CHAPTER-2
Hypertension

Physiology:

(1) The heart and blood vessels network (vasculature) is in essence a closed hydraulic pumping system and pressure. The heart is the pump, and blood is the fluid flowing through the arteries and veins.

(2) The heart is a four-chambered muscular mass whose contractions exert the pumping force. The two smaller chambers, the atria, receive blood; two large chambers, the two ventricles, force it out into the circulation. The left ventricle pumps blood to most of the body and the right atrium receives blood from the body. The left atrium receives blood from the lungs. Thus there is a continuous circulation of blood, which is kept in motion by the pumping contractions of the cardiac muscles. Arteries are the vessels that carry blood from the heart, veins are the route back.

(3) Movement of blood through the circulation occurs, because the system is under pressure. The heartbeat is a major source of this pressure. In case of hypertension the pressure in the system is too high. It occurs as a result of a variety of factors, which are not completely understood. Blood pressure means simply the amount of pressure exerted in the arterial system at any point in time.

(4) A pressure is created due to the contraction of the heart and negative pressure of sucking pressure is formed due to dilatation of the heart. This pressure is responsible for mobilization of the blood from heart to body and back to heart. Therefore this pressure is responsible for the circulation of blood from one part of the body to another part. Due to this fact this pressure is known as 'Blood Pressure', we also can say that the pressure, which is responsible for circulation as mobilization of blood, is known as blood pressure.

(5) There are so many factors, which are responsible for the quantum of this pressure and these are:

1. Pumping action of the heart.
2. Elastic & recoil of the arteriols.

3. Pressure gradient —

BP falls from the left to the right side of the heart. In the big arteries the average pressure is 120mm of Hg. In the arteriols the pressure sharply falls to about 50-60mm of Hg. In the capillaries the pressure is about 15mm Hg. In the veins the pressure falls further, while near the heart the pressures is 0mm Hg. or even negative. Due to this pressure gradient BP from the higher to the lower pressure i.e. from the left to the right side of the heart, Other factors :

4. Respiration.

5. Muscular Exercise.


(6) An adequate BP is require and if it is not maintained up to an optimum level than in that case it will harm the body in both case whether it is less or more. It will harm either by inadequate regulation of nutrition to all parts of body or by decreasing the efficiency of heart due to over exertion of heart for maintaining the higher level of BP.

(7) The heart is constructed in such a way that it will continue to beat even if it is totally denervated. The sino-atrial node (SA node) is a region of heart muscle that has the capacity to depolarize in a repetitive fashion. The normal heartbeat is formed at this locus.

(8) Hypertension is suggest that it is a state of chronically elevated blood pressure usually above a level of 140/95mm of Hg. For a small percentage of the case of hypertension 10%, a cause such as Kidney dysfunction, cerebral disease, coarctation of aorta or other physiological malfunctions can be found for the majority of cases. However there are no known aetiology, these are termed essential (Eyer, 1975; Gutmann & Bonson, 1970).

(9) Although the exact aetiology of essential hypertension is unknown. There is amply evidence to suggest that psychological factors are involved in its development.
The precise role of this psychological factor and how they influence hypertensive individual is a subject of considerable controversy. Research clearly shows that blood pressure is raised temporarily by emotional states but there is very little evidence that these states when prolonged lead to hypertension.

More and More attention is being given to the assessment of:

1. Personality
2. Individual difference &
3. Behavioural patterns associated with Such disorder.

Here there are two criteria:

(a) It involves assessing hypertension reaction to stress by studying their reactivity to various stimuli and require to compare with normotensive. Here the individuals who develop a persistently elevated blood pressure level will also exhibit a tendency to response to emotionally disturbing events with abnormal increase in blood pressure.

(b) A second group of study concerns the assessment of psychological characteristic of hypertensive such studies are psychological test and interviews to assess the personality. It has been seen that hypertensive possess certain unique personality characteristic setting them apart from normotensive. Whether their characteristic have caused the hypertensions or are a result of the disorder is not known. There is no agreement of what level of arterial pressure defines hypertensions and differentiate them from normotensive.

Probably one of the greatest concern in hypertensive research is the selection of appropriate control groups with which hypertensive should be compared is not completely clear. Saslow, Gressel, Shobe, Du Bois and Schroeder (1950) indicated a need to control for.
1. Age
2. Sex
3. Race
4. Cultural Pattern
5. Education
6. Occupation and
7. Socio-Economic Level.

Wolf and Wolf (1951) noted that resentment elevated blood pressure while despair or depression lowered it. This phenomenon was particularly prevalent among hypertensive. Mores, Daniels and Nickesson (1956) found that anxiety raised blood pressure and rage elevated it to even higher level.

Hines (1940) also felt that people who had normal but highly reactive blood pressure in response to the cold pressure test were most likely to develop hypertension later in life.

Breath holds test. It assumed that the lack of $O^2$ and increase of $CO^2$ served as a stressor for increasing breathing exercise test.

In adult males the average systolic pressure is 125-130 m.m. of Hg. ±15 (Viz from 110 to 145 m.m of Hg).

Nerves of heart and their actions:

(l) The regulation of heart is effected through the afferent and efferent nerves of the heart.

(I) Afferent nerves:

(a) From the heart and aorta through the vagus nerve.

(b) From the heart through the inferior cervical and four thorasic ganglia and 1st four thorasic nerve roots into spinal cord.

(c) From the carotid sinus through the glossopharyngeal nerve.
(II) **Efferent Nerves** :

(a) Vagus and

(b) Sympathetic

(a) **Vagus** :

Dorsal nuclei of the vagus situated in the floor of 4th ventricle in the medulla. The route is as such-vagus-Preganglionic fibres-Cardiac fibres-Separated in the neck and then towards the heart-Cardiac plexus-deep superficial-in atrial muscles make synoptic connection with ganglionic fibres arise and supply to :- SA Nodes, AV Nodes and also extended between the muscles fibres.

**Function** :

Vagus exerts a tonic inhibitory control over all parts of heart. Acetyl choline is released ganglionic fibres on stimulation. Vagal tone is of reflex origin being produced by the sino-Aortic nerves. Neutralization the action of vagal tone and vagal nerve will increase the route of the heart. Stimulation of the vagal nerve will produce the effect of :-

(i) Chronotropic by heart rate is slowed down.

(ii) Inotropic : It is due to force of contraction is diminished may be due to constriction of coronary vessels and reduction of blood supply. On the whole vagus act as the inhibitor of heart.

(b) **Sympathetic Nerves** :

The connector cells of the sympathetic nerves are situated in the lateral horn cells of the upper thoracic segments of the spinal cord.

1. **Excitor Cells** :

   Situated in the superior, middle and inferior cervical ganglion.

   Post ganglionic as exciter fibres take origin and per pass directly to the heart. Fibres supply the:-
(i) SA Nodes
(ii) AV Nodes
(iii) Atrial and Ventricular Muscles
(iv) Fibres also carry vasodilator fibres to the coronary vessels.

The sympathetic exerts a slight tonic accelerating action on the human heart.

**Sympathin**:

An eqinephrine like substance is released by the post ganglionic fibres at the sympathetic on stimulation. Stimulation will lead accelerator and increase the frequency of heart rate.

**Augmentor**:

Increase the force of contraction.

**Force of heart beat**:

Strength of contraction depends upon mainly on 3 factors.

(i) Initial length of cardiac muscles. Greater the initial length of cardiac muscles, stronger will be the contraction.

(ii) Length of diastolic pause-filling, rest and recovery take place during diastole. Hence shorter diastolic pause to inadequate for these and the force of contraction will diminish.

(iii) Nutrition and oxygen supply— An adequate supply nutrition and oxygen is essential for efficient cardiac activity.

Blood pressure is the lateral pressure exerted by blood on vessel walls.

(1) Systolic Blood Pressure
(2) Diastolic Blood Pressure
(3) Pulse Pressure

In adults the relation between the three pressure is as follows:

\[ \text{SP/DP/PP} = 3/2/1/ \]
(2) **Cardiac Centre** :

There are two nerve centres

(i) **Cardiac Inhibitory Centre** :

It is situated in the medulla and in either identical or a part of or is closely related to the dorsal nuclei of the vagus. It has the same action as the vagus.

(ii) **Cardiac Acceleratory Centre** :

It is situated in the same region and is related to the sympathetic nerves of heart. It exerts the same influence as the sympathetic Nerves. There is evidence to show that these two centre freely inter communicate and hence a reciprocal relation. Stimulation of one will depress the other and vice-verse.

(3) **Role of Hypothalamus** :

Being the highest centre of autonomic hypothalamic exert a superior control over heart. The posterior Hypothalamic nuclei are related to the sympathetic system. Stimulation of these nuclei may cause acceleration of heart. The middle nuclei related to the parasympathetic. Hence stimulation of these causes slowing of heart and average diastolic pressure is 70-90 m.m of Hg.

**Factors Controlling Arterial Blood Pressure** :

(a) Cardiac output.

(b) Elasticity of Arterial walls

(c) Peripheral Resistance :

It is the resistance which blood has to over come while passing through the periphery. The chief seat of peripheral resistance is the arteriols end to smaller extent to capillaries, peripheral resistance depends on the following :

(i) Velocity of Blood :

A Rapidly flowing stream will have more frictional effect than a slower one. Hence pressure is high in the aorta
but low in the capillaries.

(ii) Viscosity

(iii) Elasticity

(iv) Lumen of the vessels

Peripheral resistance is inversely proportional to the lumen of the vessels. One should expect therefore that the capillaries having the smallest lumen should have the highest pressure. But this is not the case. Because the velocity of blood being lowest in the capillaries the frictional effect is very low. Hence the pressure there is also low. The seat of peripheral resistance is found to be chiefly in the arterioles where the velocity is fairly high and the lumen is narrow.

(d) Blood volume.

(e) Viscosity of the blood. It will effect the diastolic blood Pressure.

BM Hegde (1988), Hypertension the other side of the Coin:

High BP is not a disease and it is possibly not even a syndrome. It only represents one end of the Gaussian curve of the mean arterial pressure. A small percent of the Hypertensive population (5-10%) have a disease which has clear-cut pathology and the elevated BP is only a consequence of the disease. This is called secondary hypertension. The rest i.e. 90-95% of hypertensive population is called primary hypertension. The aetiology, the pathophysiologic back ground, the natural history and even the necessity and response to treatment of this enigma still remains elusive.

Human beings with thin arterial and cardiac walls cannot possibly withstand very high pressure inside the arterial system for long and consequently develops target organs damage and suffer probably premature death.

Essential hypertension can be better described in four categories for better understanding and important factors of blood pressure.
Type one Hypertensions (Stress related):

Social cultural and environmental stress is probably one of the very important triggers for blood pressure to go up either acutely or on a long-term basis. Chronic sustained stimulation of the sympathetic system in rats is known to result in elevated BP, degeneration of the CVS, myocardial necrosis and death due to cardiac arrhythmia.

This type of high blood pressure needs the removal of its course for control and not the toxic antihypertensive drugs, which make life miserable for this already wretched group of individuals, prof, pickering the man who clarified some blind spots in the field of hypertension.

Rev. Stephen Hallves in 1773 was the first who measured arterial pressure in his illustrious mare. He was the one who even measures the pressure in the cardiac chambers. It was an Italian Riva Rocci in 1856 who perfected the technique of measuring the BP with the help of the mercury manometer. He was helped in his efforts by Korot coff in Russia and Mohemmed in England. (Folkow B, Physiological aspects of primary Hypertension, Physiological Rev, 1982; 62; 347-504).

Do we achieve any thing at all by forcefully lowering the marginally elevated pressure at different points in the scale where as severely elevated pressure with obvious target organs involvement when lowered by intervention would reduce morbidity and possibly mortality.

Entity of primary Hypertension is not homogenous and can be classified into various types depending on their characteristic features.

Case Study:

(1) Akkama was a Indian widow aged 64 who lives with her only son and took at her future through her son's world. She was admitted with BP reading 230/140 m.m. of Hg. Since she did not have any other worrying factors; She was left alone and given tender loving care. In the Hospital. BP steadily fell and within a week almost normalized to 150/90 m.m of Hg. When she was asked to go home, the pressure started mounting again. The thought of going home and facing the real world around her, was too much for Akkama. Her son was such a rascal.
and an antisocial rogue that the world around despised him. This very thought was enough to trigger a hypertensive mechanism in Akkama who is personification of good Indian womanhood.

(ii) Sheenappa aged 60 a retired court employee was under care 15 years ago with very high BP and all the drugs available then failed to bring his pressure down. When he was fell a sleep his BP however was always down to near normal levels. This led the discovery of a great calamity in his life. His young daughter was married to a prosperous young man in Gulf who died 6 months after the marriage due to an accident. Sheonappa lost his wife very early in life. The thought and the site of his young widow daughter kept up Sheenappa pressure at where it was and one afternoon he succumbed to his disease with a cerebral haemorrhage.

(iii) Mrs. Farnandes aged 78 was admitted with a pressure of 210/140 m.m. of Hg. Nothing seemed to bring her pressure down. After 4 days in the Hospital the pressure gradually started coming down and she remained normal ever since. Her son-in-law came back from Dubai and gave her Rs. 2000/- to be kept in safe custody while he went to see some of his own relatives. Around that time her maidservant took two days leave to go home. When the son-in-law came back Mrs. Farnandes had forgotten where she had kept the money. She was confused and thought that the servant had decamped with the booty. Her BP went up. On the 4th day of her hospitalization, the son-in-law found the money safe inside the steel almirah, Mrs. Farnandes BP started going down. Socials, cultural and environment stress is probably one of the very important triggers for BP to go up either actually or on a long term basis (Environment and cardiovascular disease, Psychosoma med, 1984, 46:33-6).

Chronic sustained stimulation of the sympathetic system in rats is known to result in elevated BP, degeneration of cardiovascular system, myocardial necrosis and death due to cardio arrhythmias (frohlich ED the adrenergic nervous systems and hyperten, myocllc, proc, 1977; 52; 361-8)
This type of high BP needs the normal of its cause for control and not the toxic antihypertensive drugs, which makes life miserable for this already some blind spots in the field of hypertension. (Pickering GW High BP second edition, London, Churchill Livingston 1968).

Richard Bright in 1836 was impressed by the vascular damage seen in many patients of the post mortem table that he exclaimed in his diary "I confess, I am afraid the Kidney is the root cause of all this trouble. A brilliant young professor of medicine in Vienna Charles Sehar Sehrmtdt wrote 1747 that he had seen a patient with a vehement agitation of the mild and spastic constriction of the vascular bed. He was definitely referring to hypertension. He had prescribed that best treatment which looks modern and physiological to-day.

-Change of mode of living
-Venesaction and
-Vasodilators like nitrates

As per 45 PARKARAN (SUSHRUTA SAMAITA)

स्रोध शोक भयायास विरद्दानातपालनाः।
कटववल्ल लवस्तु सार तीक्षोण्यातै विदाहिनाः॥
विदाहम् गलो दुष्टो रसः पिल्यं प्रवाहयेत॥
विदाहम् स्वरुपः पिल्यं विदाह्यायु म्होऽणितम॥

The above stanza very clearly discussed the vascular disease and its aetio-pathogenesis and comes from the ancient Indian medical text Sushruta Samhita.

**Type 2 Hypertension (Population Migrations)**:

Normally the Polynesians belong to the low pressure group and they rarely develop Hypertension (Patrick RC relationship between blood pressure and modernity among ponaplan Int. J. Epidemiol, 1983;12(1):36-40) when they are exposed to the stress of modern living in the western World their BP go up. Modernity in the aetio-pathogenetic factor in this type of hypertensive disease.
It is our responsibility to find out factors in the process of so-called civilization which encourage human BP to go up and how fast we can change that for the good of our future generation.

Studies on maquay Monkey have shown that if they kept in groups with members constant their BP remains normal and they do not develop degenerative disease. If they kept in groups where the members constantly change the BP go up faster and the monkeys get prone to vascular degenerative disease (Editorial environment and cardio vascular disease psycho soma, med, 1984; 46; 33-6) population studies in California have shown that people belonging to church groups have lower BP than those who do not belong to any group (Hegde Bm, Hypertension update, Bulletin VHS madras, 1982;52;6-9)

Unmarried people have higher pressure than people with happy families. The same ethnic groups have different BP based on their environment. Study of adopted children shown the BP of children following the pressure of their new environment rather than their original parental environment, Hegde BM, biweekly supervised treatment of mild-moderate hypertension proc 9th Asian pacific cong, cardiol, 1987 about 332 page 87 New Zealand)

Type-3 Hypertension (Latrogenic): Case Study:

(1) Mr. Rao has been under psychiatric treatment for mild mental depression. He had recovered from his depression and was back at work. When one day he went to see his doctor for some headache his doctor recorded his BP as 160/100 m.m. of Hg. and Started him reserpine and propronolol.

(2) Mrs. Sushila saw her doctor with a complaint of left mammary chest pain. She had stopped menstruating and was easily irritable. She could not sleep well and used to wake up very easily in the morning. She had taken oral contraceptives for a long time. Her BP was 170/104 m.m. of Hg. and she was given propranolol by her doctor. Her
problem has been ever increasing and now she gets severe suicidal tendencies

This Class of patient who have a marginally elevated BP have underlying depressive traits (Hegde BM, IMA college Bulletin CME special issue 1987;77;51-54) Antihypertensives especially belonging to the RawWolfia alkaloids or B blockers group will worsen the depression and increase their problems.

Type-4 Hypertention :

It includes to those primary hypertensive patients with various target organ involvement. As per BM Hedge, his data shows that the different target organs damage have no parallelism with either the level of blood pressure or the duration of elevated arterial pressure. His Manglore study very clearly shown that the left ventricular hypertrophy (Hegde BM Echo-cardiography—its place in clinical medicine Karnataka med Jr. 1983;60:49-55 and Richard B Devereux cardiac involvement in essential hypertension, med, clin, north AM, 1987;71;813-26) When present has no relationship to systolic, Diastolic or mean blood pressure (Hegde BM heart in hypertension bulletin VHS Madras 1987;57;1-4). Similarly it has been clearly shown that hyper cholesterolaemia claimed to have a very close relationship to vascular disease is seen in a very small percentage of hypertension (Hegde BM, fat profile in pindalol treated hypertension, proc-world cong coronary artery disease Bombay, 1984; page 36, Hegde BM- adrenergic blocking drugs. Indian Jr Pharma and Therap, 1987,1:11-6 and BM Hegde-Rao AC, Vidya Rao R and Bhat-EK fat profile in diuretic treated hypertensives, proc, international congress, cardio-vascular pharmacology, San Francis co, 1987 page 84).

Data has proved beyond all doubt that the self intake and type of food we take, have no relationship to the aetiology of hypertension in patients. (Prabhu V. Rao and Hegde BM study of salt metabolism in normotensive medical student Jr. Assoc Physi, India, 1986;34:41)

Hypertensive patients who have cardiac, renal and retinal changes have nothing in common and probably are different disease processes grouped together at the present time because of ignorance
As per Hegde BM he arrived at a clue to find out who amongst them will develop established Hypertension in later life. This hypothesis called postural Hypertension- the indicator of significance? Needs careful consideration.

When an apparently healthy person records border line mild to moderate hypertension a postural change of BP on slanting showing a significant elevation of the diastolic BP (more than 20 m.m. of Hg) with or without slight increase in systolic BP is an indication of significant Hypertension which needs Drug therapy. Similar Postural normotensives could be pointer for future hypertension developing in the individual.

If anxiety scale would have been worked out in all the cases before stopping and restarting therapy. Stress due to anxiety would have been identified. In anxiety due to environmental stress need in depth study. It is now well observed that anxiety can increase both systolic and diastolic hypertension.

Darwin a hundred years ago clearly recognized automatically medicated stimulant action of aggressive emotion.

Patients with angina and other coronary artery heart disease have many episodes of symptom-less transient myocardial ischaemia most of which can not be explained by Physical exertion alone. The association between mental activity and myocardial ischaemia may operate frequently during every day life.

Gupta et al (1989) compared the ECG changes and hoemodynamic effects of various mental stress tasks to those induced by exercise stress test. Available data shows that 68% of the study population developed ECG evidence of ischaemia on mental stress testing composed to 76.3%, during Treadmil testing of the various mental stress tasks emotionally assessing speech produced ST depression in only 8%. Magnitude of ischaemia was comparable for emotionally arousing
speech task and Treadmill test on comparing the haemodynamic changes in the patients where both exercise and speech task were performed. It was seen that ischaemic changes occurred at a lower heart rate, lower systolic blood pressure and lower doubleproduct (Peak HR x Peak S BP) during emotional speech than Treadmil test. Thus they highlighted the importance of personally relevant mental stress as an important precipitant of myocardial ischaemia. Dean field et al; silent myocardial ischaemia due to mental stress, Lancet, 1978,318,1001, Teggart P Etal: emotional catacholamine and the ECG progress in Cardiology 1978,7,119. Lepeschkin E etal: effects of epinephrine and nor epinephrine on the ECG of 100 normal subject. Amer. J. cardiol 1960,5,593. Possible pathological mechanism of ischaemia caused by mental stress is not clear. Catacholamine and cortisol the secretion of which is increased during stress are important in the genesis of ischaemia. Effect of catacholamine were believed to be caused by the changes in the repolarisation velocity of the ventricular action potential.

Funken, sen and associates put forwarded a hypothesis that epinephrine release would be specifically associated with anxious emotional state.

Non epinephrine released with aggressive emotional state. (Dea, field et al: silent myocardial ischaemia due to mental stress lancent 1978,318,1004).

Rate of rise of catacholamine level is also important in the genesis of ischaemia. In mental stress there is a transient vasoconstriction. There is little doubt that patient have flow limiting stenosis in the proximal coronary artery.

Predictable many problems remain unresolved. Why in a similar group of patients exposed to the same emotional challenge, some had ECG changes of ischaemia while others are of effected little it at all? One explanation may be the difference in the type of personality. Type A personality being CHD prone. Other possible explanation would be the degree of stress felt by each patient is related partly to his facility with numerative task.

Comparison of haemodynamic responses during speech task. As most significant changes seen during the speech task it was used or comparison in 38 patients in whom both the tests were performed. Heart rate increased during speech tasks and the magnitude of increase was significantly less than that during exercise (PL 0.01). The difference between elevation of systolic blood pressure during speech and exercise task was also significant (PL 0.01) with lesser elevations seen during mental task. The mean double product (peak heart rate & Peack systolic blood pressure was also significantly higher in the exercise group (Plo.01).

Hypertension is a common cardiovascular disease in India. Mutatic and Grant (1962) in a random stratified sample from Poona found an incidence of 0.76 percent in urban and 0.60% in Rural population. Although the causes of Hypertension are still not clear, a variety or pathological process have been known to be responsible for secondary elevation of blood pressure. Prominent among these are diseases of the Kidney, Chr. Glomerulonephritis and Chr. Pyelonephritis, tumours of adrenal gland, congenital narrowing of the aorta and toxaemia of pregnancy. Altogether these are estimated to account for about 10% or less of the cases of hypertension. In a great majority of cases primary or essential Hypertension the causes are largely unknown. Community studies and clinical observations have suggested in association of primary Hypertension with a number of factor such as heredity, diet, climate, occupation, stress and strain of modern life and over weight. Hypertensive heart disease is noticed generally speaking in people of both sexes of the age of 40 and above. In India as also in other parts of the world, persons most affected by this disease are the middle class and the rich and is relatively uncommon among the working class.

Hyper lipidaemias, cigarette smoking, hyper-uricaemia, lack
of exercise, sedentary habits, high consumption of sugar and continuing stress condition are the predisposing factor.

**Factors which produce Hypertension:**

In more than 95% of cases a specific underlying cause of hypertension cannot be found. Such patient are said to have essential Hypertension.

The pathogenesis of essential Hypertension is not clearly understood. Different investigators have proposed the Kidney, the peripheral resistance vessels and the sympathetic nervous system as the seat of the primary abnormality.

Hypertension is more common in some ethnic group, particularly American blacks and Japanese and approximately 40-60% is explained by genetic factors.

Important environmental factors include a high salt intake, heavy consumption of Alcohol, Obesity and impaired intrauterine growth.

In about 5% of unselected cases hypertension can be shown to be consequence of a specific disease or abnormality leading to sodium retention and/or peripheral vaso constriction (secondary Hypertension)

Harrison (2001), 15th edition of internal medicine, Davidson's principles and practice of medicine (1999) eighteenth edition as per them the following classification (based on causes) have been suggested for the Secondary Hypertension:

1. Alcohol
2. Pregnancy (Pre eclampsia)
3. Renal Disease
   i. Renal Vascular disease
   ii. Parenchymal renal disease particularly glomerulonephritis and Pyelonephritis
   iii. Polycystic Kidney disease
(4) Endocrine disease

(i) Pheochromocytoma
(ii) Cushing's syndrome
(iii) Primary hyperaldosteronism (conn's syndrome)
(iv) Hyperparathyroidism
(v) Acromegaly
(vi) Primary hypothyroidism
(vii) Thyrotoxicosis
(viii) Congenital adrenal hyperplasia due to 11-B-hydroxylase or 17 hydroxysteroid dehydrogenase deficiency.
(ix) Liddle's syndrome
(x) 11-B-hydroxy steroid dehydrogenase deficiency.


(6) Co-aractation of the aorta.

Physiological or other factors:

Exercise, anxiety, discomfort and unfamiliar surroundings can all lead to a Transient rise in BP. Sphygmomanometry particularly when performed by a doctor can cause an unrepresentative surge in BP which has been termed white coat hypertension, Davidson's principles and practice of medicine (1999).

Some studies suggest that home or ambulatory BP measurements can provide a better assessment of risk than casual clinic BP recordings, but these remains controversial and require confirmations.

As per the sixth report of the joint National committee on detection, education and Treatment of High blood pressure (JNC VI) Arch intern med 1997;157:2314 suggested the following risk factors:
A. **Major Risk Factors** :

1. Smoking
2. Dyslipidaemia
3. Diabetes mellitus
4. Age < 60 years
5. Sex (Men and Menopausal women)
   
   Woman : <65 years or men <55 years

B. **Other Factors** :

1. Wt.
2. Alcohol
3. Sodium intake
4. Lack of K intake
5. Lack of Ca & Mg. Intake
6. Intake of dietary saturated fat and cholesterol

Hereditary plays a part of determining, whether a patient will develop essential Hypertension. Indeed the condition has been described as an inherited tendency to develop high BP in middle life. In an investigation in families where parents had normal blood pressure and incidence of hypertension was only 3%. When one parent suffered from hypertension the incidence in the children were 45% (Ay man). In 1956 a family was reported with 3 generation of hypertensive patients (wear). All of the first generation died of stroke except one who died on high blood pressure at the age of 62. All these of the sisters married men with moderate hypertension and their 3 children, one of them a girl aged 12 years, all have BP above normal. The hereditary tendency in general and transmitted as a Mendelian dominant (William Boyd).

Shapiro et al (1982) studied the variables that influence BP and Hypertension include :
1. Family History
2. Personality and
3. Stress :
   (i) Natural disasters
   (ii) Culture
   (iii) Urbanization
   (iv) Occupation stress in individual
   (v) Type A behaviour

C. Other Factors such as :
   1. Obesity
   2. Physical activity
   3. Dietary Sodium
   4. Caffeine
   5. Alcohol

Gentry W. Doyle et al (1983) reports on an ongoing study of social, psychological and ecological factors that may combined to increase an individual's risk for essential Hypertension and findings to that indicate that anger coping style play an active role in predisposing an individual to elevated BP and/or essential hypertension.

Thomas, John et al (1983) reviewed the literature indicate that relevant precursor of hypertension are early elevated casual systolic BP, positive family history and obesity (in females). Addition predisposing a enhancing factors point to high sodium ingestion, heavy smoking and high Socio-ecologic stress. Evidence for a high-risk hypertensive personality is not conclusive.

Burch et al (1983) investigated that depression can develop in the hypertensive patient for a variety of regions, including reaction to the diagnosis to the hypertension.
either on neuroticism or extra version. But nearly half of the Psychosomatics indicated anxiety as the commonest syndrome.

M. Seth (1981) has assessed the manifest needs of hypertensive and normal subjects on Edward inwards personal preferenceschedule (EPPS). He has reported that hypertensive have greater needs to achievement, Dominance, abasement, endurance, and aggression while normal expressed the need for affiliation and change.

N. Ial, Ahuja and Madhukar (1982) have observed that hypertensive reported more number distressing life events and given higher mean distress rating than normals. This observation was more characteristics of male who were over 45 years of age.


**Stress**:

Stress has the role in conceptualization of the relationship between behaviour and disease. Body reach to threatening environmental events in a more or less stereotyped way and this response can have negative health consequences. The response is known as stress. The events giving rise to it are stressors. If stress leads to disease, methods of modifying response of removing the stimuli-giving rise to stress would have useful health consequences.

A pattern of adjustment in animals placed in a variety of aversive environments. Selye called this pattern "the general adoption syndrome" (GAS). It has three stages :-

(a) The initial alarm reaction

(b) It is followed by a period of resistance to the aversive stimulus

(c) If the stimulus not removed exhaustion ensure and finally death Selye identified this response as mediated by the
anteriot pituitary and the adrenal cortex.

Sequence of events is roughly as follows:

Stress-Pituitary-ACTH-Adrenal cortex-release the corticosteroids which act on a wide range of body site to generate defensive reactions. It persists prolonged enough, lead involution of thymus and lymph nodes, enlargement of the adrenal gland and ulceration of stomach results.

Current Definition of Stress:

Non specific response of the body to any demand. The state within a living creature, which results from the interaction of the organism with noxious stimuli or circumstances i.e. it is a dynamic state within the organism it is not a stimulus, assault, load, symbol, burden, or any aspect of the environment, internal, external, social or otherwise.

Mechanism relating life stress to coronary disease entail increased sympathetic activation cause mobilization free fatty acid in the blood, stress from adipose tissue, higher activity cause hyper triglyceride if not consumed by physical activity cause atherosclerosis, cause in-crease risk of 'myocardial infarction' or decrease efficiency of heart.

Type 'A' Personality:

Friedman and Rosenman (1969) both cardiologist they prepared a list of risk factors for myocardial infarction. As per friedman-those individuals who are engaged in a relatively chronic struggle to obtain an unlimited number of poorly defined things from their environment in the shortest period of time and if necessary against the opposing efforts of other things or persons in this same environment (1969). Jenkins (1971) added some description including extreme competitiveness, achievement, motivation, impatience, restlessness and time sense urgency (1971). Reviewed by friedman and Rosenman. Type 'B's described as easy going and relaxed. 'A'. behaviour speed and impatience, job involvement and hard driving conscientiousness.

Ratio of cardio vascular disease between A and B is 1.7 to 4.5 times high for 'A', frustration is considered a Key aspect of the
Psychosomatic Symptoms:

Insomnia, fatigue, dizziness and headache. Persons with coronary disease score high on these symptoms than did normals. Incidence of coronary disease, angina and myocardial infarction.

Like wise type 'B' S do some times appear in coronary care units. It is suggestive that detailed analysis and more study is require. Carruther's original hypothesis presented in 1969 stated that the mechanism relating life stress to coronary disease entail increased sympathetic nervous system activity which leads to mobilization from adipose tissues of free fatty acids in to the blood stream. If those fatty acids are not utilised via metabolic activity they are converted to 'Triglycerides' by the liver and are then available for formation of atheroma. Hyper-tri-glyceridaemia is statistically associated with increased incidence of coronary artery disease (Lewis et all 1974). Release of catachol-amines particularly norepinephrine in emotion thus is the precipitating event. In an attempt to elucidate the relationship among emotion, exertion and tryglyceride. Taggart and corruthers studied racing car drivers on the ground that they work in a highly emotionally arousing stimulus situation that entails relatively little Physical exertion. A possible analogy to the occupational stress in less romantic circumstances is reactive. (In absence of recreational environment, the occupational stress will exert more adversely).

1st Study:

In one study plasma samples well taken at various time during the 3 hrs. Following a race (Taggart and Corruthers 1971). Analysis show a linear relationship between circulating catacholamines and free fatty acids and a dramatic rise in each at the end of the race. Triglycerides rose on free fatty acids fell during the following hours lending support to the conversion hypothesis.

2nd Study:

Taggart and associates (1973) assessed catacholamines and
free fatty acids levels before and after public speaking (1973). Nor-
epinephrine rose while epinephrine held constant. Free fatty acids also 
rose, supporting the idea that nor-epinephrine is the critical catacholamine 
in the aroused fat release phenomena.

3rd Study:

Oxprenolol (B adrenergic blocking drugs), it selectively 
suppresses nor-epinephrine rise. Fatty acids level actually fell after public 
speaking in persons given oxpranolol. It is B adrenergic blocking drug and 
has the general effect of blocking sympathetic activation. They lower 
pulse and release anxiety especially when somatic factors play a major 
role in the patients complaints (Tryer and lader 1973). This would suggest 
that emotional leads to increase fatty acids level and this effect can be modified by blocking the activity of the sympathetic nervous system.

Wolf and colleagues (1964) examined the relationship 
between a personal way of coping with a stressful environment and 
measures of 17 OHCS, a secretion of the adrenal cortex (1964).

Bourne and associates (1968) who compared officers and 
enlisted men in Vietnam during a period of expected enemy attack (1968). 
The officers showed higher 17-OHCS levels. In a similar vein several studies 
reported dramatic increases in resting heart rate among executive given increased responsibility (Moods, 1975).

The data suggest that stressful environments interact with 
personality attributes like denial and with specifics of the subjects role 
in the situation to yield different Neuro-endocrino-logical effects and presumably different degrees of disease rises. Type 'A' behaviour involves sympathetic discharge and this method of dealing with environmental events in turn has a variety of effects some of which may be atherogenic.

Crowding:

As noted earlier epidemiologic evidence shows that a variety of illnesses increase in frequently as human populations become increasingly dense through urbanization. Deaths from coronary disease were reported as being higher in urban than rural VS populations as
long ago as 1960 (Enterline et al. 1960), and a comparison of prison inmates found those housed in dormitory group to have BP than inmate housed alone (Ostfeld and Atri 1975). Harleuly and Coworkers (1975) compared urban demographic distributions differing in population density and found BP to be higher in the dense, more stressful living condition (1970).

The result of Malmo's research on symptom stereotypy indicate that under conditions of stress individuals with psychosomatic complaints respond maximally with disturbances in certain critical symptom areas. In 1939 Alexander also noted that blood pressure among hypertensive patients were markedly elevated during sessions which particularly stressful or disturbing to them. Schneider and Zangari (1951) revealed that whenever hypertensives become anxious during an interview their systolic and diastolic blood pressure rose. By introducing conflictive topics Wolf, Pfuffer, Ripley, Winter and Wolf (1948) were able to produce sharp increases in the blood pressure of hypertensives. While moderate blood pressure increases occurred in a group of normotensives both the intensity and duration of their reactions of the hypertensives.

Observing hypertensives during interviews Vander Valk (1957) noted marked blood pressure increases in response to topics of rejection by others hostility or ambivalence towards parents. When patients were discussing materials with hostile content blood pressure was reported by Kaplan, Gothchalk, Macidocloox Robovit and Ross (1961) to rise in hypertensives and fall in normotensives.

During discussions of disturbing interpersonal problems Wolf and Wolf (1951) noted that resentment elevated blood pressure while despair or depression lowered it. The phenomenon was particularly prevalent among hypertensives. Recording the BP of essential hypertensives undergoing psychoanalysis, Mores, Daniel's and Nickerson (1956) found that anxiety raised BP and rage elevated it to even higher levels.

Although Sachachter (1957) did not produce emotional changes by means of an interview technique, his results substantiated interview studies by showing that hypertensives respond to both fear (fear
of electric shock) and anger (the blundering and badgering of a technicians) with significantly greater increase in BP than did normotensives.

Adler, Hermann, Schafer, Schmidt, Schonecke and Rexkulla (1976) found that blood pressure increased during the discussion of topics such as personal failure or loss of status.

Wolf indicated that both hypertensives and controls showed a rise in blood pressure during the discussion of personal topics but that the return to base line occurred more rapidly in the controls than in the hypertensives.

Hypertensive patients usually over react in their BP response to interview situations.

The hyper reactivity of hypertensives to cold pressure have been confirmed by Alam and Smirk (1938); Ayman and Gold shine (1938); Smithwick and Robertson (1951) and Thacker (1940).

Hines (1940) also felt that people who had normal but highly reactive blood pressure in response to the cold pressure test were most likely develop Hypertension later in life.

**Scope of Hypertension in BSF Personnel**

The details of the scope of stress in BSF Jawan for hypertension is as follows:

BSF : Means Border Security Force assigned with the duty to guard the International border and line of control of Jammu and Kashmir region and prevent crimes on Border. Therefore duty on the front line where fear of the enemy/smuggler/infiltrator remains all the time. Even the border tension is there due to the presence of force strength and activity of the force personnel on opposite side to the border. Even during peace they are require to guard the border with the motive to control the activity of infiltrator and smugglers. More over during the patrolling of border area there is risk to cross the border due to desert thick vegetation and hilly area where no line of demarcation is clear. With the result there is fear of unknown cause. There is no fix hours of duty. On the border there is no concept of Sunday or holiday. Sources for reduction of tension
are very less. Sources of Physical as well as mental recreation are limited.

Life is very monotonous. They are living in a very limited number of 15-20 personnel for long time. Place is also very remote and the human population in nearby area is negligible. Weather condition of desert, plane area of Punjab or Kashmir of the border area is hostile and extreme.

Service conditions are involving high mobility and frequently require to shift in contrast weather e.g. desert to snow belt area of Kashmir, or humid heat area of North Eastern, to plane dry heat area of Punjab. It provides less opportunity for proper acclimatization of wealthier and area. It creates the difficulty in performing the assigned duty which gives the sense of fatigue and burden.

In force, there is a one man show and democratic environment can not be maintained. Boss is right. Psychological environment depend upon the boss what kind he is. All the time one is require to live in discipline and no freedom of union, no freedom to raise the voice. There is a hierarchy of ranks and this require to be maintained with strict code of conduct and high discipline.

Because of hardship and prolong duration of duty, personnel are not aware regarding their perks and privileges, leaves can not be granted as per planning. It depends upon the disturbance causing activity and also depends upon the strength of troops available. What ever the facilities are on paper not available in the ground. It is difficult for him to solve the home problems. Even fresh vegetable, milk not available inmost of the border area.

He is living away from modernisation and civilization. he is unable to keep him self in main stream. It creates a psychological pressure and make him maladjusted in his society. Not able to provide guidance and proper supervision to his family regarding children education, health, maintenance of day to day requirement of family, social relations and growth of the family.

If we just give a thought on world scenario to terrorism; in India also most of the states facing the problem of terrorism, violence,
anarchy and human killing activities. With the result security forces are require to deploy there. It also has experienced that the disturbance once started in any state is continue, rather it is on increasing side; with the result the security forces which are deploy there, are not coming back to their posting place. It has broken the concept of family in troops. Due to this deployment in counter insurgency role (CI role) or internal security duty (is duty) or in Anti infiltration role (AI role) they are working far away from their base and from where family contact is not possible. Perforce they are require to maintain three; establishment; one, where they are performing duties; two where they have kept their family; and three where parents are living. More over he is getting very less pay to meet the requirement of his liability of all these establishments. In such circumstances he is bound to under go high psychological pressure.

He is not able to maintain the proper relation with his family, relatives and society due to inability to show his presence in all the social activities whether of happiness or sadness. To maintain a proper contact through correspondence, mail, telephone etc is again a matter of problem.

Force personnel are having very tough life even from the beginning of the training to retirement. They are getting the training to face the death. A very strict discipline monotonous life, very tough physical as well as mental exhausting training. Badly fatigue. No one is there to listen them. During training no relaxation, no freedom, no time to get relaxed. From early morning to late evening they are busy in one or other kind of job, which involves physical exertion and mental fatigue.

After the training; they get posting either in field or in peace area. In field, again they are away from the family. They themselves and families are depend upon the salary what they get per month. He perforce is require to maintain two establishments and according to that whatever salary he gets is very less.

(1) He has no contact with his kin and kith, relatives, Peers and family. Only in a year when he comes on leave then only it is possible to have interaction with his near and
(2) He is away from the touch of modernization, civilization. In force there is monotonous life. Every morning he sees the same face, same food and same activities, which are responsible to make him, tired. When he goes on leave he looks stranger in his behaviour and has poor personality factor to establish properly in his society, he is ignorant in absence of the social touch for his social growth.

(3) All these factors are responsible to bring him in frustration, insecure all the time tenseful, because of high risk of life; lack of confidence to be settled in his society; economic inadequency; unsocial behaviour; rude and uncultured. Because of these poor psychological and environmental factors he is prone to be unsuitable in his civil life and bring him under high pressure and made him to indulge in smoking and drinking habit. Now World is advancing very fast and to be in the modern world main stream is difficult for him. All these factors are responsible to destabilize his emotional behaviour. Hence in this modern and advancing world, force personnel are more prone to be hypertensive in comparison to others.

Now a days he is all the time facing the challenge of bullet if he is on border; there is exchange of fire every day if he is little lazy or relaxed may face the dare-consequences. If he is away from the border than he is in terrorist prone area where risk is much. Because he does not know who is terrorist and who is not terrorist. Even during the journey when he is on leave or otherwise again he is on high risk and occasions of bomb blast on railway stations are its witness. He is a source of receiving difficulties, frustration, conflict without getting any clue to solve them amicably with the result the pressure is gradually accumulated making him restlessness. This unresolved pressure cause high tension and responsible for his miserable life. Although the number of volunteer retirements are increased but even after retirement he is not comfortable, his children whom he could not arranged good education
facility, could not given them guidance and guardianship are not properly settled. After the retirement the source of income is pension. It is also a great tension and conflict whether to go on retirement or not. Both the places becomes uncomfortable for him.

**Consequences of Hypertension**

The most vulnerable organs because of the hypertension are:

(i) Heart
(ii) Kidney
(iii) Retina and
(iv) Brain

It represents a combination of adaptive and degenerative changes in the heart and circulation.

**Heart**

There is a general Hypertrophy of the left ventricle as result of increased work. Slowly and gradually it cause the heart failure.

**Circulation**

The adaptive changes of work hypertrophy are seen in the arterial tree, particularly in the small arteries and arterioles. Here, there is thickening of the muscular media, which has important consequences, since in these small vessels a minor increase in wall thickness has large effect on peripheral resistance.

These changes may be gradually progressive over many years. Eventually degenerative or wear and tear lesions develop in both heart and arteries and lead to the lethal consequences of 'Hypertension'. The consequences of this is the development of hypertensive heart failure.

**Kidney**

The consequence of medial and intimal hypertrophy is most important in the kidney and brain. Thickening of the renal arterial branches was one of the earlist recognized Pathological lesions of hypertension. The lesion which ultimately so impairs renal blood flow that renal failure
occurs, is a combination of medial hypertrophy, elastic proliferation and intimal proliferation.

**Retina**:

The changes which leads to a severe reduction in the lumen of the vessel is responsible for the rapid development of micro infarcts in retina and kidney. It cause 'Hypertensive Retinopathy'. Papilloedema or swelling of the optic disc may be present with or without haemorrhages and exudates. It may be the result of intracranial hypertension caused by general brain oedema secondary to breakdown of cerebral auto regulation in the face of high pressure, gradually vision diminish.

**Brain**:

It causes Hypertensive encephalopathy. In hypertension fits and coma may be associated. There may be brain stroke and cerebral haemorrhage. In severe stroke death may be followed or if stroke is moderate with mild haemorrhage or blood leakage paralysis may occurred. It depends upon the severity of haemorrhage and involvement of vital centre of brain.

The outcome in any subject is determined by the interplay of the actual level of arterial pressure and the duration for which it is raised causing wear and tear on vessels which are variably resistant to this stress.

There is clear linear correlation between arterial pressure and risk of death thus a subject with arterial pressure of 100 m.m Hg. Will be less of risk than one with 120 m.m. Hg. A man with a systolic arterial pressure of 170 m.m. Hg will have about twice the mortality risk of the man whose pressure is 120mm Hg. If other risk factors are present, the hazard may be even greater. A rise in systolic pressure might be expected when the peripheral arterioi tree is stiff. The increase in morbidity with increase in arterial pressure is more striking for stroke and heart failure than for coronary or peripheral artery disease.

The risk of morbidity or death at any level of arterial pressure will vary many times, depending on the presence of one more of these other adverse factors such as cholesterol, Cigarette smoking and diabetes.