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REVIEW OF LITERATURE

High blood pressure, or hypertension, is not a disease. Rather, it is a symptom complex which may manifest itself in the course of many disorders, and the development of which may be based on one of several mechanisms (Fig 1) (Mukherjee, 1978). A new systemic approach to our as yet incomplete understanding of the complex physiologic and pathogenetic mechanisms which control arterial pressure in health and disease is required to be developed.

Our modern pathophysiologic explanation is that hypertension and cardiac hypertrophy are the result of either or both increased peripheral resistance and effective blood volume expansion. This is also a gross oversimplification, and we do not have as yet, adequate explanations of the intimate renal and other mechanisms which control the blood pressure in primary and secondary hypertension. Secondary hypertension is a symptom developing in the course of a number of renal, endocrinologic, vascular and nervous disorders.

Primary or essential hypertension is perhaps the major health problem of modern man living in industrialized societies. Nearly all who have studied the problem agree that it occurs as a result of environmental and genetic or familial factors.
CONTROL CIRCUITS IN BLOOD PRESSURE REGULATION

SYSTEM ———— NEURAL CONTROL LOOPS ———— EFFECCTOR ORGANS ———— HUMORAL CONTROL LOOPS ———— VESSEL WALLS
COMPONENT VIA BRAIN VIA KIDNEY

TRANSDUCERS CEREBRAL CORTEX Receptors
TRANSMITTERS CEREBRAL CORTEX
TRANSFORMER CNS
TRANSMITTERS SYMPATHEIC VS VAGUS
TRANSFORMERS ADRENAL MEDULLA
TRANSMITTERS NORMOMELINE EPINEPHRINE
TRANSFORMER

Fig. 1 Diagram to show the interconnections of components on the control loops which regulate the circulation. In the central column are the effector units which input or receive the energy carried by the blood flowing under pressure. At each side are the circuits which receive information on the state of the circulation based on the transmural pressure differential and the elastic and viscous properties of the vessel walls. This information is transformed and transmitted back to the effector units to regulate the blood pressure.
Possible Etiologies:

It is generally accepted that susceptibility to high blood pressure is genetic in nature, but the method of transmission is unclear. It has been suggested that inheritance is through a single incomplete dominant autosomal gene, but polygenic inheritance has also been postulated. We believe that it is also possible that all men react normally to an environmental factor, such as abnormally high salt intake, by developing increasing blood pressure with age and that the small percentage of those who do not have inherited a genetic resistance to high blood pressure. Be that as it may, primary hypertension appears to have roots early in life. Blood pressures in young children from families with history of hypertension are higher than those from families free of hypertension (Goodhart and Shils, 1980).

As industrialized populations grow older, all but 15 to 20 per cent have increases in blood pressures. These latter may be genetically resistant to the development of hypertension. At some time or other in the life of each individual, an upward gradient of blood pressure may or may not eventually pass through those arbitrary levels defined by the World Health Organization and other medical organizations as "above normal". If it does and if it is maintained for a time, the individual will be declared "hypertensive". Rising blood pressure with age is typical of primary
hypertension and differentiates it from the secondary forms of hypertension.

Environmental factors considered to be acting in populations with high blood pressure are: the stresses of industrial life, overcrowding, air pollution, psychosocial and psychologic perturbations and dietary disturbances. Among the latter, studies have incriminated "soft" drinking water (Morris et al. 1961); high cadmium intake (Sherrett & Feinleib, 1975); Calcium consumption (Kesteloot & Geboergs, 1982); potassium/sodium ratio (Langford & Watson, 1975) and salt (sodium) intake (Dahl, 1972).

Early manifestations of hypertension:

A high initial blood-pressure level is the strongest, but still a weak, predictor of future hypertension. The frequency of established hypertension at the age of 40 years in persons whose blood pressure was in the "borderline" range at the age of 20 is about 20% (Julius & Schork, 1978). This figure is three times higher than the prevalence of later hypertension in the rest of the population. This borderline elevation of blood pressure in youth (blood pressure oscillating above and below 139/89 mm Hg (18.6/11.9kPa) or an average of multiple readings between 135-145/85-90 mm Hg (18.0-19.4/11.3-12.0 kPa) (Julius & Schork 1978) increases the risk of the development of hypertension. However, the majority of these patients with borderline elevation of blood
pressure at 20 years of age do not develop hypertension by the age of 40. Consequently additional methods to identify future hypertensive individuals have been sought. These are (a) provocative tests and (b) descriptive characteristics.

(a) Provocative tests are based on the notion that hyper-responsiveness of blood pressure to a pressor stimulus reflects an overall tendency to excessive blood-pressure fluctuation and that cumulatively such pressor episodes lead to sustained hypertension. Blood-pressure hyper-responsiveness during mental arithmetic tests and tilting has been described in persons with borderline hypertension, but the response to static exercise, dynamic exercise, volume expansion, and injection of pressor agents was found to be normal (Julius & Schork 1971). The overall, naturally occurring variation in blood pressure in borderline hypertension is known to be normal (Wåsson et al 1980; Mancia et al 1980). The suggestion that repeated pressor episodes lead to established hypertension has not been adequately proved in experimental models.

(b) Descriptive characteristics. The following characteristics, in addition to raised blood pressure, are predictors of future hypertension: family history, racial background, overweight and increased resting heart rate. Specific personality characteristics have been described in borderline hypertension, but their predictive value is doubtful.
It has been calculated from the literature that in a group of individuals having both borderline hypertension and all known risk factors at the age of 40 years the annual incidence rate of hypertension is up to 5.5% (Julius & Schork 1978). Such individuals with all risk factors for hypertension are rare and in this multiple-risk factor prediction, the level of blood pressure still remained the strongest risk factor. The order of importance of the contributions of the other risk factors was family history, race, weight, and heart rate.

It is important to note that in many studies one or only a few baseline blood pressure readings have been used. It is clear that the predictive value of pressure measurements is increased if more readings are taken.

Haemodynamics of early hypertension:

The so-called borderline hypertension, which may be a precursor of later hypertension, has been extensively investigated in selected populations. Most of the data have been obtained in white, middleclass males. In some patients, cardiac output is elevated, heart rate is very high, stroke volume and cardiopulmonary blood volume are increased, and peripheral resistance is inappropriately high for the prevailing cardiac output (Julius & Schork 1971). However, in the majority of subjects the haemodynamic pattern resembles that in established hypertension: normal cardiac output decreased
stroke volume, increased heart rate, and elevated vascular resistance (Lud-Johansen, 1967). Plasma volume is decreased and plasma renin concentration may be normal or low. The increased total vascular resistance is detected in the forearm vessels; the resistance at maximal vasodilation is elevated, and the responsiveness to vasoconstrictors is increased both indicative of structural changes in the arterioles (Sivertson 1976).

Minerals and Hypertension:

Sodium:

Increased salt intake leads to expansion of extracellular fluid, increased cardiac output, overperfusion of the tissues; this by autoregulation leads to higher peripheral vascular resistance and high blood pressure with normalization of cardiac output. While there is no clear evidence of increased total body sodium or extracellular fluid sodium in primary hypertension, it has been shown that the arteries from hypertensives have an increased sodium content per unit of dry weight. This may add to the thickening of the artery wall, with a resulting increase in vascular resistance. It may also be related to enhanced vasoconstriction.

The degree to which sodium intake influences the development of hypertension is still debatable. Several communities whose daily intake of sodium chloride is 3 g (or less) have low average blood pressure and show little tendency for pressures
to rise with age. When people migrate from such communities to areas where the daily salt intake is around 7–8 g, their blood pressure increases proportionately although there are several confounding factors such as social changes and altered overall nutritional status. Dahl (1962) has plotted population sodium intakes against the incidence of hypertension (Fig. 2). It remains unclear if the change in salt intake is solely or partially responsible (WHO 1978).

Epidemiological studies among adult men and women in the USA, Europe and New Zealand have failed to demonstrate a clear relationship between salt intake or exertion and blood pressure.

Macgregor et al (1982) have demonstrated in a double blind study that moderate sodium restriction alone without alteration in potassium intake caused a 6% fall in mean arterial blood pressure. The urinary excretion of sodium was also reduced considerably suggesting that moderate sodium restriction achieved by not adding salt or avoiding sodium laden foods should, if not already, become a part of the management of essential hypertension.

Reduction in sodium intake to a range of 50–100 g. Eq. per day often prevents the development of hypertension in susceptible persons and makes treatment much easier and more effective if hypertension develops (Holden et al 1983 and Seribner, 1983).
Fig. 2: Salt intake and prevalence of high blood pressure in different populations.
Potassium:

The importance of potassium in blood pressure regulation was suggested by Addison in 1928. Some years later, McQuarrie et al. (1936) achieved reduction in blood pressure by adding potassium chloride to the diet of salt eating hypertensive patients.

In the following years further evidence was gathered that potassium has a definite role in blood pressure regulation through several mechanisms.

Potassium was shown to have natriuretic properties (Berliner 1960; Kahn & Böhm, 1967). Experimental studies (Young et al. 1970) demonstrate that small increases in plasma potassium within the physiological range have long-term effects on sodium balance, potent enough to produce sustained extracellular volume contraction.

Increasing dietary potassium causes a significant decrease in plasma renin activity (Young et al. 1976). This may influence blood pressure through a reduction in angiotension II generation.

In vitro studies with renomedullary cells indicate that an increase in potassium concentration in the medium produces an increase in prostaglandin F release (Knapp et al. 1980).
Potassium also has a direct effect on vascular peripheral resistances. A small increase in serum potassium concentration produced local vasodilatation in experiments conducted in both humans and animals (Glover et al 1962, Simonsen et al 1959). It seems that this effect results from an action of potassium on sodium-potassium ATPase of the vascular smooth muscle (Chen et al 1972).

Lately several authors have raised the possibility that a large potassium intake has a protective effect against the hypertensiogenic properties of excessive salt. There is also epidemiological evidence that communities which eat small amount of sodium usually eat large amount of potassium and hence a low prevalence of hypertension. The fall in blood pressure was found to be independent of urinary excretion of sodium. Potassium supplementation may reduce sympathetic activity on vascular sensitivity to nor-adrenalin, both of which could be dependent on the effect of external potassium concentration on the Na⁺K⁺ pump. Meneely and Batterbee (1976) believe that the high sodium, low potassium diet ingested in Western civilization, combined with a genetic susceptibility is a principal factor in the genesis and perpetuation of essential hypertension.

A study on the racial difference in blood pressure between blacks and whites in Georgia showed a negative
correlation between blood pressure and dietary potassium. Blacks had higher average blood pressure and greater prevalence of hypertension, while ingesting the same quantities of sodium as whites but significantly less potassium (Grim et al 1980). Sullivan et al (1980) reported that urinary potassium was higher in borderline hypertensive patients whose blood pressure did not increase in response to dietary salt loading than in those who did and concluded that potassium blunts the prohypertensive effect of sodium.

In a randomised double blind cross-over trial of increased oral potassium (64 mEq per day) versus placebo in normal healthy adults with no history of hypertension and living on normal sodium diet, it was observed that a significantly greater proportion of the subjects on potassium supplementation had lower systolic and diastolic pressure than that on placebo. The mean diastolic pressure was significantly lowered by 2–4 mm Hg during potassium supplementation. Change in diastolic pressure correlated negatively with change in 24 hour urinary potassium and positively with change in 24 hour urinary sodium/potassium ratio in individual subjects (Khan and Thom 1982).

Kincaid et al (1971) and Mickelsen et al (1977) have proved that in the group of subjects using sodium chloride + potassium chloride in the ratio of 1:1, the total intake of sodium was only 40–55% of that of the subjects using regular
table salt. Thus sodium restriction is found to be achieved more easily when potassium chloride is mixed with common salt in desirable proportions. Furthermore, the disagreeable flavour of potassium chloride can also be masked by this technique.

Contrary to this, Richards (1984) found no hypotensive effect of potassium supplementation probably due to the short period of intervention as the hypotensive effect of sodium restriction takes at least one year. The enormous amount of chloride may also have some impact on the results. As both potassium and phosphorus (energy) decrease with age in the intracellular space any supplementation of potassium should probably be a phosphate.

Calcium:

Recently blood pressure has been claimed to be related to calcium intake (Kesteloot & Geboers, 1982). Calcium has effects on blood pressure at several physiological levels. Intracellular calcium mediates vascular smooth muscle contractions, external calcium has other actions on the cell membrane. The cell membrane binding to calcium is reduced in clinical and some forms of experimental hypertension. Calcium is initially important in stabilising the cell membrane i.e. reducing permeability to sodium and potassium.
maintaining membrane polarity.

Higher blood pressure was associated with a lower intake of calcium (McCarrion et al 1981). This relation was independent of other important confounding factors such as body mass, race and sex. McCarrion et al found the higher intake of sodium and potassium also to be associated with a lower systolic hypertension.

The inverse relationship between calcium intake and blood pressure has been supported by Belizan et al (1983). The calcium supplemented (1 g per day) young adult group showed a significant decrease in diastolic pressure, this effect being stabilized after nine weeks in women and six weeks in men. The mean reduction in diastolic pressure was 5.6% and 9% for women and men respectively.

On the subjects with usual calcium intakes (either low or high) sodium supplementation was found to have no effect on blood pressure but the mean systolic and diastolic pressures in the high calcium group were lower (Jacqueline et al 1985).

The U.S. National Health and Nutrition Examination Survey (1976) reported a direct relation between serum calcium and blood pressure with a tendency for patients with high blood pressure to have high serum calcium. Similar trends have been
The effect of magnesium on blood pressure may be direct or through an influence on the internal balance of potassium, sodium and calcium. Apart from being a necessary activator of Na-K-ATPase, magnesium is also a calcium antagonist. A decrease in magnesium leads to increased cellular calcium concentrations and vasoconstriction. Magnesium may also have a direct effect on vascular smooth muscle. Dyekner and waster (1983) reported a significant decrease in both systolic and diastolic pressure with magnesium supplementation. These results indicate that treatment with magnesium should be considered in arterial hypertension at least as an additional treatment in patients already receiving diuretics.

Dietary fat and Hypertension:

Although there are very few studies on direct etiologic relationship, the observations, that obesity is an important determinant of the precursors of high blood pressure which in turn is a major factor in the development of coronary heart diseases, have led the WHO Expert Committee to consider weight reduction and intake of polyunsaturated fats to have favourable effects in inhibiting thrombus formation.
Pekka et al (1983) found the composition of dietary fat to influence blood pressure. Low fat diet group (23% of fat (calories) brought a fall in the diastolic pressure from 88.9 to 81.3 mm Hg and in the systolic pressure from 138.4 to 129.5 mm Hg. This fall was greater among hypertensive than among normotensive subjects.

Several studies (Ophir et al 1983, White 1958, Prior 1968, Seile 1930, Sacks et al 1964 & 1981, Armstrong et al 1977) have reported a lower average blood pressure in vegetarians as compared to the nonvegetarians living in the same urban environment. While some presume that the low blood pressure may be due to a low sodium intake by the vegetarians, some studies have suggested an adverse effect of beef, meat, eggs etc. on plasma lipids and blood pressure (Sacks et al 1981, McGill 1979).

The indirect relationship between cardiovascular diseases and saturated fat by way of increasing the blood cholesterol, triglycerides etc. have been established by several studies. Keys et al (1957) found that gram for gram, saturated fats were about twice as effective in raising the blood cholesterol as polyunsaturated fats were in lowering it. A positive correlation between the iodine value of dietary fats and their cholesterol lowering properties has been reported (Ahrens et al 1959). The consumption of diets low
in fat calories or high in vegetable oils (higher proportion of unsaturated fatty acids) has been found to keep the serum triglyceride levels low and vice-versa (Antonis and Bersohn 1961). Keys et al (1965) concluded that the saturated fatty acids with more than 12 but less than 16 carbon atoms raise plasma cholesterol concentration and that the polyunsaturated fatty acids have an opposite but weaker effect than that of saturated fat and that 2 g of polyunsaturated fats were required to counteract the effect of 1 g of saturated fat. Hegsted et al (1965) showed a moderate reduction of 8-10% in serum cholesterol levels by reasonable changes in dietary saturated fatty acids.

The mechanism by which polyunsaturated fats lower blood cholesterol remains controversial. Wood et al (1966) and Moore et al (1968) have postulated an increase in excretion, while Spritz and Mishkel (1969) believed that the greater volume occupied by unsaturated fatty acids reduces the number of cholesterol molecules that can be accommodated in the lipoprotein.

Stamler (1976) established the epidemiologic associations between diet and heart diseases from his studies on the decrease in mortality due to coronary heart diseases in European countries after World War II. He speculated that the reduction in mortality due to coronary heart diseases
was associated with a sharply reduced intake of total Calories, total fat Calories (primarily saturated) and dietary cholesterol.

Glueck (1979) found that the level of plasma cholesterol is determined only partially by the intake of dietary cholesterol, saturated and polyunsaturated fat and total Calories. Genetic factors play appreciable role to plasma lipid. It is a biological truism that dietary and genetic factors interact along with other factors, in the determination of plasma lipid and lipoprotein levels. The development, progression and regression of atherosclerosis are closely related to the resultant plasma cholesterol level.

Robertson et al (1979) found that two hundred grams of raw carrot eaten at breakfast each day for 3 weeks significantly reduced serum cholesterol by 11%, increased fecal bile acid and fat excretion by 50%, and modestly increased stool weight by 25%. This suggests an associated change in bacterial flora or metabolism. The changes in serum cholesterol, fecal bile acids and fat persisted 3 weeks after stopping treatment.

Wittum and Schonfeld (1979) reported that in populations having habitual diets relatively rich in carbohydrates and low in fat (and usually low in cholesterol and saturated fats), there are not only lower Low Density Lipoprotein (LDL)
cholesterol levels than in "westernized urbanized" populations but also considerably lower High Density Cholesterol Lipoprotein (HDL) cholesterol levels.

The most marked reduction was observed in the high density lipoprotein-cholesterol fraction. It is concluded that these results could have therapeutic consequences for the dietary management of hyperlipidemia. However, the lowering of high density lipoprotein-cholesterol could also be interpreted as unfavourable since an inverse relationship between high density lipoprotein cholesterol levels and the occurrence of coronary heart disease has been established.

Researchers from the Royal Free Hospital Medical School in London have found that smoking, obesity, blood pressure and presence of non-HDL cholesterol to be more important in determining the risk of developing heart diseases (Times of India 1986).

Body weight and Hypertension:

In a large number of cross-sectional observational studies, it appears that blood pressure and relative weight levels are highly correlated not only in adults but also at younger ages (Chiang et al 1969 and Kotchen & Havlik 1980). In prospective observational studies, it is found that persons who gain weight show a greater rise in blood pressure.
over time than those who maintain their weight, while pressure falls with weight loss (Miall et al 1968, Kannel et al 1967, Paffenberger et al 1968). Among such studies, the most persuasive data come from Evans County, Georgia, USA, indicating that those who are obese from the start and gain further weight are at 6 times higher risk of becoming hypertensive than thin people who stay thin (Tyroler et al 1975). In several experimental population studies, it has likewise been observed that weight loss is accompanied by blood-pressure reduction (Heyden et al 1973, Stamler et al 1980). While, in some of these trials, dietary factors other than restriction of energy intake might also have been involved, there is at least one investigation in which it was shown that hypertension was reduced if only energy intake, but not salt, was restricted (Reisin et al 1978). The relationship between weight and blood pressure has been explained by the suggestion that obese people eat more salt (Dahl 1972).

In evaluating cross-sectional data, the need to distinguish between overweight and obesity has to be kept in mind. In the studies reviewed by the Scientific Group, the weight indices used largely reflect body fat, though some contribution by body mass cannot always be excluded. It is also recognized that the application of insufficiently large sphygmomanometer cuffs to obese arms may lead to an overestimate of blood
pressure readings, thus causing fictitiously high correlations between body weight and blood pressure; nevertheless, it is unlikely that this correlation results entirely from such artefacts because the problem has been recognized for some 20 years in epidemiological studies. Furthermore, weight reduction has been shown to cause lowering of arterial pressure when the latter has been measured directly, using an intra-arterial catheter. However, in less-well-controlled situations and in clinical practice there remains a need to pay attention to the difficulties of making accurate blood pressure measurements.

A paramount need is to determine how much weight reduction is needed at various ages to lower blood pressure by a given amount and, even more important, to what degree avoidance of weight gain will prevent an undue rise in blood pressure with age. Furthermore, the mechanisms responsible for the association between obesity and blood pressure require more through investigation in order to provide plausible evidence for a causal relationship and more effective means of intervention.

On the basis of present knowledge, it should be expected that avoidance of weight gain with age in adulthood and of undue weight gain in youth may prevent, diminish or postpone the rise of blood pressure with age in many populations, especially in hypertension-prone individuals.
People who are more than 20% overweight have three times the risk of heart disease than those whose weight is normal or near normal. High blood pressure is twice as common in overweight people.

Among those aged 40 to 65 years, the overweight group had a 50% higher hypertension prevalence rate than the normal weight group and 100% higher than the underweight group. With each higher degree of blood pressure elevation, relative frequency of hypertension with overweight was larger. Since both hypertension and overweight are mass phenomena in the United States, even a partial achievement might favourably affect millions of persons (Stamler et al 1978).

Ramsay et al (1978) found that successful weight loss was associated with a highly significant and substantial improvement in blood pressure control and with less frequent increase in anti-hypertensive treatment. The highly significant correlations between weight loss and fall in blood pressure could not be ascribed to observer bias to encouragement by a dietitian.

The investigation of Paderberg et al (1984) on the separate and combined efforts of dietary energy and sodium restriction on regulation of blood pressure in 30 middle aged obese men with essential hypertension showed that in hypertensive obese men a moderate weight reducing diet decreases indices
of sympathetic nervous system activity. Reduction of blood pressure to the normotensive range was observed only when there was a concomitant restriction of sodium intake.

Langford (1985), showed that patients with mild hypertension who have successfully controlled blood pressure medication for several years can discontinue antihypertensive drug therapy and remain normotensive for at least one year if they are willing to lose weight or restrict intake of sodium. On the other hand, patients with severe hypertension (defined by initial blood pressure or number of medications required for control) are likely to experience relapse after discontinuing medication.

MacMahon et al (1985) found that the weight reduction produced significant and clinically important reduction in blood pressure but not the adverse effects on plasma lipids commonly associated with antihypertensive drug therapy.

Weight control could thus result in a decreased prevalence of hypertension, and also in lower blood pressures in the nonhypertensive range. It can be calculated, on the basis of reasonable assumptions, that shifting the weight distribution in the population to the left could reduce the prevalence of hypertension by as much as 25% (Epstein 1979). This theoretical estimate gives an indication of the magnitude of the effect that might be expected.
The relationship between physical activity and blood pressure has been little studied in large, well controlled population samples. In the Framingham Study no significant relationship was detected (Dawber et al 1967). In studies in Gothenburg relationships were not found between either leisure time or occupational activity and blood pressure and also there was no association between activity level and change of pressure with time (Svärdsudd et al 1980, Wilhelmsen 1976, Berglund & Wilhelmsen 1975, Svärdsudd & Wilhelmsen 1980).

A more sensitive method of detecting a possible effect of physical activity on blood pressure would be to perform intervention studies. All the studies undertaken so far have been small and most of them have not used untrained, randomized control groups. In general, physical activity appears to have little or no effect on blood pressure in normotensive subjects but in the hypertensive groups some patients have shown a decrease in at least diastolic pressure. Effects on body weight and other variables have not been sufficiently reported (Ekblom et al 1968, Hansen et al 1968, Boyer & Kasch 1970).

Boyer and Kasch (1970) determined the effect of exercise programme (walk joy type) on blood pressure (taken while the men were resting). There was no other change in therapeutic
management during the exercise training period. A drop in mean diastolic pressure of 11.8 mm Hg and in mean systolic pressure of 13.5 mm Hg occurred in the hypertensive group. There was a mean decrease of 6 mm Hg in the diastolic pressure of the normotensive group but no significant change in the mean systolic pressure.

Pickering et al (1982) found that the blood pressure was the highest at work and the lowest during sleep. The situational blood pressure changes were generally similar but hypertensive group showed consistently higher pressure in the physician's office than at home, whereas normal subjects showed little difference. During exercise, the hypertensive groups showed a similar rise of systolic pressure to that of normal subjects. Pressures recorded in physician’s office gave good predictions of the average 24 hour pressure in the normal and established hypertensive subjects, but not in the borderline group; in such patients, 24 hour monitoring may be of particular value in establishing the need for treatment.

Peters et al (1983) concluded that poor physical fitness may be an important risk factor for heart disease especially when conventional risk factors are also present.

Choquette & Ferguson (1973) reported significant reductions in both systolic and diastolic blood pressure from a 6 months' training programme. Body weight remained unchanged.
The differences in resting systolic and diastolic pressures were considerably more pronounced in hypertensives (16/8 mm Hg, 2.1/1.1 kPa) than in normotensives (4/2 mm Hg, 0.5/0.3 kPa), but it is not possible to determine how much of the change was due to the phenomenon of regression to the mean.

Krotkiewski et al (1979) found that blood pressure decreased after training in 27 obese women and was not related to decrease in body fat.

A controlled clinical trial of physical training of post-infarction patients showed that pressures decreased significantly at rest in the training group, that the rise seen from 3 months to 1 year in the non-trained patients during submaximal exercise was not seen in the training group, and that there was no difference in body weight (Wilhelmsen et al 1975).

Both in the study of post-infarction patients and in that of obese women it was found that insulin levels decreased in those who trained effectively (Krotkiewski et al 1979, Björntorp et al 1972), and in the latter there was a correlation between the decrease in plasma insulin on training and the decrease in blood pressure. It has been suggested by Horton (1981) that this change in plasma insulin level may result in altered renal handling of sodium, which could explain at least part of the decrease in blood pressure. An additional blood pressure lowering effect of training might be decreased plasma concentrations of noradrenaline and adrenaline, probably with a
decreased peripheral resistance. (Trap Jansen et al 1973).

A metabolic disturbance with obesity, impaired glucose tolerance and higher fasting insulin levels compared with normals is seen in some hypertensive patients (Berglund et al 1976) and may be one factor that could be influenced by physical training in efforts to prevent increased blood pressure.

Blair et al (1984) used multiple logistic risk analysis to estimate the independent contribution of physical fitness to risk of becoming hypertensive. After adjustment for sex, age, follow-up, interval, baseline blood pressure and baseline body-mass index, persons with low levels of physical fitness (72% of the group) had a relative risk of 1.52 for the development of hypertension when compared with highly fit persons. Risk of developing hypertension also increased substantially with increased baseline blood pressure.

A regular moderate daily exercise (a brisk walk for 30 min/day) along with a reduction in caloric intake is indicated in the treatment of obese patients with uncomplicated hypertension.

Alcohol and Hypertension:

Many cross-sectional studies in different populations have shown a positive relationship between blood pressure and reported alcohol consumption or various indices of it.
(Clark et al 1967, Dyer et al 1977). Studies of the dose-response relationship have given conflicting results; it appears that alcohol consumption raises systolic pressure more than diastolic. Adjustments for body weight had little effect on the associations.

Wallace et al (1981) have summarised studies on the acute effects of alcohol in humans, which may be a relative decrease, a relative increase or no change in blood pressure. Alcohol cannot be given in a double blind fashion in humans, which makes the pharmacological effect difficult to evaluate.

Henningsen et al (1980) reported a decrease in blood pressure following alcohol withdrawal which indicated that reduction or cessation of anti-hypertensive drug treatment might be possible in alcoholic hypertensive patients if drinking stops. Saunders et al (1981) studied 132 alcoholic patients whose daily consumption exceeded 80 g. They found a significant correlation between blood pressure and mean daily alcohol intake, and also between the level of blood pressure and the severity of alcohol withdrawal symptoms. In most patients blood pressure fell to normal after abstinence, and remained so for at least a year in those who continued to abstain, but rose in those who started to drink again.
Several mechanisms may be involved: increased blood cortisol levels, increased catecholamine levels, and effects on the renin-angiotensin system or on antidiuretic hormone. Alcohol withdrawal induces excess adrenergic discharge, which increases blood pressure transiently. Many studies of blood pressure have required some fasting and non-drinking prior to the examination so that many moderate to heavy drinkers may have been in a withdrawal state when examined.

The finding that blocked pressure returns to normal with abstinence suggests that alcohol-induced elevations are not fixed, and do not necessarily lead to a continuing rise of blood pressure over a long term period. Longitudinal studies of factors associated with blood pressure increases over a well-defined follow-up period, suggest that alcohol is not a major cause of the increases (Svärdsudd 1980). However, this finding cannot be taken as an indication of fewer long-term sequelae in patients with alcohol-induced hypertension, since the age-specific death rate is high in heavy drinkers, and a direct relationship with the higher blood pressure in alcoholics has been reported in other studies (Dawber et al 1967).

Potter and Beavers (1984) demonstrated a pressor effect of alcohol in patients with hypertension and confirmed the link between alcohol and blood pressure. The mechanism of alcohol
induced hypertension is still uncertain and is more likely to be due to an effect of alcohol rather than to the pressure response produced by alcohol withdrawal.

Psychological and Social Influences:

Included under this heading are external and environmental influences that are understood or presumed to operate through mental processes, whether consciously or unconsciously, to produce physiological effects that may include blood-pressure elevation. Examples are the processes of migration, rapid modernization, and the experience of major life events. Such influences have been the subject of much research and continue to be entertained as possibly having an important bearing on the development of sustained elevation of blood pressure. Their relevance to primary prevention is reviewed briefly in the light of the scientific evidence available.

Studies of populations under circumstances of environmental change have been undertaken in several areas of the world, for changes involving either migration to a new environment or rapid transition of local conditions for a stationary population. In such circumstances, many conditions may change within a given period, this requires careful assessment of all factors, such as dietary change, that may be plausible related to any blood pressure changes that might be observed. Not the least difficulty lies in measurement
of the presumed psychological influences themselves, whether this be through independent documentation of the board social processes in themselves or through structured interview or questionnaire assessment of individual subjects. Thus the effects of confounding variables may be assessed and assurance given that conditions likely to constitute psychological influences are in fact present and perhaps impinging in different degree on subgroups of individuals. Many of the studies in this area have been reviewed by Cassel (1975) with the conclusion that the degree of adaptation of one’s niche in society remains an important factor, alongside diet in general and salt intake in particular, in explaining differences in patterns of blood pressure among populations.

Acute emotional experiences certainly elevate blood pressure but the evidence that sustained, chronic stress, however, defined, contributes to hypertension in man is controversial (Zanchetti & Bartorelli 1977). In mice, however, the psychosocial stress of crowding causes sustained hypertension (Henry, 1977). Personality traits that may be conducive to susceptibility to psychosocial stress have been described in hypertensives, but the matter is unclear (Stephens 1981). Although there is important potential for behavioural interventions to lower blood pressure, the degree and duration of their effect have so far been small (Shapiro et al 1978).
Evidence for the part played by psychological stimuli is derived from the finding that chronically stressed animals may become hypertensive. It has also been suggested that along with a hereditary make-up, intermittent bombardment of cortico-hypothalamic discharge provoke sympathetic vaso-constriction and the out-pouring of chemical agents such as catecholamines, cortisol, A.D.H. etc. which lead to medical hypertrophy of the resistance vessels (Fig. 3).

This body of evidence, taken as whole, stimulates continuing interest in psychological influences on blood pressure, leading both to recommendations for further research and to some comments on implications for primary prevention.

Several considerations bear on recommendations for further research in this area. For population studies there is a need to employ prospective designs whenever possible to help distinguish between the psychological influences that are antecedents of blood pressure elevation and those that are possible maintaining factors or even the consequences of high blood pressure. It is desirable to choose situations with naturally-occurring population changes, wherein psychological influences may be studied independently of geographical relocation; a secondary choice would be situations in which subgroups experience different degree of concomitant change in physical environment, diet, and other influences, so that
Fig. 3. Stress and Hypertension

Vasomotor Center → Stress → Hypothalamus

ADH

Aldosterone

Renin

Norepinephrine

Hydrocortisone

Sodium and H₂O Retention

Vasoconstrictor Substances

Medulla
psychological effects may be isolated analytically.

For clinical and laboratory research, emphasis should be concentrated on hypertension-prone normotensive subjects rather than on established hypertensives (in whom causative and maintaining factors are inextricably interwoven). Prospective studies of children of hypertensive parents can include investigation of personality traits, living conditions, cardiovascular reflexes, cardiovascular responsiveness, plasma catecholamines, etc. and should compare the measurements in offspring who develop and those who do not develop hypertension over the years. Help might also come from parallel measurement of some of the so-called membrane markers of essential hypertension, but it should be kept in mind that the meaningfulness of these markers is not yet fully elucidated. Finally, behavioural interventions require special training and substantial contact time; their wide-spread use cannot be recommended before their feasibility and efficacy have been reliably demonstrated.

nevertheless, given the pathophysiology of the early hypertension, it is eminently sensible to investigate whether reduction of stress, modification of individual responses to stress, or measures to decrease central sympathetic out-flow voluntarily may be useful in the primary prevention of hypertension.

One conclusion to be drawn from the foregoing review is that it is quite possible that modification of psychological
influences may be important as an approach to the primary prevention of essential hypertension. This approach is suitable for application now in carefully designed studies, although the further research suggested above may in time raise or lower its priority. Psychological influences may also require attention in four other respects.

1. Measurement of blood pressure and consequent decisions may be influenced by transient psychological factors; these should be controlled as far as possible in the measurement procedures employed.

2. Intervention to alter blood pressure levels may generate psychological influences as a side-effect; these can be avoided or diminished to a large extent, but in any case they warrant appropriate concern and should be monitored to the extent that circumstances permit.

3. If psychological influences, for some persons or groups, do in fact have a strong influence on blood pressure levels, then overlooking this fact may lead to under-estimation of the efficacy of other approaches, such as dietary change, to primary prevention.

4. Compliance by participants in any form of intervention programme depends upon development of a favourable psychological approach; without this an otherwise beneficial strategy may appear to be ineffective. This attention to psychological factors in compliance with intervention programmes is an essential component (WHO 1983).

Social Influences:

Hypertension has been shown to be more prevalent in
persons of lower social status, and in those with lower income and less education in many though not all studies. There are also difference according to whether residence is in an urban or a rural area.

It is not known how far these relationships are accounted for by differences in frequency of obesity, and in alcohol, sodium and potassium intake or whether they may be partly due to psychosocial stress of various kinds or other habits in low-income groups. There is a great need to find the determinants of these relationships.

It is also not known how social class and blood pressure are related in developing countries. Studies in these populations are required, especially at this time when marked social and cultural changes are in progress.

In the long term, knowledge resulting from such studies could, in theory at least, permit protection of populations undergoing such changes from the development of hypertension. From a practical point of view, the need is to improve social conditions and, in the interim, make special efforts to reach and motivate people in unfavourable environments to take better care of their health.

Other Influences:

There has also been a belief for many years that certain
kinds of animal protein raise blood pressure but these claims have never been substantiated. In contrast, inverse inter-community and intra-community correlations between dietary animal-protein intake (estimated by urinary-sulfate/urea-nitrogen ratios) and blood-pressure levels have been demonstrated in communities where the incidence of hypertensive complications is high. (Yamori et al 1979). Recently, polyunsaturated fatty acids have been reported to lower blood pressure, presumably mediated by prostaglandin derivatives (Iacono et al 1975); however, the effect is not marked. In another study, the blood pressure of young, normotensive persons was not influenced by the types of fat, protein, or dietary fibre.

Several other environmental factors have been implicated in affecting blood pressure levels but none of them, with the possible exception of noise and conceivably, some constituents of air and water, has a bearing on the primary prevention of hypertension.

In the physical environment, water and temperature have been related to blood pressure. An inverse relationship between cardiovascular disease and water hardness, if confirmed, could be mediated in part by blood pressure, but in the British Regional Heart Study, blood pressure and water quality were found to be unrelated (Shaper et al 1980). Cadmium in particular has been said to raise blood pressure but the evidence
is controversial (Sharrett, 1975). Blood pressure varies inversely with the temperature in the examination setting (Heller et al, 1978) but it is not likely that his effect has a major influence on geographical differences in blood pressure distributions.

Similarly, it is difficult to separate the effect of altitude from other factors in explaining population differences in the average blood-pressure level among people living at different heights above sea-level. One such study has been reported from New Mexico (Mortimer et al, 1977) but it has been criticised because it failed to account adequately for social differences.

Lately, it has been suggested that noise plays a role in contributing to elevated blood pressures in exposed population groups (Andriam, 1982). This possible factor should be given further consideration.

Approaches to Primary Prevention of Hypertension:

Although it is obviously better to prevent than to cure a disease, interest has focused on primary prevention of high blood pressure only in very recent years. Hypertension is of importance mainly as a risk factor for heart and brain disease and, to date, efforts have been made to reduce high blood pressure levels rather than to prevent their development. Therapy of high blood pressure, whether by drugs or in other ways,
is a primary preventive measure as regards cerebrovascular or coronary heart disease.

There are two approaches to the primary prevention of disease, and these are usually complementary, the high-risk approach or strategy and the mass approach or strategy.

The high-risk strategy may be especially appropriate in two situations, first, when there is still uncertainty as to whether the relationship between the risk factor and the corresponding disease is causal, and second if the risk factors occurs with very low prevalence in the population. By contrast, the mass strategy is directed at the whole population, irrespective of individual risk levels. In the early history of risk-factor intervention in cardiovascular diseases, as recently as two to three decades ago, the total population approach was deliberately given a lower priority, because it was not considered defensible to subject every person in the community to preventive measures for which many had only a small need and for which benefit had not been established. The relative merits of the high-risk and mass strategies have been discussed by Rose (1981) and a strong case for the mass strategy has been made in the report of a recent WHO Expert Committee (WHO Technical Report Series No. 678, 1982).

With respect to blood pressure, the concept of the mass strategy is based on the fact that the risk of vascular complications and death rises continuously with increasing blood
pressure, and a considerable number of events occur when blood pressure is moderately elevated. This conclusion is based upon several prospective epidemiological studies whose data can be viewed separately and collectively in the reports on the United States National Pooling Project and the Framingham Study (Pooling Project Research Group 1978, United States Department of Health, Education and Welfare 1976). In addition, the risk of cardiovascular complications is known to depend on a combination of several risk factors, including moderately elevated blood pressure, lipid abnormalities, smoking and others. Thus it appears appropriate at least theoretically, to try to reduce blood pressure, even in persons for whom antihypertensive drug treatment would not be recommended. It is not known whether blood pressure reduction in this group will also reduce the incidence of vascular events, but results from clinical drug trials using different regimens point towards such a possibility.

Detecting persons with elevated blood pressure in the population involves cost and effort, persuading them to change long-established unfavourable living habit is difficult; and compliance in taking medication is not easy to achieve. The mass strategy, if effective, would prevent blood pressure from rising to a point where treatment, with these and other difficulties, becomes necessary.

The aim of the primary prevention of hypertension may
thus be expressed in both of the following ways, which are regarded as complementary: a) in high-risk individuals, to prevent the attainment of levels of blood pressure at which the institution of management and treatment would be considered, b) in the general population, to delay or arrest further progression of blood pressure levels beyond those attained upon reaching adulthood.

In summary, the need for primary prevention is clear. For it to have an adequate impact on the population, both the high-risk and mass strategies must be implemented. Questions requiring immediate answers are: a) On the basis of existing knowledge, what can be done now?, b) What kinds of new study are required to provide a firmer scientific base for effective and efficient action?

Anti-hypertensive Agents:

Anti-hypertensive agents fall into four main categories:

1. Agents that interfere with the activity of the sympathetic system,
2. Vaso dilators that relax vascular smooth muscle,
3. Diuretics, and
4. Drugs that interfere with the renin – angiotensin system.

Drugs acting on the sympathetic nervous system:

These drugs affect the central sympathetic areas in the
brainstem where alpha-adrenoceptors are located. Some of them may deplete the central stores of catecholamines.

Methyldopa acts on alpha-adrenoceptors and lowers arterial pressure. Clonidine reduces the central sympathetic outflow to reduce arterial pressure. Reserpine also acts centrally, depleting hypothalamic and other central stores on noradrenaline, dopamine and serotonin. Ganglionic blocking drugs are competitive antagonists to acetyl choline at the ganglionic synapses. They cause a marked, mainly orthostatic hypotension.

Ghanethidine, betanidine and debrisoquine interfere with the storage and release of noradrenaline at adrenergic nerve endings. These drugs are generally reserved for severely hypertensive patients where condition is not readily controlled by other means.

Beta adrenoceptor blocking agents: Numerous products are available differing in their relative cardio-selectivity, intrinsic sympathomimetic (agonist) activity, metabolism and duration of action. Despite these variations all appear to have a similar anti-hypertensive effect. These agents should not (unless accompanied by digitalis and/or diuretics) be used by patients with congestive heart failure, atrioventricular block, obstructive lung disease or peripheral arterial insufficiency.
Alpha-adrenoceptor blocking agents such as phentolamine, phenoxy-benzamine and the hydrogenated ergot-alkaloids have no useful hypotensive effect in phaeochromocytoma.

Vasodilators: Hydralazine and dihydralazine both produce a fall in pressure and a reflex increase in heart rate through a peripheral action on vascular smooth muscle. Dissanoxide, chemically related to the thiazide diuretics but causing sodium retention, produces marked peripheral vasodilation.

Minoxidyl is one of the most effective drugs available.

Nitroprusside is used only in hypertensive emergencies.

Diuretics: The mechanism by which diuretic drugs lower the blood pressure is complex. Given intravenously, they may have a prompt and sometimes powerful pressure reducing effect. Given orally thiazides have a mild anti-hypertensive effect that is associated initially with reduced plasma volume and cardiac output. With prolonged treatment there is a reduction in total peripheral resistance, associated with a maintained contraction of plasma and extra-cellular fluid volume. It seems likely that part of the mode of action results from a mechanism comparable to that achieved by low dietary sodium intake.

Thiazides: Drugs of this group produce a slight but distinct lowering of arterial pressure and for many years they have been the basis therapy for mild hypertension, because
they enhance the excretion not only of sodium but also of potassium.

Potassium conserving diuretics: Drugs in this group which include spironolactone, amiloride and triamterene have a weak natriuretic effect and produce a small reduction of blood pressure.

Loop diuretics: These powerful diuretics (Furosemide, Etacrynic acid and Bumetanide) produce a rapid natriuresis and an immediate vasodilation when given intravenously.

Drugs selectively interfering with the renin-angiotensin system: In case of renin-dependent hypertension, pressure may be lowered by administering antagonists or inhibitors of the action of the renin-angiotensin system. Two main types of agent are being investigated—(1) Synthetic analogues and competitive antagonists of angiotensin II and (2) Inhibitors of the enzyme responsible for converting the inactive angiotensin I into the active peptide angiotensin II. Enzyme inhibitors may also cause an accumulation of circulating bradykinin, which may contribute to their antihypertensive effect. (WHO Technical Report Series 1978).

Drug Treatment:

A wide variety of drugs are available for treatment of hypertension. Diuretics as a sole therapy can control blood pressure in more than 40% of patients. If a diuretic alone proves inadequate a beta-adrenergic blocking agent, vasodilator and central acting sympathetic inhibitor may be added,
as needed, in the familiar three step program (Table 1). A Calcium channel blocker, with its vasodilating effects of an angiotensin converting enzyme inhibitor may be considered in appropriate situations. Selection among these agents should be based on knowledge of their pharmacologic action, duration of action and side effects. With careful selection, blood pressure can be better controlled, side effects minimized and patient compliance improved (Orme and Hegstrom, 1984).

Simple drug regimes (beta-adrenergic blockade and a diuretic plus a third agent when required) are often effective in the treatment of even the most severe hypertension (Marshall & Berritt 1984).

Side effects of drug treatment:

The goal of treatment is not only normalization of blood pressure but more importantly prevention of cardiovascular disease. The hypertension detection and follow-up program unequivocally demonstrated the risk of mortality associated with pre-existing cardiovascular end-organ damage, regardless of treatment. Since immediate risk of complications is minimal in mild hypertension drug therapy should be instituted only after nonpharmacologic measures have been tried. Weight control, moderate salt restriction and an individualised exercise programme may be effective management in many cases of mild hypertension without complications. This regimen avoids the
## TABLE I

### Drug Regimen for Hypertension
(Diastolic Pressure below 95 mm Hg)

<table>
<thead>
<tr>
<th>Hypertension — Severity</th>
<th>Drug Regimen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild to moderate (Diast. Pr. 95-110 mm Hg)</td>
<td>Step 1: Diuretic + Reserpine, propranolol, methyldopa, clonidine</td>
</tr>
<tr>
<td>Moderately severe (Diast. Pr. 111-125 mm Hg)</td>
<td>Step 2: Diuretic + Hydralazine, reserpine, propranolol, methyldopa, clonidine</td>
</tr>
<tr>
<td>Severe (Diast. Pr. &gt; 125 mm Hg)</td>
<td>Step 3: Diuretic + Guanethidine, propranolol, hydralazine, methyldopa, clonidine</td>
</tr>
</tbody>
</table>

*Step 2 is indicated when Step 1 fails and Step 3 when Step 2 fails.*
the potential hazards, inconvenience and expense of long-term drug use (Wilburn, 1984, An Interim Analysis WH0/1.S.H. Mild Hypertension Liaison Committee 1982).

Chemical trials have failed to show that treatment prevents complications. The adverse side-effects of antihypertensive drugs (Cranston et al 1963) may diminish the quality of life for patients. It is not yet clear to what extent the said effects of medication are subjectively more or less troublesome than the symptoms of untreated hypertension (McAlister 1983).

Although the drug treatment of mild hypertension has been proved to prevent strokes and congestive failure (UNI report, Times of India 1985, Veterans Administration Cooperative Study Group on Antihypertensive Agents, 1970), the numerous undesirable side effects like gout, diabetes, impotence, cold hands, dizziness, diabetic coma, dehydration and death have been a constant threat for the physicians in selecting the drugs.

Nonpharmacologic treatment like weight and/or salt reduction and exercise has been proposed as an alternative for drug therapy as the same is inexpensive and safe (Black, 1979).

Even with the wide range of drugs now available, the cost
of treatment is another important factor to be considered especially in the case of hypertension which is a lifetime commitment (Orme 1984). It gives a sad illustration of the fact that drug treatment too often converts a symptom free person into a handicapped symptomatic patient (McAlister 1983). Hyynes et al (1978) verified on 80% increase in absenteeism from work among newly diagnosed hypertensives.

Alternative remedy:

Under these circumstances it seems worthwhile to scan the literature available on indigenous medicines or herbs as a remedy for Indians have a long tradition of depending on 'nature cure'. Interestingly quite a number of these remedies suggested by the age old people but forgotten in the middle in the wake of allopathy, have been proved to be scientifically sound. For instance, fenugruk seeds as antianaemic (Subhulakshmi et al 1965) ginger as hyperglycemic and hypercholesteremic (Gujral et al 1978, Giri et al 1984) turmeric as carminative and inhibitor of gastric secretion (Chopra et al 1941).

_Tulsi (Ocimum Sanctum)_ is one such herbal plant claimed to be beneficial in curing hypertension by Sandesara (1979).

_Ocimum Sanctum:_

It is an annual erect, softly hairy herb, 30-60 cm high
much branched and purplish in colour sub-squadrangular sometimes woody below and clothed with soft spreading hairs.

It is found throughout India and is grown purposely by Hindus in houses and temples. It is interesting to note its various characteristics:

- **Tulsi**: matchless, the best plant
- **Suras**: with best juice
- **Gramya**: available in villages
- **Sulabha**: easily available
- **Bahu Manjari**: with plenty of flowers
- **Apeta-Rakshasi**: by its sight, demonical diseases or sins run away.
- **Deva-Dundubhi**: generating joy amongst the godly and learned.
- **Shulanghani**: pain killer.

It is slightly bitter and astrigent but aromatic and appetising and is used as a remedial drug (Sandesara 1979).

**Composition of the leaves**:

<table>
<thead>
<tr>
<th>Component</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Essential oil</td>
<td>0.7</td>
</tr>
<tr>
<td>Eugenol</td>
<td>71.3</td>
</tr>
<tr>
<td>Carvacrol</td>
<td>3.2</td>
</tr>
<tr>
<td>Methyl Eugenol</td>
<td>20.4</td>
</tr>
<tr>
<td>Cryophylline</td>
<td>1.7</td>
</tr>
</tbody>
</table>
Seeds : % Composition

<table>
<thead>
<tr>
<th>Component</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ocimum S. oil</td>
<td>17.8</td>
</tr>
<tr>
<td>Ocimum B. oil</td>
<td>16.8</td>
</tr>
<tr>
<td>Linoleic Acid</td>
<td>56.3</td>
</tr>
<tr>
<td>Palmitic Acid</td>
<td>3.3</td>
</tr>
<tr>
<td>Stearic Acid</td>
<td>6.4</td>
</tr>
<tr>
<td>Oleic Acid</td>
<td>15.4</td>
</tr>
<tr>
<td>Carbohydrates</td>
<td>23.0</td>
</tr>
<tr>
<td>Fibre</td>
<td>18.1</td>
</tr>
<tr>
<td>Moisture</td>
<td>6.9</td>
</tr>
<tr>
<td>Protein</td>
<td>16.4</td>
</tr>
</tbody>
</table>

Use of Ocimum Sanctum:

Jain (1968) observed that leaves and seeds of Ocimum Sanctum are used as traditional medicine. Oil obtained from leaves has bactericidal property and kills certain maggots.

The leaves of Ocimum Sanctum are found to be antiseptic in nature (Sharma 1978). Its uses in skin diseases have also been described by Dey (1980).

Kirtikar and Basu (1935) prescribed the juice of leaves of Ocimum Sanctum to be applied on the skin in ring worm and other cutaneous diseases. The leaves also offer effectual means of dislodging maggots.
Decoction of root of Tulsi has a property to destroy bacteria and insect poison. Tulsi yields essential oil containing eugenol, carvacrol, methyl eugenol and cryophyllene which is used as an expectorant, antiseptic and insect repellent (Dey 1980).

Sharma (1951) has mentioned that irrigating the wound with infusion of leaves of Ocimum Sanctum destroys the worms of wound and hastens the healing process. It has been stated that leaves of Ocimum Sanctum ground with water are applied on bad boils. Persons effected with bad skin diseases such as itches, ring worm, leprosy etc. should drink the juice of basil leaves and also apply the same by itself or preferably mixed with lime juice as a paste for radical cure (Nadkarni 1954).

According to Dastur (1977) the leaf juice of Ocimum sanctum is locally used for relief or earache.

Sandesara (1979) gives a long list of its therapeutic value (Appendix-I).

Chopra et al (1956) have listed Tulsi as one of the Indian medicinal plants which is useful in variety of ailments. The juice of leaves is a diaphoretic, antiper and stimulating expectorant used in catarrh and bronchitis, and is a remedy for earache. It is also useful in gastric disorders in children and in hepatic affections. The powder from dried
leaves is used as snuff in Ogaena. The seeds are used in disorders of genito-urinary system while the roots in the form of decoction is used in malarial fevers. This plant also finds its uses as a musquite repellent and in snake bites and scorpion stings.

Formulations with Zinc Oxide base ointment prepared from chloroform extracts of Ocimum Sanctum were found to have healing property on infected wounds comparable to that of Furacin Vet Ointment (SK & F) and better than that of M & B antiseptic cream (Thaker (1983)). The antibacterial action of Ocimum Sanctum on the pathogenic organisms Streptococcus, Staphylococcus, E.Coli, Pseudomonas Pyogenes and Corynebacterium has also been proved by Thaker (1983). He showed that the chloroform extract inhibited their growth to the extent of 75 to 99%.

On the basis of the available information on the threat of hypertension, the need for further research on the factors causing elevation of blood pressure, the need for working on simpler, cheaper and indigenous remedies and the tall claim of the folklore for the hypertensive effects of Ocimum Sanctum, the present investigation has been carried out as per the work plan mentioned in chapter III.