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Essential Hypertension

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Abstract
Cardiovascular disease is the world's biggest cause of mortality and is caused by hypertension, a modifiable risk factor. About 85% of hypertensive cases are essential or primary with no identifiable cause. Therefore, it becomes even more important to identify the etiology and causes of essential hypertension to develop novel treatments. There are multiple alleles associated with essential hypertension because it is a complex, multifactorial, and polygenic condition. Around 30% to 45% of the people in the general population have hypertension. Women make up 20% of the world's hypertensive population, compared to men's 24%. Numerous factors, such as social, genetic, and environmental influences, might affect hypertension. Too far, numerous investigations have been conducted to identify the genes and polymorphisms associated with essential hypertension. None of the studies have shown conclusive evidence associating a certain polymorphism exclusively with essential hypertension. Most people are unaware if they have hypertension, it is frequently referred to as a "silent disease." It is important that people with hypertension adopt a healthy lifestyle combined with medications if required.

Keywords- Essential Hypertension, Polymorphism, Blood Pressure, Cardiovascular Disease

1. Introduction

1.1 Hypertension

The ratio of the diastolic blood pressure, which happens when the heart relaxes, to the systolic blood pressure, which shows the force that blood applies to artery walls during cardiac contraction, is known as blood pressure (BP).[1] It simply refers to the state of the circulatory system when the entire system is calibrated for a certain individual. As the heart pumps blood throughout the body, it presses up against the walls of the arteries.

When the artery walls exert a stronger opposing force, the heart must work harder to circulate blood throughout the body, which results in high blood pressure. Therefore, the product of peripheral resistance and cardiac output can likewise be used to quantify blood pressure. Thereby, higher cardiac output or increased peripheral resistance might be considered the cause of hypertension.[2]A systolic blood pressure of more than 140 mm/Hg and a diastolic blood pressure of more than 90 mm/Hg are considered hypertension, according to the Joint National Committee (JNC). In addition to being one of the most common modifiable risk factors for cardiovascular diseases such as coronary artery disease, heart failure, stroke, myocardial infarction, and chronic renal disease, HT is one of the major causes of morbidity and mortality in the world.[3]Although it affects only one-third of individuals worldwide, it is responsible for about 45% of deaths.[4]

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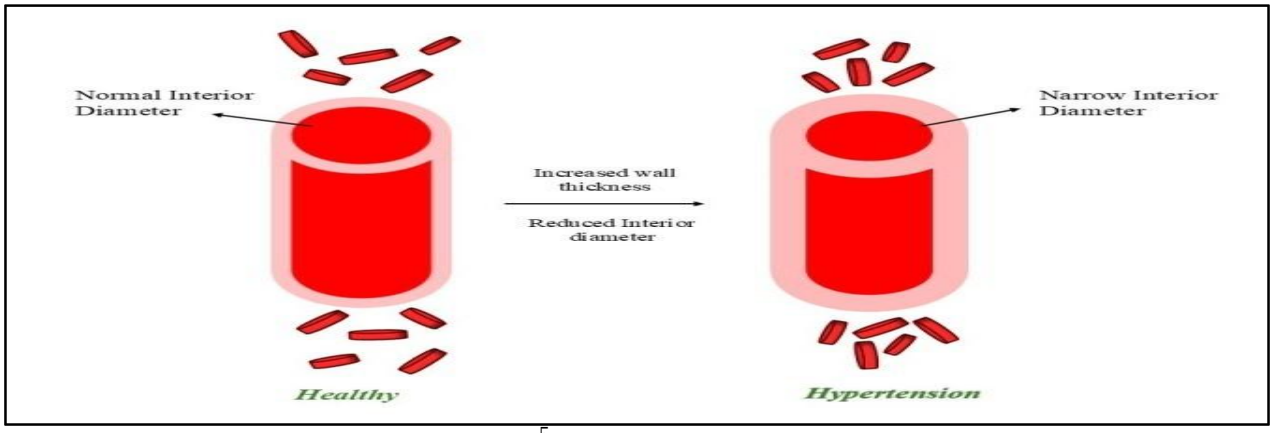

Fig 1- High Blood Pressure- Hypertension[5]

1.1.1 Classification

A disease's staging or classifying, like that of hypertension, is an evaluation of how far along it isataparticularpointintime.[5]An individual's blood pressure classification is determined by (i) the average of two or more accurately recorded measurements and (ii) various factors that influence blood pressure.[6]

(i) Classification based on measured BP readings

The 7th JNC report has recently classified hypertension into multiple categories based on recorded blood pressure levels, as illustrated in Table 1. In most adults, a normal resting blood pressure typically falls between 100-130 mmHg for systolic pressure and 60-80 mmHg for diastolic pressure.[7]Prehypertension is often referred to as borderline hypertensive and raised normal blood pressure. This medical condition occurs when a person's blood pressure is above the optimal level but not high enough to be categorized as hypertension. According to the JNC, prehypertension is defined as a systolic blood pressure ranging from 120 to 139 mmHg and a diastolic pressure between 80 and 90 mmHg.[8]It isavery common condition. Pre hypertension affects 1 in 3 people, on average.[9]At the time of diagnosis, it frequently exhibits no symptoms. The degree to which pre hypertension poses a severe health risk is still up for debate. Stage I hypertension is identified by a systolic blood pressure ranging from 140 to 159 mmHg or a diastolic pressure between 90 and 99 mmHg. This stage is marked by consistently elevated blood pressure levels, which increase the risk of cardiovascular disease (CVD).Making life style adjustment is crucial at this stage ofhypertension.[6]Stage II hypertension is characterized by a systolic blood pressure of 160 mmHg or higher and a diastolic pressure of at least 100 mmHg. At this stage, lifestyle changes may need to be combined with medication to manage blood pressure effectively .[10]

BP Classification	SBP (mm/Hg)	DBP (mm/Hg)	Condition	Life style Modification
Normal	<120	<80	Normal blood pressure or rarely there is BP elevation And no identifiable CVD risk.	Persuade

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Pre hypertension	120-139	80-90	Occasional or intermittent Blood pressure elevation and early CVD.	Yes
StageI	140-159	90-99	Sustained blood pressure Elevation or progressive CVD.	Yes
StageII	≥160	≥100	Marked and sustained blood Pressure elevation or advanced CVD.	Yes

Table1*-Classification of Blood Pressure*[6]

(ii) Classification based on factors affecting BP

Hypertension can be categorized into two types—Primary and Secondary—based on the factors influencing blood pressure.

Primary Hypertension- Primary hypertension is a type of High blood pressure that, by definition, has no other known causes. This type, which is the most common, is present in 85% of patients with high blood pressure. Primary hypertension frequently runs in families and is probably the result of how environmental and genetic factors interact. Primary hypertension becomes more prevalent with age, and individuals with moderately elevated blood pressure earlier in life are at a higher risk of developing it as they grow older.[6]

Secondary Hypertension- Secondary

Hypertension refers to high blood pressure that is caused by a specific, identifiable condition or factor. Only 5-10% of hypertensive people have this kind of hypertension, which is far less prevalent than the other variety. Endocrine disorders, renal conditions, and malignancies are only a few of their many causes. Additionally, several drugs may cause it as a side effect.

1.2 Essential Hypertension

As the cause of hypertension is unclear in most cases, it is classified as "Essential Hypertension."[4]It happens when your blood pressure is abnormally high without being the result of a medical problem. It affects 85% of hypertensive patients. The majority of individuals with hypertension have essential hypertension, making it the most common form of elevated blood pressure. It is a complex and varied condition that can result from a combination of genetic, environmental, and epigenetic factors.[12] It is also referred to as primary or idiopathic hypertension.[13] Different etiologies of essential hypertension are caused by dynamic interactions between environmental and genetic factors, which affect biological pathways and, eventually, result in hypertension. Although significant treatments have made a difference; it is still unclear what causes essential hypertension.

1.2.1 Path physiology

The path physiological events that lead to hypertension are diverse. Hypertension is influenced by a range of factors, including environmental, genetic, and social influences.1 The inter action between several factors has continued to be more fully comprehended. Additionally, more information is being gained regarding the pathogenesis of hypertension. Along with common environmental factors such as

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physical inactivity, obesity, excessive sodium intake, and chronic stress, other contributors to the condition include air and noise pollution, as well as low birth weight or preterm birth. In addition, the path physiology of hypertension has been shown to involve immunological processes and systemic inflammation. 3 Essential hypertension is influenced by various genes, including specific allelic variants that increase the risk of developing primary hypertension and are often linked to a positive family history.2

The physiological mechanisms responsible for regulating blood pressure and contributing to hypertension include the following:

(i) Cardiac Output and Peripheral Vascular Resistance- Maintaining normal blood pressure requires a balance between peripheral vascular resistance and cardiac output. While individuals with essential hypertension often have a normal cardiac output, they typically exhibit increased peripheral resistance. Maintaining normal blood pressure requires a balance between peripheral vascular resistance and cardia output. While individuals with essential hypertension often have a normal cardiac output, they typically exhibit infby Peripheral resistance is regulated by small arterioles, which contain smooth muscle cels in their walls, rather than by large arteries or capillaries.[14]An increase in intracellular calcium levels is believed to be linked to the contraction of smooth muscle cells. Prolonged smooth muscle constriction is thought to lead to structural changes, possibly influenced by angiotensin, resulting in artery wall thickening and a permanent rise in peripheral resistance.

One theory suggests that in the early stages of hypertension, peripheral resistance remains unchanged, while an increased cardiac output, driven by sympathetic over activity, is responsible for the rise in blood pressure.[15]Tomaintain normal blood pressure, cardiac output and PVR are two crucial factors. It is believed that elevated cardiac output resulting from sympathetic dysfunction acts as a driving force in onset of hypertension, while an increase in peripheral vascular resistance (PVR) functions as the body's adaptive mechanism to regulate pressure changes and maintain homeostasis.[16]

(ii) Renin, angiotensin and Aldosterone System (RAAS)- The RAAS system is crucial in coordinating the maintenance of normal blood pressure levels. Numerous organs contain the RAAS at the cellular level, but the kidney's pressure-volume homeostasis is where it plays the most significant function.[1]

Just aglomerular cells in the kidney produce and store renin and prorenin, which are synthesized and released in response to various stimuli. The primary role of renin is to convert angiotens inogen into angiotensin I.[17]

The role of the renin-angiotensin-aldosterone system (RAAS) in hypertension is largely determined by the conversion of angiotensin I into angiotensin II by the angiotensin-converting enzyme (ACE). Angiotensin II enhances the activity of the sodium/hydrogen exchanger 3, the electrogenic sodium bicarbonate co transporter 1, and the Na+/K+-ATPase while also stimulating aldosterone production in the adrenal glomerulus. Additionally, its profibrotic and pro-inflammatory effects, mainly driven by increased oxidative stress, contribute to endothelial dysfunction and damage to the kidneys, heart, and blood

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vessels. Through these mechanisms, angiotensin II plays a key role in hypertension-related target organ damage.[18]ACE2 has become a key regulator in the development of hypertension, cardiovascular disease, and kidney disorders due to its function in converting angiotensin II into angiotensin.[19] Vascular smooth muscle cells, cardiac my ocytes, fibroblasts, and glomerular and proximal tubular cells are all affected by angiotensin,which also causes systemic and localized vasodilatation, dieresis, andnatriuresis. Angiotensin also exerts protective effects on the heart and kidneys, mediated through the proto-oncogene Mas receptor. These effects are facilitated by signaling pathways involving mitogen-activated protein kinases (MAPKs), PI3K-AKT (phosphoinositide 3-kinase-RAC serine/threonine-protein kinase), NADPH oxidase, TGFβ1, the epidermal growth factor (EGF) receptor, and nuclear factor-κβ (NF-κβ) activity.[20]

Aldosterone plays a key role in the development of hypertension. When it binds to the minera locorticoid receptor, it triggers non-genomic effects such as activating the amiloride-sensitive sodium channel and the epithelial sodium channel (ENaC), which enhance sodium reabsorption in the renal cortical collecting duct. aldosterone also induces vasoconstriction, hypertension, and endothelial dysfunction through several non-epithelial mechanisms. These include increased oxidative damage, smooth muscle cell proliferation in the blood vessels, extracellular matrix deposition, vascular remodeling, and the development of vascular fibrosis.[21],[22]

The RAAS system activates the kidney's epithelial cells to enhance the reabsorption of salt and water, leading to an increase in blood volume and blood pressure.[23]

(iii) Sympathetic Nervous System (SNS)- Over the past decade, extensive research has focused on the role of the SNS in regulating the development and maintenance of blood pressure. additionally, it has been proven that SNS is crucial to the etiology of essential hypertension.[24]Sympathetic activation of the heart, peripheral blood vessels, and kidneys leads to higher cardiac output, increased vascular resistance, and fluid retention, all of which play a crucial role in development of hypertension.[25]Furthermore, individuals with elevated heart rates in the early stages of clinical hypertension often exhibit a combination of sympathetic over activity and parasympathetic dysfunction. Renal hemodynamics, salt balance in the tubules, and renin secretion in the kidney are all extensively influenced by the sympathetic nervous system.[26] The efferent and afferent pathways of the renal sympathetic nervous system, which alter blood pressure, are two mechanisms. via which HTN develops and is maintained. The efferent pathway transmits signals from the SNS to the kidneys, stimulating renin release, which activates the RAAS system and increases sodium and water retention. This raises circulation volumes, contributing to higher blood pressure. Additionally, the efferent pathway decreases renal blood flow, prompting the kidney to activate the afferent pathway. This pathway sends signals to the SNS, amplifying sympathetic over activity and helping sustain high blood pressure.[27]

Endothelial Dysfunction - It remains uncertain whether endothelial dysfunction is a cause or a result of hypertension, but there is considerable evidence linking the two. Research shows a positive correlation between the severity of hypertension and the degree of endothelial dysfunction. The endothelium has a significant role in

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controlling vascular tone and, through NO, plays a significant role in salt sensitivity. Numerous vaso active chemicals are produced by endothelial cells, but NO is the most crucial for controlling blood pressure. In response to flow-induced shear stress, endothelial cells continuously release NO, which causes guany late cyclase to be activated and produces intracellular cyclic GMP, which relaxes vascular smooth muscle. In humans, inhibiting the constitutively expressed endothelial NO synthase, which disrupts NO synthesis, leads to an increase in blood pressure and the development of hypertension. Endothelial dysfunction in hypertensive patients has also been shown to be fundamentally influenced by impaired vascular flow.

Activation of the RAAS, along with inflammation and oxidative stress, drives vascular remodeling, the progression of atherosclerotic plaques, and increases the risk of cardiovascular events.

(iv) Excess Na Intake - This process suggests that the primary issue in hypertension is the kidney's inability to remove excess sodium burden caused by a high-salt diet. It is based on research showing that people in developing nations who consume less sodium have mild to non-existent hypertension. In a few trials, lowering salt intake was linked to lower blood pressure. By volume-dependent mechanisms, salt consumption raises blood pressure in the following ways:

- Consuming too much sodium raises fluid volume and preload, which raises cardiac output and produces hypertension.
- Increased cardiac output is also aided by renal salt retention.
- **Volume independent mechanisms:**
  - Angiotensin-mediated CNS effects
  - Increasing sympathetic activity.

The pressure- natriuresis phenomenon (where sodium balance is maintained through an increase in arterial pressure) is marked by higher pressure leading to greater excretion of sodium and water. This occurs in normotensive individuals who have a high salt intake and experience an increase in blood pressure.[34]This mechanism involves a slight increase in glomerular filtration along with a reduction in the renal tubules ability to reabsorb sodium. In individuals with primary hypertension, the pressure-natriuresis curve resets, hindering the normalization of blood pressure.[35]

Inflammation and Immune System- Inflammation is a key factor in the development of hypertension and the resulting damage to target organs. It is associated with increased vascular permeability and the production of potent mediators such as reactive oxygen species, nitric oxide, cytokines, and metal loproteinases. Cytokines contribute to the formation or thickening of the arterial intima, narrowing the lumen of resistance vessels. This in turn leads to greater vascular stiffness and increased resistance. Resistance vessels, which include small arteries and arterioles, are highly innervated by autonomic nerves and play a crucial role in blood pressure regulation. Cytokines influence renal tubular function by increasing local angiotensinogen and angiotensin II production, as well as promoting salt and fluid retention in hypertension.[1,]36Various factors can contribute to high blood pressure, and emerging evidence suggests that the immune system plays a role in pathophysiology of essential hypertension. The development and progression of hypertension can be viewed as a sequence of immune system

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responses.37
In hypertension, both innate and adaptive immune responses contribute to the generation of reactive oxygen species and inflammatory changes in the kidneys, blood vessels and brain.37
Innate immune responses, particularly those driven by macrophages, have been linked to hypertension induced by angiotensin II, aldosterone, and nitric oxide antagonists. Reduced macrophage infiltration in the kidney or the periadventitial region of the aorta and medium-sized arteries leads to lower salt sensitivity and decreased blood pressure.38T cell-mediated adaptive immune responses have also been linked to the development of hypertension and the resulting organ damage. In angiotensin II-dependent hypertension, T cells expressing the type 1 angiotensin II receptor play a key role.39

1.2.2 Risk Factors

(i) In general, there are two main categories of risk factors: factors like age, sex, ethnicity, and genetics that cannot be changed, and factors that could be altered that could reduce or even stop hypertension.

(ii) Genetic Variation- A person is more likely to acquire hypertension if they have a personalfamily history of it. Over 50 genes have been studied in association with hypertension, and this number continues to grow. Genome-wide association studies (GWAS) have revealed a link between blood pressure and specific genetic loci. Additionally, mutations in a single gene inherited in a Mendelian pattern can lead to hypertension. SNPs are enhanced for variationslinked tohypertensioninmanyconditions.[40]

(iii) High Sodium Intake- The evidence suggests a clear connection between sodium consumption and high blood pressure. The pathophysiological link between salt consumption and increased blood pressure has been widely debated.[41] An elevated flow in arterial vessels can occur due to increased salt consumption, as it may lead to water retention. One theory suggests that pressure natriuresis is a physiological process in which elevated blood pressure in the renal arteries triggers increased excretion of salt and water.[42]Besides blood pressure inhypertension individuals, reducing salt may also improve vascular function, all ofwhich arepositiveeffectsonthecardiovascularsystem.[43]

Low Potassium Level- Potassium, an essential mineral, plays a key role in maintaining the resting membrane potential and intracellular osmolarity. Moreover, numerous studies have shown that increased potassium intake lowers blood pressure to a more optimal level by affecting the functions of vascular smooth muscle and endothelial cells. The cytoskeleton of the vascular endothelium may become more versatile due to potassium, which may then cause NO to be released. In slightly hypertensive patients, increasing potassium consumption could considerably enhance endothelial function and raise arterial resistance. Potassium may promote vascular smooth muscle relaxation and enhance blood flow by facilitating norepinephrine uptake into sympathetic nerve terminals and inhibiting the sympathetic nervous system. Therefore, it's crucial to maintain adequate potassium levels because it poses an elevated risk of developing hypertension.

UnhealthyDiet- An I m proper diet, which includes eating healthy food in excess, is known to increase the chance of developing hypertension. For both its prevention and management, a balanced diet is encouraged.

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Consuming plenty of fruits, vegetables and low-fat dairy products, while keeping sodium and saturated fats low, is strongly supported by evidence indicating its ability to reduce bloodpressure.[46,47]

(iv) Alcohol Consumption- It is evident that drinking alcohol and hypertension are directlyrelated.[48]The primary factors behind alcohol-induced hypertension include impaired relaxation due to inflammation and oxidative damage to the endothelium by angiotensin II, which inhibits the production of endothelium-dependent nitric oxide.[49]

(v) Lack of physical exercise and Obesity- Exercise regularly lowers blood pressure. When compared with individuals of normal weight, obesity can significantly increase one's chance of developing hypertension, and up to two-thirds of cases of hypertension can be directly linked toobesity.[50] Individuals with a Body Mass Index (BMI) greater than 25 represent over 85% of cases. With the help of animal and clinical investigations, a clear connection between obesityand hypertension was discovered. As a result, it has become clear that a wide range of processes could be to blame for obesity-induced hypertension. These mechanisms include the activation of the sympathetic nervous system and the renin-angiotensin-aldosterone system.[51]

(vi) Diabetes- Insulin resistance and/or hyperinsulinemia, which are aspects of syndrome X, or the metabolic syndrome, can also contribute to hypertension. Additionally, insulin has vasodilatory characteristics. Insulin may increase sympathetic activity in those with normal Blood pressure without increasing mean arterial pressure. The vasodilatory effects of insulin may be overcome in more severe situations, such as metabolic syndrome, by the increased sympathetic neuronal activity.

Other Factors- Additional potential risk factors for hypertension include noise exposure, air pollution, psychological stress and smoking.[54]Smokingcausesanabrupt, acute rise in blood pressure, mostly by stimulating the sympathetic nervous system. However, its long-term impact on blood pressure and the occurrence of hypertension remains unclear.[55]According to several findings, both short- and prolonged exposure to air pollution may raise the chance of developing hypertension. Future research is required to determine how chronic air pollution exposure affects hypertension disparities over the world. Over a brief period, exposure to gaseous pollutants such as O_3 and NO_2 led to an increase in systolic and diastolic blood pressure.[56] There have also been reports associating the risk of hypertension with psychological stress and rotating shift work. Overall, the evidence currently indicates that these possible risk factors have little impact on hypertension in the broader population.

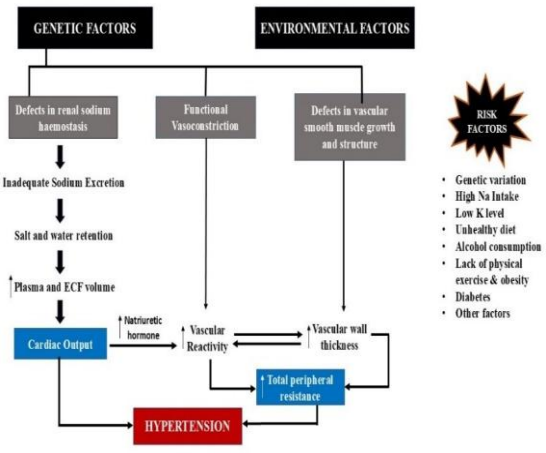

Figure 2*- Pathophysiology and Risk Factors of Hypertension*

1.2.3 Demographics & Prevalence

While the estimated global mean blood pressure seems to remain relatively stable,

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the prevalence and overall burden of hypertension are increasing worldwide. Elevated blood pressures significantly, independently, and is proportionally related to the risk of CVD, CKD and several othercomplications.[57] Around 30% to 45% of the people in the general population have hypertension. Women make up 20% of the world' shy extensive population, compared to men's 24%.[58] 27 million adults with hypertension, or one in four, have it under control. Regardless of the income status of countries, whether lower, middle, or higher, there is an increased occurrence of hypertension everywhere in the world.

According to Age-

With age, the incidence of hypertension increases, being >60% in those over the age of 60. The prevalence is 22.4% for those aged 18 to 39, 54.5% for those aged 40 to 59, and 74.5% for those aged 60 and above. Compared to non-Hispanic white (43.6%) or Hispanic adults (43.7%), non-Hispanicblackadults(57.1%)hadahigheroccurrenceofhypertension.[59]

Population Studies-

- The incidence of hypertension differs significantly by geography.
- Unlike patients from South Asia and Sub-Saharan Africa, who only have a40%awareness rate, Americans and Europeans with hypertension are 70% more likely to be aware of their disease.
- In a sample of 1.7 million adults in China, 44.7% were hypertensive, and only those individuals were aware of their condition.
- A thorough review and meta-analysis of data from 25 studies in the African population found a combined prevalence of 5.5% for high blood pressure in children and adolescents, and 12.7% for mildly elevated blood pressure in the same group.
- Another study found that individuals of African descent are at a higher risk of developing hypertension compared to those of European descent.
- According to a US study, young African Americans, and boys of white ethnicity experience prehypertension at ages 8 and 25, respectively. This shows how heterogeneity is evident in hypertension.
- According to a different study, eastern Germany showed a higher prevalence of hypertension than Western Germany. Numerous explanations for the population's varying hypertension have been put forth in research of a similar nature conducted in European nations.
- In general, European countries have a higher incidence of hypertension than Northern American nations.By2025, the global number of people with hypertension is expected to reach nearly 1.5 billion, representing an increase of15%and 20%.60
- **1.2.4 Diagnosis**

Since the majority of people does not recognize and are unaware that they have hypertension, it is frequently referred to as a "silent disease." Nevertheless, it harms the body and could ultimately result in complications. Therefore, it is essential to diagnose it at the right time. It's essential to frequently check your blood pressure to detect hypertension, especially if it has recently been higher than the "normal range" or if you have a genetic predisposition to the condition. A device known as a ‘sphygmomanometer’ is frequently used to measure blood pressure. It can be assessed at a medical facility, a pharmacy, or at home. One elevated blood pressure reading does not necessarily indicate hypertension. It is essential to measure your blood pressure

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multiple times while remaining comfortably still for at least five minutes. A diagnosis of hypertension usually requires at least three elevated readings.[62] Individuals are encouraged to take at least two readings, ideally one minute apart, twice a day for four to seven days. The average of the subsequent readings should be used after excluding the first day's results. Additional screening and confirmation tests may be necessary to verify the diagnosis if the average blood pressure is near the diagnostic threshold, particularly in younger people, as the effects of hypertension can be more severe.[63]

The diagnostic tests for hypertension include the following –

(i) BP Measurement- An inflatable cuff is placed over the arm to take blood pressure, which records both the systolic and diastolic pressure. If the readings are continuously high, based on the severity of hypertension and if there are other medical conditions, there might be a diagnosis right away.

(ii) Heart Exam- With a stethoscope, the doctor assesses the quality and intensity of heart sounds, along with the rate and regularity of the heart rhythm. Abnormal sounds may indicate high blood pressure.

(iii) Eye Exam- Chronic high blood pressure can elevate pressure in the small blood vessels at the back of the eyes, and any damage to the retina, the light-sensitive tissue that lines the eye's rear, could signal hypertension.

(iv) Blood and Urine Tests-Blood tests are performed to identify and underlying conditions associated with high blood pressure. Once the blood is collected, levels of potassium, sodium and cholesterol are examined. Urine samples may be used to check for liver or renal issues.

(v) ECG- Images from echocardiography can be used to detect bigger cardiac chambers or thicker heart muscles, which might be signs of damage from high blood pressure.

The specific screening tests for essential hypertension include the following-

(i) Raised Sodium level

(ii) Low Potassium level

(iii) Raised Bicarbonate

(iv) Combination of high sodium and bicarbonate levels

(v) Low levels of potassium

The confirmatory test for essential hypertension is the Aldosterone: Renin ratio greater than 850.[65]

1.2.5 Treatment

It is recommended that people with hypertension adopt healthy lifestyle adjustments combined with medications if required. The blood pressure reading and the likelihood of experiencing problems like a heart attack or stroke will determine whether additional medication is advised. A healthcare professional will create a treatment strategy that can include modifications to a healthy lifestyle or prescription drugs. A risk calculator can also be used to determine the likelihood of complications and select the best course of action. A risk calculator takes age, sex, race, cholesterol, blood pressure, and several other factors into account. The important lifestyle modifications recommended to treat hypertension include-

- Make heart-healthy eating choices- A well-balanced diet and reducing salt intake can be just as effective as prescription medication in lowering high blood pressure.

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Exercise regularly and be physically fit- Engaging in the recommended amount of Physical activity each week offers numerous health benefits. Research has shown that regular exercise can help lower and regulate high blood pressure levels.

• **Avoid consumption of Alcohol and Avoid Smoking** - Alcohol use or smoking can have an impact on the blood vessels as they might get narrower. Thus, it increases the likelihood of developing hypertension.
• **Reduce Stress** - Long-term emotional stress may contribute to high blood pressure. Further research is needed to determine whether stress management techniques effectively reduce blood pressure levels.

Medications may be used when healthy lifestyle modifications alone fail to control or lower high blood pressure. These medications reduce blood pressure in various ways. It is crucial to take other diseases, including kidney or heart disease, into consideration when using medications. For the treatment of hypertension, several kinds of drugs are accessible and are referred to as anti-hypertensive drugs. The several anti-hypertensive drugs used are-

- Angiotensin-converting enzyme(ACE)inhibitors- These work by preventing blood vessels from narrowing, as they block an enzyme responsible for producing angiotensin II, a substance that causes blood vessel constriction.
- Angiotensin II receptor blockers (ARBs)- Angiotensin II receptor inhibitors help lower blood pressure and improve heart function by relaxing arteries and veins. These medications block angiotensin II from taking effect, allowing blood vessels to widen (dilate).
- Calcium channel blockers-These blockers prevent calcium from entering the muscle cells of the heart and blood vessels, allowing the blood vessels to relax.
- Diuretics- These reduce the amount of fluid in your blood by eliminating excess water and sodium (salt) from the body. Thiazide is the main diuretic used to manage high blood pressure. Diuretics are often combined with other blood pressure medications, sometimes in a single dose.
- BetaBlockers- Theseaidinmakingtheheartbeatmoreslowlyandwithlessforce.This reduces the heart's workload, making it easier to pump blood through the vessels. Beta-blockers are typically used as a last resort or when additional complications are present.[70]

2. Conclusion

"High blood pressure," also known as "hypertension," refers to blood pressure that exceeds the normal range. Hypertension is a major cause of death and illness worldwide and is the most common modifiable risk factor for cardiovascular diseases. A type of high blood pressure, called essential or primary hypertension, occurs without any underlying medical conditions or identifiable causes. This disease is diverse and multifaceted, with potential genetic, environmental, and epigenetic causes.

The primary causes of essential hypertension include cardiovascular output, peripheral vascular resistance, the RAAS system, the sympathetic nervous system, excessive salt intake, inflammation, and the immune system.

Risk factors for essential hypertension can be broadly classified into two groups: those that cannot be changed, such as age, sex, ethnicity, and genetics, and those that can be adjusted to lower or even prevent

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hypertension. Just one high blood pressure measurement does not always signify hypertension. It is important to measure your blood pressure several times while lying comfortably and staying still for at least five minutes. To diagnose hypertension, at least three elevated values are typically needed.It is advised that individuals with hypertension make appropriate lifestyle choices and, if necessary, take medication. The decision to prescribe additional medication will depend on the blood pressure reading and the risk of complications, such as a heart attack or stroke.

According to current theories, hypertension is a "highly complex" genetic trait that is influenced by interactions between genes and their surroundings as well as between genes.

Finding the reasons and etiology of essential hypertension becomes increasingly more crucial in order to create innovative therapies.

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