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PRESCRIPTION PATTERN OF ANTIHYPERTENSIVE MEDICATION AMONG HYPERTENSIVE OUTPATIENTS IN COMMUNITY PHARMACY

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ABSTRACT

Aim: To study the prescription pattern of antihypertensive medication among hypertensive patients in community pharmacy. **Methodology:** A cross sectional study was conducted for a period of 2 months in community pharmacy of Kanyakumari district. A total of 100 patients of either gender was included in the study. Patients were asked for their age, gender, previous medication, current medication for the accurate analysis of antihypertensive drugs prescribed o the patients. **Result:** A total of 100 patients were analyzed for determining the prescription pattern of antihypertensive drugs. Males (69%) were the major hypertensive patients compared to that of females (31%). The major antihypertensive agents were Calcium Channel Blockers (42%). Drugs such as amlodipine, telmisartan were frequently sold drugs. **Conclusion:** Calcium channel blockers were the frequently used drugs by the patients. 90% of the drugs were used in monotherapy as well as combination therapy according to the JNC 8 guidelines.

KEYWORDS: Hypertension, JNC 8 (joint national committee), Prescription.

1. INTRODUCTION

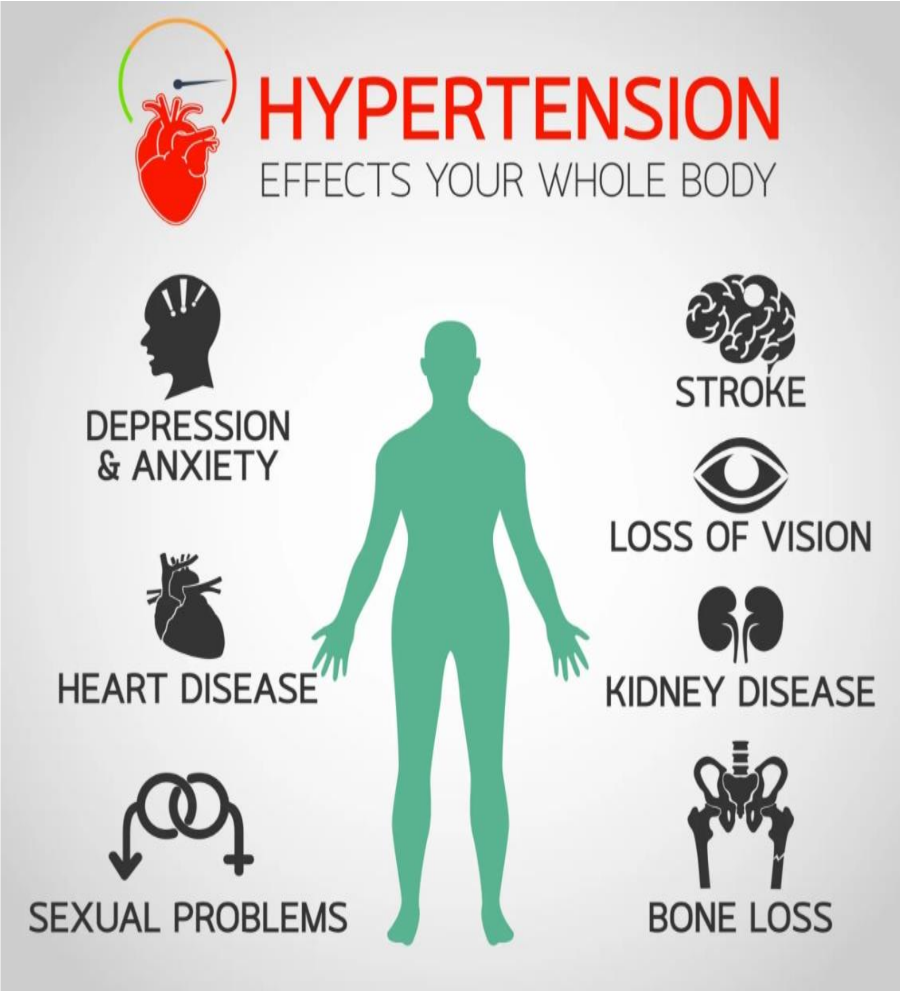
Hypertension, also known as high blood pressure, is a condition where the force of the blood against the walls of the arteries is consistently too high. This increased pressure can strain the heart and damage blood vessels over time, leading to serious health problems like heart disease, stroke, kidney damage, and other complications.

Blood pressure is typically measured in two numbers.

- **Systolic pressure**: the higher number, which measures the pressure in the arteries when the heart beats.
- **Diastolic pressure**: the lower number, which measures the pressure in the arteries when the heart rests between beats.

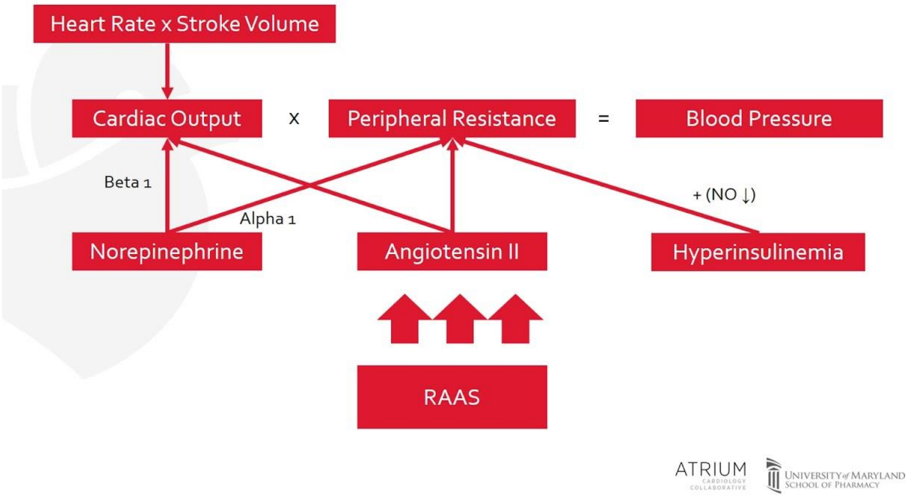
Normal blood pressure is usually around 120/80 mm Hg. Hypertension is diagnosed when blood pressure readings consistently exceed 130/80 mm Hg. There are two main types.

1. **Primary (essential) hypertension**: This type develops gradually over many years and has no clear cause. It's the most common form of hypertension.
2. **Secondary hypertension**: This type is caused by an underlying condition, such as kidney disease, hormonal disorders, or the use of certain medications.



1.1 PATHOPHYSIOLOGY OF HYPERTENSION

The pathophysiology of hypertension involves complex mechanisms that lead to sustained high blood pressure. Understanding these mechanisms helps in explaining how hypertension develops and progresses. Here's an overview of the key processes.



1.1.1. Increased Cardiac Output

- **Cardiac output** is the amount of blood the heart pumps per minute. When cardiac output increases (due to factors like increased heart rate or stroke volume), blood pressure rises. Conditions like hyperthyroidism or stress can increase heart rate and thus cardiac output, contributing to hypertension.

1.1.2. Increased Peripheral Vascular Resistance (PVR)

- Peripheral resistance is the resistance to blood flow in the small arteries and arterioles. If these blood vessels constrict, it increases resistance and raises blood pressure. This can occur due to:
  - **Vasoconstriction**: Constriction of the blood vessels, often due to the action of hormones like angiotensin II, norepinephrine, or endothelin.
  - **Endothelial Dysfunction**: The endothelium (the lining of blood vessels) becomes impaired, reducing its ability to produce nitric oxide (a vasodilator), leading to vasoconstriction and increased vascular resistance.

1.1.3. Renin-Angiotensin-Aldosterone System (RAAS)

- The **RAAS** plays a crucial role in regulating blood pressure. In situations where blood pressure drops or there’s decreased blood flow to the kidneys, the kidneys release **renin**.

Renin activates **angiotensin I**, which is converted into **angiotensin II** by the enzyme ACE (angiotensin-converting enzyme). Angiotensin II:

- Constricts blood vessels (raising PVR).
- Stimulates the release of **aldosterone**, which causes the kidneys to retain sodium and water, increasing blood volume and thus blood pressure.
- Stimulates the release of **antidiuretic hormone (ADH)**, promoting water retention by the kidneys.

1.1.4. Sympathetic Nervous System Activation

- Chronic activation of the sympathetic nervous system (SNS) can contribute to hypertension. The SNS releases neurotransmitters like **norepinephrine**, which causes:
  - **Vasoconstriction** in peripheral vessels, increasing resistance.
  - Increased **heart rate** and **contractility**, which raises cardiac output.
- Stress, obesity, and certain diseases can enhance SNS activity, contributing to sustained high blood pressure.

1.1.5. Salt Sensitivity and Sodium Retention

- The kidneys' ability to excrete sodium is critical in regulating blood pressure. In some individuals, **salt sensitivity** exists, where the body retains excess sodium, leading to increased blood volume and pressure. This occurs due to impaired renal function or increased sodium retention in response to hormonal signals like aldosterone and angiotensin II.

1.1.6. Vascular Remodeling

- Over time, sustained high blood pressure leads to **vascular remodeling**, where the structure of the blood vessels changes. The walls of arteries become thicker and stiffer (due to smooth muscle hypertrophy and collagen deposition), which increases **vascular resistance** and worsens hypertension.

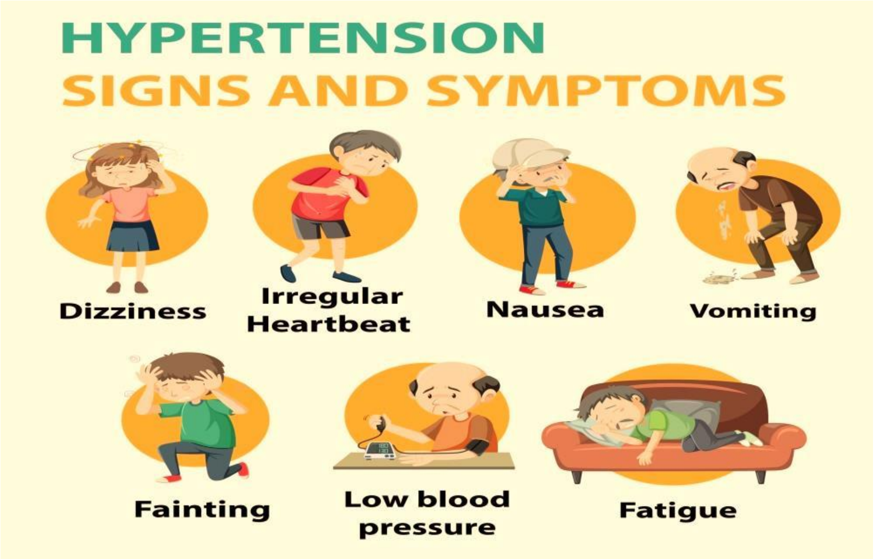
1.1.7. Genetic and Environmental Factors

- There is a hereditary component to hypertension, where genetic factors may influence how the body responds to salt, fluid balance, or stress. Environmental factors such as obesity, high salt intake, lack of physical activity, and chronic stress can exacerbate these genetic predispositions, contributing to the development of hypertension.

1.1.8. Endothelial Dysfunction

- In hypertension, the endothelial cells lining the blood vessels become dysfunctional. Normally, endothelial cells release nitric oxide (NO), a potent vasodilator that helps maintain blood flow and blood pressure. In hypertension, this ability is reduced, leading to **impaired vasodilation**, further increasing resistance and blood pressure.

1.2 SIGNS AND SYMPTOMS



Common Symptoms

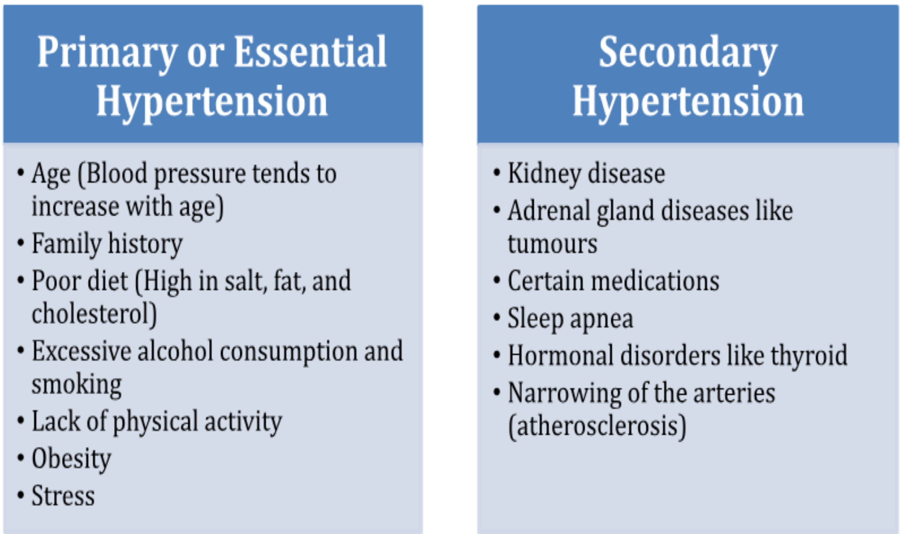
1. **Headaches**: Particularly in the morning, headaches can occur, and they may become more severe as blood pressure increases.
2. **Dizziness or Lightheadedness**: A feeling of dizziness may occur when standing up too quickly due to changes in blood flow.
3. **Shortness of Breath**: Difficulty breathing, especially during physical activity or even at rest, can be a sign of hypertension-related heart issues.
4. **Blurred Vision**: High blood pressure can damage the blood vessels in the eyes, leading to vision problems.
5. **Chest Pain**: Severe hypertension can strain the heart, leading to chest pain or angina. This could also be a sign of an impending heart attack.
6. **Nosebleeds**: Frequent or unexplained nosebleeds can sometimes be a result of high blood pressure.
7. **Fatigue**: Feeling tired or weak may indicate that your heart and other organs are struggling to function properly due to the strain of elevated blood pressure.

Severe or Uncontrolled Hypertension

When hypertension becomes very severe, the symptoms can become more noticeable:

- **Confusion or Cognitive Issues**: High blood pressure can affect brain function, leading to confusion, difficulty concentrating, or memory problems.
- **Nausea and Vomiting**: This can occur due to increased pressure on the brain.
- **Severe Chest Pain or Heart Attack**: This can indicate that the hypertension is causing damage to the heart or blood vessels.
- **Blood in Urine**: Kidney damage due to uncontrolled hypertension can lead to blood in the urine.

1.3 Causes



1.3.1. Genetic Factors

- Family history and genetic predisposition play a significant role in the development of hypertension. People with a family history of hypertension are more likely to develop high blood pressure themselves.

1.3.2. Obesity

- Excess weight puts extra strain on the heart and blood vessels, leading to an increased risk of high blood pressure.

1.3.3. Physical Inactivity

- Lack of regular physical activity can contribute to weight gain and higher blood pressure. Regular exercise helps in maintaining normal blood pressure levels.

1.3.4. Excessive Salt Intake

- High sodium intake leads to fluid retention, which increases blood volume and pressure within the blood vessels.

1.3.5. Alcohol and Tobacco Use

- Chronic alcohol consumption and smoking contribute to the development of hypertension by damaging the blood vessels and increasing heart rate.

1.3.6. Chronic Kidney Disease

- Impaired kidney function can lead to fluid retention and the production of hormones that increase blood pressure.

1.3.7. Age

- As people age, their blood vessels become less elastic, and the risk of developing high blood pressure increases.

1.3.8. Stress

- Chronic stress can lead to temporary increases in blood pressure, and if prolonged, may contribute to sustained hypertension.

1.3.9. Dietary Factors

- Diets high in fats, cholesterol, and refined sugars are linked to hypertension, as these can lead to weight gain, poor blood vessel function, and increased blood pressure.

1.3.10. Sleep Apnea

- Sleep apnea can cause fluctuations in blood oxygen levels during sleep, leading to increased blood pressure over time.

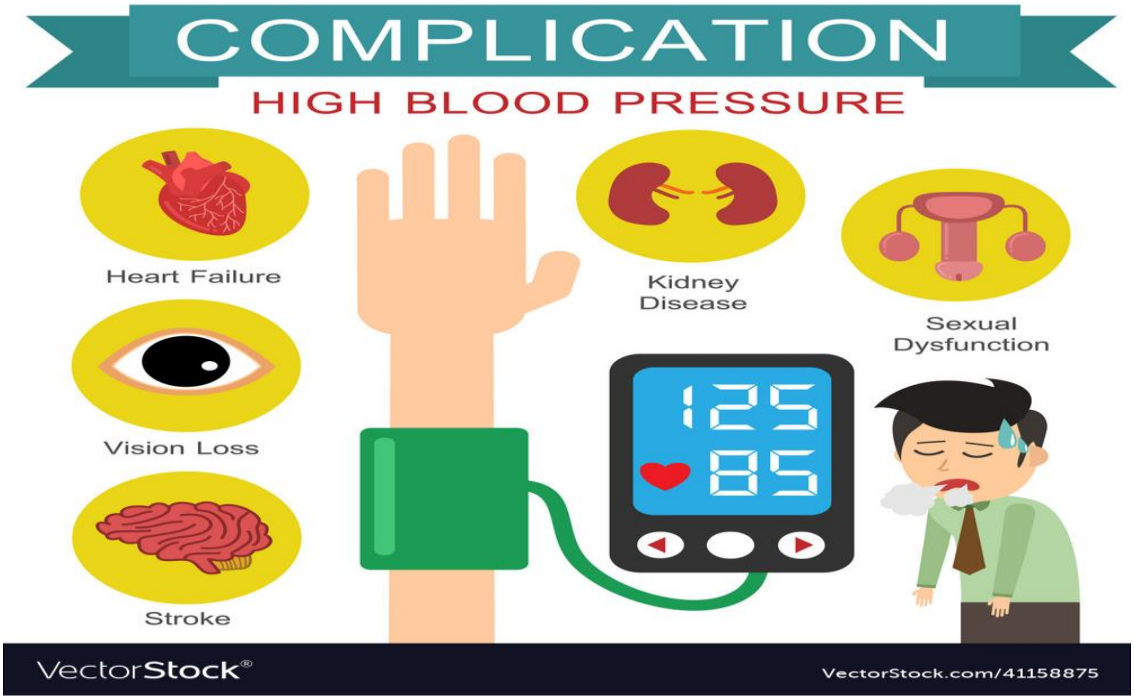
1.3.11. Endocrine Disorders

- Certain hormonal conditions, such as hyperthyroidism, Cushing's syndrome, or pheochromocytoma, can lead to secondary hypertension.

1.3.12. Medications

- Certain medications, such as no steroidal anti-inflammatory drugs (NSAIDs), steroids, and oral contraceptives, can elevate blood pressure.

1.4. COMPLICATION



1.4.1 Cardiovascular Disease

- **Coronary Artery Disease (CAD)**: Chronic high blood pressure can damage the blood vessels, increasing the risk of atherosclerosis (plaque buildup in the arteries), which can lead to heart attacks.
- **Heart Failure**: The heart must work harder to pump blood against the increased pressure in the arteries, potentially leading to heart failure over time.
- **Stroke**: High blood pressure can damage blood vessels in the brain, increasing the risk of ischemic stroke (caused by blockage) or hemorrhagic stroke (caused by bleeding).
- **Aneurysm**: High blood pressure can weaken blood vessels, leading to the formation of an aneurysm, which is a bulge that can rupture and cause life-threatening bleeding.

1.4.2 Kidney Damage (Chronic Kidney Disease)

- The kidneys filter excess waste and fluid from the blood, but high blood pressure can damage the blood vessels in the kidneys, impairing their ability to function and leading to kidney disease or kidney failure.

1.4.3 Vision Loss

- Hypertension can damage the blood vessels in the eyes, leading to conditions like retinopathy (damage to the retina) or even blindness.

1.4.4 Metabolic Syndrome

- High blood pressure is a key component of metabolic syndrome, a cluster of conditions that include elevated blood sugar, excess body fat around the waist, and abnormal cholesterol levels, which increases the risk of cardiovascular diseases and diabetes.

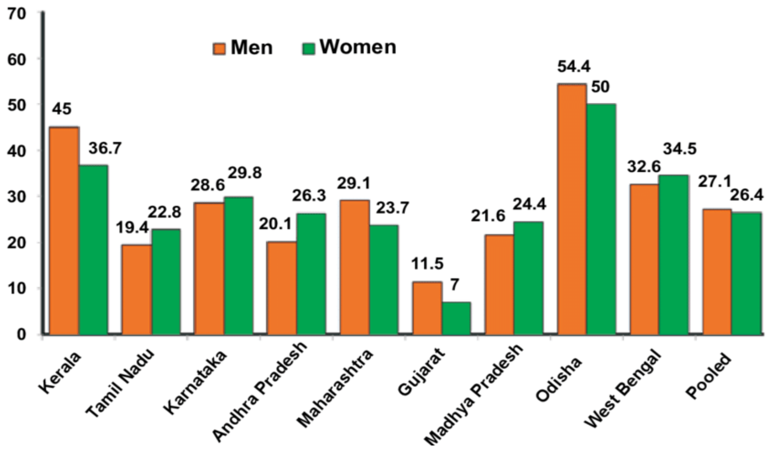
1.4.5 Cognitive Decline and Dementia

Chronic high blood pressure can contribute to cognitive decline and increase the risk of developing dementia, including Alzheimer's disease, as it can impair the blood flow to the brain.

1.4.6 Sexual Dysfunction

- Hypertension can lead to reduced blood flow, which may cause erectile dysfunction in men and lower libido in both men and women.

1.5 EPIDEMOLOGY



1.5.1. Global Prevalence

- **World Health Organization (WHO)** estimates that over **1.13 billion people** globally suffer from hypertension.
- Hypertension is responsible for **9.4 million deaths** each year due to complications like heart disease, stroke, and kidney failure.
- It affects approximately **30% to 45% of adults worldwide**, with rates varying by region, age, gender, and socioeconomic factors.

1.5.2. Age and Gender Distribution

- The prevalence of hypertension increases with age. It is more common in people over the age of **60**. In fact, by age 60, about **60% of people** are affected.

- Men tend to develop hypertension at an earlier age than women, but post-menopausal women experience a higher prevalence, approaching the rates seen in men.

1.5.3. Geographic Variations

- **Developed countries** generally have higher rates of hypertension due to lifestyle factors such as high salt intake, obesity, physical inactivity, and smoking.
- In **low- and middle-income countries**, hypertension is increasingly becoming a concern due to urbanization, dietary changes, and greater access to healthcare that improves diagnosis.

  According to the **Global Burden of Disease Study**, the highest rates of hypertension are found in **Eastern Europe, Sub-Saharan Africa, and the Middle East**.

1.5.4. Risk Factors

- **Modifiable risk factors** include high salt consumption, obesity, lack of physical activity, smoking, alcohol intake, and poor diet.
- **Non-modifiable risk factors** include age, family history, gender (male), and certain ethnic backgrounds (e.g., African Americans have a higher prevalence).

1.5.5. Hypertension and Comorbidities

- Hypertension is often accompanied by conditions such as **diabetes**, **high cholesterol**, and **obesity**, which amplify the risk of heart disease and stroke.
- It's also closely associated with chronic kidney disease and can lead to kidney failure if untreated.

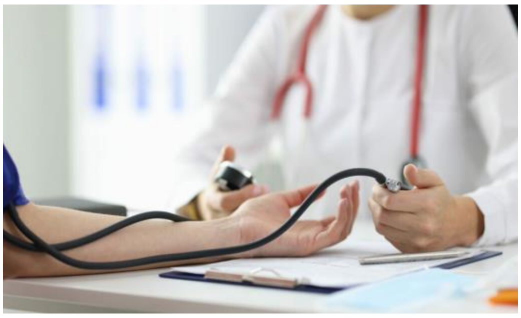
1.5.6. Trends Over Time

- The prevalence of hypertension has been increasing globally, mainly due to **population aging** and the **rise of lifestyle-related risk factors**.
- In many countries, **awareness, treatment, and control rates** for hypertension are still suboptimal, contributing to the burden of cardiovascular diseases.

1.5.7. Screening and Public Health Importance

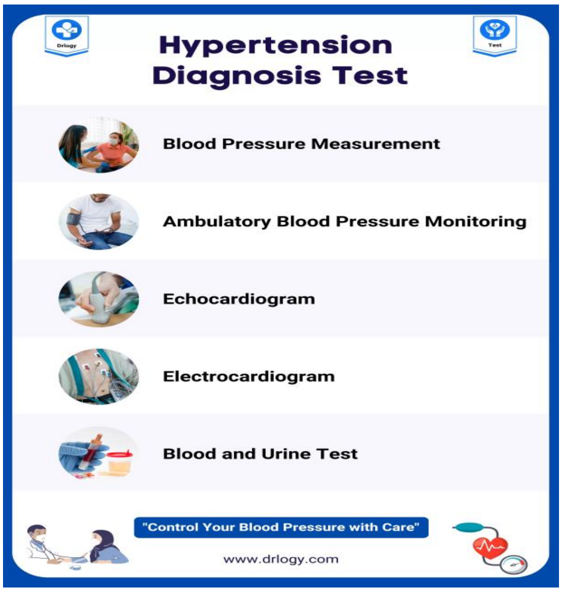
- Screening for hypertension is critical for early detection and management.
- According to studies like those from the **American College of Cardiology**, hypertension control rates are still below targets in many populations, emphasizing the need for better awareness and treatment programs.

1.6 WHEN TO SEE A DOCTOR?



- **Consistently High Readings:** If your blood pressure readings are consistently higher than normal (above 130/80 mmHg), it's time to consult a doctor. They can help you manage it and prevent complications.
- **Severe Symptoms:** If you experience symptoms like severe headaches, chest pain, difficulty breathing, vision changes, or dizziness, seek medical attention right away. These could be signs of a hypertensive emergency.
- **New or Worsening Symptoms:** If you've been diagnosed with hypertension and notice new or worsening symptoms, such as swelling, headaches, or fatigue, you should get checked by a healthcare provider.
- **Lack of Control with Medication:** If you're already on medication for hypertension but your blood pressure remains high, your doctor may need to adjust your treatment plan.
- **Other Risk Factors:** If you have additional risk factors, such as diabetes, obesity, or a family history of heart disease, you may need more frequent checkups.

1.7 DIAGNOSIS



1. **Blood Pressure Measurement:** The primary method for diagnosing hypertension is through repeated blood pressure measurements. Blood pressure is measured in millimeters of mercury (mmHg) and is recorded with two numbers:

- **Systolic blood pressure (the upper number):** The pressure in your arteries when your heart beats.
- **Diastolic blood pressure (the lower number):** The pressure in your arteries when your heart rests between beats.

According to the American College of Cardiology (ACC) and the American Heart Association (AHA), blood pressure categories are defined as:

- **Normal :** Systolic <120 mmHg and Diastolic <80 mmHg
- **Elevated :** Systolic 120-129 mmHg and Diastolic <80 mmHg
- **Hypertension Stage 1 :** Systolic 130-139 mmHg or Diastolic 80-89 mmHg
- **Hypertension Stage 2 :** Systolic ≥140 mmHg or Diastolic ≥90 mmHg
- **Hypertensive crisis :** Systolic >180 mmHg and/or Diastolic >120 mmHg

Reference: American College of Cardiology (ACC), American Heart Association (AHA) Hypertension Guidelines, 2017.

2. **Confirming the Diagnosis:** To confirm the diagnosis, blood pressure measurements are typically taken on at least two separate visits to ensure accuracy. It's important to measure blood pressure at different times of the day and under different conditions, as stress, caffeine, and physical activity can affect readings.

3. **Ambulatory Blood Pressure Monitoring (ABPM):** In certain cases, healthcare providers may recommend 24-hour ambulatory blood pressure monitoring. This device measures blood pressure at regular intervals throughout the day and night, giving a more accurate picture of how blood pressure fluctuates over time.

4. **Home Blood Pressure Monitoring:** Patients may be instructed to take their blood pressure at home using a digital blood pressure cuff to ensure consistency in the measurements.

Risk Factors for Hypertension include

- Age
- Family history

- Obesity
- Sedentary lifestyle
- Poor diet (especially high salt intake)
- Excessive alcohol consumption
- Smoking
- Chronic stress.

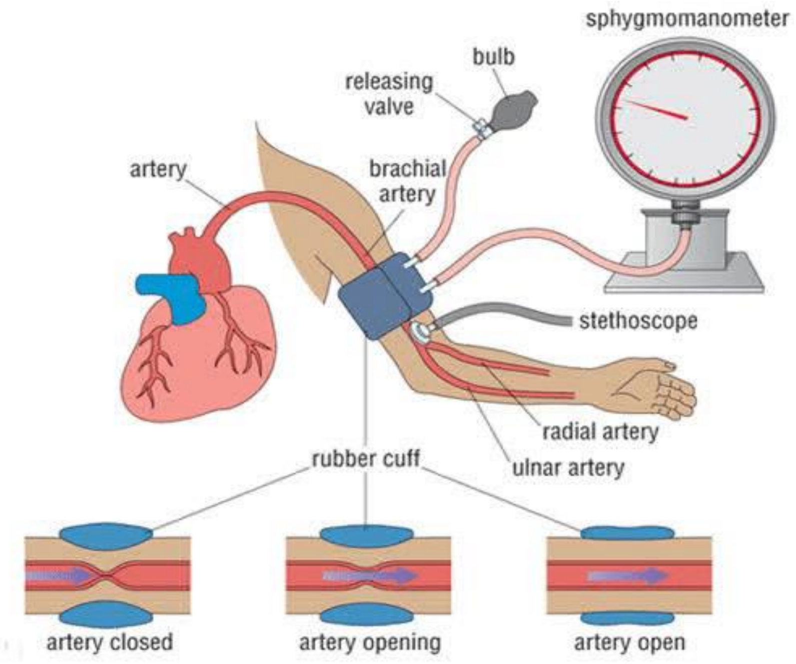
Associated Complications

- Cardiovascular diseases, such as heart attack and stroke
- Kidney damage
- Vision loss
- Cognitive decline.

Further Testing: If hypertension is diagnosed, additional tests may be performed to assess organ damage and identify the underlying cause (if secondary hypertension is suspected). These may include:

- Blood tests (to check kidney function and cholesterol levels)
- Urinalysis
- Electrocardiogram (ECG)
- Echocardiogram (ultrasound of the heart).

1.8 BLOOD PRESSURE MEASUREMENT



Blood pressure measurement is crucial in diagnosing and managing hypertension. Hypertension, or high blood pressure, is a condition where the force of the blood against the walls of the arteries is consistently too high. It is typically diagnosed when blood pressure readings are consistently above 130/80 mmHg.

Blood Pressure Measurement

Blood pressure is usually measured in millimeters of mercury (mmHg) and consists of two numbers:

1. **Systolic Pressure**: The higher number, representing the pressure in the arteries when the heart beats (contracts).
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Categories of Blood Pressure

According to the American College of Cardiology (ACC) and American Heart Association (AHA) guidelines:

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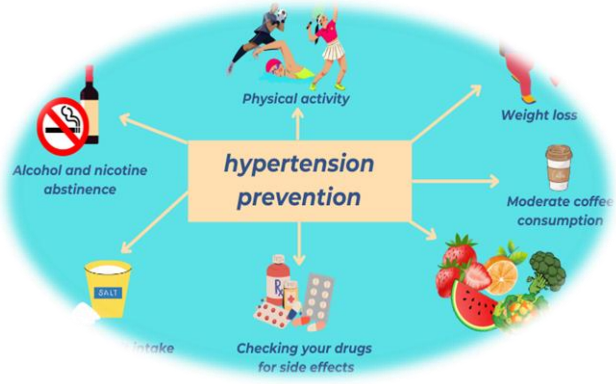
- **Hypertension Stage 1:** Systolic 130–139 mmHg or Diastolic 80–89 mmHg
- **Hypertension Stage 2:** Systolic ≥ 140 mmHg or Diastolic ≥ 90 mmHg
- **Hypertensive Crisis:** Systolic > 180 mmHg and/or Diastolic > 120 mmHg (requires immediate medical attention).

Measurement Technique

1. **Device:** Blood pressure is typically measured using a **sphygmomanometer** (either an aneroid or digital model) and a stethoscope for manual readings.
2. **Position:** The patient should be seated comfortably, with their back supported and feet flat on the floor. The arm should be at heart level.
3. **Procedure:** The cuff is placed around the upper arm, and air is pumped into it to temporarily restrict blood flow. As the cuff deflates, the systolic pressure is noted when the pulse first becomes audible, and the diastolic pressure is recorded when the pulse sound disappears.

1.9 PREVENTION



1. Maintain a Healthy Diet

- **Eat plenty of fruits and vegetables:** Rich in potassium, these help balance the negative effects of sodium.
- **Limit sodium intake:** Reduce salt in your diet by avoiding processed foods and eating fresh, unprocessed foods.
- **Increase whole grains:** Foods like oats, brown rice, and quinoa are beneficial.
- **Limit alcohol consumption:** Drinking too much alcohol can raise blood pressure.
- **Choose healthy fats:** Focus on unsaturated fats, like those in olive oil, nuts, and avocados, and limit saturated and trans fats.

2. Exercise Regularly

- Aim for at least 30 minutes of moderate exercise most days of the week, such as walking, swimming, or cycling. Regular physical activity can help lower blood pressure and strengthen the heart.

3. Maintain a Healthy Weight

- Being overweight or obese increases the risk of hypertension. Losing even a small amount of weight can help reduce blood pressure.

4. Manage Stress

- Chronic stress can contribute to hypertension. Practice relaxation techniques such as deep breathing, meditation, yoga, or spending time in nature to reduce stress.

5. Avoid Smoking

- Smoking damages the walls of blood vessels and increases the risk of high blood pressure. Quitting smoking can have immediate health benefits.

6. Monitor Blood Pressure

- Regularly check your blood pressure to ensure it remains within a healthy range. Early detection can help prevent further complications.

7. Get Enough Sleep

- Poor sleep or sleep deprivation can increase the risk of high blood pressure. Aim for 7-8 hours of quality sleep each night.

8. Limit Caffeine

- Vintake.

9. Stay Hydrated

- Drinking enough water helps maintain normal blood volume, which in turn helps regulate blood pressure.

2. TREATMENT

- The treatment of hypertension (high blood pressure) typically involves a combination of lifestyle changes and medications. The goal is to reduce the risk of heart disease, stroke, and other complications.

2.1 Non-Pharmacological Treatment

Lifestyle changes can play a critical role in the management of hypertension and may even reduce the need for medication in some cases. Non-pharmacological treatments are typically recommended as the first line of defense.

2.1.1. Dietary Modifications (DASH Diet)

- **What to Do**: The **DASH (Dietary Approaches to Stop Hypertension)** diet emphasizes fruits, vegetables, whole grains, lean proteins, and low-fat dairy while reducing sodium intake.
- **Why It Works:** A diet low in sodium and high in potassium, calcium, and magnesium can help relax blood vessels and improve blood pressure.

2.1.2. Weight Management

- **What to Do**: Losing excess weight is one of the most effective ways to lower blood pressure. A reduction of 5-10% of body weight can significantly reduce systolic blood pressure.

- **Why It Works**: Weight loss reduces strain on the heart and lowers blood volume, resulting in lower pressure on the arterial walls.

2.1.3. Regular Physical Activity

- **What to Do**: Engage in aerobic exercise, such as walking, cycling, or swimming, for at least 30 minutes most days of the week.
- **Why It Works**: Regular exercise strengthens the heart, allowing it to pump more efficiently and reduce the pressure on the arteries.

2.1.4. Limiting Alcohol Intake

- **What to Do**: Limit alcohol consumption to no more than one drink per day for women and two drinks per day for men.
- **Why It Works**: Excessive alcohol can increase blood pressure by affecting the heart and blood vessels. Reducing alcohol intake can help lower blood pressure.

2.1.5. Quit Smoking

- **What to Do:** Cease smoking, as nicotine and other chemicals in cigarettes can temporarily raise blood pressure and damage blood vessels.
- **Why It Works:** Smoking accelerates the development of atherosclerosis (narrowing of the arteries), leading to increased blood pressure.

2.1.6. Reduce Stress

- **What to Do:** Engage in relaxation techniques such as yoga, meditation, or deep breathing exercises.
- **Why It Works:** Stress triggers the release of hormones that can temporarily increase blood pressure. Long-term stress contributes to chronic hypertension.

2.1.7. Sleep Improvement

- **What to Do:** Ensure sufficient and quality sleep, typically 7-9 hours per night.
- **Why It Works:** Poor sleep quality or insufficient sleep can increase blood pressure over time, especially in those with sleep apnea.

2.1.8. Monitor Blood Pressure at Home

- **What to Do:** Regularly check blood pressure using a home blood pressure cuff.
- **Why It Works:** Monitoring helps identify patterns and assess the effectiveness of treatment, especially for individuals managing hypertension at home.

2.2 Pharmacological Treatment

Pharmacological treatments aim to lower blood pressure through various mechanisms. Medications are typically used when lifestyle changes alone aren't enough to control hypertension, especially in moderate to severe cases. Below are the main classes of antihypertensive drugs:

2.2. 1. Diuretics

- **Examples**: Hydrochlorothiazide, Chlorthalidone
- **Indications**: Often used as first-line treatment for mild to moderate hypertension.

2.2.2. Angiotensin-Converting Enzyme (ACE) Inhibitors

- **Examples**: Lisinopril, Enalapril
- **Indications**: Common for patients with heart disease, chronic kidney disease, or diabetes.

2.2.3. Angiotensin II Receptor Blockers (ARBs)

- **Examples**: Losartan, Valsartan **Indications**: Used when ACE inhibitors are not tolerated due to side effects like coughing.

2.2.4. Calcium Channel Blockers

- **Examples**: Amlodipine, Diltiazem
- **Indications**: Often used in older adults or in patients with angina or arrhythmias.

2.2.5. Beta-Blockers

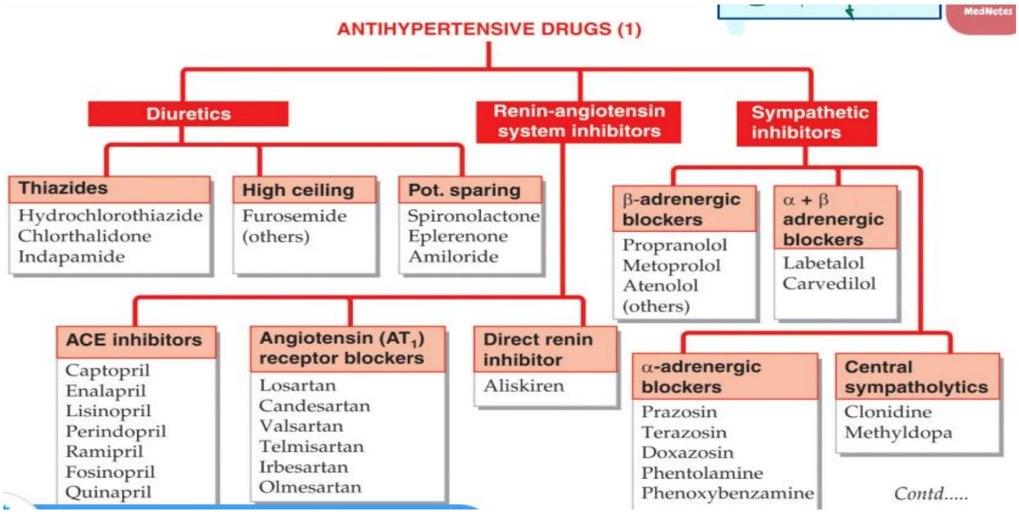
- **Examples:** Metoprolol, Atenolol
- **Indications:** Useful in patients with heart disease, arrhythmias, or post-heart attack care.

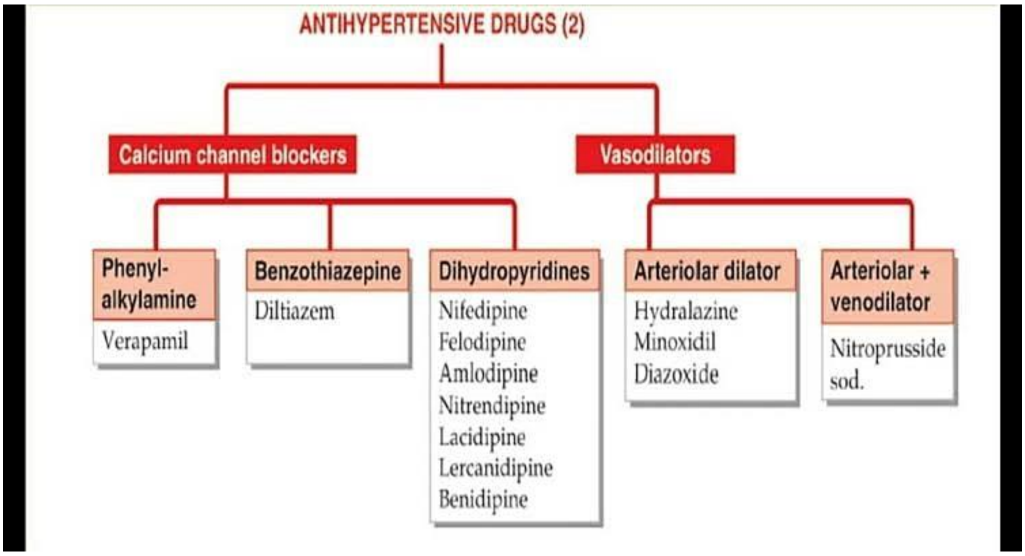
2.2.6. Direct Vasodilators

- **Examples:** Hydralazine
- **Indications:** Often used in severe hypertension or hypertensive emergencies.

2.2.7. Alpha-Blockers

- **Examples:** Doxazosin
- **Indications:** Sometimes used when other medications are ineffective or in cases of enlarged prostate.





3. MECHANISM OF ACTION

3.1 DIURETICS

Diuretics are medications that increase the production of urine, helping the body get rid of excess salt (sodium) and water. This action is useful in managing conditions like high blood pressure, heart failure, kidney disease, and edema (fluid retention). The mechanism of action of diuretics varies depending on the class of diuretic, but generally, they work by affecting the kidneys' ability to reabsorb sodium and water.

3.2 THIAZIDE DIURETICS

Thiazides are a class of diuretics that primarily work by inhibiting sodium reabsorption in the kidneys. Their mechanism of action occurs mainly in the distal convoluted tubule of the nephron.

Here's how it works

- **Inhibition of the Na+/Cl- symporter**: Thiazides block the sodium-chloride symporter (Na+/Cl- cotransporter) in the distal convoluted tubule. This prevents the reabsorption of sodium and chloride ions back into the bloodstream.
- **Increased sodium and water excretion**: Because sodium is not reabsorbed, water follows sodium osmotically. As a result, thiazides increase the excretion of sodium, chloride, and water, which reduces blood volume and leads to a decrease in blood pressure.
- **Potassium and magnesium loss**: Thiazides also cause a loss of potassium and magnesium in the urine. This is a notable side effect, as hypokalemia (low potassium levels) and hypomagnesemia (low magnesium levels) can occur.
- **Calcium retention**: Unlike some other diuretics (e.g., loop diuretics), thiazides tend to increase calcium reabsorption, which can be beneficial in conditions like osteoporosis, as they help preserve calcium in the body.

3.3 HIGH CEILING DIURETICS

High-ceiling diuretics, such as **furosemide** (Lasix) and **bumetanide**, are potent diuretics used primarily to treat conditions like heart failure, renal disease, and edema. Their mechanism of action involves inhibiting sodium, chloride, and potassium reabsorption in the **thick ascending limb of the loop of Henle** in the kidneys.

- **Inhibition of Na+/K+/2Cl- co transporter:** High-ceiling diuretics block this co transporter, which normally moves sodium, potassium, and chloride ions from the tubular lumen (inside the nephron) back into the bloodstream. This blockage prevents the reabsorption of these ions.
- **Increased excretion of sodium, chloride, potassium, calcium, and magnesium:** With this co transporter inhibited, more sodium stays in the tubular fluid, drawing water along with it, leading to increased urine output. Similarly, potassium, chloride, calcium, and magnesium are also excreted in larger amounts than with other types of diuretics.
- **Water follows solutes:** As sodium and other ions remain in the nephron and are not reabsorbed, water follows them due to osmosis, leading to a significant diuretic effect (increased urine production).
- **Effect on renal blood flow and glomerular filtration rate (GFR):** High-ceiling diuretics can also lead to an increase in renal blood flow and GFR initially, although they do not change the overall renal function long-term when used appropriately.

3.4 POTASSIUM SPARING DIURETICS

Potassium-sparing diuretics work primarily by either inhibiting sodium reabsorption in the kidneys or blocking the effects of aldosterone, which helps retain potassium in the body. There are two main classes of potassium-sparing diuretics: **aldosterone antagonists** and **sodium channel blockers**.

- **Aldosterone Antagonists (e.g., Spironolactone, Eplerenone)**
- These drugs block aldosterone, a hormone that promotes sodium retention and potassium excretion in the kidneys.
- By blocking aldosterone receptors, they reduce sodium reabsorption in the distal tubules and collecting ducts while preventing potassium from being excreted, thus conserving potassium.

- **Sodium Channel Blockers (e.g., Amiloride, Triamterene)**
- These drugs block sodium channels in the epithelial cells of the distal tubules and collecting ducts.
- By inhibiting sodium reabsorption directly, they reduce the driving force for potassium secretion, thereby sparing potassium.

3.5 RENIN ANGIOTENSIN SYSTEM INHIBITORS

Renin-angiotensin system (RAS) inhibitors are a group of medications that work by targeting different parts of the renin-angiotensin system, a hormone system that regulates blood pressure and fluid balance in the body. These inhibitors are commonly used to treat conditions like hypertension (high blood pressure), heart failure, chronic kidney disease, and sometimes post-myocardial infarction (heart attack).

3.6 ACE INHIBITORS

Angiotensin-converting enzyme (ACE) inhibitors work by blocking the enzyme ACE, which plays a key role in the renin-angiotensin-aldosterone system (RAAS). Here's how they function:

- **Inhibition of ACE**: ACE normally converts angiotensin I (a relatively inactive precursor) into angiotensin II, a potent vasoconstrictor.
- **Reduction in Angiotensin II**: By blocking ACE, ACE inhibitors decrease the levels of angiotensin II.

- **Effects of Reduced Angiotensin II**
  - **Vasodilation**: Angiotensin II typically causes blood vessels to constrict, so reducing its levels leads to relaxation of blood vessels, lowering blood pressure.
  - **Decreased Aldosterone Secretion**: Angiotensin II also stimulates the release of aldosterone, which promotes sodium and water retention by the kidneys. By lowering angiotensin II, ACE inhibitors reduce aldosterone levels, leading to less sodium and water retention and thus further helping to reduce blood pressure.
  - **Reduced Blood Volume**: With lower aldosterone and reduced water retention, the overall blood volume is decreased, which also helps in lowering blood pressure.

3.7 ANGIOTENSIN RECEPTOR BLOCKER

Angiotensin Receptor Blockers (ARBs) work by blocking the action of a hormone called angiotensin II, which is a part of the renin-angiotensin-aldosterone system (RAAS). Angiotensin II is a potent vasoconstrictor, meaning it narrows blood vessels, which raises blood pressure. It also stimulates the release of aldosterone, a hormone that increases sodium and water retention, leading to increased blood volume and further elevation of blood pressure.

ARBs specifically target and bind to the angiotensin II type 1 (AT1) receptor. By blocking this receptor, ARBs prevent angiotensin II from exerting its effects on blood vessels, adrenal glands, and kidneys. This results in:

- **Vasodilation** – Blood vessels relax and widen, which lowers blood pressure.
- **Reduced aldosterone secretion** – Less sodium and water retention, helping to reduce blood volume and lower blood pressure.
- **Improved kidney function** – By reducing the constriction of blood vessels in the kidneys, ARBs can help protect kidney function in conditions like hypertension and diabetic nephropathy.

3.8 DIRECT RENIN INHIBITORS

Direct renin inhibitors (such as aliskiren) act by directly inhibiting renin's activity. By binding to the active site of renin, these inhibitors prevent the conversion of angiotensinogen to angiotensin I. This leads to a decrease in the production of angiotensin II, thereby reducing its effects on blood pressure, vasoconstriction, aldosterone secretion, and fluid retention.

- **Renin release**: Triggered by factors like low blood pressure or low sodium levels.

- **Inhibition**: Direct renin inhibitors block renin from converting angiotensinogen into angiotensin I.
- **Outcome**: Reduced levels of angiotensin II and its downstream effects, leading to lower blood pressure and reduced fluid retention.

3.9 SYMPATHETIC INHIBITORS

Sympathetic inhibitors refer to substances or agents that block or reduce the activity of the sympathetic nervous system (SNS). The sympathetic nervous system is part of the autonomic nervous system and is responsible for the "fight or flight" response, increasing heart rate, blood pressure, and blood flow to muscles in times of stress or danger.

Sympathetic inhibitors can include.

- **Beta-blockers**: These drugs block the action of the sympathetic neurotransmitters (like norepinephrine) on beta-adrenergic receptors, leading to a reduction in heart rate and blood pressure. They are commonly used to treat conditions like hypertension, arrhythmias, and anxiety.
- **Alpha-blockers**: These inhibit alpha-adrenergic receptors, which can lead to vasodilation (widening of blood vessels) and a reduction in blood pressure.
- **Sympatholytics**: A broader class of drugs that block sympathetic nervous system activity, including both alpha and beta blockers, as well as drugs that affect the release or action of neurotransmitters like norepinephrine.

3.10 BETA ADRENERGIC BLOCKERS

- Beta-adrenergic blockers, also known as beta-blockers, work by blocking the effects of adrenaline (epinephrine) and norepinephrine on beta-adrenergic receptors in the body. There are two main types of beta-adrenergic receptors: **beta-1** (found primarily in the heart) and **beta-2** (found in other tissues, such as the lungs and blood vessels).
- **Beta-1 receptor blockade**: Most beta-blockers primarily target **beta-1 receptors** in the heart. When these receptors are blocked, the heart rate slows down (negative chronotropy), the force of contraction is reduced (negative inotropy), and the conduction of electrical impulses through the heart is slowed (negative dromotropy).
- **Beta-2 receptor blockade (in some cases)**: Some beta-blockers also have some activity at **beta-2 receptors**, which are mainly found in smooth muscle (such as in the lungs, blood vessels, and gastrointestinal tract). Blocking beta-2 receptors can cause

bronchoconstriction, which is why non-selective beta-blockers should be used with caution in people with asthma or chronic obstructive pulmonary disease (COPD).

3.11 ALPHA + BETA ADRENERGIC BLOCKERS

Alpha and beta adrenergic blockers, commonly known as alpha-beta blockers, work by blocking the effects of catecholamines (such as norepinephrine and epinephrine) on alpha and beta receptors in the body.

Alpha Blockade (α-blockade)

- **Alpha-1 receptors** are found primarily in smooth muscle (e.g., blood vessels). When these receptors are activated by catecholamines, they cause vasoconstriction leading to an increase in blood pressure.
- **Alpha-1 blockers** inhibit this vasoconstriction by preventing catecholamines from binding to the alpha-1 receptors. This leads to vasodilation which can reduce blood pressure.

Beta Blockade (β-blockade)

- **Beta-1 receptors** are mainly located in the heart. Activation of these receptors increases heart rate and the force of contraction, which increases cardiac output and blood pressure.
- **Beta-1 blockers** inhibit this effect, leading to a slower heart rate (negative chronotropy), reduced force of contraction (negative inotropy), and lower blood pressure.
- **Beta-2 receptors** are found in smooth muscle, especially in the lungs, and their activation causes bronchodilation. Some beta blockers can block beta-2 receptors, leading to constriction in the airways, which is a consideration in patients with respiratory conditions like asthma.

3.12 ALPHA ADRENERGIC BLOCKERS

Alpha-adrenergic blockers, also known as alpha blockers, work by blocking the alpha-adrenergic receptors (α-receptors) in the smooth muscle of blood vessels and other tissues. These receptors are part of the sympathetic nervous system, and when activated by norepinephrine or other catecholamines, they typically cause vasoconstriction leading to increased blood pressure.

- **Blockade of Alpha-1 Receptors**: Alpha-1 receptors are primarily found in the smooth muscle of blood vessels. When alpha-1 receptors are activated by norepinephrine, the smooth muscle contracts, leading to vasoconstriction and an increase in blood pressure.

Alpha blockers inhibit the action of norepinephrine at these receptors, causing the smooth muscle to relax.

- **Reduction in Peripheral Vascular Resistance**: By blocking alpha-1 receptors, these drugs reduce the peripheral vascular resistance. This can be especially useful in treating conditions like hypertension (high blood pressure) and benign prostatic hyperplasia (BPH), as it helps to lower blood pressure and ease urinary symptoms in men.
- **Possible Effects on Alpha-2 Receptors**: Although less pronounced, some alpha blockers may also have an effect on alpha-2 receptors, which typically inhibit the release of norepinephrine. This could contribute to additional mechanisms, but the primary effect is seen at the alpha-1 receptors.

3.13 CENTRAL SYMPATHOLYTICS

Central sympatholytics are a class of drugs that work by inhibiting the sympathetic nervous system. The sympathetic nervous system is responsible for the "fight or flight" response, including raising heart rate, blood pressure, and other physiological changes. Central sympatholytics act primarily on the central nervous system (CNS), particularly in the brainstem, to reduce sympathetic outflow.

- **Alpha-2 adrenergic agonists** (e.g., clonidine, methyldopa): These drugs bind to alpha-2 adrenergic receptors in the brainstem, leading to decreased sympathetic tone. This results in lowered norepinephrine release from nerve terminals, which ultimately reduces heart rate and blood pressure.
- **Imidazoline receptor agonists** (e.g., rilmenidine): These drugs may also target imidazoline receptors in the brainstem, leading to decreased sympathetic outflow and thus lowering blood pressure.
- **Alpha-2 adrenergic agonists** (e.g., clonidine, methyldopa): These drugs bind to alpha-2 adrenergic receptors in the brainstem, leading to decreased sympathetic tone. This results in lowered norepinephrine release from nerve terminals, which ultimately reduces heart rate and blood pressure.
- **Imidazoline receptor agonists** (e.g., rilmenidine): These drugs may also target imidazoline receptors in the brainstem, leading to decreased sympathetic outflow and thus lowering blood pressure.

3.14 CALCIUM CHANNEL BLOCKERS

Calcium channel blockers (CCBs) are a class of medications that primarily treat conditions like high blood pressure, angina (chest pain), and certain arrhythmias (irregular heartbeats). They work by blocking calcium from entering cells of the heart and blood vessel walls, which causes the blood vessels to relax and widen, improving blood flow and reducing the workload on the heart.

PHENY-ALKYLAMINE

Phenylalkylamines are a class of compounds that include various neurotransmitters and psychoactive substances, such as amphetamines and certain trace amines. Their mechanisms of action can vary based on the specific compound, but they generally affect the central nervous system (CNS) through interactions with neurotransmitter systems.

- **Dopamine, Norepinephrine, and Serotonin Release/Release Enhancement**
- **Amphetamines** and related phenylalkylamines typically increase the release of dopamine, norepinephrine, and serotonin. They achieve this by reversing the action of the respective neurotransmitter transporters on presynaptic neurons, allowing neurotransmitters to accumulate in the synapse.
- This increase in neurotransmitter levels results in heightened alertness, euphoria, and increased energy.

- **Inhibition of Reuptake**
- These compounds also inhibit the reuptake of dopamine, norepinephrine, and serotonin into the presynaptic neuron. This prevents the neurotransmitters from being recycled, leading to prolonged activation of receptors on the postsynaptic neurons.

- **Monoamine Oxidase (MAO) Inhibition**
- Some phenylalkylamines, like certain amphetamines, also inhibit the enzyme monoamine oxidase (MAO). MAO normally breaks down monoamine neurotransmitters (like dopamine and serotonin), so inhibiting it further increases the levels of these neurotransmitters.

- **Receptor Interaction**
- Many phenylalkylamines can interact directly with specific receptors in the brain, including adrenergic receptors (for norepinephrine), serotonergic receptors (for

serotonin), and dopaminergic receptors (for dopamine), contributing to their stimulant effects or other psychological outcomes.

3.15 BENZOTHIAZEPINES

Benzothiazepines are a class of drugs that primarily act on the central nervous system (CNS) by modulating the activity of certain receptors in the brain. Their mechanism of action is mainly through their interaction with **calcium channels** and **GABA receptors**.

- **Calcium channel blockade**: Benzothiazepines are thought to block certain types of calcium channels. This results in the inhibition of calcium influx into cells, which has a calming effect on the nervous system, as it reduces neuronal excitability. This action is one of the key mechanisms behind their ability to treat conditions like hypertension, arrhythmias, and anxiety.
- **GABAergic modulation**: They also interact with **gamma-aminobutyric acid (GABA)** receptors, which are the primary inhibitory neurotransmitter systems in the brain. By enhancing the effects of GABA, benzothiazepines promote CNS depression, leading to sedative, anxiolytic, and muscle relaxant effects.

3.16 DIHYDROPYRIDINES

- **Calcium Channel Blockade**: Dihydropyridines bind to L-type calcium channels on the smooth muscle cells in blood vessel walls. These channels are responsible for calcium influx, which plays a key role in muscle contraction and vascular tone regulation.
- **Vasodilation**: By blocking calcium entry, DHPs reduce the intracellular calcium concentration, leading to relaxation of smooth muscle. This causes vasodilation, particularly in the peripheral arteries, and lowers blood pressure.
- **Minimal Effect on Heart**: Unlike some other calcium channel blockers, DHPs have a more significant effect on vascular smooth muscle than on cardiac tissue. This makes them more effective for treating hypertension and angina without causing significant bradycardia.

3.17 VASODILATORS

Vasodilators are substances or medications that relax and widen the blood vessels, which helps to improve blood flow and reduce blood pressure. By dilating the blood vessels, vasodilators decrease the resistance that the heart has to work against, which can be helpful in treating conditions like hypertension (high blood pressure), heart failure, and angina (chest pain).

3.18 ARTERIAL DILATOR

- **Increased Nitric Oxide (NO) Production**: Some arteriolar dilators, like hydralazine, work by increasing the production of nitric oxide in the endothelium. NO is a potent vasodilator that relaxes smooth muscle in the blood vessels, leading to vasodilation.
- **Inhibition of Calcium Ion Influx**: Many arteriolar dilators, such as calcium channel blockers (e.g., nifedipine), inhibit the entry of calcium into smooth muscle cells. Since calcium is crucial for muscle contraction, inhibiting its influx leads to muscle relaxation and vasodilation.
- **Sodium Nitroprusside and Nitroglycerin**: Some vasodilators, like sodium nitroprusside and nitroglycerin, release nitric oxide directly or through metabolic processes, causing smooth muscle relaxation and vasodilation.
- **Sympathetic Nervous System Inhibition**: Certain agents, like alpha-1 blockers (e.g., prazosin), inhibit the alpha-1 adrenergic receptors, which are normally activated by norepinephrine to cause vasoconstriction. Blocking these receptors leads to relaxation of the smooth muscle in arterioles.
- **Direct Smooth Muscle Relaxation**: Drugs like hydralazine can also directly relax vascular smooth muscle by interfering with intracellular signaling pathways that regulate muscle contraction.

3.19 ARTERIAL AND VENODILATOR

- **Arterial Dilators**
- These drugs primarily act on the **arteries**, causing them to relax and widen (dilate).
- **Mechanism of action**: Arterial dilators work by relaxing the smooth muscle in the arterial walls, which reduces the resistance to blood flow, leading to a decrease in **systemic vascular resistance (SVR)** and thus reducing **afterload**.
- Common examples include **hydralazine** and **minoxidil**.

- **Venodilators**
- These drugs primarily act on the **veins**, causing them to dilate and hold more blood.

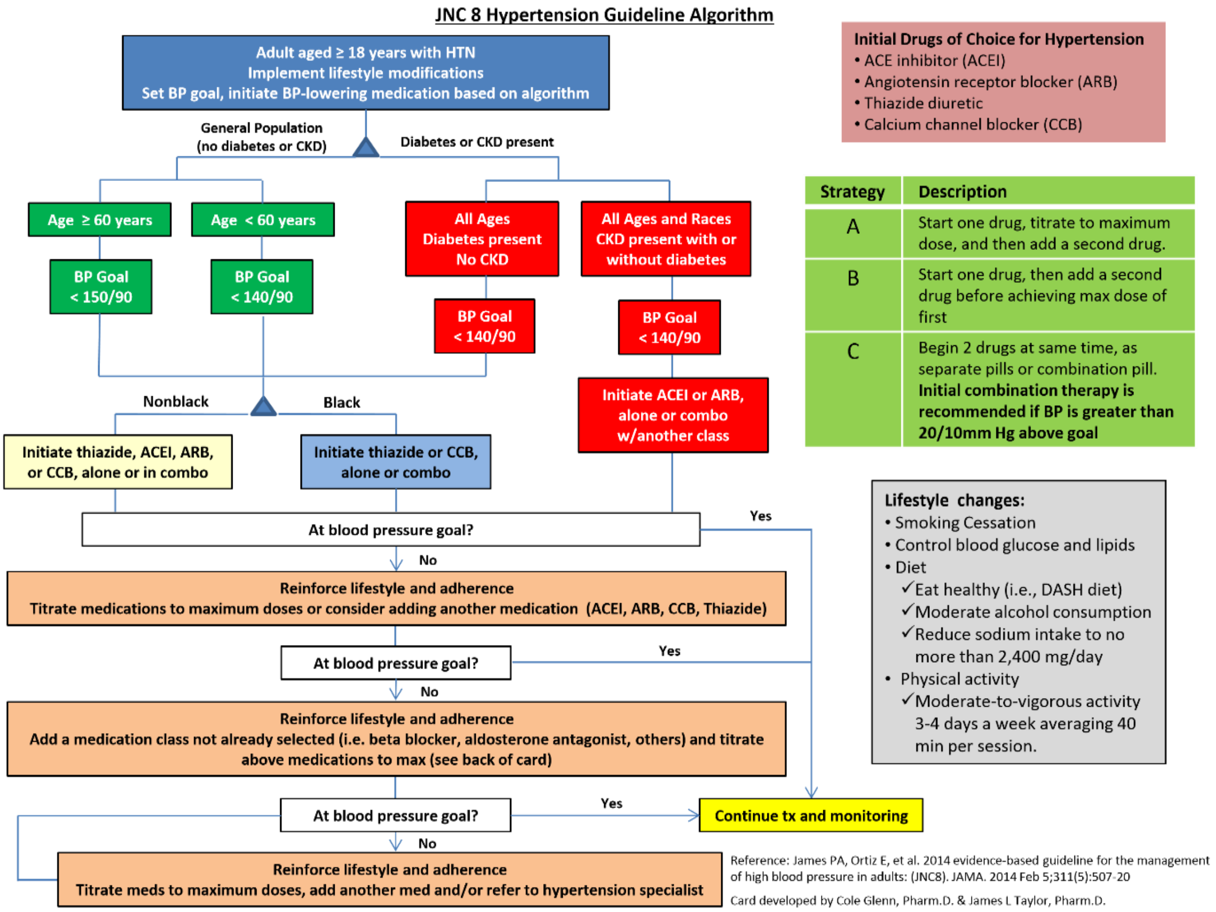
- **Mechanism of action**: Venodilators relax the smooth muscle in the veins, increasing the venous capacitance (the ability to hold blood). This leads to a **reduction in preload**, which is the amount of blood returning to the heart.
- Common examples include **nitroglycerin** and **isosorbide dinitrate**.

4 LITERATURE REVIEW

AUTHORS NAME	NAME OF THE STUDY	STUDY DESIGN	CONCLUSION
Taklo Simeneh Yazie., et.al	Prescription pattern of anti-hypertensive medication among hypertensive outpatients at selective hospitals of south Gondar zone, Amhara, Ethiopia:A hospital based cross sectional study	Cross-sectional Study	Diuretics were the most frequently prescribed drug in monotherapy and in combination with calcium channel blocker as dual therapy. On average more than 90% of prescription was in accordence with who guidelines.
Renren yang., etal	Analysis of prescription status of antihypertensive drugs in Chinese patients with hypertension based on real-world study	Cross-sectional retrospective study	CCBs and ARBs were the two most frequently prescribed for patients with hypertension. The prescription pattern of antihypertensive medications in the study largely complied with recommended Chinese hypertension guidelines.
T Philipp[1], M Anlauf., et.al	Randomised, double blind, multicentre comparison of hydrochlorothiazide, atenolol, nitrendipine, and enalapril in antihypertensive treatment: results of the HANE study. HANE Trial Research Group.	Cross-sectional population based study	According to the survey first choice calcium channel blockers and angiotensin converting enzyme inhibitors are used as the antihypertensive drugs. Moreover further studies confirming the reduction of morbidity and mortality rate.
C Ineke Neutel., et.al	Antihypertensive medication use by recently diagnosed hypertensive Canadians.	Cross-sectional study	The most commonly used antihypertensive medication for men was angiotensin-converting enzyme inhibitors (beta-blockers were second), but the most common medication for women was diuretics.
Atle Fretheim., et.al	International variation in prescribing antihypertensive drugs: Its extent and possible explanations.	Cross-sectional study	Calcium channel blockers and ACE-inhibitors are generally the most widely used drugs for the treatment of hypertension. In the UK thiazides account for 25% of consumption.
Juvenal Soares Dias da Costa, Sandra	Cost-effectiveness of hypertension treatment: a	Cross-sectional study	Diuretics and beta blockers were the drugs most frequently used in

Costa Fuchs., **et.al**	population-based study		monotherapy, while the most common associations were diuretics and beta blockers or diuretics and angiotensin-converting enzyme (ACE) inhibitors.
Aleksander Prejbisz [1], Francesca Donnaloja., et.al	Physicians' attitudes toward beta-blockers for the treatment of hypertension in Italy, Poland, and Turkey	Cross-sectional study	. β-blockers, as monotherapy/dual combination therapy (i.e., with diuretics or with CCB or with ACEi or with ARB) are mainly used.
Timothy S Anderson, John Z Ayanian., et.al	National Trends in Antihypertensive Treatment Among Older Adults by Race and Presence of Comorbidity, 2008 to 2017	Cross-sectional study	The most common antihypertensive classes filled in 2008 were ACEI/ARBs, beta blockers thiazide diuretics, and CCBs.
Martin C S Wong [1], Wilson W S Tam	Initial Antihypertensive Prescription and Switching: A 5 Year Cohort Study from 250,851 Patients	Cross-sectional study	Thiazide diuretics were significantly more likely to be switched when compared to other major antihypertensive drug classes

JNC 8 HYPERTENSION GUIDELINE ALGORITHM



Compelling Indications	
Indication	**Treatment Choice**
Heart Failure	ACEI/ARB + BB + diuretic + spironolactone
Post –MI/Clinical CAD	ACEI/ARB AND BB
CAD	ACEI, BB, diuretic, CCB
Diabetes	ACEI/ARB, CCB, diuretic
CKD	ACEI/ARB
Recurrent stroke prevention	ACEI, diuretic
Pregnancy	labetolol (first line), nifedipine, methyldopa

Beta-1 Selective Beta-blockers – possibly safer in patients with COPD, asthma, diabetes, and peripheral vascular disease:

- metoprol
- bisoprolol
- betaxolol

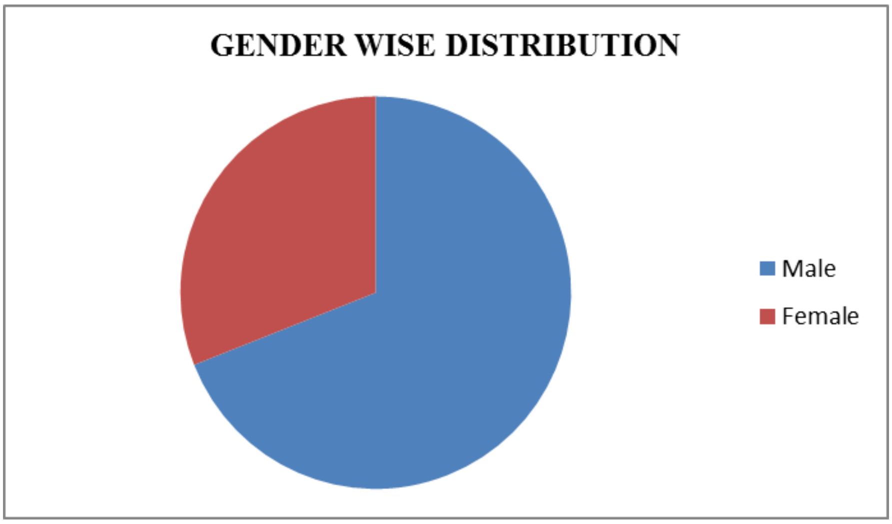
Drug Class	**Agents of Choice**	
Diuretics	HCTZ 12.5-50mg, chlorthalidone 12.5-25mg, indapamide 1.25-2.5mg triamterene 100mg *K+ sparing* – spironolactone 25-50mg, amiloride 5-10mg, triamterene 100mg furosemide 20-80mg twice daily, torsemide 10-40mg	Monitor for hypokalemia Most SE are metabolic in nature Most effective when combined w/ ACEI Stronger clinical evidence w/chlorthalidone Spironolactone - gynecomastia and hyperkalemia Loop diuretics may be needed when GFR <40mL/min
ACEI/ARB	*ACEI:* lisinopril, benazapril, fosinopril and quinapril 10-40mg, ramipril 510mg, trandolapril 2-8mg *ARB*: candesartan 8-32mg, valsartan 80-320mg, losartan 50-100mg, olmesartan 20-40mg, telmisartan 20-80mg	SE: Cough (ACEI only), angioedema (more with ACEI), hyperkalemia Losartan lowers uric acid levels; candesartan may prevent migraine headaches
Beta-Blockers	metoprolol succinate 50-100mg and tartrate 50-100mg twice daily, nebivolol 5-10mg, propranolol 40-120mg twice daily, carvedilol 6.25-25mg twice daily, bisoprolol 5-10mg, labetalol 100-300mg twice daily,	Not first line agents – reserve for post-MI/CHF Cause fatigue and decreased heart rate Adversely affect glucose; mask hypoglycemic awareness
Calcium channel blockers	*Dihydropyridines*: amlodipine 5-10mg, nifedipine ER 30-90mg, *Non-dihydropyridines*: diltiazem	Cause edema; dihydropyridines may be safely combined w/ B-blocker

	ER 180-360 mg, verapamil 80-120mg 3 times daily or ER 240-480mg	Non-dihydropyridines reduce heart rate and proteinuria
Vasodilators	hydralazine 25-100mg twice daily, minoxidil 5-10mg terazosin 1-5mg, doxazosin 1-4mg given at bedtime	Hydralazine and minoxidil may cause reflex tachycardia and fluid retention – usually require diuretic + B-blocker Alpha-blockers may cause orthostatic hypotension
Centrally-acting Agents	clonidine 0.1-0.2mg twice daily, methyldopa 250-500mg twice daily guanfacine 1-3mg	Clonidine available in weekly patch formulation for resistant hypertension

6 RESULT

6.1 GENDER WISE DISTRIBUTION AMONG THE STUDY POPULATION

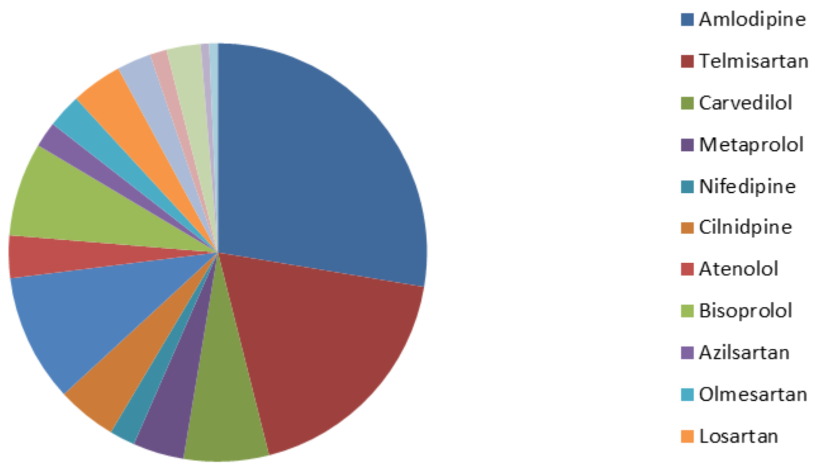
Among 100 patients male 69%(69) where more prone to the disease then the female patients 31% (31) as shown in the table.



GENDER	NO.OF PATIENTS	PERCENTAGE
MALE	69	69%
FEMALE	31	31%
TOTAL	100	100%

6.2 PRESCRIPTION PATTERN OF HYPERTENSIVE DRUG AMONG THE STUDY POPULATION

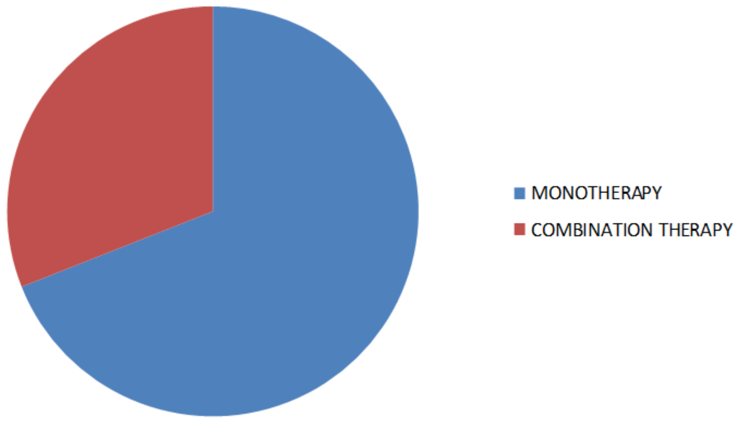
On basis of the prescription pattern of hypertensive drug; 42 prescriptions contains Amlodipine, 28 prescription contains Telmisartan, 15 prescription contains Cilnidipine, 11 prescriptions contains Bisoprolol, 10 prescriptions contains Carvedilol, 6 prescriptions contains Metoprolol, 5 prescriptions contains Atenolol, 4 prescriptions contains Olmesartan, 3 prescriptions contains Nifedipine, 3 prescriptions contains Azilsartan.



NAME OF THE DRUGS	NO OF DRUGS	PERCENTAGE
Amlodipine	42	42%
Telmisartan	28	28%
Cilnidipine	15	15%
Bisoprolol	11	11%
Carvedilol	10	10%
Metaprolol	6	6%
Atenolol	5	5%
Olmesartan	4	4%
Nifedipine	3	3%
Azilsartan	3	3%

6.3 PRESCRIPTION PATTERN FOR MONOTHERAPY AND COMBINATION THERAPY

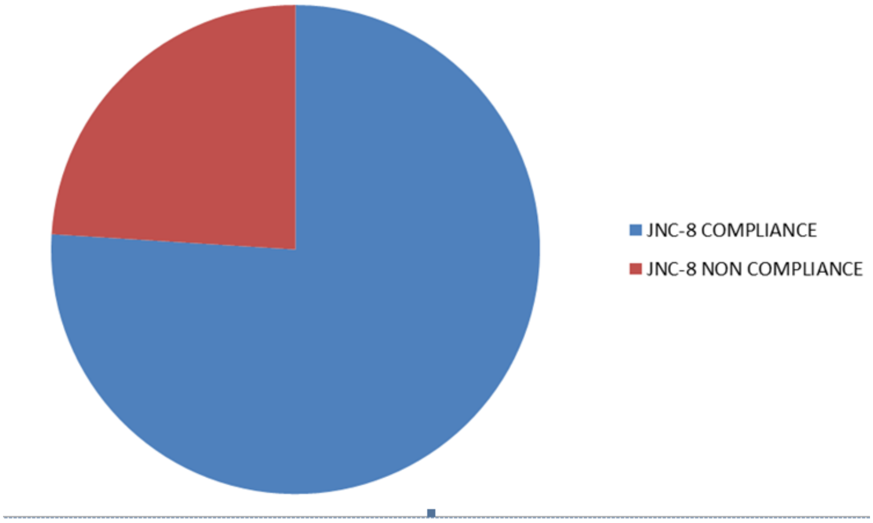
Among 100 prescriptions 69 were on monotherapy (69%) and 31 were on combination therapy (31%) as shown in table.



TYPE OF THERAPY	NO: OF PRESCRIPTION	PERCENTAGE
MONOTHERAPY	69	69%
COMBINATION THERAPY	31	31%
TOTAL	100	100%

6.4 PERCETAGE OF PRESCRIPTIONS ON COMPLIANCE AND NON COMPLIANCE WITH JNC-8 GUIDELINES

Among 100 prescription 76 (76%) prescriptions were on compliance with JNC-8 guidelines and 24 (24%) prescriptions were not in compliance with JNC-8 guidelines as shown in the table.



JNC-8 GUIDELINES	NO: OF PRESCRIPTION	PERCENTAGE
COMPLIANCE	76	76%
NON-COMPLIANCE	24	24%

7. DISCUSSION

Hypertension, also known as high blood pressure, is a condition where the force of the blood against the walls of the arteries is consistently too high. This increased pressure can strain the heart and damage blood vessels over time, leading to serious health problems like heart disease, stroke, kidney damage, and other complications.

World Health Organization (WHO) estimates that over **1.13 billion people** globally suffer from hypertension.

Hypertension is responsible for **9.4 million deaths** each year due to complications like heart disease, stroke, and kidney failure.

It affects approximately **30% to 45% of adults worldwide**, with rates varying by region, age, gender, and socioeconomic factors.

The prevalence of hypertension increases with age. It is more common in people over the age of **60**. In fact, by age 60, about **60% of people** are affected.

Men tend to develop hypertension at an earlier age than women, but post-menopausal women experience a higher prevalence, approaching the rates seen in men.

The study comprises of 100 patients out of which most patient where in the age group between 48 – 80. In the study male patients 69 (69%) where more in number then the female patients.

Calcium channel blocker (Amlodipine) was the most frequently prescribed class of antihypertensive drug accounting for 42 (42%) prescription.

Monotherapy was used for majority of patients 69 (69%), 31 (31%) receiving two or more antihypertensive drugs as combination therapy.

76 (76%) Prescriptions was in adherence with the JNC hypertensive guideline 8 (Joint National Committee) and 24 (24%) were in non adherence with JNC-8 guidelines.

8. CONCLUSION

According to our study males were more prone to hypertension and Calcium channel blocker (Amlodipine) where the most frequently prescribed drug in monotherapy as well as in combination therapy. Most of the patients were prescribed with single hypertensive drug. 76 (76%) prescriptions was in accordance with JNC 8 hypertensive guidelines.

9. LIMITATION

Our study concluded calcium channel blocker (Amlodipine) most frequently prescribed drug. But we had few limitations of short study period and few patient where not cooperative.

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