HYPERTENSION

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Hypertension, or high blood pressure, is a very common and serious condition that can lead to or complicate many health problems. The risk of cardiovascular morbidity and mortality is directly correlated with blood pressure. Risks of stroke, MI, angina, heart failure, kidney failure or early death from a cardiovascular cause are directly correlated with BP. The roles of drug treatment, diet control, exercise, etc are discussed in this review.

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HYPERTENSION

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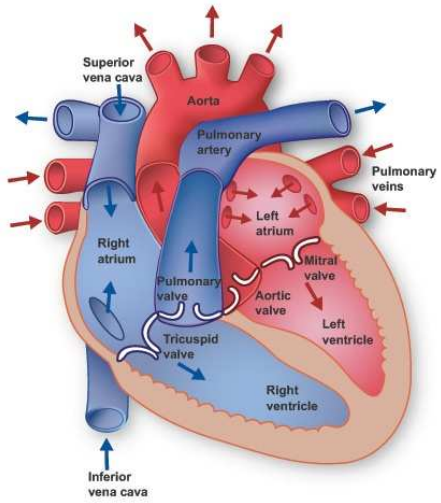

Hypertension [1-16, 46-64], or high blood pressure, is a very common and serious condition that can lead to or complicate many health problems. The risk of cardiovascular morbidity and mortality is directly correlated with blood pressure. Risks of stroke, MI, angina, heart failure, kidney failure or early death from a cardiovascular cause are directly correlated with BP.

Hypertension is often called "the silent killer" because it generally has no symptoms until serious complications develop.

There are three general types of hypertension. Essential or primary hypertension occurs when the condition has no known cause. This form of hypertension cannot be cured, but it can be controlled. More than 90% of individuals with hypertension have essential hypertension. Genetic factor may play an important role in the development of essential hypertension. When hypertension is caused by another condition or disease process, it is called secondary hypertension. Fewer than 10% of patients have secondary hypertension; where either a co-morbid disease or drug is responsible for elevating BP. In most of these cases renal dysfunction resulting from sever chronic kidney disease or renovascular disease is the most common secondary cause.

Hypertension has a variety of causes. Blood pressure generally tends to rise with age. Hypertension can also be caused by other medical conditions, such as thyroid disease or chronic kidney disease. Hypertension may also be a side effect of certain medications, such as over-the-counter cold medications and oral contraceptives and other hormone drugs.

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Obesity, heredity and life style also play a role in the development of hypertension. When symptoms do occur, they can differ between individuals depending on such factors as the level of blood pressure, age, underlying cause, medical history, the presence of complications and general health. For more information on symptoms and complications, refer to symptoms of hypertension.

Diagnosis of hypertension includes performing a complete evaluation that includes a medical history and physical examination and a series of blood pressure readings. Systolic blood pressure is a stronger predictor of cardiovascular diseases than diastolic blood pressure in adults' ≥ 50 year of age and is the most important clinical blood pressure parameter for most patients. Patient with diastolic blood pressure value less than 90 mmHg and systolic blood pressure value ≥140 mmHg have isolated systolic hypertension. Many people think of a reading of 120/80mmHg as "normal". In fact there are many variations of normal that are dependent on a variety of factors. As a very general guide, adults should keep their blood pressure below 140/90mmHg. In addition, current guidelines consider consistent readings over 120/80mmHg as a condition called pre-hypertension, which should be monitored and addressed to ensure that blood pressure does not rise higher over time. It is very possible that a diagnosis of hypertension can be missed or delayed because there are generally no symptoms in the early stages. Patient compliance with a good treatment plan generally results in a normalization of blood pressure and also minimizes complications.

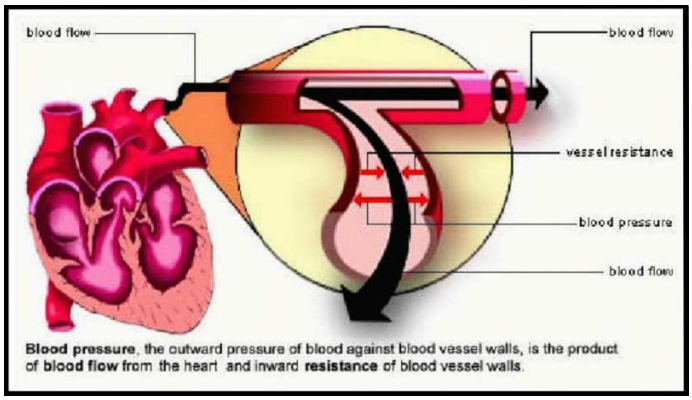
Heart is relatively small, roughly the same size as your closed fist. Heart rest on the diaphragm, near the midline of the thoracic cavity. It lies in the mediastinum, a mass of tissue that extends from the sternum to the vertebral column between the lungs.Human heart is covered by double walled covering called pericardium. The membrane that surrounds and protects the heart is the pericardium. It confines the heart to its position in the mediastinum, while allowing sufficient freedom of movement for vigorous and rapid contraction. The pericardiums consist of two parts; the fibrous pericardium and the serous pericardium. The superficial fibrous pericardium is composed of tough, inelastic dense irregular connective tissue. The fibrous pericardiums prevent overstretching of heart, provide protection

BLOOD PRESSURE (BP)

Blood pressure (BP) is defined as lateral pressure exerted by the blood on the walls of the blood vessels while flowing through them. Blood pressure in a blood vessel depends upon two things.

1) Distance from the heart and 2) Nature of the blood vessel.

Blood pressure is more in blood vessels close to the heart. Blood pressure is more in arterial system than in the venous system. This is because walls of arteries are thicker and less elastic; the walls of the veins are thinner and more elastic.



Normal blood pressure is 120/80 mmHg,Systolic BP (SBP) is the maximum BP during the ventricular systole- 120 mmHg. Range: 110-130 mmHg.Diastolic BP (DBP) is the minimum pressure during the ventricular diastole. It is 80 mmHg. Range: 70-90 mmHg

Plus pressure (PP) means the difference between systolic BP and diastolic BP. ie, 40 mmHg., SBP: DBP: PP = 3:2:1

Mean arterial blood pressure

It is not the arithmetic mean but it is less than that. It is because most of the time BP is closer to diastolic value than systolic value. It because duration of ventricular diastole is longer than duration of systole.
Mean arterial BP=Diastolic BP+1/3 of pulse pressure i.e., 80+13=93 mmHg.

Physiological Variations

Age: BP more in adult than in children.
Sex: BP more in male than females.
Pregnancy: During the later stages of pregnancy BP usually increase.
Altitude: BP is higher in people living at higher altitude.
Exercise: Systolic BP increases during exercise.
Emotion: BP rises during emotional expressions.
Sleep: BP falls during sleep

Factor Effecting Blood Pressure

1) Volume of blood.
2) Force of contraction of the heart.
3) Heart rate and BP are inversely proportional.
4) Viscosity of blood.
5) Nature of the blood.
6) Elasticity of blood vessel

REGULATION OF BLOOD PRESSURE

It means maintaining a constant blood pressure within a narrow variation. Both increase in blood pressure (hypertension) and decrease in blood pressure (hypotension) are harmful in the body.

The mechanism of regulation of BP is divided in to two groups.

1) Rapidly acting mechanism 2) Slow acting mechanism

I. **Rapidly acting mechanism**

This includes both nervous regulations as well as endocrine or hormonal regulation.

a) Nervous Regulation of BP

The smooth muscles of blood vessels will always remain in a state of contraction. Because of this the blood vessels remain in a state of constriction- vasoconstriction. The degree of vasoconstriction depends upon the sympathetic tone. When sympathetic tone increase the degree of vasoconstriction will also increase. When vasoconstriction increases total peripheral resistance (TPR) wills increases which will in turn increase BP. Suppose the BP increases that will be detected by the baroreceptors situated at the aortic arch and carotid sinus. These baroreceptors will send impulses to the medulla oblongata. In the medulla oblongata there is a group of nervous concerned with control of BP. It is known as vasomotor centre. There are two different area, pressor area and Depressor area. These impulses coming from baroreceptors will inhibit the pressor area; this will decrease the sympathetic tone. This will increase vasodilatation. TPR decreases so BP decreases to normal level. This mechanism operated very fast. It corrects BP within few second.

Baroreceptors and the sympathetic nervous system

Baroreflexes involving the sympathetic nervous system are responsible for the rapid moment to moment regulation of blood pressure. A fall in blood pressure causes pressure-sensitive neurons (baro-receptors in the aortic arch and carotid sinuses) to send fewer impulses to cardiovascular centers in the spinal cord.

This prompts a reflex response of increased sympathetic and decreased parasympathetic output to the heart and vasculature, resulting in vasoconstriction and increased cardiac output. These changes result in a compensatory rise in blood pressure.

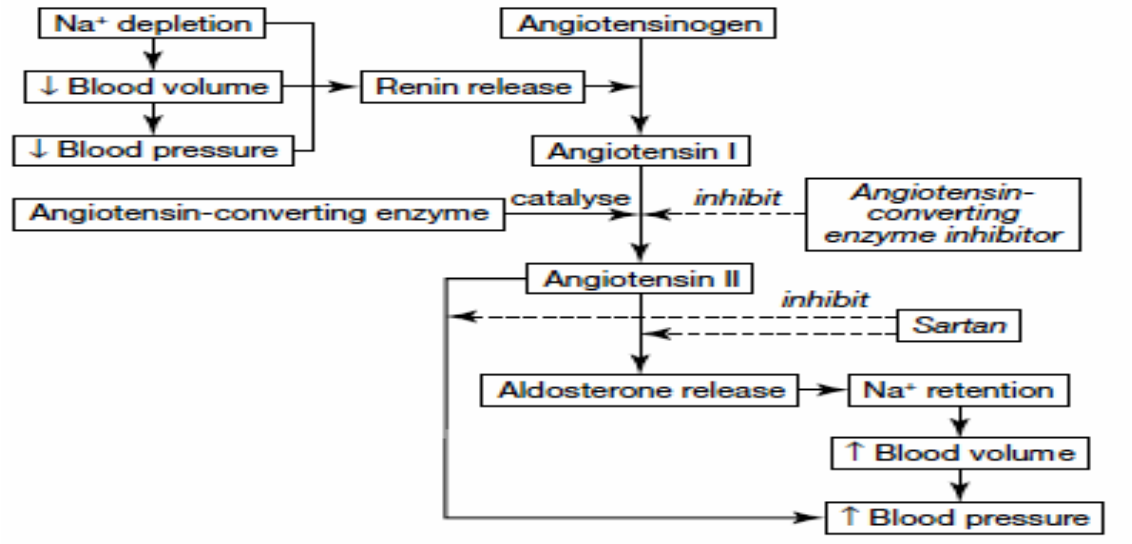
b) Endocrine or Hormonal Regulation of BP

There are three important hormones taking part in regulation of BP.

1) Renin- Angiotensin- Aldosterone mechanism or system.
2) Regulation of BP by Vasopressin or ADH.
3) Adrenalin (Epinephrine) and noradrenalin (nor epinephrine).

1) Renin- Angiotensin- Aldosterone mechanism or system

Suppose BP falls, it will stimulate the kidney. The juxta glomerular apparatus of the kidney will secrete renin. Renin acts as an enzyme. It acts on a plasma protein, angiotensin substrate and converts in to angiotensin I. The angiotensin I is converted in to angiotensin II by the action of converting enzyme. Angiotensin II is a vasoconstrictor in action. It acts on the walls of blood vessels and increases the degree of vasoconstriction. TPR will increase; this will in turn increase the BP to normal. In addition to that angiotensin II stimulates adrenal cortex. This will increase the secretion of the hormone aldosterone. Aldosterone acts as kidneys. It increases the reabsorption of sodium and water. This will increase blood volume. When blood volume increases that will in turn increase BP



2) Regulation of BP by Vasopressin or ADH

Suppose BP falls, that wills stimuli hypothalamus. Hypothalamus in turn stimulate posterior pituitary. Posterior pituitary secrete vasopressin. It acts on the wall of blood vessels. It increases vasoconstriction. TPR increases and BP will increases to the normal level. In addition to this, ADH acts at the kidneys. It increases the reabsorption of water. That will increase blood volume, so BP increases to the normal level

3) Adrenaline (Epinephrine) and noradrenaline (nor epinephrine)

Suppose BP falls that will stimulate hypothalamus. Hypothalamus in turn stimulates sympathetic nervous system. This will in turn stimulate adrenal medulla. It secretes more adrenaline. Adrenaline acts at the wall of the blood vessels. It increases vasoconstriction. This will increase TPR, this will in turn increase BP to normal level.

II. **Long term regulation of BP**

Suppose the BP increases. This will increase G.F.R, this will increase urine output and water loss from the body. This will decrease blood volume, this will in turn decease the BP.

HYPERTENSION

Hypertension is defined as abnormally high blood pressure (more than 120/80 mm Hg) in the arteries. Persistent increase in systemic arterial blood pressure is known as hypertension. Usually a mean arterial pressure greater than in 110mm Hg under resting conditions is considered to be hypertensive; this level normally occurs when the diastolic blood pressure is greater than 90 mm Hg and the systolic pressure is greater than about 135-140 mm Hg. Hypertension is generally symptom less, but increases the risk of various other cardiovascular diseases like stroke, heart attack and non-cardiovascular diseases like renal damage, end stage of renal failure, etc.

Although hypertension is a common health problem with some times devastating consequence, it often remains asymptomatic until late in its course. A sustained diastolic pressure greater than 90 mm Hg, or a sustained systolic pressure in excess of 140 mm Hg, is considered to constitute hypertension90-95% of hypertension is idiopathic (essential hypertension), which is compatible with long life, unless a myocardial infarction, cerebrovascular accident, or other complication supervenes. Most of the reminder of "benign hypertension" secondary to renal disease or, less often to narrowing of the renal artery, usually by an atheromatous plaque (renovascular hypertension). Infrequently, hypertension is secondary to diseases of the adrenal glands, such as primary aldosteronism, cushing syndrome, pheochromocytoma, or other disorders. Various determinants play important role of hypertensive condition and in causation of premature cardiovascular risk over and beyond hypertension.

THE COMMON DETERMINANTS

a) **Age and sex**: BP generally rises with age in both male and female. The rise is more steep in the middle age and thereafter. At initial phase, pressure is more in men while in later phase rise is more in women.
b) **Weight**: the rise in BP with respect to weight is found to be directly proportional.
c) **Alcohol consumption**: it is also reported that alcohol intake than positively increase the BP, but the reason is not clear
d) **Geographic variation**: geographical variation can affect BP via variable contributing factors like TPR, hypoxia, primitive condition, etc.
e) **Smoking**: tobacco combustion results in nicotine and carbon monoxide production, a potent vasoconstrictor leading to development of hypertension.
f) **Salt consumption**: Salt intake can promote rigidity to vascular smooth muscle and therefore excessive salt intake (more than 8 -10 gm per day) may result in hypertension.
g) **Genetic predisposition**: Based upon survey it is now believed that hypertension may be the result of typical genetic makeup.

TYPES OF HYPERTENSION

Category pressure	Systolic pressure mm Hg	Diastolic pressure mm Hg
Normal	90–119	60-79
Pre-hypertension	120–139	80–89
Stage 1	140–159	90–99
Stage 2	≥160	≥100
Isolated systolic Hypertension	≥140	<90

It is divided into two types

1) Primary hypertension (Essential hypertension)
2) Secondary hypertension (Non-essential hypertension)

PRIMARY HYPERTENSION

It results when arterial blood pressure is increased due to increased peripheral resistance. It is further divided in to two types namely: benign and malignant hypertension

- **Benign hypertension**

  Here, there is a moderate increase in blood pressure with systolic pressure of 200 mm Hg and the diastolic pressure of above 100 mm Hg. However, in resting condition and sleep, the blood pressure returns to normal level. Later, if there is increase in blood pressure it will not come back to normal level in resting conditions.

- **Malignant hypertension**

  Here, the blood pressure elevated to a great extends of about 250 mm Hg of systolic pressure and 150 mm Hg of diastolic pressure. It produces severe symptoms like renal disease, retinal disease, and being a fatal disease, it causes death within few years.

Some of the characteristics of primary or essential hypertension are,

1) The mean arterial pressure is increased 40-60 %.
2) The renal blood flow in the later stages is decreased about one half of normal.
3) The resistance to blood flow through the kidney is increased 2-4 fold.
4) The kidneys will not excrete adequate amounts of salt and water unless the arterial pressure is high.

SECONDARY HYPERTENSION

The different forms of secondary hypertension are

- **Cardiovascular hypertension**

  It is produced due to

  a) Atherosclerosis- hardening and narrowing of blood vessels
  b) Coarctation of aorta- narrowing of aorta.

- **Renal hypertension**

  It is produced due to

  a) Stenosis renal arteries- narrowing of one or both renal arteries, so that the renal function is impaired.
  b) Glomerulonephritis- nephritis with inflammation of the capillary loops in the renal glomeruli.

- **Endocrine hypertension**

  It occurs due to

  a) Pheochromocytoma- tumer in adrenal medulla
  b) Hyperaldosteronism- excess secretion of aldosterone from adrenal cortex Conn's syndrome.
  c) Cushing's syndrome- excess secretion of cortisone.
  d) Gigantism or Acromegaly- excess secretion of growth hormone.

- **Neurogenic hypertension**

  Acute hypertension can be caused by strong stimulation of the sympathetic nervous system.

  a) Section of the baroreceptors nerves.
  b) Lesions in tractus solitarius.
  c) Increased intracranial pressure.

ETIOLOGY OF HYPERTENSION

Although hypertension may occur secondary to other disease processes, more than 90% of patients have essential hypertension, a disorder of unknown origin affecting blood pressure regulating mechanism. A family history of hypertension is increases the likelihood that an individual will develop hypertensive disease. Essential hypertension occurs four times more frequently amoung blacks than amoung whites, and it occur more often amoung middle-aged males than amoung middle-aged females. Environmental factors such as stressful lifestyle, high dietary intake of sodium, obesity, and smoking all further predispose an individual to the occurrence of hypertension.

PATHOGENESIS OF HYPERTENSION

The multiple mechanism of hypertension constitutes aberrations of the normal physiologic regulation of blood pressure.Regulation of normal blood pressure: The blood pressure level is a complex trait that is determined by the interaction of multiple genetic, environmental, and demographic factors that influence to hemodynamic variables: cardiac output and total peripheral resistance. Cardiac output affected by the blood volume, itself greatly dependent on body sodium homeostasis. Total peripheral resistance is predominantly determined at the level of the arterioles and depends on the effect of neutral and hormonal influence. Normal vascular tone reflects the balance between humoral vasoconstriction influences (including angiotensin II and catecholamine) and vasodilators (including kinins, prostagladins, and nitric oxide). Resistance vessels also exhibit autoregulation, whereby increased blood flow induced vasoconstriction to protect against tissue hyper perfusion. Other local factors such as pH and hypoxia, as well as neural interactions (α- and β- adrenergic systems), May important.

The kidneys play an important role in blood pressure regulation, as follows:

- Through the renin-angiotensin system, the kidney influences both peripheral resistance and sodium homeostasis. Renin elaborated by the juxta glomerular cells of kidney transforms plasma angiotensinogen to angiotensin I, which is then converted to angiotensin II by angiotensin converting enzyme (ACE). Angiotensin II raises blood pressure by increasing both peripheral resistance (direct action on vascular SMCs) and blood volume (stimulation of aldosterone secretion, increase in distal tubular reabsorption of sodium).
- The kidney also produced a variety of vasodepressor or antihypertensive substances (including prostaglandins and nitric oxide) that presumably counterbalance the vasopressor effects of angiotensin.
- When blood volume is reduced, the glomerular filtration rate falls, leading to increased reabsorption of sodium and water by proximal tubules and thereby conserving sodium and expanding blood volume.
- Glomerular filtration rate- independent natriuretuc factors including atrial natriuretic peptide, secreted by heart atria in response to volume expansion, inhibit sodium reabsorption in distal tubules and cause vasodilation
- When renal excretory function is impaired, increased atrial pressure is a compensatory mechanism that helps restore fluid and electrolyte balance.

CAUSES

1. Essential Hypertension
2. Renal
    a) Acute nephritis
    b) Interstitial nephritis and pyelonephritis
    c) Polycystic kidneys
    d) Renal artery stenosis
3. Vascular: Arteriosclerosis, coaractation of aorta.
4. Endocrine: Pheochromocytoma, Cushing's syndrome, thyrotoxicosis, myxedema.

5. Neurological: Raised intracranial tension, lead encephalopathy, etc.
6. Miscellaneous: Polycythemia, aortic incompetence, toxemia of pregnancy, periarteris nodosa, gout, etc.

EFFECT OF HYPERTENSION

The common organ damage by long standing hypertension are heart, blood vessels, retina and central nervous system.

1. CVS: Increased myocardial work leads to concentric hypertrphy of left ventricle, angina pectoris and accelerated coronary artery diseases. There is systolic as well as diastolic dysfunction.
2. Kidneys: Progressive arteriosclerosis involves both the efferent and afferent renal arteriols and capillaries of glomerular truft. This leads to compromise in renal function, shrinkage of kidneys, proteinuria.
3. CNS: Hypertention may cause microaneurysms, which may rupture and cause cerebral hemorrhage. Accelerated atherosclerosis may cause cerebral thrombosis, embolism and infection. Cerebral arteriolar spasm may cause hypertensive encephalopathy.
4. Fundus: The following changes may occure:
    - Grade I: Arteriolar narrowing leading to copper wire and silver wire appearance.
    - Grade II: Arteriovenous nipping where arteries cross the vein.
    - Grade III: In addition to Grade II changes, superficial flame shaped and deep dot like hemorrheges and cotton wool exudates.
    - Grade IV: Grade III change with papilledema.

SYMPTOMS [17,18]

The clinical features may be due to the elevated BP itself, target organ involvement or due to underlying diseases, as in secondary hypertension.

Symptoms due to hypertension:

1. Head ache: This occurs usually in morning hours. It is throbbing and usually frontal.
2. Dizziness: The patients feel unsteadly.
3. Epistaxis: This occurs due to increased pressure, causing repture of the capillaries of the nose. The bleeding reduces circulating volume, and lowers the BP

Symptoms due to affection of organs

1. CVS:
    a) Dyspnea on exertion (insipient LVF)
    b) Anginal chest pain (IHD)
    c) Palpitation
2. Kidneys: Hematuria, nocturia, polyuris
3. CNS:
    a) Transient ischemic attacks (TIA or strocke) with focal neurological deficit.
    b) Hypertensive encephalopathy (head ache, vomiting, convulsion, unconciousness, focal neurological deficit).
    c) Dizziness, tinnitus and syncope.
4. Retina: Blurred vision or sudden blindness.

Symptoms due to underlying diseases

1. Edema and puffy face- Acute nephritis.
2. Weight gain, hirsutism and stira- Cushing's syndrome.
3. Weight loss, tremors, palpitation and sweating.
4. Hyperthyroidism/ pheochromocytoma.
5. Weakness- hyperaldosteronism.
6. Joint pain, bronchospasm and peripheral vascular disease.
7. Symptoms- polyarteritis nodosa.

SIGNS

General Examination

1. Moon face, buffalo hump and truncal obesity- Cushing's syndrome.
2. Puffy face, rough skin, obesity- Myxedema.
3. Tremors, tachycardia, exopthalmos and goitre- Hyperthyroidism.
4. Prognathism, clubbed hand, coarse features- Acromegaly.
5. Pigmentation- neurofibromatosis.
6. Radiofemoral delay and collanteral vessels over the chest wall- Coarction of aorta
7. Weaker left radial- Preductal coarctation.
8. Waterhammer pulse- Aortic incompetence.

Cardiovascular System

1. Cardiomegaly.
2. Third and fourth heart sound gallop.
3. Loud second hearr sound.
4. Early diastolic murmur- due to AI.

Respiratory System

1. Basal crepitations- LVF.
2. Rhonchi- LVF, polyarteritis nodosa.

Abdomen

1. Hepatomegaly- cardiac failure.
2. Palpable kidney lump- Polycystic kidney, hypernephroma.
3. Bruit over renal artery- Renal artery stenosis.
4. Bruit over abdominal aorta- Abdominal aortic aneurysm.

INVESTIGATIONS

To assess target organ damage

X-ray of chest for heart size **,** ECG for LV hypertrophy and evidence of IHD.**,** Echocardiogram for LV systolic and diastolic functions.**,** Urinalysis- protrinuria > 200 mg/ day and hematuria suggest renal involvement.

To detect the cause of hypertension

1. X-ray chest-

Rib notching suggests coarctation of aorta. ,Mediastinal widening suggests aortic dissection.

2. Imaging of abdomen (Sonography, CT scan, MRI)

To detect-Polycystic kidney,Tumours of kidney,Renal calci ,Adrenal tumours, Pheochromocytoma ,Urinary catecholamines or breakdown products (Metanephrine or VMA),Pheochromocytoma.,Echocardiogram- coarctation of aorta,IVP- for renovascular hypertension, kidney tumours and stones.

3. Aortography- for aneurysm and coarctation of aorta.

TREATMENT [31, 32, 33, 38, 41] FOR HYPERTENSION

ANTIHYPERTENSIVE DRUGS

As arterial pressure is product of cardiac output and peripheral vascular resistance, it can be lowered by the action of drug on either the peripheral resistance or cardiac output, or both. Drugs may be reduced cardiac output by either inhibiting myocardial contractility or decreasing ventricular filling pressure. Reduction in ventricular filling pressure may be achieved by action on the venous tone or on blood volume via renal effect. Drugs can reduce peripheral resistance by acting on smooth muscle to relaxation of resistance vessel or by interfering with the activity of systems that produced constriction of resistance vessel (e.g., the sympathetic nervous system)

The simultaneous use of drug with similar mechanism of action and hemodynamic effects often produces little additional benefit. However, concurrent use of drug from different classes is strategy for achieving effective control of blood pressure while minimising the dose related adverse effects.

NON-PHARMACOLOGICAL MANAGEMENT OF HYPERTENSION

In the initial phase of hypertension (high, normal or mild hypertension), the non-pharmacological measures can lower the B.P. in most of individuals. in some patients, who do not show any reduction even after 04-06 months need drug therapy. it is harmless treatment and helpful either to eliminate the requirement of drug or reduce the dose as well as dose regiment.Non pharmacological approaches to the reduction of blood pressure generally are advisable as the initial approach to treatment of patients with diastolic blood pressure in the range of 90 to 95 mmHg. Further, these approaches will augment the effectiveness of pharmacological therapy in patients with higher level blood pressure. Non pharmacological methods to lower blood pressure allow the patient to participate actively in the management of his or her disease. Reduction of weight, restriction of salt, and moderation in the use of alcohol may be reduced blood pressure and improve the effect of drug treatment. In addition, regular isotonic exercise also lowers blood pressure in hypertensive patients.

Smoking per se does not cause hypertension. However, smokers do have a higher incidence of malignant hypertension, and smoking is major risk factor for coronary heart disease. Hypertensive patients have an exceptionally great incentive to stop smoking. Consumption of caffeine can raise blood pressure and elevate plasma concentrations of nor epinephrine, but long term consumption of caffeine causes tolerance to these effects and has not been associated with the development of hypertension.

Reduction of Body Weight

Obesity and hypertension are closely associated, and the degree of obesity is positively correlated with the incidence of hypertension. Obese hypertensives may lower their blood pressure by losing weight regardless of a change in salt consumption. The mechanism by which obesity causes hypertension is unclear, but increases the secretion of insulin in obesity could result in insulin mediated enhancement of renal tubular reabsorption of Na^+ and an expansion of extracellular volume. Obesity is also associated with increased activity of the sympathetic nervous system. A combination of aerobic physical exercise and dietary counseling may enhance compliance.

Sodium Restriction

Severe restriction of salt will lower the blood pressure in most hospitalised hypertensive patients; this treatment method was advocated prior to the development of effective antihypertensive drugs. However severe salt restriction is not practical from a standpoint of compliance.

Several studies have shown that moderate restriction of salt intake to approximately 5 g per day (2 g Na^{+}) will, on average, lower blood pressure by 12 mm Hg systolic and 6 mm Hg diastolic. An additional benefit of salt restriction is improved responsiveness to some antihypertensive drugs.

Alcohol Restriction

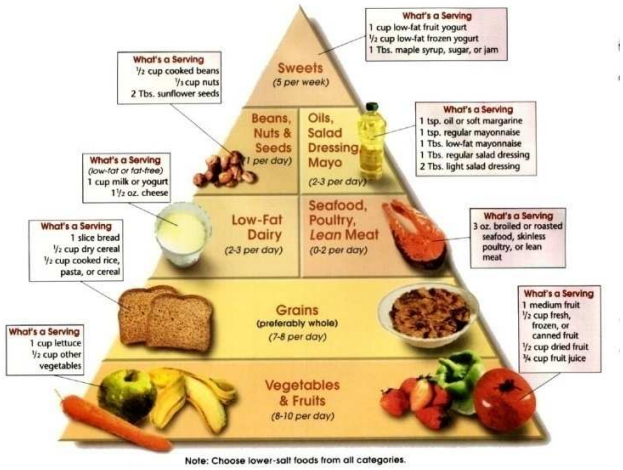
Consumption of alcohol can raise blood pressure, but it is unclear how much alcohol must be consumed to observe this effect. Heavy consumption of alcohol increases the risk of cerebrovascular accidents but not coronary heart disease. In the fact, small amount of ethanol have been found to protect against the development of coronary artery disease. The mechanism by which alcohol raises blood pressure is unknown, but it may involve increased transport of Ca^{2+} into vascular smooth muscle cells. Excessive intake of alcohol also may result in poor compliance with antihypertensive regimens. All hypertensive patients should be advised to restrict consumption of ethanol to more than 30 ml per day.

Physical Exercise

Increased physical activity lowers rates of cardiovascular disease in men. It is not known if this beneficial effect is secondary to an antihypertensive response to exercise. Lake of physical activity is associated with a higher incidence of hypertension. Regular isotonic exercise reduces blood volume and plasma catecholamines and elevates plasma concentration of atrial natriuretic factor. The beneficial effect of exercise can occur in subjects who demonstrate no change in body weight or salt intake during the training period.

Relaxation and Biofeedback therapy

The fact that long term stressful stimuli can cause sustained hypertension in animals has given credence to the possibility that relaxation therapy will lower blood pressure in some hypertensive patients. Only those few patients with mild hypertension who wish to use this method should be encouraged to try, and these patients should be closely followed and receive pharmacological treatment if necessary.



Diet

Lacto vegetarian diet and high intake of polyunsaturated fish oils lower BP due to high content of potassium and vegetable diet high content of fiber. Natural vegetables containing high levels of potassium, which lower the BP by:

a) Increased sodium excretion.
b) Decreased sympathetic activity.
c) Decreased renin angiotensin secretion and direct dilation of renal arteries.

DRUG CATEGORY

Sl.no.	Class	Example	Dose
1.	Angiotensin Converting Enzyme (ACE) Inhibitors	Captopril,	12.5-50 mg
		Enalapril,	5-40 mg
		Lisinopril,	2.5-10 mg
		Perindopril,	4-8 mg
		Ramipril,	1.25-10 mg
		Benazepril,	2.5-10 mg
		Trandolapril,	2-4 mg
		Fosinopril,	10-40 mg
		Imidapril.	5-20 mg
2.	Angiotensin (AT_1 Receptor) Antagonists:	Losartan,	25-100 mg
		Valsartan,	80-320 mg
		Telmisartan,	40-80 mg
		Candesartan,	8-32 mg
		Irbesartan.	75-300 mg
		Olmesartan medoxomil	10-40 mg
3.	Calcium Channel Blockers:	Verapamil,	120-480 mg
		Diltiazem,	60-36- mg
		Nifedipine,	10-90 mg
		Felodipine,	2.5-20 mg
		Amlodipine,	2.5-10 mg
		S (-) Amlodipine,	2.5-5 mg
		Nitrendipine	5-20 mg
		Lacidipine	2-6 mg
		Benidipine	4-8 mg
		Lercanidipine.	10-20 mg
4.	Diuretics:	**Thiazides** Hydrochlorothiazide	12.5-50 mg
		Chlorthalidone	25-100 mg
		Indapamide	1.25-2.5 mg
		Loop Diuretics Furosemide	40-80 mg
		K^+ sparing Spironolactone	50-400 mg
		Triamterene	150-250 mg
		Amiloride	5-10 mg
		Metalazol	1.25-5 mg
		Torasemide	2.5-5 mg
		Xipamide	20-80 mg
5.	β- Adrenergic blockers:	Propranolol	40-640 mg
		Metoprolol	50-400 mg
		Atenolol,	25-100 mg
		Bisoprolol	2.5-10 mg
		Nebivolol	5-40 mg
		Oxprenolo	40-480 mg
6.	β + α Adrenergic blockers:	Labetalol	50-400 mg
		Carvedilol	12.5-50 mg
		Sotalol	80-320 mg
7.	α Adrenergic blockers:	Prazosin	0.5-20 mg
		Terazosin	1-20 mg
		Doxazosin	1-4 mg
8.	Central sympatholytics	Clonidine	0.1-2.5 mg
		Methyldopa	250 mg
9.	Vasodilators:	**Arteriolar** Hydralazine	25-50 mg
		Minoxidil	10-25 mg
		Diazoxide	10-25 mg
		Arteriolar + venous Sodium nitroprusside	50 mg

DIET FOR A HYPERTENSIVE PATIENT

ECLAMPSIA

Hypertension in pregnant women is known as pre-eclampsia. Pre-eclampsia can progress to a life-threatening condition called eclampsia, which is the development of protein in the urine, generalized swelling, and severe seizures. Other symptoms indicating that brain function is becoming impaired may precede these seizures such as nausea, vomiting, headaches, and vision loss.

It may be of two types:

a) A hypertensive woman becomes pregnant.
b) Pregnancy induced hypertension

In both cases, drugs which are safe in pregnancy must be used.

The development of hypertension, accompanied by protein uria and edema in the third trimester of pregnancy, is referred to as preeclampsia. This syndrome occurs in 5% to 10% of pregnancies, particularly with first pregnancies in women older than age 35 year. In those severely affected, convulsive seizure may appear which are then termed as eclampsia. By long historical precedent, preeclampsia and eclampsia have been referred to as toxemia of pregnancy. No bloodborn toxin has ever been identified, however, and so the historically sanctified term (still in use) is clearly misnomer. Full blown eclampsia may lead to disseminated intravascular coagulation (DIC), with all of its attendant widespread is chemic organ injuries, and so eclampsia is potentially fatal. However, recognition and treatment of preeclampsia has now made eclampsia and, particularly, fatal eclampsia rare.

The triggering events initiating these syndromes are unknown, but the basic feature underlying all cases inadequate maternal blood flow to the placenta secondary to inadequate development of the spiral arteries of the uteroplacental bed. In the third trimester of normal pregnancy, the musculoelastic walls of spiral arteries are replaced by a fibrinous material, permitting them to dilate into wide vascular sinusoids. In preeclampsia and eclampsia, the musculoelastic walls are retained and channels remain narrow. The basis of these vascular abnormalities remains unknown, but a number of consequences ensue:

- Placental hypoperfution with an increased predisposition to the development of infarction.
- Reduced elaboration by the throphoblast of vasodilators: prostacyclin, prostaglandin E_2, and nitric oxide, which in normal pregnancies oppose the effect of renin-angitensin- hence the hypertension of preeclampsia and eclampsia.
- Production by the ischemic placenta of thromboplastic substances such as tissue factor and thromboxane, which probably account for the development of DIC.

Clinically, preeclampsia appears insidiously in the 24^{th} to 25^{th} weeks of gestation, with the development of edema, proteinuria, and rising blood pressure. Should the condition evolve into eclampsia, renal function is impaired, the blood pressure mounts, and convulsions may appear. Prompt therapy early in the course aborts the organ changes, with clearance of all abnormalities.

COUNSELING

Recommendations

- Maintain a healthy weight
- Exercise regularly
- Follow a healthy eating plan and limit the amount of salt in your food.
- Avoid smoking. If you already smoke, try to stop

- If you drink alcohol, try to limit the amount the you drink
- Avoid bush medicines as these could affect the treatment you are taking.
- Read food labels to see how much sodium or salts is in the food

In addition you need to

Check your blood pressure regularly,Take the medications that your doctor prescribes regularly Take time to relax and learn, how to manage stress

Eating Habits

Avoid

Salted potato or corn chips,Reduction in saturated fat,Salted peanuts,Fried chicken or other fried foods,Canned foods such as luncheon meats, chopped ham, Vienna sausages and corned beef,High salt seasonings such as garlic salt, onion salt, season-all etc.,Preparing foods with lots of oil,Salt or corned fish,Pigtail, salt beef, salted pig snout,Salami, bolgna sausage, ham or other salted meats,Soft drinks,Adding salt to your food at the table

You should eat

Five servings of fresh fruits and vegetables every day

Also try to have regular servings of

whole wheat bread, whole grain cereals such as brown rice**,**Pasta, potatoes**,**Increase in take of fiber**,**Beans, nuts
Low fat cheeses, low fat milk**,**Lean meats, fish, chicken without the skin

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