REVIEW OF LITERATURE
CHAPTER I

HYPERTENSION AND SPECIFIC HYPERTENSIVE RISK FACTORS
Blood pressure is the force exerted by the blood pumped from the heart against the walls of the arteries. Or, it is the amount of force required to circulate the blood round the body and is dependent on the energy of the heart action, elasticity of the arterial walls and volume and viscosity of the blood. The human heart pumps intermittently by means of a sudden contraction of the entire ventricular musculature, then it relaxes and remains quiescent for a brief period until the next contraction. Hence, the cardiac cycle is divided into two main parts, the contraction phase being called systole and the resting phase of the cycle, diastole. The duration of these periods is unequal, the diastolic period, at normal heart rates, being considerably longer than the systolic. The maximum pressure is during systolic as it occurs near the end of the stroke output of the left ventricle and the minimum pressure during diastole (Youmans, 1954).

Haemodynamics of Hypertension:

Arterial pressure mainly reflects the interaction of two factors, cardiac output and total peripheral resistance. An increased heart rate is a fairly consistent feature of hypertension, and is observed with different levels of
arterial pressure. Early in the course of essential hypertension, cardiac output is elevated while total peripheral resistance may be within the normal range or only slightly raised. Together with mild arteriolar constriction in early hypertension there may be peripheral venoconstriction, which serves to redistribute the circulating blood from the periphery to the cardiopulmonary area. As constrictor influences on the capacitance and resistance vessels persist and progress, arterial pressure and vascular resistance increase further. Ultimately, the heart shows evidence of adaptive hyperfunction and left ventricular hypertrophy; and with advancing hypertension and further increase in total peripheral resistance the cardiac output, together with stroke volume, gradually falls until cardiac failure supervenes (WHO, 1978).

The increase in cardiac output in early hypertension may be due to an intrinsic intrarenal defect. Such a defect impairs renal excretion of sodium and water at normal arterial pressures to maintain normal intravenous and extracellular volumes. This defect permits development of an expanded plasma volume that increases the cardiac output and arterial pressure. However, with time, the total circulation adapts to the increased volume, output and pressure by increasing the total peripheral pressure (Prohlick,
Role of Adrenergic Nervous System in Blood Pressure Regulation:

The acute changes in the blood pressure are controlled through the baroreceptors and the autonomic nervous system. Carotid sinus and aortic arch baroreceptors consist of stretch receptors located in vessel walls and send information reflecting the level of blood pressure to the central nervous system. Nerve impulses are generated from various centres and are transmitted through postganglionic nerves to the blood vessels and the heart. Central neural impulses to the cardiovascular system are mediated by neurohumoral substances, the commonest being norepinephrine. When norepinephrine is released from the postganglionic sympathetic nerve ending, it traverses a synaptic cleft and stimulates the effecter cardiovascular adrenergic receptors. These receptors either produce contraction or relaxation (Frohlick, 1983; Covell, 1985).

Whether hypertension is primarily caused by a defect in the adrenergic nervous system or whether this mechanism is reinforced by or exaggerated by such factors as sodium excess, the renopressor system or other mechanisms still remains to be seen.
Role of Renin-angiotensin System in Blood Pressure Regulation:

Argument still continues as to whether participation of the renopressor system is a cause or an effect of hypertension. Participation of the renopressor system is initiated with the release of renin from juxtaglomerular apparatus of the kidney, from various stimuli like decreased renal perfusion pressure, flow or volume; reduced delivery of sodium to the maculadensa (heavily nucleated area in the distal renal tubule); enhanced adrenergic stimulation of kidney; and altered concentration of circulating electrolytes (including potassium) or hormones (such as aldosterone). As a result of all or any of these factors, renin is released from the kidney and acts in the blood on a protein substrate angiotensinogen that has been synthesized within the liver to free a decapeptide-angiotensin I. This loses two peptides from its molecule and is converted to angiotensin II. This is a rather stable molecule and has several target organs on which it acts

i) It constricts arteriolar smooth muscle raising arterial pressure,

ii) it stimulates the release of catecholamines and aldosterone from the adrenal medulla and cortex respectively. Aldosterone, in turn, stimulates the distal tubule of the kidney to actively retain sodium and water, thereby
3. Liver produces angiotensinogen.

1. Decreased perfusion pressure in the afferent arteriole stimulates secretion of renin by the juxtaglomerular cells.

Renin reacts with angiotensinogen to form angiotensin I.

5. Activation of angiotensin I to angiotensin II occurs in the pulmonary capillary bed by a converting enzyme.

Angiotensin I is converted to angiotensin II by an enzyme.

6. Angiotensin II stimulates release of aldosterone from the adrenal cortex. Aldosterone causes increased sodium and water reabsorption by the tubules of the kidney. Result is increased blood volume.

Figure 1: RENIN-ANGIOTENSION SYSTEM IN BLOOD PRESSURE REGULATION.
further increasing the arterial pressure. Also, potassium excretion is increased and hypokalemic alkalosis can result. The process has been represented diagramatically in Figure 1.

iii) It also acts directly in the brain at different centres to increase the outflow of sympathetic impulses, thereby further elevating arterial pressure and promoting thirst, which serves to insure greater fluid intake for volume retention.

Thus, whether angiotensin II is primarily generated as an effector cause of some stimuli, a vicious cycle can be initiated or maintained that will permit continued elevation of arterial pressure (Peart, 1967; WHO, 1978; Frohlick, 1983; Satoskar and Bhandarkar, 1986).

Technique of Blood Pressure Measurement:

Blood pressure depends on the position of the person in whom blood pressure is being measured, on his physical and mental state, time, temperature, eating or smoking preceding the measurement and on the person who is recording. Hence certain specifications are given by W.H.O. (1978) which are to be strictly followed during blood pressure measurement.
The mercury sphygmomanometer that is usually used to record blood pressure should be kept in good working order without any dust in rubber tubes linking the inflation bulb with the mercury reservoir and without any foreign matter in the space above the mercury column. The deflation valve must be in good working order; the cuff must be in good condition.

The standard cuff is 12.5 cms wide and sufficiently long to surround at least 2/3rd of the upper arm. Different widths are required for blood pressure measurement in children and obese adults. The following are recommended by the American Heart Association.

<table>
<thead>
<tr>
<th>Age</th>
<th>Width of the cuff</th>
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<tbody>
<tr>
<td>Under one year</td>
<td>2.5 cms</td>
</tr>
<tr>
<td>1 - 4 years</td>
<td>5 or 6 cms</td>
</tr>
<tr>
<td>4 - 8 years</td>
<td>8 or 9 cms</td>
</tr>
<tr>
<td>Average adults</td>
<td>12.5 cms</td>
</tr>
<tr>
<td>Obese adults</td>
<td>14 cms</td>
</tr>
</tbody>
</table>

The antecubital fossa should be left free while tying the cuff. If a choice has to be made between a cuff which is too small and one which is too large, the larger one should be chosen.
Measurement should be made on the seated subject at a comfortable temperature. There should be no severe exertion, eating, smoking or exposure to cold immediately preceding the measurement. The manometer should be placed on horizontal surface at the level of the heart of the subject. The examiner locates the brachial pulse in the antecubital fossa and places a stethoscope over the artery. The cuff is inflated rapidly to 20-30 mm Hg above the pressure at which the radial pulse disappears to palpitation. The cuff is then gradually deflated at a constant rate of 2.3 mm Hg per second. The mercury column is watched continuously and carefully. Systolic pressure is taken as the pressure at which the ear distinguishes the first arterial sound. The point at which the last arterial sound disappears (Korotkoff Phase 5) is usually taken as diastolic pressure. The SI units are kilo pascal but the general units are millimeters of mercury (WHO, 1978).

RISK FACTORS IN HYPERTENSION

Age

There is a positive relationship between age and the incidence of hypertension, even though hypertension is not an essential aspect of aging. The increase in systolic pressure appears to continue throughout life, whereas there is a tendency for diastolic pressure to level off around
the age of 55 to 60 (Indrayan et al., 1972; W.H.O., 1978; Waldron et al., 1982; Siconolli et al., 1985).

The mean blood pressure levels are usually lower in women than in men in the early decades of life but the position reverses after the age of 45 - 50 (WHO, 1978; Gupta et al., 1979). The mean blood pressure levels in urban and rural areas were observed to be around 135/90 and 115/76 mm Hg respectively in subjects under 40 years and 160/100 and 128/81 mm Hg respectively after the age of 40 years (Gupta et al., 1979).

Table 1 gives the distribution of blood pressure according to age in men and women (Holden et al., 1983).

Table 1

Distribution of blood pressure according to age and sex

<table>
<thead>
<tr>
<th>Age</th>
<th>Systolic Pressure</th>
<th>Diastolic Pressure</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(mm Hg)</td>
<td>(mm Hg)</td>
</tr>
<tr>
<td></td>
<td>Men</td>
<td>Women</td>
</tr>
<tr>
<td>99</td>
<td>205</td>
<td>213</td>
</tr>
<tr>
<td>65</td>
<td>145</td>
<td>140</td>
</tr>
<tr>
<td>53</td>
<td>135</td>
<td>128</td>
</tr>
<tr>
<td>37</td>
<td>125</td>
<td>117</td>
</tr>
<tr>
<td>27</td>
<td>117</td>
<td>109</td>
</tr>
<tr>
<td>21</td>
<td>110</td>
<td>102</td>
</tr>
<tr>
<td>18</td>
<td>90</td>
<td>85</td>
</tr>
</tbody>
</table>

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The levels that are considered to be hypertensive also vary with age and are given in Table 2 (Barbara and Rosendalm, 1984).

The gradual increase in systolic pressure with age may be due to decreased aortic elasticity. The increase in diastolic pressure results from an increased resistance in the peripheral blood vessels. The baroreceptors in the aorta and carotid arteries become less sensitive to pressure changes with age. This results in a slow response to postural changes and sudden change in position may cause dizziness or syncope. Pressure on the carotid sinus may cause serious slowing of the heart rate and may be elicited by twisting and turning of the head (Street and Earles, 1984) Body mass index also increases with age and may further effect the onset of hypertension in the elderly (Jalkanen et al, 1989).

Table 2

Hypertension as it relates to different age groups

<table>
<thead>
<tr>
<th>Age group years</th>
<th>Blood Pressure (Systolic/diastolic mm Hg)</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Normal</td>
</tr>
<tr>
<td>Infants</td>
<td>80/40</td>
</tr>
<tr>
<td>7 - 11</td>
<td>100/60</td>
</tr>
<tr>
<td>12 - 17</td>
<td>115/70</td>
</tr>
<tr>
<td>20 - 45</td>
<td>120-125/75-80</td>
</tr>
<tr>
<td>45 - 65</td>
<td>135-140/85</td>
</tr>
<tr>
<td>Over 65</td>
<td>150/85</td>
</tr>
</tbody>
</table>
Heridity

The concept that genetic factors play an important role in blood pressure regulation has been well established. Hypertension is common among relatives of hypertensives (Harlan et al, 1962; Brown et al, 1976; Smith, 1977). A screening programme comprising more than half of a million adults revealed that a positive family history of hypertension was associated with a prevalence of hypertension which was twice that found in persons with negative histories (Stamler et al, 1979).

Urinary sodium to potassium ratio, weight, body-mass index were positively correlated to blood pressure in subjects with a positive family history of hypertension and no such correlations were found in those with no family history of hypertension (Pietinen et al, 1979). Offspring of hypertensive parents of the age group 18-37 years were found to have higher diastolic pressures (Ibsen, 1984).

Thus, periodical check ups by persons with a positive family history of hypertension would help in early detection of blood pressure elevations before hypertensive damage to heart, kidney and brain.
Obesity

The heart is the target organ affected by both obesity and arterial hypertension—two disorders frequently co-existing in the same patient. Weight is an indirect measure of the adequacy of nutrition as well as the expenditure of energy through physical activity. If the balance of calories consumed is greater than that expended, weight will increase to obesity (Bavlik et al, 1983). Congestive heart failure, sudden death and coronary heart disease are common sequelae of obesity hypertension (Messerli, 1984).

Hypertension detection and follow-up programme (1977) found that 60 per cent of the participants were more than the ideal weight by 20 per cent. On screening one million Americans it was observed that hypertension was twice as prevalent in younger over-weight individuals and 50 per cent more prevalent in older obese subjects than in normal weight controls (Stamler et al, 1978).

In the national health and nutrition examination survey, Body mass index was the nutritional factor most strongly and consistently related to blood pressure (Harlan et al, 1984). Intersalt study group (1988) also reported that body mass index was strongly, positively and independently associated with blood pressure.
Obesity may tend to mitigate the harmful effects of a chronically elevated total peripheral and renal vascular resistance and lessen end organ damage such as nephrosclerosis in essential hypertension. A decrease in risk of cardiovascular death was observed with increasing body mass index (Cambien et al, 1985). However, since both obesity and hypertension increase cardiac work load by different mechanisms, their presence in the same patient results in a double burden to the left ventricle (Messerli, 1984). An excess of body fat requires a high cardiac output to meet the higher metabolic demands. Because of an increment in cardiac output and stroke volume, left ventricular stroke work is augmented in obesity and left ventricular end-diastolic pressure may become elevated (Messerli et al, 1983).

Compared to lean patients, obese patients had greater left arterial, ventricular and aortic root diameters; posterior and septal wall thickness and ventricular mass, cardiac output, stroke volume and stroke work. Hypertensive patients had increased posterior wall thickness, end diastolic wall stress, stroke work and a lower radius to posterior wall thickness ratio indicating concentric hypertrophy when compared to normotensive patients (Messerli et al, 1983).
Endocrine and metabolic changes in obesity include hyperinsulinemia, insulin resistance, decreased carbohydrate tolerance, increased serum triglycerides and cholesterol, increased cortisol secretion rate and metabolic clearance without increased plasma cortisol or urinary free cortisol and possibly sympathetic nervous activity (Dustan, 1983).

Benefits of Weight Reduction:

Weight reduction is one of the few therapeutic strategies for hypertension. It reduces arterial pressure in both normotensives and hypertensives and in obese hypertensive persons, blood pressure falls before normal weight is achieved (Dustan, 1983). A weight loss of 12 kg gives a blood pressure fall of 21/13 mm Hg and a weight loss of 3 kg gives a fall of 7/4 mm Hg (Hovell, 1982).

Decrease in arterial pressure is significantly correlated with fall in body weight. Total circulating and cardio pulmonary blood volumes were significantly reduced and these changes permitted decreases in venous return and cardiac output. This fall in cardiac output was directly related to a contracted total blood volume and decrease in cardio-pulmonary blood volume. Patients
who did not loose weight showed no changes in any of these hemodynamic measurements. In addition weight loss was associated with reduced resting circulating levels of plasma norepinephrine, suggesting that diminished adrenergic function may also be related to weight reduction and its associated fall in arterial pressure (Reisin et al, 1983).

On modest weight reduction, there were profound changes in the lipid and lipoprotein composition. Nelives et al (1982) found that after four weeks of weight reducing diet, total cholesterol decreased on the average by 44 mg per cent and serum triglycerides by 65 mg per cent while low density lipoproteins declined by 30 mg per cent and very low density lipoproteins by 10 mg per cent high density lipoproteins showed a small decrease of 7 mg per cent. Similar results were reported by Stamler et al (1987) and Berglund et al (1989) except that there was a slight increase in high density lipoproteins. They also observed that the lipid changes were in the opposite direction on drug treatment.

Dahl et al (1958) reported that hypertension of obesity is salt dependent as they found that salt restriction alone and combined salt and calorie restriction normalized arterial pressure. But Reisin et al (1978)
found lowering of blood pressure on weight reduction of obese hypertensives irrespective of sodium intake.

Experiments were conducted to observe whether the blood pressure reduction on loss of body weight was due to calorie restriction or has an independent effect. The fall in blood pressure with weight loss has been associated with a fall in cardiac output (Ramsay et al, 1978; Reisin et al, 1983) probably due to a reduction in vascular sympathetic nerve activity which reflects body's normal response to calorie restriction i.e., a diminution of catecholamine mediated metabolic activity (Jung et al, 1979) Reduced sympathetic nerve activity may also help to lower blood pressure less directly. Plasma renin activity falls progressively with weight loss (Juck et al, 1981). The changes in sodium and fluid balance are likely to stimulate renin rather than reduce it and hence this effect may be due to reduced vascular efferent sympathetic activity, which ascertain the blood pressure lowering effect of weight reduction exclusively to calorie restriction. But Weinsier et al (1991) reported after achieving a weight loss of 13 kgs in their subjects that weight loss has a blood pressure lowering effect that is distinct from energy restriction and that it independently reduces plasma volume, cardiac output and plasma renin activity, insulin, resting metabolic rate and heart rate declined and sodium and
potassium balances were negative during energy restriction. Catecholamines, renin, aldosterone, plasma volume, cardiac output and blood pressure showed no consistent response to changes in energy intake.

Weight reduction, a non-drug treatment of hypertension was compared with drug therapy. Reisin et al (1978) observed that weight reduction alone had reduced blood pressure to a greater extent than weight reduction combined with drug therapy. Similarly, Stamler et al (1987) found that reduction of over weight and salt intake was more effective than drug therapy. But Berglund et al (1989) reported that while dietary therapy (weight reduction and sodium restriction) brought diastolic blood pressure to 90 mm Hg in only 29 per cent of the subjects, drug treatment produced the same result in 73 per cent of the subjects and conclude that drug therapy was more effective, but the lipid profile changes were better on weight reduction.

Thus, weight reduction is one of the cheapest and harmless methods of reducing blood pressure. The obese borderline hypertensives have been recommended to first reduce their over weight before commencing drug therapy (Wasir and Mohan, 1989).
Alcohol

Alcohol has recently been recognised as an important cause of hypertension. It is responsible for at least 10 per cent of hypertension in men and 1 per cent in women (Kaplan, 1985). Klatsky et al (1977) described a positive association between alcohol intake and blood pressure in about 84,000 subjects, independent of age, sex, race, smoking and adiposity. The percentage of persons having hypertension in drinkers and non-drinkers was 11.2 per cent and 4.7 per cent respectively in white men and 15.1 per cent and 10.2 per cent respectively in black men. When compared to non-drinkers, those taking 6 or more portions each day and had strikingly significant elevation of blood pressure.

Gruchow et al (1985) on analysing the data from the first Health and Nutrition Examination Survey found that alcohol and sodium intakes were the strongest dietary predictors of blood pressure. Similar results were observed in the Zutphen study which was carried out over a period of 10 years; alcohol intake was significantly and positively related to systolic and diastolic blood pressure (Kromhout et al, 1985). The Intersalt study (1988) reported that high alcohol intake was strongly, positively and independently associated with blood pressure. In the Scottish Heart Health Study, alcohol consumption, body mass index
and potassium excretion had significantly independent effects but not sodium excretion (Smith et al, 1988).

More direct evidence for the pressor effect of alcohol comes from investigations where alcohol has been given experimentally. Consumption of 80 g alcohol daily by untreated hypertensive patients was followed by gradual increases in systolic and diastolic blood pressure (Fortter and Beevers, 1984; Malhotra et al, 1985); both fell when drinking stopped and rose in those who started again (Saunders et al, 1961; Fortter and Beevers, 1984). No significant changes were observed in normotensive people (Malhotra et al, 1985).

Mechanism for the association of stroke death and alcohol consumption is blood pressure (Kozararevill et al 1980). Thus curtailing of alcohol intake may lead to improved blood pressure control and may reduce the need for antihypertensive drugs. Among individuals who reduced their alcohol intake from 452 ml ethanol/week to 64 ml/week by switching to low alcohol beer, mean blood pressure fell by 5/3 mm Hg (Puddey et al, 1987). Among alcohol dependent patients who abstain, more substantial falls in blood pressure - 15 to 20 mm Hg systolic and 10 to 15 mm Hg diastolic are to be expected (Saunders, 1987).

Baghurst and Dwyer (1981) found no relationship
between high alcohol consumption and elevated blood pressure in young men and suggest that the relationship between these two factors become clinically obvious either with longer exposure to alcohol or in an older age group in whom the mechanisms of control of blood pressure may have already been altered.

Moderate alcohol intake was said to have protective action against ischaemic heart disease. But Cook et al (1983) found a progressive increase in blood pressure even at low levels of consumption (10-60 g ethanol/week). Shaper et al (1987) found no significant relation between reported alcohol intake and incidence of ischaemic heart disease.

Etiology of Alcohol Induced Hypertension

The biological mechanisms by which alcohol raises blood pressure have not been clarified. Alcohol causes irregularities in the heart beat by interfering with the hearts conduction system. Physiological studies have shown that moderate doses of alcohol has transiently increased cardiac output and blood pressure. The intensity of the effect and the mechanism responsible seem to vary according to the tissue alcohol level. Alcohol reduces
cardiac contractility which may explain its harmful effect on patients already suffering from heart disease (West et al, 1984).

There seems to be two distinct mechanisms by which alcohol may be related to hypertension a 'slow pressor' effect of alcohol that is seen after two or more days of regular drinking and a withdrawal related one (Saunders, 1987).

Porter and Beevers (1984) and Puddey et al (1987) are of the opinion that alcohol induced hypertension is more likely to be due to an effect of alcohol rather than the pressor response produced by alcohol withdrawal. Porter and Beevers (1984) found no changes in plasma renin activity and a significant drop in plasma cortisol with a fall in blood pressure after stopping alcohol. A significant rise was observed on resuming alcohol intake and it is possible that cortisol may have caused a rise in blood pressure by enhancing the hemodynamic effects of circulating catecholamines.

Bannan et al (1984) are of the opinion that alcohol withdrawal hypertension is more clearly linked with pressor substances and that clinically the syndrome resembles
a heightened sympathetic state. Plasma noradrenaline concentrations, renin activity and concentrations of aldosterone and cortisol were raised during withdrawal, though only cortisol concentration correlated significantly with blood pressure. But as raised blood pressure is seen in those apparently having only one or two drinks daily, this may be a less likely cause (Saunders et al, 1981).

Liver-dysfunction was observed in the hypertensives and was related to alcohol consumption, heavy body weight, male sex, young age and higher diastolic blood pressure. It was also suggested that alcohol and obesity were the principal causal factors probably due to fatty intilibration of the liver (Ramsay, 1977; Beevers, 1977).

Ethanol induces structural and chemical disorganisation of membranes, interferes with iron transport and various biochemical functions are deranged which may possibly allow calcium to accumulate in the cell. Calcium accumulation in vascular smooth muscle may increase sensitivity to circulating vasopressors and account for the increased incidence of hypertension in chronic alcoholics (Knochel, 1983).

In view of the deleterious effects of chronic
alcoholism, reduction and preferably abstinence from alcohol has been recommended in hypertensives.

Smoking

Smoking is a major cause of premature coronary heart disease, peripheral vascular disease and stroke. In addition, it has other serious effects on health. In many countries, the widespread adoption of smoking has impeded improvements in health and length of life that could otherwise have been expected from the control of mass infectious diseases. Smoking control is a primary objective of preventive medicine and is expected to yield extensive and diverse benefits (WHO, 1986).

In the Australian Therapeutic Trial in mild hypertension (1980), out of 3427 hypertensive subjects, 25 per cent were smokers. In the hypertension detection and follow-up programme study (1982) of 10,940 hypertensive subjects, 26 per cent were smokers. In the Medical Research Council Trial of mild hypertension (1985) 25.32 per cent of patients were smokers. A significant interaction between smoking and cardiac events was observed by International Prospective Primary Prevention study in hypertension (1985) and by Anand et al (1990).
Dewan and Howlands (1986) found that 16 per cent of the smokers had hypertension whereas the prevalence of high blood pressure was only 3.8 per cent in non-smokers. In another study of 918 hypertensive patients, 28 per cent were smokers (Anand et al., 1990). Malhotra (1971) found that cigarette smoking was three times more in North India when compared to South India but found no correlation with blood pressure.

Dewan and Howlands (1986) found a positive correlation between number of cigarettes smoked per day and blood pressure levels. The mean blood pressure levels were 122/86 mm Hg in those who smoke 1-5 cigarettes a day, 131/86 mm Hg in those smoking 10, 138/89 mm Hg and 140/91 mm Hg in those smoking 25 and 40 cigarettes per day.

Freestone and Ramsay (1982) reported that blood pressure reduced by 15/8 mm Hg on abstinence from smoking and drinking coffee. They also observed that smoking 2 cigarettes (3.4 mg nicotine) elevated blood pressure by 10/8 mm Hg for 15 minutes and drinking coffee (200 mg caffeine) increased blood pressure by 10/7 mm Hg between 1 and 2 hours.

There was significant increase in serum cholesterol and decrease in high density lipoproteins along with
increase in blood pressure in smokers (Dewan and Rowlands, 1986).

Baer and Radichevich (1985) studied the blood pressure and endocrine responses on cigarette smoking. There was increase in pulse rate and no change in plasma renin activity. In contrast, plasma aldosterone and plasma cortisol levels increased significantly after smoking, suggesting pituitary adrenal mechanism activation. An increase in plasma adrenocorticotropic hormone and plasma catecholamine was also observed. The long-term significance of these acute hormonal changes in regard to blood pressure homeostasis and vascular disease in cigarette smoking remains to be determined.

**Caffeine**

Coffee is drunk widely by majority of the population around the world. There has been speculation that caffeine might contribute to the development of disorders of the cardiovascular and gastro-intestinal systems and that it might promote certain types of cancer (Curatolo and Robertson, 1983).

Short term administration of Caffeine has several noteworthy effects on the cardiovascular system in
Caffeine-naive subjects. Blood pressure is increased, cardiac output and stroke volume are raised and heart rate tends to decline (Robertson et al, 1978).

Prolonged administration of caffeine is not associated with significant elevation in blood pressure, plasma catecholamines or plasma renin activity in patients with borderline hypertension (Robertson et al, 1984).

Hafiner et al (1985), confirmed a positive relationship between coffee consumption and both total and low density lipoprotein cholesterol in both sexes after adjusting for age, ethnicity, obesity, smoking and alcohol intake. But the authors conclude that the positive relationship between coffee and cholesterol may also be due to confounding effects of other aspects of diet like intake of lipids.

Burr et al (1989) conducted a randomized controlled trial to examine the effects of coffee on blood pressure and plasma lipids and found that coffee appeared to cause a small rise in systolic blood pressure and though not statistically significant, a decrease in high density lipoprotein and apolipoprotein AI and increase in total cholesterol and low density lipoprotein cholesterol and apolipoprotein B were observed.
However, the effect of coffee on raising blood pressure is not very significant.

**Stress**

Acute psychological stress can elicit sudden and transient elevations of blood pressure but the role played by chronic emotional stress in the development of hypertension remains to be defined.

Ecological stress has been defined in terms of socio-economic level, crime rate, residential change and over crowding. Blacks living in areas of Detroit with low ecological stress had less hypertension than their counterparts living in high stress areas (Mustacchi, 1977).

Blood pressure were higher in groups which have greater involvement in a money economy, more economic competition, more contact with people of different cultures or beliefs and more unfulfilled aspirations for a return to traditional beliefs and values. Blood pressure levels were also higher in groups for which the predominant family type was a nuclear or father absent family. For Negroes, groups who were descended from slaves had higher blood pressure than other groups (Waldron et al, 1982).
Schnall et al (1990) found that the echocardiographically determined left ventricular mass index was, on average, 10.8 g/m² greater in subjects aged 30-40 years with job strain than in subjects without job strain suggesting structural changes in heart of working men.

Even television viewing can induce pressure response in both hypertensive and normotensive persons. Watching television for a long time proved particularly unhealthy for hypertensives. In individuals without hypertension, blood pressure returned to normal shortly after watching television while in some with hypertension, significant elevations in blood pressure were observed 10-15 hours after the programmes were over. The mental engagement, emotion, concentration or television viewing were considered the factors that influenced vasopressor responses. Thus blood pressure rose more steeply with crime plays, less with sports and news (Mustacchi, 1977).

The hypothesis connecting stress and blood pressure is that stress leads to increased blood pressure which in turn leads to increased weakening and tearing of the linings of the blood vessels, thus providing a focal point for the deposition of cholesterol plaques on the vessel lining. The end result is narrowing of the lumen of vessels, leading to an increased peripheral vascular resistance and further
increase in blood pressure levels. To compensate for the elevated blood pressures, the left ventricle undergoes hypertrophy and requires greater blood and oxygen supply (Bullock and Gelein, 1984) which is in part confirmed by Schnall et al (1990).

As age increases blood pressure increases and those with a positive history of hypertension tend to become hypertensive. When weight increases to obesity, the risk of turning hypertensive is more and weight reduction has been strongly recommended especially in borderline hypertensives. As alcohol intake and smoking are associated with increased risk of hypertension and cardiovascular events, curtailing both the habits are advised in patients with elevated blood pressure and methods to reduce stress like meditation when followed would further reduce blood pressure.

When each factor that augments blood pressure is controlled, the chances of an individual to become hypertensive are decreased and the need for early drug treatment is reduced.