.

Mechanism of Action (MoA): NAD⁺ boosting compounds, such as nicotinamide mononucleotide (NMN) and nicotinamide riboside (NR), increase NAD⁺ levels in cells. This leads to improved vascular function, reduced oxidative stress, and lower blood pressure.

Example: NMN (nicotinamide mono nucleotide) and NR (nicotinamide riboside) are being investigated for their potential to improve cardiovascular health and reduce blood pressure.

5. Gut Microbiome-Targeted Therapies

The gut microbiome plays a significant role in blood pressure regulation and modulating the gut microbiome may help reduce blood pressure. Gut microbiome-targeted therapies aim to promote a healthy gut microbiome and reduce blood pressure.

Mechanism of Action (MoA): Gut microbiome-targeted therapies modulate the gut microbiome, leading to changes in the production of short-chain fatty acids (SCFAs) and other metabolites. SCFAs can promote vasodilation, reduce inflammation, and lower blood pressure.

Example: Prebiotic fiber can increase the production of ShortChainFattyAcids, such as acetate and butyrate, which can help regulate blood pressure. Probiotics and other gut microbiome-modulating therapies are also being explored for their potential to reduce blood pressure.

CONCLUSION

The management of hypertension has evolved significantly with the development of various antihypertensive drugs, including traditional agents such as diuretics, beta blockers, ACE inhibitors, and calcium channel blockers. These medications have been effective in reducing blood pressure and cardiovascular risk. Despite the challenges posed by economic factors, existing generic medications (i.e. traditional drugs) and effective combination therapies the development of innovative antihypertensive drugs represents a new better outcome in treating hypertension by developing new medications like novel therapeutic strategies, such as RNA-based therapies (e.g., antisense oligonucleotides), natriuretic peptide-based therapies (e.g., sacubitril/valsartan), endothelin receptor antagonists (e.g., bosentan), NAD⁺ boosting compounds (e.g., NMN, NR), and gut microbiome-targeted therapies, are emerging as promising approaches to treat hypertension.

The integration of these traditional and novel agents may provide a more comprehensive and personalized treatment approach, enabling healthcare providers to tailor therapy to individual patient needs and improve blood pressure control. Fixed-dose combination pills (FDCs) incorporating both traditional and emerging therapies are expected to simplify treatment regimens, enhance patient adherence, and ultimately achieve better cardiovascular outcomes.

REFERENCES

1. Rang H. P., Dale M. M., Ritter J. M., Flower R. J., Rang and Dale's Pharmacology.
2. Churchill Livingstone Elsevier.
3. Katzung B. G., Masters S. B., Trevor A. J., Basic and clinical pharmacology, Tata Mc Graw-Hill.
4. Goodman and Gilman's, The Pharmacological Basis of Therapeutics.
5. Basic Knowledge of Pharmacology BY Roland Seifert, Springer.
6. Marry Anne K. K., Lloyd Yee Y., Brian K. A., Robbin L.C., Joseph G. B., Wayne A. K., Bradley.
7. R.W., Applied Therapeutics, The Clinical use of Drugs. The Point Lippincott Williams & Wilkins.
8. Mycek M. J, Gelnet S. B and Perper M.M. Lippincott's Illustrated Reviews-Pharmacology.
9. K. D. Tripathi. Essentials of Medical Pharmacology, JAYPEE Brothers Medical Publishers (P) Ltd, New Delhi.
10. Sharma H. L., Sharma K. K., Principles of Pharmacology, Paras medical publisher Modern Pharmacology with clinical Applications, by Charles R. Craig & Robert.
11. Ghosh MN. Fundamentals of Experimental Pharmacology. Hilton & Company, Kolkata.

12. Kulkarni SK. Handbook of experimental pharmacology. Vallabh Prakashan.
13. N. Udupa and P.D. Gupta, Concepts in Chronopharmacology.
14. Essentials of Experimental Pharmacology, General Concepts by Sunil Bothra, Pharmamed Press.
15. Opie LH. Drugs for the Heart. 8th ed. Philadelphia, PA: Elsevier, 2013.
16. Katzung BG, Masters SB, Trevor AJ. Basic & Clinical Pharmacology. 14th ed. New York, NY: McGraw-Hill, 2018.