CHAPTER-II

DIET AND ITS RELATION TO HYPTERTENSION
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The importance of diet in the etiopathogenesis of hypertension has been repeatedly emphasized in the literature. Salt intake and one's weight are still considered to be the most important diet related factors affecting the onset, evolution and treatment of hypertension.

Nature of diet - Vegetarian vs Non-vegetarian Diet and Hypertension

In 1930, Saile first hinted that vegetarians have lower blood pressure than most meat eaters of the same age (Ophir et al, 1983). Epidemiological observations on primitive populations eating a vegetarian diet showed lower blood pressure compared to similar meat eating populations. Subsequent reports described lower blood pressures in acculturated vegetarians in Trappist Monks, American macrobiotic vegans, Seventh-day Adventists in the United States and Australia and vegetarians in Israel (Beilin et al, 1988).

The difference in blood pressure among the vegetarians and non-vegetarians has been attributed to various reasons based on the weight and difference in nature of dietary ingredients consumed like difference in sodium, potassium, fibre, fat and animal protein intake.
Difference in Body Weight:

Ophir et al (1983) in their study found very low prevalence (2%) of hypertension in a group of vegetarians whereas the control group had 26 per cent hypertensives. On comparison of groups with same relative weight, it had emerged that the blood pressure of vegetarians was still significantly lower. This shows that weight is not responsible for the lower prevalence of hypertension in vegetarians.

Sacks et al (1985) found low blood pressure in vegetarians and were concerned whether the low body weight of the vegetarians was responsible for it. Many of the converted vegetarians had reported that they had lost weight during the first few months on vegetarian diet. Weight adjusted blood pressure of the lacto and strict vegetarians were significantly lower. Thus the authors do not rule out the influence of body weight in vegetarians on blood pressure.

Difference in Sodium Intake

It was presumed that the low blood pressure was due to a low sodium intake by the vegetarians. Armstrong et al (1979) found no significant difference in urinary sodium excretion between the two groups and concluded that dietary
sodium does not explain the blood pressure differences between vegetarians and non-vegetarians. Studies by Margetts et al (1986) and Beilin et al (1988) also revealed similar results.

Difference in Potassium Intake:

Meneely and Batterbee (1976) believe that the high sodium, low potassium diet ingested in Western civilisation, combined with a genetic susceptibility is principal factor in the genesis and perpetuation of essential hypertension.

Armstrong et al (1979) found that urinary potassium excretion was high in vegetarians (62.9 mmol/day) than non-vegetarians (54.8 mmol/day) thus giving a lower mean sodium to potassium ratio. Ophir et al (1983) also reported higher urinary potassium and presumably potassium intake in vegetarians and opines that high potassium is the possible cause for low blood pressure in vegetarians. However, Margetts et al (1986) observed no consistent changes in urinary potassium levels.

Difference in Fat Intake:

Meat intake increases the dietary intake of proteins, saturated fatty acids and cholesterol at the expense of
carbohydrates. Sacks et al (1981) tested the effect of 250 g/day of lean beef on blood pressures of 21 strict vegetarians, for 4 weeks followed by two weeks period of iso-caloric strict vegetarian diet. By the end of 3rd week of meat eating period, systolic blood pressure increased significantly by 3 mm Hg but not diastolic blood pressure. Plasma cholesterol rose by 19 per cent and returned to base level during vegetarian diet. There was no change in body weight.

A reciprocal design was also conducted by Sacks and Kass (1988). Non-vegetarians were taught to eat a low-fat, lacto vegetarian diet and were kept on it for three months. The low-fat diet decreased the subjects intake of protein, saturated fatty acids and cholesterol and increased carbohydrate. Plasma cholesterol and urinary creatinine declined on the vegetarian diet confirming the decrease in dietary saturated fatty acids and cholesterol. Blood pressure in these subjects did not decrease during the study.

Changing from saturated to poly-unsaturated fatty acid and addition of fish oil to diet has lowered systolic and diastolic pressure in mild hypertension (Norris et al, 1986). The effect of polyunsaturated fat appears to be due to the vasodilatory action of prostaglandins that are
formed from linoleic acid which is the major constituent of polyunsaturated fatty acid (Berry and Hirsch, 1986).

Several studies have shown that an increase in polyunsaturated/saturated ratio (P/S ratio) would lower blood pressure. However the blood pressure reduction achieved by change to a vegetarian diet still could not be ascribed to change in dietary P/S ratio (Margetts, 1988).

Differences in Protein Intake:

As the blood pressure lowering effect could not be attributed to sodium and fat intake, the role of total protein and animal protein was studied by Sacks and Kass (1988). A double blind study of egg intake by vegetarian subjects was conducted. The eggs were mixed in custards, muffins cakes that had counterparts without egg that were indistinguishable in taste and appearance. At the end of three weeks, low density lipoprotein cholesterol and apolipoprotein B both increased significantly but there were no significant changes in blood pressure.

Next, a study was conducted to see if low total protein intake in vegetarians was responsible for their low blood pressure. High protein, say wheat patties and low protein rice patties, were given for 6 weeks to
strict vegetarians. Blood pressure did not change with the variation in dietary proteins from 63 to 119 grams daily. Blood pressure did not differ when given either casein (animal protein) or soy protein isolate (vegetable protein) which amounted to 82 g/day.

Thus, it seemed unlikely that the low protein intake of vegetarians or the high ratio of vegetable to animal protein was affecting blood pressure.

Differences in Fibre Intake

Vegetarians have a higher fibre intake than the general population. High plant fibre diet has been reported to decreases blood pressure in mildly hypertensives and normotensives (Wright et al, 1979, Anderson, 1983; Jenner et al, 1988) whereas Silman (1980) found no such correlation. It is however not clear as to how the vegetarian fibre complex could be related to blood pressure.

Margetts et al (1987) conducted a randomized controlled trial where they altered the usual fibre intakes of typical omnivores to that eaten by vegetarians. The subjects were given low fibre (15 g/day) or high fibre (50 g/day) biscuit. There was decrease in blood pressure but could not be attributed to the high fibre diet.
Margetts et al (1986) also found an increase in calcium and magnesium intake and a reduction in vitamin B₁₂ in vegetarian diet.

These findings offer no support for the view that a vegetarian diet affects blood pressure by virtue of its higher fiber content. In summary, vegetarians have lower blood pressure than do non-vegetarians. Perhaps combination of nutrients may be needed to produce enough of a blood pressure lowering effect and long-term studies will be needed.

Nature of Fat-Effect of Linoleic Acid and P/S Ratio on Hypertension

Epidemiological and clinical studies have implicated a relationship between dietary fat and blood pressure. Data from observational studies suggest that in populations consuming diets rich in sea food, the prevalence of hypertension is low compared with Western societies.

A study of blood pressure in two religious groups revealed that Benedictive monks had greater prevalence of hypertension (51%) than Trappists (12%) which was attributed to differences in polyunsaturated fat intake among these groups (Groen et al, 1962).
Oster et al (1979) found that higher concentration of linoleic acid in adipose tissue correlated with lower blood pressures levels in a survey of 650 German men. Whereas Berry and Hirsch (1986) showed that adipose linoleic acid was not associated with blood pressure but an absolute 1 per cent increase in linoleic acid was associated with a decrease of 5 mm Hg in the systolic, diastolic and composite mean arterial blood pressure.

Iacono et al (1983) described a pilot epidemiologic study in three countries and showed that of the three, Finns had the highest mean blood pressure levels which was associated with relatively low intake of polyunsaturated fatty acid. Total fat intake did not relate to blood pressure level in these three groups.

A low prevalence of hypertension was reported in a Japanese fishing village where total dietary fat was low and intake of n-3 polyunsaturated fatty acid high. A lower prevalence of hypertension was also found in Crete and in Yugoslavia Cohort where intake of total fat was high and monounsaturated fat and polyunsaturated fat of the n-6 series were dominant sources of unsaturated fat (Bonaas, 1989).
Role of Polyunsaturated Fatty Acid

Dietary polyunsaturated fatty acid intake as a source of linoleic acid may be important in the production of prostaglandins which are known to modify blood pressure levels. Available evidence indicates that an increased intake of polyunsaturated fat relative to saturated fat (P/S ratio) may have a hypotensive effect in individuals with borderline hypertension (Weinsier and Norris, 1985).

Production of prostaglandins like PGE\textsubscript{1}, PGD\textsubscript{2} and PGX in the kidney and in vessel walls operate to increase sodium excretion and induce peripheral arteriolar dilation and chronic exposure to an excess of sodium stimulates prostaglandins synthesis (Heinemann and Lee, 1976). But prostaglandins PGI\textsubscript{2}, TXA\textsubscript{2} and PHE\textsubscript{2} stimulate platelet adhesion and aggregation (Vangsness, 1977).

Since prostaglandins are synthesized from arachidonic acid and the major precursor of arachidonic acid is dietary linoleic acid, it has been postulated that increasing dietary intake of linoleic acid may modify sodium induced hypertension (Weinsier and Norris, 1985).

Polyunsaturated fatty acid were found to be
hypocholesterolemic. Replacement of saturated by polyunsaturated fatty acids in the diet may lower serum very low density and low density lipoprotein concentrations because the liver preferentially converts polyunsaturated fatty acid into ketone bodies instead of into very low density lipoprotein and triglycerides. Thus, unlike saturated fatty acids, polyunsaturated fatty acids are transported to the tissues for oxidation without leaving a trail of lipoprotein remnants in the form of low density lipoproteins (Beynen and Katan, 1985).

Systolic blood pressure in patients with mild essential hypertension was significantly reduced after supplementation of the normal Western diet which is rich in n-6 fatty acids, with fish oil containing predominantly n-3 polyunsaturated fatty acid (Norris et al., 1986).

The mechanisms of the antihypertensive active action of fish oil is unclear. Hypotensive effects of fish oil in normotensive subjects are not associated with any change in plasma renin concentration or sodium balance and may be due to an effect on platelet prostaglandin metabolism. Eicosapentaenoic acid derivative of marine fish oil, competes with arachidonic acid as a substrate for platelet cyclo-oxygenase shifting the balance from thromboxane
A₂ to thromboxane A₃, which causes less vascular contraction and platelet aggregation and increases endothelial production of prostaglandins I₃, a more potent vasodilator than prostaglandin I₂ (Dyerberg et al, 1978).

Studies Modifying Polyunsaturated and Saturated Fatty acid Content of Diet

In a pilot study of 8 mildly hypertensive men, Comberg et al (1978) noted an average reduction of 10 mm Hg in diastolic BP after 3 weeks on linoleic acid enriched diet (2 to 4% of total calories). There was a strong correlation between dietary linoleic acid intake and changes in diastolic blood pressure. After subjects resumed their normal diet, blood pressure returned to baseline level. There appeared to be no significant change in sodium intake or body weight during the study.

Rao et al (1980) compared the effects of supplements containing 15-20 cc of refined groundnut oil, refined safflower oil or starch (as a placebo). After 6 weeks of treatment, there was no significant change in systolic blood pressure. Diastolic pressure decreased by 9 per cent with safflower oil and 6 per cent with groundnut oil but was unchanged in the placebo group.
Iacono (1983) compared the blood pressure response of men to diets containing either 43 or 25 per cent fat and a P/S ratio of 0.3 or 1.0. It was found that the blood pressure was lower for subjects on the diet with a P/S ratio of 1.0 when compared to those with a P/S ratio of 0.3. There was no evident effect of total fat intake on blood pressure levels.

Studies Modifying Total Fat and P/S Ratio:

Iacono et al (1983) found that 10 hypertensive individuals had a lower systolic blood pressure on a low fat intake with a high P/S ratio than on a diet high in fat with a low P/S ratio.

Puska et al (1983) demonstrated that high polyunsaturated fatty acid in diet is more effective in reducing blood pressure than dietary sodium restriction. On giving a diet low in fat (23% of energy) with a high P/S ratio (1.0) a reduction of 9 mm Hg in both systolic and diastolic pressures was observed and the reduction was greater in hypertensives. However, there was very little change on reduction of sodium intake from 192 mmol to 77 mmol. On switching back to normal diet, an increase in blood pressure was observed.
Margetts et al (1988) increased the P:S ratio (1.0 and 0.3 in a double blind fashion) while maintaining the rest of the diet, including total fat intake in 54 healthy normotensive subjects aged 20-59 years. The results showed that the change in P/S ratio affected blood pressure when total fat intake was reduced at the same time.

Iacono et al (1975) studied normotensive men and hypertensive men and found to have essentially no differences in blood pressure levels on diets containing 25 per cent and 43 per cent of total calories as fat with the same P/S ratio. Dodson et al. (1985) found a significant reduction in blood pressure after 3 months on a low fat, high fibre and low sodium dietary regime and reported similar results even after 3½ years.

Mensink et al (1988) did not find an effect on blood pressure of a high fat, olive oil rich diet relative to a low-fat, carbohydrate rich diet with the same level of saturated and polyunsaturated fatty acid. This suggests that total fat is not a determinant of blood pressure in normotensives and that the observed effects of fat modified diets on blood pressure are attributable to an increase in linoleic acid intake.
Summerizing these trials, it appears that increasing the P/S ratio of the diet is associated with a reduction of blood pressure among mildly and overtly hypertensive patients, a combination of a reduction of total fat and an increase in P/S ratio results in blood pressure lowering among hypertensives and a reduction of total fat alone has no effect in either normals or borderline hypertensives.

Salonen et al (1988) observed low plasma ascorbic acid and serum selenium concentrations which reflect low vitamin C and selenium intake with elevated blood pressure, which are new findings that need to be confirmed. Antioxidants could theoretically elevate the activity of vasodilating prostacyclin in both systemic and renal circulation through scavenging peroxides, which inhibit prostacyclin synthetase. Prostacyclin is an intermediate in the metabolic pathway of arachidonic acid formed from prostaglandin endoperoxides in the walls of arteries and veins.

Role of Minerals in Hypertension:

Control of blood pressure is multifactorial and thus the causes of hypertension are heterogeneous. Many dietary minerals have been implicated in the pathogenesis or maintenance of elevated blood pressure in humans, the foremost of which have been dietary intake of sodium, potassium and calcium.
Role of Sodium in Hypertension

Development of the sodium hypothesis in human hypertension has been based on epidemiologic evidences, studies manipulating dietary sodium and experimental hypertension.

Ambard and Beaujard in 1904 were the first researchers to report that salt deprivation may be associated with a decline in the blood pressure of hypertensive patients. Later, in 1944 Kempner advocated rice-fruit diet for hypertensives and were successful in reversing hypertension in 66 per cent of the patients studied (Porter, 1983). Murphy (1950) reported that when blood pressure fell in patients on the rice-fruit diet, a measurable decrease in both plasma volume and extracellular volume also occurred. In 1950, Watkins emphasized that persistent restriction of dietary sodium was required for the antihypertensive effect of sodium depletion to be sustained. Subsequent studies have shown that modern Western societies eat more sodium chloride and have higher blood pressure than primitive societies (Lever et al, 1981).

Epidemiological Studies

Bushmen of the Kalahari desert (Kaminer and Lutz 1960), the Easter Islanders (Cruz Coke et al, 1964) the Pupa-Pukans
of the Cook Islands (Prior et al, 1968), the Solomon Islanders (Page et al, 1974), the Yanomamo Indians (Oliver et al, 1975) and Papua New Guinea highlanders (Kikimaru et al, 1986) have low prevalence of hypertension. They represent different races and have different climates, diets, customs etc. They are generally lean and active, have little tendency for weight to increase with age and usually have diets that are low in sodium and high in potassium. Absence of obesity may not be the decisive factor in preventing hypertension, since Prior et al (1968) studied two polynesian communities which are similar in all other aspects except sodium consumption and found that the Rarotongans whose sodium consumption is higher (120-140 meg/day) exhibit an increase in blood pressure with age (131/84 to 155/92 mm Hg) when compared to Puka-Pukans whose sodium consumption is half and no increase in blood pressure with age (114/71 to 119/80 mm Hg).

Tobian (1979) cites observation on an acculturated Solomon Island group that cooks its food in sea water. This population has a 10 per cent incidence of hypertension which is not found in a similar island group that uses water from fresh water stream thereby having a low sodium intake.
Rikimaru et al (1988) gave a high sodium diet to Papua New Guinea highlanders who do not usually take dietary salt. Sodium loading resulted in positive sodium balance and significant increase in blood pressure from 92/56 mm Hg to 102/60 mm Hg.

Studies Manipulating Diet and Drugs in Hypertension:

Parijs et al (1973) suggested that a reduction of the daily sodium chloride intake from 10 to 5 gms could produce a decrease in blood pressure of about 10/5 mm Hg.

Morgan (1978) studied 31 patients with diastolic blood pressure between 95 and 109 mm Hg and have been treated for two years with moderate restriction of salt and found that the diastolic blood pressure was reduced by 7.3 ± 1.6 mm Hg and an increase of 1.8 ± 1.1 mm Hg was observed in control group.

Mac Gregor et al (1982) conducted a carefully controlled trial on patients with mild to moderate hypertension, whose average supine blood pressure after two months observation and on no treatment was 156/98 mm Hg. They were on low sodium diet and given either sodium chloride capsule to return sodium intake to the pre-study level or given identical appearing placebo capsules. The
average blood pressure fell significantly during the period of lower sodium intake by 6.1 per cent and on addition of sodium chloride, blood pressure returned to the pre-study level.

Dodson et al (1989) similarly found that dietary restriction of sodium has hypotensive effect and that blood pressure rose on sodium supplementation in their randomised blind controlled and cross over study.

Beard et al's (1982) study combined drug treatment with sodium restricted diet. It involved 90 hypertensive patients who remained on anti-hypertensive drug therapy for 12 weeks during which half of the subjects sharply reduced their dietary sodium intake to an average of only 37 mmol/day. This group had a greater fall in blood pressure despite the purposeful discontinuation of more than half of their daily total number of antihypertensive tablets whereas the control group had to remain on almost the full amount of medication to achieve a lesser fall in blood pressure. Andreas et al (1984) compared the effects of diuretic therapy and low sodium intake in isolated systolic hypertension and found that both are equally effective in lowering hypertension.
Heterogenic Response of Dietary Sodium Restriction to Blood Pressure:

The evidence suggesting that moderate salt intake would significantly lower blood pressure is weak. In the initial experiments, as those of Kempner’s rice diet, all the variables were not controlled. The rice diet was somewhat unpalatable and weight loss was common (Schroeder et al., 1949), independent of salt intake (Tuck et al., 1981) and weight loss was not controlled in these studies (Laragh and Puker, 1983).

Holden et al. (1983) found on a sample representative of 2.1 million adults in Connecticut that dietary salt is unlikely to have a clinically significant effect on blood pressure in majority of individuals in a large defined population but they do not exclude the possibility of a clinically significant effect in a small sub-group of salt sensitive individuals.

When Grobber and Hofman (1986) reviewed 13 randomised trials on the effect of sodium restriction on blood pressure they noted that the hypotensive effect of sodium restriction was small and restricted largely to systolic blood pressure which fell by an average of 3.6 mm Hg. It was also observed that the reduction increased with age and
in those with higher blood pressure. Thus sodium restriction seems to be of limited use in young patients with mild hypertension.

In Scotland 7354 men and women aged 40-59 years were studied (Smith et al, 1988). The association between sodium and blood pressure was shown to be weak and did not have any real independent role in explaining blood pressure. The effect of potassium intake and alcohol on blood pressure seemed to be greater.

In an International study by Intersalt Co-operative research group (1988), subjects from 52 centres in 32 countries were recruited. Subjects from 4 centres who had very low sodium intake (estimated from 24 hour urinary sodium excretion) had low blood pressure and no increase in blood pressure with age. As data from these cultures weigh the results, the Intersalt study group presented separate analyses excluding these four centres. In the remaining 48 centres neither the median blood pressure nor the prevalence of high blood pressure were related to sodium excretion. On the other hand, the rate of increase of both systolic and diastolic pressure with age was significantly related to sodium excretion. In addition, there was a significant tendency for sodium intake and systolic
(but not diastolic) blood pressure to be correlated when individual centres were looked at separately.

In view of the heterogenic response of dietary sodium restriction in lowering blood pressure, it is obvious that human beings have different reactions to a high sodium chloride diet. It is possible that 4 to 20 per cent of the population may be genetically susceptible to developing essential hypertension by middle life. The remaining of the population may be genetically resistant to developing essential hypertension. A person genetically resistant to hypertension can ingest as much as 200 meq/day of salt without developing elevated blood pressure. However, in persons genetically susceptible to hypertension, a life long restriction of salt intake may prevent hypertension indefinitely and subsequent hypertensive complications (Tobian, 1983).

Pathophysiological Mechanisms of Hypertension:
Evidence from Animal Experiments:

Goldblatt et al (1934) showed that reducing renal perfusion pressure in dogs caused hypertension. This finding rejuvenated interest in renin. Further, definition of renin action as pressor substance was provided. Angiotensin was identified as the vasoactive substance formed by renin's action (Porter, 1983).
Selkurt (1951) showed a direct relation between acute elevation of blood pressure and increased urine volume originating the concept of pressure diuresis. Later reports showed that sodium content of arteries increased and potassium content decreased in hypertensive rats bringing an integration of sodium with elevated peripheral resistance (Tobian and Binion, 1952).

On a moderately high salt intake, some humans are hypertensive and others are not. On a very low sodium intake (less than 60 meq/day) few develop hypertension. Dahl et al (1962) selectively bred two strains of rats that mimic the human response. On a low salt diet, the rats have normal pressure. But when the two strains are challenged with a high salt intake, the sensitive strain gradually become severely hypertensive. The resistant strain has no rise in blood pressure.

Tobian et al (1978) showed that isolated kidneys of salt sensitive Dahl rats excrete only half as much sodium as kidneys from resistant rats when compared at equal levels of in-flow pressure. Thus, in the sensitive strain prone to hypertension there appears to be a resetting of the pressure natriuresis curve, in that a higher inflow pressure is needed to achieve a given sodium excretion. This action was observed to occur in kidneys without any obvious lesions.
Role of Prostaglandins:

The renal papilla in sensitive rats, in both the prehypertensive as well as the hypertensive state, have a 50 per cent lower prostaglandin concentration than that of the resistant rats. This reduction in E₂ prostaglandins will enhance sodium reabsorption in the ascending loop of Henle, collecting tubule and collecting duct, thereby encouraging sodium retention and hypertension. The reduction in renal papillary plasma flow in sensitive rats on any salt diet also promoted sodium retention with its concomitant rise in blood pressure (Tobian, 1983).

Role of Central Nervous System:

Takeishi and Mark (1978) found an increased vascular resistance in Dahl sensitive rats that have become hypertensive after one month of a high sodium chloride intake. When sympathetic nerves to the hind quarters of these hypertensive rats were cut, half of the increase in vascular resistance was abolished. This finding points to sympathetic nerve participation in the sodium induced increase in the vascular resistance. After the peripheral sympathetic nerves in the newborn Dahl salt sensitive rats were destroyed, the feeding of a high salt diet did not cause a rise in blood pressure, again pointing to participation of sympathetic nervous system in hypertension.
Evidence from Human Experimentation:

In 1969, the pathogenesis of hypertension was centered around abnormal extracellular fluid volume: a dysfunction of renal sodium excretion was thought to cause initial disturbance in regulation of human sodium balance in hypertension (Guyton and Coleman, 1969). It was later proposed that abnormal salt retention and subsequent extracellular fluid expansion and hypertension were mediated humorally (Haddy and Overbeck, 1976).

Dewardner and Mac Gregor (1980) proposed that an endogenous natriuretic hormone was secreted in response to pathologic renal sodium retention in hypertension. The natriuretic hormone through an effect on cellular ATPase activity, modified sodium content of vascular tissue and thereby vascular tone.

In a review by Lever et al (1981) it was cited that patients with essential hypertension have excess of sodium in their blood cells. A similar excess of sodium in the cells of vascular smooth muscle produce vasoconstriction and hypertension.

In the arteriolar smooth muscle, the inhibition of sodium transport across the cell wall causes a rise in
the intracellular sodium concentration which in turn rises the intracellular calcium concentration and thus increases vascular reactivity. This hypothesis also suggests that the abnormality of sodium transport in circulating cells in vivo is directly due to the increased secretion of the circulating sodium transport inhibitor (Mac Gregor, 1982).

Kidney-salt Hypothesis

In patients with essential hypertension, there is an inhibited defect of the kidney's ability to excrete sodium that becomes increasingly obvious as the sodium intake increases. A central point of some theories is that pressure natriuresis is reset in essential hypertension; higher pressure being needed to maintain a given sodium excretion.

One view of the pathogenesis of essential hypertension is that two mechanisms are at work. Blood pressure is raised first by a process that is not primarily a resetting of pressure natriuresis. Later, and as a possible consequence of the earlier hypertension, a renal lesion develops that is characterised by resetting and cause a further increase in blood pressure. Interaction of the two mechanisms then produce progressive hypertension (Lever et al, 1981).
Role of Renin-angiotensin System:

Renin-aldosterone system regulates sodium - volume and blood pressure simultaneously. When the sodium intake is low, plasma renin activity rises and vice versa. Renin converts angiotensinogen to Angiotensin II which is a vasoconstrictor. Angiotensin stimulates aldosterone which causes retention of sodium and excretion of potassium. Low renin and high renin hypertension has been recognised. One possible explanation for the heterogenic response of persons with high blood pressure to dietary sodium restriction is the disparate patterns of plasma renin activity (Brunner et al, 1972).

For a particular sodium intake, the renin secretion is abnormally low in some patients and in few it was observed to be abnormally high. This phenomena is termed as low renin hypertension and high renin hypertension respectively.

In high renin hypertension, excess renin is the cause of hypertension as more of angiotensin and aldosterone are produced. Angiotensin being a potent vasoressor increases blood pressure and aldosterone retains more sodium. Therefore patients with a high renin secretion do not respond to salt restriction as any given
amount of sodium is perceived as a shortage by the kidneys and more renin is produced.

But the situation is reversed in low renin hypertension. A given sodium intake is perceived as excess sodium balance by the kidney and renin production is further suppressed. Hence these patients respond best to sodium deprivation (Laragh and Pecker, 1983).

If susceptible human beings become hypertensive only when they eat generous amounts of sodium chloride, first there must be some initial accumulation of sodium or chloride in the body and secondly this initial accumulation of body sodium must lead to rise in arterial pressure. While Kawasaki et al (1978) and Fujita et al (1980) recorded greater sodium retention and increase in cardiac output on a high sodium diet, Parfrey et al (1981) did not observe any sodium retention. However, secondary hypertension observed with disease of the renal parenchyma tend to retain body sodium and develop hypertension. Patients with an adenoma that causes a rise in aldosterone were observed to have a tendency to retain sodium and frequently become hypertensive (Tobian, 1979).

Role of Adrenergic Nervous System:

Nervous impulses are generated from various centres
in the central nervous system and are transmitted through post ganglionic nerves to the blood vessels and the heart mediated by neurohumoral substances, the commonest being nor-epinephrine. Nor-epinephrine is secreted by adrenal medulla being predominantly released in response to hypotension. It is a powerful vasopressor. In a study by Fujita et al (1980), mean plasma norepinephrine concentrations on low sodium diet did not differ between salt sensitive and non-sensitive hypertensive patients but on an increase in sodium intake from 9 meq/day to 249 meq/day it significantly decreased in all patients. On continuation of the high sodium diet, plasma nor-epinephrine increased significantly in the salt sensitive patients whereas it showed a decrease in the non-sensitive patients. Thus, the relative retention of sodium by the salt sensitive patients could depend in part upon a relatively higher activity of the adrenergic nervous system.

Is Sodium Ion Alone Important in the Pathogenesis of Hypertension?

The view now prevalent is that the capacity of sodium chloride to increase blood pressure depends only on its sodium component. Kurtz et al (1987) investigated whether the anionic component of an orally administered sodium salt can influence the salts capacity to increase blood pressure. They observed that on giving salt
sensitive hypertensive patients 240 m mol of sodium as sodium chloride per day, induced significant increases in systolic and diastolic pressure (increase of 16/8 mm Hg) whereas an equimolar amount of sodium given as sodium citrate induced no change in blood pressure. Further it was noted that on replacing supplemental sodium chloride with an equimolar amount of sodium as sodium citrate abolished the increase in blood pressure induced by sodium chloride. It was also observed that both salts induced substantial and comparable sodium retention, weight gain and suppression of plasma renin activity and plasma aldosterone but supplemental sodium chloride increased plasma volume and urinary excretion of calcium whereas sodium citrate did not.

The mechanism whereby changes in extracellular fluid volume were induced by sodium chloride is not clear. Chloride may influence blood pressure independently of sodium. Actions of chloride have been demonstrated in the brain, kidney and vascular smooth muscle (Luft and Ganten, 1987).

From a practical viewpoint, the overwhelming majority of ingested sodium in human diets is in the chloride form. Thus, a clearer understanding of the
mechanisms for the effects of sodium chloride on blood pressure would have important implications for health (Weinburger, 1987).

Risks and Benefits of Reducing the Intake of Sodium:

Sodium is an essential nutrient, but the amounts commonly consumed exceed the minimum requirement. Wasir (1989) opines that there is no harm in restricting sodium intake in diet and that it must be given a trial in all cases of high blood pressure.

Lowering salt intake is associated with an improved health status and an increased life expectancy at the population level without discernible health hazards. The effect of high salt intake is not limited to increasing blood pressure and stroke mortality alone and increasing evidence indicates that salt is a caustic product with properties that promote atrophic gastritis and stomach cancer (Joosens and Geboers, 1987).

There are also risks of lowering sodium diet. Occasionally, intense sweating, secondary to physical excretion or salt loss with diarrhoea or vomiting may lead to severe depletion in persons maintained on intakes as low as 60 meq/day (Tobian, 1979). It becomes essential
to know what level of salt restriction is needed and whether it applies to the whole population evenly or only to a subsection.

Another major problem with sodium restriction is it makes food unpalatable and patient may find it difficult to adhere to it life long.

Despite major advances in the past 50 years, role of dietary sodium in hypertension is not clearly understood as to how it exerts pressor action, when it does so and why it does only in certain people. Whether the action of sodium is a direct effect of the ion itself or induced by changing the amount of activity of other ions such as potassium, calcium, chloride or magnesium also needs to be clarified. Clinical trials are needed in which a moderate or modest reduction of sodium in the diet is maintained over a reasonably long period and in which the dietary regimen is planned to avoid large concurrent changes in potassium, calcium or magnesium or a change in other ingredients or calories that might affect the blood pressure.
Role of Potassium in Hypertension:

Physiological Significance of Potassium:

Potassium is the most abundant cation in the body. It exists primarily within the cells at concentrations of 140-150 meq/litre while in contrast, sodium is the principal cation of the extra-cellular fluid. Potassium plays an important part in the regulation of acid/base balance. The relative concentration of potassium in and out of cells determine to a large extent the potential difference across cell membranes. In addition, the stimulus necessary to cause cell depolarization as well as the cells' subsequent ability to repolarize are a function of the intra and extra-cellular potassium concentrations.

Potassium is important for the transmission of nerve impulses to muscle fibres and to the contractility of the muscle itself. Hence, disturbances in transmembrane and total body potassium homeostasis may directly affect blood pressure by influencing the ability of cardiac and smooth muscle as well as nerve cells to function (Luft and Weinberger, 1987).

Epidemiological Inferences Associating Potassium with Blood Pressure:

Potassium intake has been linked to arterial hypertension since Addison in 1928 first suggested that a diet
high in potassium could exert an antihypertensive effect. Several within population studies have been carried out since then to determine if there is any relation between potassium and blood pressure.

Northern Japanese ingest a very high salt intake and exhibit a correspondingly high prevalence of hypertension. In two neighbouring northern Japanese villages, in which individuals ingested similar amounts of sodium, a lower blood pressure was identified in the village with a higher potassium intake (Sasaki, 1962).

Meneely and Battarbee (1976) believed that the high sodium, low potassium diet ingested in Western civilization combined with a genetic susceptibility was a principal factor in the genesis and perpetuation of essential hypertension.

An epidemiologic investigation was conducted on 2500 Japanese and Yamori et al (1981) found an inverse correlation between blood pressure and potassium. They also found a significant gradual increase in the prevalence of hypertension and mean arterial blood pressure with increasing urinary sodium to potassium ratio. Lever et al (1981) also observed that total body potassium
correlated inversely with arterial pressure in hypertensive patients.

Moderate potassium supplementation was observed to have caused a significant fall in blood pressure in patients with mild to moderate essential hypertension. The increase in potassium intake could be achieved with a potassium based salt substitute and a moderate increase in vegetable and fruit consumption. Moderate dietary sodium restriction with dietary potassium supplementation may reduce the need for drug treatment in some patients with mild to moderate hypertension (Parfney et al, 1981; Mac Gregor et al, 1982; Khaw and Connor, 1984).

In a study, Parfrey et al (1981) observed that blood pressure rose in hypertensive as well as normotensive subjects on a high sodium diet by $8.9 \pm 12.6$ mm Hg and $5.3 \pm 9.0$ mm Hg respectively but noted a significant fall in blood pressure by $8.9 \pm 11.2$ mm Hg in hypertensives on supplementation with potassium.

Mac Gregor et al (1982) recorded a 4 per cent fall in blood pressure in mild to moderate hypertensives on potassium supplementation ($60$ mmol/day or $2345$ mgs of potassium) for four weeks. There was increase in sodium excretion and plasma potassium concentration. The results are given in Table No. 3.
<table>
<thead>
<tr>
<th>Parameter</th>
<th>Pre-Treatment</th>
<th>Placebo</th>
<th>After Treatment</th>
<th>Placebo</th>
<th>After Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Plasma cortisol</td>
<td>63 ± 2.7</td>
<td>63 ± 2.7</td>
<td>63 ± 2.7</td>
<td>63 ± 2.7</td>
<td>63 ± 2.7</td>
</tr>
<tr>
<td>Blood urea</td>
<td>4.2 ± 0.2</td>
<td>4.2 ± 0.2</td>
<td>4.2 ± 0.2</td>
<td>4.2 ± 0.2</td>
<td>4.2 ± 0.2</td>
</tr>
<tr>
<td>Plasma sodium</td>
<td>140 ± 3.4</td>
<td>140 ± 3.4</td>
<td>140 ± 3.4</td>
<td>140 ± 3.4</td>
<td>140 ± 3.4</td>
</tr>
<tr>
<td>Plasma potassium</td>
<td>69 ± 2.3</td>
<td>69 ± 2.3</td>
<td>69 ± 2.3</td>
<td>69 ± 2.3</td>
<td>69 ± 2.3</td>
</tr>
<tr>
<td>Plasma aldosterone</td>
<td>10 ± 0.2</td>
<td>10 ± 0.2</td>
<td>10 ± 0.2</td>
<td>10 ± 0.2</td>
<td>10 ± 0.2</td>
</tr>
<tr>
<td>Plasma renin activity</td>
<td>150 ± 1.8</td>
<td>150 ± 1.8</td>
<td>150 ± 1.8</td>
<td>150 ± 1.8</td>
<td>150 ± 1.8</td>
</tr>
<tr>
<td>24 hour urinary creatinine</td>
<td>0.2 ± 0.1</td>
<td>0.2 ± 0.1</td>
<td>0.2 ± 0.1</td>
<td>0.2 ± 0.1</td>
<td>0.2 ± 0.1</td>
</tr>
<tr>
<td>24 hour urinary sodium</td>
<td>11 ± 0.4</td>
<td>11 ± 0.4</td>
<td>11 ± 0.4</td>
<td>11 ± 0.4</td>
<td>11 ± 0.4</td>
</tr>
<tr>
<td>24 hour urinary potassium</td>
<td>20 ± 0.8</td>
<td>20 ± 0.8</td>
<td>20 ± 0.8</td>
<td>20 ± 0.8</td>
<td>20 ± 0.8</td>
</tr>
<tr>
<td>24 hour urinary aldosterone</td>
<td>3.2 ± 0.6</td>
<td>3.2 ± 0.6</td>
<td>3.2 ± 0.6</td>
<td>3.2 ± 0.6</td>
<td>3.2 ± 0.6</td>
</tr>
<tr>
<td>24 hour sodium pulse</td>
<td>78 ± 2.3</td>
<td>78 ± 2.3</td>
<td>78 ± 2.3</td>
<td>78 ± 2.3</td>
<td>78 ± 2.3</td>
</tr>
<tr>
<td>Metformin</td>
<td>7 ± 0.2</td>
<td>7 ± 0.2</td>
<td>7 ± 0.2</td>
<td>7 ± 0.2</td>
<td>7 ± 0.2</td>
</tr>
</tbody>
</table>

**Table No. 3**

Effect of potassium supplementation in essential hypertension

(\( n = 20 \))
Ophir et al (1983) found significantly lower blood pressure in vegetarians and concluded that the protective antihypertensive factor in the vegetarians was due to potassium. They also observed that sodium excretion remained the same in vegetarians as well as non-vegetarians.

A long term randomised double blind placebo controlled trial by Siani et al (1987) showed that moderate oral supplements were associated with a long term reduction in blood pressure by 14/10 mm Hg in patients who had mild hypertension. The blood pressure changes are presented in Table No. 4.

In an International study by Intersalt Co-operative Research Group (1988), potassium excretion was negatively correlated with blood pressure and significant positive relation between urinary Na:K ratio and blood pressure was noted in 10,079 men and women.

Racial differences were also observed in urinary electrolyte excretion. Several studies showed that Blacks had significantly higher blood pressure than the whites, even though sodium consumption by the Blacks was significantly less when compared to the whites (Grim et al, 1980; Voors et al, 1983). It was observed that dietary potassium
**Table No. 4**

Mean Blood pressure changes on potassium supplementation  
(Food Pressure as mm Hg)

<table>
<thead>
<tr>
<th></th>
<th>Placebo group (n=19)</th>
<th>Potassium group (n=18)</th>
<th>Mean Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Week 0</td>
<td>Week 15</td>
<td>Week 0</td>
</tr>
<tr>
<td><strong>Supine</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>145.1±2.9</td>
<td>145.8±2.6</td>
<td>144.8±2.2</td>
</tr>
<tr>
<td>Diastolic</td>
<td>91.5±1.6</td>
<td>92.5±2.1</td>
<td>91.6±1.4</td>
</tr>
<tr>
<td><strong>Standing</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>145.5±3.4</td>
<td>145.9±3.2</td>
<td>145.0±2.4</td>
</tr>
<tr>
<td>Diastolic</td>
<td>96.8±1.5</td>
<td>98.5±1.6</td>
<td>97.2±1.5</td>
</tr>
</tbody>
</table>

* P < 0.001, 15 Weeks vs baseline + P < 0.001 Potassium vs Placebo treatment.
was less in Blacks. Urinary excretion of sodium and potassium was also less in Blacks when compared to Whites (Grim et al, 1980; Watson, 1980).

Skrabal et al (1981) on giving 'calculation stress test', observed equal maximum rise in blood pressure whether the subject was on the usual diet, a low sodium diet or a high potassium diet, but a combined low sodium-high potassium diet reduced the rise in blood pressure during mental stress. The results are presented in table No. 5.

However, a few studies have not documented a relationship between blood pressure levels and dietary potassium intake.

Holbrook et al (1984) found no significant correlation between absolute sodium or potassium intake and blood pressure in a one-year study. Richards et al (1984) showed that moderate restriction of sodium intake or supplementation of dietary potassium has variable effects on arterial pressure in individuals with mild essential hypertension and that the overall blood pressure changes induced were very small.
Table No. 5

Effects of sodium restriction and/or high potassium intake in normotensive subjects on body weight, Electrolytes, Pentence hormones and response to mental stress

<table>
<thead>
<tr>
<th></th>
<th>Usual diet</th>
<th>Low sodium diet</th>
<th>High Potassium diet</th>
<th>Low sodium/ High potassium diet</th>
</tr>
</thead>
<tbody>
<tr>
<td>Change in body weight (kg)</td>
<td>75.8 ± 2.66</td>
<td>-1.02 ± 0.07**</td>
<td>-0.69 ± 0.43*</td>
<td>-1.20 ± 0.10***</td>
</tr>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>125.0 ± 2.39</td>
<td>122.3 ± 2.32</td>
<td>123.3 ± 2.54</td>
<td>122.7 ± 1.81</td>
</tr>
<tr>
<td>Diastolic blood pressure (mm Hg)</td>
<td>73.1 ± 2.17</td>
<td>70.1 ± 1.66</td>
<td>68.6 ± 1.56</td>
<td>69.6 ± 1.67</td>
</tr>
<tr>
<td>Heart rate (beats/min.)</td>
<td>62.2 ± 2.44</td>
<td>67.4 ± 3.25</td>
<td>67.4 ± 2.06</td>
<td>71.0 ± 6.88</td>
</tr>
<tr>
<td>Serum sodium (mmol/l)</td>
<td>141.2 ± 1.75</td>
<td>144.3 ± 1.29</td>
<td>142.6 ± 0.90</td>
<td>149.1 ± 0.49**</td>
</tr>
<tr>
<td>Serum potassium (mmol/l)</td>
<td>4.69 ± 0.13</td>
<td>4.64 ± 0.09</td>
<td>4.51 ± 0.10</td>
<td>4.75 ± 0.20</td>
</tr>
<tr>
<td>Serum creatinin (mg/dl)</td>
<td>1.07 ± 0.02</td>
<td>1.13 ± 0.03**</td>
<td>1.13 ± 0.03**</td>
<td>1.28 ± 0.04**</td>
</tr>
<tr>
<td>Plasma renin (pg/ml/h)</td>
<td>326.3 ± 32.02</td>
<td>602.8 ± 72.20**</td>
<td>774.8 ± 46.10</td>
<td>1439.6 ± 413.87***</td>
</tr>
<tr>
<td>Plasma aldosterone (ng/dl)</td>
<td>5.3 ± 1.34</td>
<td>15.9 ± 1.79</td>
<td>11.1 ± 1.54**</td>
<td>43.1 ± 9.70**</td>
</tr>
<tr>
<td>Plasma noradrenaline (ng/ml)</td>
<td>0.353 ± 0.075</td>
<td>0.605 ± 0.177***</td>
<td>0.440 ± 0.104</td>
<td>0.418 ± 0.096</td>
</tr>
<tr>
<td>Basal</td>
<td>0.413 ± 0.104</td>
<td>0.580 ± 0.169</td>
<td>0.401 ± 0.078</td>
<td>0.448 ± 0.091</td>
</tr>
<tr>
<td>Plasma adrenaline (ng/ml)</td>
<td>0.053 ± 0.009</td>
<td>0.062 ± 0.017</td>
<td>0.062 ± 0.011</td>
<td>0.044 ± 0.009</td>
</tr>
<tr>
<td>After mental stress</td>
<td>0.087 ± 0.009</td>
<td>0.071 ± 0.019</td>
<td>0.070 ± 0.015</td>
<td>0.078 ± 0.009</td>
</tr>
<tr>
<td>Plasma vasopressin (pg/ml)</td>
<td>10.1 ± 0.76</td>
<td>11.6 ± 1.49</td>
<td>10.5 ± 0.88</td>
<td>Not done</td>
</tr>
<tr>
<td>Basal</td>
<td>11.9 ± 1.41</td>
<td>11.9 ± 1.00</td>
<td>Not done</td>
<td></td>
</tr>
<tr>
<td>After mental stress</td>
<td>210.5 ± 23.6</td>
<td>40.9 ± 7.5**</td>
<td>155.1 ± 20.9</td>
<td>210.4 ± 5.3***</td>
</tr>
<tr>
<td>Urinary potassium (mmol/day)</td>
<td>71.4 ± 5.8</td>
<td>65.4 ± 5.6</td>
<td>115.5 ± 11.5**</td>
<td>72.4 ± 4.1***</td>
</tr>
<tr>
<td>Response to mental stress</td>
<td>83.50 ± 5.23</td>
<td>93.60 ± 5.98**</td>
<td>88.30 ± 5.25*</td>
<td>100.00 ± 5.41**</td>
</tr>
<tr>
<td>Systolic blood pressure at 2 min (mm Hg)</td>
<td>134.50 ± 4.92</td>
<td>134.50 ± 4.27</td>
<td>134.30 ± 1.70</td>
<td>124.40 ± 2.25***</td>
</tr>
</tbody>
</table>

* Significant at 0.05 level  
** Significant at 0.01 level  
*** Significant at 0.001 level
Mechanisms for Antihypertensive Action of Potassium:

The blood pressure lowering characteristics of potassium have been attributed to its

1) diuretic action,
2) natriuretic action,
3) altered activity of renin-angiotensin system,
4) direct alteration of peripheral resistance and
5) effect on central or peripheral nervous system.

Diuretic and Natriuretic Action of Potassium:

Addition (1928) opined that the effect of potassium salts on blood pressure was induced by diuresis. More recently Morino et al (1978) observed a reduction of 10 mm Hg systolic and 5 mm Hg diastolic blood pressure along with natriuresis producing a total sodium loss of 250 meq over three days, on supplementation of 250 meq potassium to moderately hypertensive subjects.

Experimental studies (Young et al, 1976) demonstrated that small increase in plasma potassium within the physiological range have long term effects on sodium balance, potent enough to produce sustained extracellular contraction.
Skrabal et al (1981) observed a loss of about one kilogram body weight on moderate salt restriction or a high potassium intake and a reduction in sodium space from 25.75 ± 1.79 to 21.83 ± 1.24 litres. It was therefore inferred that when on the usual high sodium/low potassium diet, the subjects live with a larger extracellular fluid volume (about one litre) than when on a low sodium diet.

The statutory effect of an increased potassium intake may be related to stimulation of the Na⁺ K⁺ pump. On stimulation, the cellular Na⁺ K⁺ ATPase increases the efflux of sodium from the cellular pool and increases sodium excretion. In blood vessels pump stimulation results in vasodilation.

The natriuretic effect of potassium does not seem to be related to alterations in renal hemodynamic variables although data in normal humans do suggest that a high oral potassium intake increased renal blood flow. There is considerable debate concerning the precise nephron site where a high potassium level inhibits sodium reabsorption. Some data points to the proximal nephron while others, the ascending limb (Tannen, 1983).
Altered Activity of the Renin-angiotensin System:

A high potassium level might influence blood pressure through a decrease in plasma renin activity by a reduction in angiotensin II generation (Ophir et al, 1983). On the other hand, the natriuretic effects of potassium salts may tend to increase renin activity.

Skrabal et al (1981) found the greatest stimulation of the renin-aldosterone system after the low sodium/high potassium intake, during which urinary sodium excretion was lower and urinary potassium higher than when sodium intake was reduced and potassium intake was increased separately. Whereas, Parfrey et al (1981) found no significant changes in plasma renin activity in hypertensive patients on high potassium intake despite a fall in blood pressure.

The variable response may be explained by the dual effect of high potassium levels on renin levels. The natriuresis and volume contraction which accompany a high potassium intake stimulate renin release. But high potassium level itself seems to be inhibitory. The net result on plasma renin activity depends on which effect predominates (Tannen, 1983).
Alteration of Peripheral Resistance:

The potassium ion may act as a vasodilator and thus influence arteriolar smooth muscle directly. A small increase in serum potassium concentration produced local vasodilation in experiments conducted in both humans and animals. This effect seems to result from an action of potassium on Na–K ATPase of the vascular smooth muscle (Chen, 1972).

Effect on Central or Peripheral Nervous System:

The effect of potassium to lower blood pressure may be related to action on the nervous system. Medulla oblongata has the vasomotor area which controls the vasomotor tone. The smooth muscle of the arterioles and veins receive nerve impulses that keep the lumen of the vessels more or less constricted. The transmitter substance is nor epinephrine. The re-uptake of nor-epinephrine by sympathetic nerve terminals is influenced by the relative concentration of sodium and potassium.

A low sodium, high potassium diet has been associated with a decrease in resting blood pressure as well as an increase in baro-receptor sensitivity (Mickelson et al, 1977; Skrabal et al, 1981). Further, the pressor
and heart rate responses to intravenously administered norepinephrine were attenuated when patients were pretreated with potassium salts (Battarbee et al., 1979).

Studies by Parfrey et al. (1981) on the progeny of hypertensive and normotensive parents showed that in the progeny of hypertensive persons, plasma nor-epinephrine levels reduced significantly and blood pressure fell in response to a high potassium intake; whereas in the progeny of normotensive persons, norepinephrine levels increased but blood pressure did not change in response to a high potassium intake.

Any influence of potassium on blood pressure are probably the results of multiple simultaneous actions. The data for potassium supplementation are not firm enough to allow guidelines for their general use. A palatable and inexpensive potassium supplement has not been developed. It is possible that with increased potassium intake through natural means (as vegetables and fruits) the incidence of vascular complications could be reduced.

Role of Calcium in Hypertension:

Calcium is an essential element in normal cellular physiology. Normal cardiovascular function is critically
dependent on both extra and intracellular calcium concentration. The constant influx and efflux of calcium in the cells is said to be a major responsible factor in maintaining vascular tone. Abnormalities in calcium metabolism have been identified in both human and experimental hypertension (McCarron, 1985).

At present, contradicting results have been reported about calcium in the pathogenesis and therapy of human essential hypertension.

Dietary Calcium and Blood Pressure:

McCarron et al (1982) reported significantly less dietary calcium ingestion (670 mg compared to 890 mgs) in subjects with essential hypertension when compared to normotensive controls, while typical values were reported for other nutritional components i.e., calories, protein, fat, sodium and potassium. McCarron et al (1984) again analysed data from the first National Health and Nutrition Examination Survey (NHANES-I) and found similar results. Ackley et al (1983) showed a significant association between low calcium intake from dairy products to elevated blood pressure independent of age, obesity and alcohol consumption.
Castenmiller et al (1985) divided normotensive subjects into a low and high calcium group and on sodium supplementation to both groups found that sodium supplementation had no effect on blood pressure but mean systolic and diastolic blood pressure in the high calcium consuming group were lower, suggesting that calcium might have a role in the regulation of blood pressure. Kok et al (1986) extended similar evidence to an earlier time period. They found a significant negative trend with systolic blood pressure for calcium intake in both males and females from data gathered in the Netherlands in the early 1950s.

Using NHANES-I data, Gruchow et al (1988) studied the relationships of dietary sodium, potassium and alcohol to blood pressure in relation to levels of dietary calcium intake. At low calcium intakes (less than 400 mg/day for men and 600 mg/day for women) the ratio of sodium to potassium was significantly related to blood pressure after controlling for age, body mass index, race and gender. At higher calcium intakes neither sodium to potassium ratio nor any other nutrient (with the exception of alcohol) was related to blood pressure. Sodium to potassium ratio was more strongly related to blood pressure and low calcium intakes were necessary for the sodium: potassium blood pressure relationship to be evident.
These data suggest a link between reduced calcium intake in the pathogenesis of human hypertension. But Sowers et al (1985) and Johnson et al (1985) found no significant relationship between estimated current intake of calcium and blood pressure but reported a significant inverse relationship between estimated dietary intake of vitamin D and systolic blood pressure.

In an analysis of the relation between blood pressure and twenty dietary factors among 8,000 Japanese men in Hawaii, calcium intake from dairy sources alone was inversely related to blood pressure but the high degree of intercorrelation between calcium intake and potassium and protein intake precluded assignment of an independent role to any specific nutrient (Reed et al, 1985).

Serum Calcium and Blood Pressure:

A number of biochemical abnormalities associated with hypertension and calcium metabolism have been reported.

McCarron (1982) and Strazzullo et al (1983) found no significant difference in serum total calcium levels between normotensives and hypertensives. On the other hand, Kesteloot and Geboers (1982), Sangal and Beevers (1982) and Robinson et al (1982) found a positive correlation between blood pressure and serum total calcium. Similarly, Harlan et al (1984) studied subjects of NHANES-I and reported that blood pressure in general was related directly to serum calcium even among subjects with an inverse relationship to dietary calcium intake.

In contrast, Erne et al (1984) found significant inverse relationship between blood pressure and total plasma calcium. Data showing lower total serum calcium levels are however not common particularly for large populations.

While McCarron (1982) reported a lower serum concentration of ionized calcium in hypertensives when compared to normotensive subjects, Kesteloot et al (1982) and
Strazzullo et al (1982) found no such relationship. The levels of ionized calcium have been said to differ directly with levels of plasma renin activity (Resnick et al, 1986) but Freeman et al (1985) have not confirmed that relationship.

Urinary Excretion of Calcium in Hypertension:

There seems to be urinary calcium leak in hypertension as urinary excretion of calcium in patients with primary hypertension is higher when compared to normotensives (Kesteloot and Geboers, 1982; Staessen et al, 1983). Strazzullo et al (1983) also found urinary calcium to be significantly different between normotensives and hypertensives with normal renal function, the levels being 3.58 ± 0.20 and 4.60 ± 0.26 mmol/24 hours respectively even when sodium excretion was not significantly higher in hypertensive patients. Calcium/sodium ratio was however significantly increased in the hypertensive group. However, Breslau et al (1982) and Castenmiller et al (1985) found correlation between higher excretion of calcium and increased intake and excretion of sodium.

Calcium and Regulation of Blood Pressure:

Intra-arterial blood pressure is a function of two cardiovascular parameters - cardiac output and peripheral
Calcium is necessary for contraction as well as relaxation of cells. In the resting phase of the cell, intracellular calcium is maintained at extremely low levels ($10^{-7}$ mol). Upon cell activation, there is a sudden increase in cytosolic ionized calcium ($10^{-6}$ mol) which reflects the rapid release of vesicle stored ion (Entry phase). The free cation is then quickly bound to calcium specific binding protein (binding phase). Metabolic events are provoked that are needed for cell specific physiological function. The binding phase is followed by the storage and extrusion phase i.e., the initiation of vesicle uptake and storage of calcium ($10^{-4}$ mol) as well as efflux of calcium across the cell membrane. Once this is accomplished the cell returns to its resting metabolic state. Thus calcium regulation is required for both contraction and relaxation of cells (Mc Carron, 1985).

Free cytosolic calcium in large measures determine the development of tension in vascular smooth muscle.
cells and hence arteriolar resistance. An increase in intracellular free calcium may result from enhanced influx, reduced efflux or alteration in mechanisms for calcium homeostasis. Calcium channel blockers which are found very effective in reducing blood pressure are based on the same principle (Davidson, 1989).

Erne et al (1984) studied free calcium levels in blood platelets, which have many features in common with vascular smooth muscle cells and found free calcium concentration in platelets to be elevated in hypertensives.

Mc Carron (1985) postulate that reduced dietary exposure to calcium may cause depletion of calcium from its membrane storage cites and may produce enhanced calcium fluxes which could be similar to inherent metabolic defect. As a consequence, the vascular smooth muscle cell membrane is less stable and vascular tone and reactivity are increased with a resultant elevation of peripheral resistance.

Kaplan and Meese (1986) are of the opinion that there is an inherent fallacy in this hypothesis because on exposure of cells to low concentration of extracellular calcium, flux rate of calcium across the cell membrane...
might be increased but just enough to maintain intracellular calcium levels at a normal level. The authors see no apparent reason for a lower extracellular concentration of calcium that would mediate vaso constriction. However, there is no experimental evidence that less dietary or extracellular calcium would favour vasoconstriction.

The hypothesis of McCarron and Morris (1982) is that increased exposure of vascular tissue to calcium has membrane stabilizing vaso relaxing effect. Kaplan and Meese (1986) object to this theory as experimental evidence shows that calcium above physiological levels by about 8 times and higher are necessary for vasorelaxing effect and hence are of the opinion that the vaso relaxing hypothesis of calcium is based on extrapolation of experimental data far beyond reasonable limits.

Calcium Regulating Hormones in Hypertension:

Resnick et al (1986) investigated calcium metabolism in human essential hypertension by using the renin aldosterone system. Serum ionized calcium levels were found to be lower among hypertensive subjects with inappropriately low levels of renin than normotensive individuals.
Conversely, in high renin hypertensive subjects, serum ionized calcium values were higher than normotensive or other subjects.

Parathyroid hormone and 1,25 dihydroxy vitamin D levels were higher in low-renin hypertensive subjects whereas calcitonin levels were suppressed confirming a lower average serum ionized calcium levels and suggesting a calcium deficiency in the low renin hypertensives. In contrast, high renin subjects had calcium regulating hormone values appropriate for the higher serum ionized calcium levels, suggesting an endogenous extracellular calcium excess. In salt sensitive individuals, dietary salt loading was observed to produce identical calcium deficient profile as that found in the low renin hypertensive patient (Resnick et al, 1986).

Breslau et al (1982) found sodium induced renal hypercalciuria to be accompanied by increased 1,25 dihydroxy vitamin D synthesis and enhanced intestinal calcium absorption.

Strazzullo et al (1983) observed increasing plasma parathyroid hormone with increased urinary calcium excretion in hypertensives when compared to normotensives.
Parathyroid hormone has been known to increase intracellular calcium concentration in several types of cells due to its influence on cell membrane. Parathyroid hormone stimulates adenyl cyclase activity for the formation of cyclic adenosine monophosphate (cAMP) which increases the efflux of calcium from mitochondria stores increasing cytoplasmic calcium levels thereby causing smooth muscle contraction (Belizan et al, 1983). Strazzula et al (1983) also observed increased cAMP levels in hypertensives.

In contrast to the vasoconstricting effect of parathyroid hormone, Mc Carron et al (1985) stated that parathyroid hormone has vasodilating action. Kaplan and Meese (1986) reject this hypothesis as secondary hypertension is frequently observed in hyperparathyroidism. But Bukowski and Kremer (1991) are of the opinion that parathyroid hormone has vasodilating effect as it can block both basal and agonist activated flux of calcium into aortic smooth segments.

In the healthy cell, vitamin D is required for absorption of calcium across the brush border membrane and synthesis of calcium binding protein is also vitamin D dependent. Calcium binding protein serves as a carrier of calcium to the serosal side of the membrane where it
is actively transported out of the cell via a sodium calcium-ATPase pump (Clark, 1989).

In human hypertension, calcium-ATPase activity was reported to be impaired in the erythrocytes (Postnov et al., 1984) and platelets (Resink et al., 1986) when compared to normotensives.

The blood pressure of spontaneously hypertensive rats fed on a calcium deficient diet was higher than in those fed on a calcium adequate diet. Blood pressure of normotensive rats was not affected. Reduction in activities of sodium-potassium-ATPase, calcium ATPase and alkaline phosphatase were observed on calcium reduction. Alkaline phosphatase activity was strongly inhibited in the basolateral and brush border membranes of spontaneously hypertensive rats indicating that there is decreased calcium absorption by the active transport mechanism (Blakelrough et al., 1990).

The significant reduction in sodium-potassium ATPase activity in the rats may be due to a defect in the membrane itself. It is hypothesized that this reduction would result in an accumulation of sodium in the enterocytes which in turn would tend to reduce calcium absorption by way of sodium calcium exchange (Blakelborough et al., 1990).
Parathyroid hypertensive factor has been reported in plasma of humans with essential hypertension and spontaneously hypertensive rats. The serum concentration of this factor increases during calcium restriction and suppressed on calcium loading (Lewanczuk and Pang, 1989).

Another calcium regulating hormone, calcitonin, is without vascular effects (Bukoski and Kremer, 1991) and calcitonin gene-related peptide was found to be a extremely potent, endothelium independent vasodilator. It is a 37 amino acid neuropeptide, known to stimulate cAMP in vascular smooth muscle cell membrane. It was also demonstrated that calcitonin gene-related peptide is capable of activating potassium-ATP channels of vascular smooth muscle which results in vasodilation secondary to membrane hyperpolarization (Nelson et al, 1988).

Effect of Calcium Supplementation on Blood Pressure:

Belizan et al (1983) found reduction of diastolic blood pressure in normotensives by 5.6 per cent in women and 9 per cent in men after 9 weeks of supplementing 1 g/day of elemental calcium.

A decrease of 13 mm Hg in systolic pressure was
reported by Johnson et al (1985) in medicated hypertensive women on calcium supplementation of 1.5 g/day and an increase of 7 mm Hg in unsupplemented women and no change was observed in normotensives.

A variable response in lowering of blood pressure was found by Alberts et al (1988) on calcium supplementation and are of the opinion that normotensive subjects with high basal calcium intake may not be sensitive to calcium supplementation.

Similarly, Lyle et al (1988) also reported a variable response in blood pressure reduction and thereby classified the subjects as responders and non-responders to calcium supplementation of 1500 mg/day. The results are given in Table No. 6. They found that responders were older and exhibited high mean arterial pressure, higher serum parathyroid hormone and lower serum total calcium. A stepwise discriminant function analysis revealed that mean arterial pressure and serum total calcium were the most important determinants of blood pressure response to supplemental calcium.

Mc Carron and Morris (1985) found a reduction of 4/2 mm Hg supine blood pressure on 8 weeks supplementation
Table No. 6

Baseline characteristics in calcium supplemented group and in responders vs non-responders within the calcium group

<table>
<thead>
<tr>
<th></th>
<th>Calcium supplemented group (n=37)</th>
<th>Responders (n=14)</th>
<th>Non-responders (n=23)</th>
<th>Probability*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (years)</strong></td>
<td>30.5 ± 8.3</td>
<td>35.6 ± 8.6</td>
<td>27.3 ± 6.5</td>
<td>0.002</td>
</tr>
<tr>
<td><strong>Height (cms)</strong></td>
<td>179.98 ± 8.6</td>
<td>178.0 ± 10.6</td>
<td>181.3 ± 6.7</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Weight (cms)</strong></td>
<td>83.0 ± 13.2</td>
<td>87.3 ± 13.2</td>
<td>80.3 ± 12.7</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Blood Pressure (mm Hg)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>115.5 ± 8.5</td>
<td>120.2 ± 6.3</td>
<td>112.7 ± 8.5</td>
<td>NS</td>
</tr>
<tr>
<td>Diastolic</td>
<td>73.2 ± 8.2</td>
<td>80.1 ± 5.5</td>
<td>69.0 ± 6.6</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Serum Analysis</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ionized calcium (mmol/l)</td>
<td>1.28 ± 0.03</td>
<td>1.27 ± 0.04</td>
<td>1.29 ± 0.02</td>
<td>NS</td>
</tr>
<tr>
<td>Total calcium (mmol/l)</td>
<td>2.45 ± 0.08</td>
<td>2.39 ± 0.09</td>
<td>2.48 ± 0.06</td>
<td>0.001</td>
</tr>
<tr>
<td>Parathyroid hormone (ng/l)</td>
<td>390.2 ± 123.4</td>
<td>452.3 ± 130.6</td>
<td>352.4 ± 104.3</td>
<td>0.01</td>
</tr>
<tr>
<td><strong>Overnight urine</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calcium</td>
<td>0.24 ± 0.12</td>
<td>0.22 ± 0.11</td>
<td>0.25 ± 0.13</td>
<td>NS</td>
</tr>
<tr>
<td>Sodium</td>
<td>9.19 ± 3.46</td>
<td>9.58 ± 3.74</td>
<td>9.96 ± 3.35</td>
<td>NS</td>
</tr>
<tr>
<td>Potassium</td>
<td>2.28 ± 0.91</td>
<td>2.47 ± 0.84</td>
<td>2.26 ± 1.24</td>
<td>NS</td>
</tr>
</tbody>
</table>

* Responders vs Non-responders. NS = Not significant (Lyle et al. 1986).
of 1 g/day calcium in hypertensives. Grobber and Hofman (1986) also observed a lowering of diastolic blood pressure by 2.3 mm Hg on 12 weeks supplementation of 1g/day calcium in a section of their subjects with lower serum ionized calcium levels.

Studies by Resnick (1987) reveal that increased dietary calcium reverses the hypertensive effect of dietary sodium loading and salt sensitivity of the subject indicates the antihypertensive efficacy of calcium supplementation.

McCarron et al. (1991) suggest that there is a threshold of the potential protective effect of adequate calcium intake, below which the risk of hypertension increases at a greater rate. Calcium intake at or above this threshold, estimated at 700-800 mg/day could be of potential benefit to certain racial groups, individuals ingesting excessive alcohol and pregnant women, all of whom generally consume low amounts of calcium and who are at higher risk of developing hypertension.

Thus calcium supplementation appears to have a variable response on blood pressure reduction in both normotensives and hypertensives.
Role of Magnesium in Hypertension:

Magnesium salts were first shown in 1925 to lower blood pressure in hypertensive patients. The incidence of hypertension is high in geographic areas with soft drinking water or magnesium poor salt (Altura et al, 1984). However hard water is also rich in calcium salts and thus decrease in blood pressure cannot be attributed only to magnesium in areas of hard water (Hoechst International Service, 1990).

Dawson et al (1978) found that water calcium, magnesium, lithium, strontium and silicon may protect against cardiovascular mortality; possibly by competing with sodium and potassium for transport in the intestinal lumen, increasing excretion of sodium or by other mechanisms.

In a study by Dyckner and Wester (1983) patients receiving long term diuretic treatment were supplemented with magnesium as aspartate hydrochloride, 15 m mol/day for 6 months. A reduction of 12/8 mm Hg was observed at the end of the study with no significant changes in plasma or urinary electrolytes. These results were confirmed by Henderson et al (1986) in a similar experimental design. Though a reduction of 7 mm Hg diastolic pressure was
observed, the authors are of the opinion that magnesium supplementation does not exert a clinically important effect on blood pressure when given to hypertensive patients receiving long term diuretic treatment.

The effect of magnesium on blood pressure may be direct or through influences on the internal balances of potassium, sodium and calcium.

Increase in potassium concentration lowers blood pressure possibly through an increase in the activity of Na-K-ATPase and thus in the transport of electrolytes over the cell membrane. Na-K-ATPase is magnesium dependent (Hoschst International Service, 1990).

Magnesium is also a calcium antagonist. A decrease in magnesium leads to increased cellular calcium concentrations and vasoconstriction (Weinsier and Morris, 1985).

Serum magnesium levels were higher in patients with low renin hypertension and lower in patients with high renin hypertension than in those with normal renin hypertension or in normotensive controls. In contrast, serum levels of ionized calcium were lower in patients with
low-renin hypertension and higher in patients with high-renin hypertension (Resnick et al, 1983).

Diuretics have an effect on electrolyte metabolism. In the long term, thiazides may give rise to potassium and magnesium deficiencies and an increased cellular sodium content, all of which in turn results in increased cellular calcium content and possible vasoconstriction. The blood pressure lowering effects of thiazides have been estimated to be around 15/8 mm Hg in several studies on hypertensive patients. An additional reduction of 12/8 mm Hg was achieved with magnesium supplementation to patients already on diuretics (Dyckner and Wester, 1983).

Artificial lowering of the magnesium content in isolated coronary, cerebral and peripheral blood vessels from rats, rabbits, piglets and dogs as well as man induces rapid, contractile responses and potentiates the actions of a variety of neuro-humoral constrictor agents, including adrenergic amines and angiotensin. It has been suggested that some forms of hypertension could be due to the direct effects of a hypomagnesemic state on arteriolar and venular tone. The hypomagnesemia could produce progressive vasoconstriction of arterioles, pre-capillary
sphincters and venules in the microcirculation and this would eventually increase overall systemic vascular resistance, curtail capillary blood flow and result in hypertensive disease (Altura et al, 1984).

With so many other causes of hypertension, magnesium levels in certain circumstances may play a role in development of hypertension. However, further studies are required in order to shed light on this issue.

Diet Survey:

In recent years there has been renewed interest in the role of nutritional factors in the etiology of a variety of chronic diseases. The ability to firmly establish these relationships requires appropriate methodology for measuring the nutritional factors in question. Dietary assessment is central in the evaluation of nutritional status, at the individual or national level. Diet surveys either alone or as part of nutritional surveys provide the relevant information.

Methods of Dietary Survey:

The dietary surveys are classified according to
ICMR (1963) in the following manner.

Diet Surveys

Family or Institutional Survey
- Checking the stock by inventory method
- Weighing of raw food
- Oral questionnaire method

Individual Survey
- Checking the food list
- Weighing of cooked food before consumption
- Recording by using household measures

Family or Institutional Survey:

The unit of enquiry can either be a family or a residential institution depending on the specific objectives of the enquiry. Families are popular units particularly for the detailed study of dietary trends and inclination in any area. Various methods used for such family surveys are discussed (ICMR Special Reports, 1951 and Usha and Devadas, 1964).

a. Checking the Stock by Inventory:

Here the investigator takes an inventory of the food
existing in the house at the time of his visit by actual weighment. The housewife or the head of the family is required to make the necessary entries of all purchases of the food made till the end of the survey period. At the end, the investigator makes a final visit to the house and again takes an inventory of the food stuffs present in the house. During this period, the investigator makes two or more visits to the house to ensure that the entries are properly and regularly made.

b. Weighing of Raw Foods:

In this method the weights of raw foods in daily use are recorded prior to cooking. This method demands personal attention of the investigator to record any alterations or modifications occurring as a consequence of the subjectivity of the housewives.

c. Checking the Food List:

The foods available in the community and used in the household are listed during the preceding week of survey. The quantities of various foods consumed in the household during the period of survey are recorded as given by the housewife. The honesty of the housewife is an important criterion. McHenry (1963) found the method unreliable
though the method is rapid and economical.

Individual Survey:

Individual dietary surveys are difficult to make, time consuming for both subjects and investigators and costly. But from precision point of view, the survey of individual food intakes yields the best results. Various methods in these individual surveys are as follows:

a. Survey by Questionnaire and Interview:

The most commonly used method of diet survey is the oral questionnaire method, the investigator using this method goes from door to door and collects information on the kind and amount of foods consumed. The advantages of this method are that it is not a time consuming procedure and a large number of families can be covered in a short time. The method is suitable where quick information on the general dietary pattern in the group studied is desired.

The answer orally given by the subject may not however be accurate as many housewives do not have reliable estimates of say, what a kilogram is in terms of household measures. A more reliable practice is to get
the housewife to show how much she uses and either measure it or weigh it. Another approach to the problem is for the investigator to take a set of containers of assorted sizes and get the housewife to identify the measure corresponding to the amount she uses. It is desirable to repeat the survey after some interval to check the reliability of the data obtained. Where only a rapid impression of the dietary pattern prevailing in the locality is desired, a day's survey properly carried out should be enough (Rajyalakshmi, 1969).

The disadvantages of this method are that it relies on the responses made by the housewife. Rajyalakshmi (1969) found that prestige considerations affect the responses made, particularly in the middle and upper classes. She found that cereal intakes reported for individual members of the family do not add up to the total amount consumed by the family. Similarly items consumed which are of low prestige value such as kesari dhal and different varieties of animal foods like small fish, crabs etc., are not mentioned.

D. Weighing of Cooked Foods Before Consumption:

This method consists of determining the quantity of each food item taken for cooking in the kitchen and also
the weight of the cooked food. The cooked food left after consumption is also weighed. A simpler method is to issue known quantities of all foods used for cooking or consumption and request the housewife to help herself only from these stores for the day's cooking and to keep a record of any additional foods used. The amount left at the end of the day can be measured and the amount used determined. It is necessary to take into account any leftovers from the previous day. To simplify the procedure, grains, sugar, fats and legumes can be issued as described, whereas milk and vegetable intake can be recorded orally, as in most households, definite quantities of the latter are purchased for the day.

c. Recording by Using Household Measures:

When an individual is surveyed, he can give the information only in terms of foods consumed. A knowledge of the consumption of the foods prepared is therefore necessary. The same can be obtained in terms of number of servings and the amount of raw ingredients used in a known amount of food without weighing before and after cooking. Some of the factors which vary from household to household are size, as in the case of idli, chapatis etc., moisture as in the case of rice, dhal, vegetables,
milk, butter milk etc., and fat in the case of butter milk and milk. The investigator can make a reasonable guess about the composition of foods from their size and appearance and taste if he has first tried out different recipes of the same food.

Recall method: With this method (often called the 24 hour recall), the interviewee is asked to name the kind and amount of all food consumed during the preceding 24 hour period. The amounts are estimated in common household measures or servings. The interviewer helps the interviewee to determine the amounts by showing him food models and by providing him with measuring cup.

One significant advantage of the recall method is that it can be widely used. The period of recall is sufficiently short to accommodate most memories. The interviewer writes down the foods and the quantities so that the interviewees inability to read or write, will not exclude a person from the survey.

Analysis of Cooked Food:

This method involves the actual analysis of a composite sample of cooked foods consumed by the family or
members of an institution. All the items are mixed and mashed to a fine paste in a blender or grinding machine. Aliquots are used for the assay of vitamins, proteins, fat, calcium, phosphorus, iron etc. This method yields accurate data regarding the nutrient intakes, providing allowances for losses in cooking etc. But it is time consuming and costly to be carried out on a large scale.

Castenmiller et al (1985) calculated the daily food intake for each subject by analysis of seven day pooling of the daily food and by calculations from food composition tables and found that the results obtained were similar (Table No. 7).

Table No. 7
Mean daily food intake calculated from two different methods

<table>
<thead>
<tr>
<th>Nutrient (per MJ)</th>
<th>Calculated*</th>
<th>Analysed†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy intake (MJ)**</td>
<td>13.4</td>
<td>14.3 ± 0.1</td>
</tr>
<tr>
<td>Protein (g)</td>
<td>7.0</td>
<td>6.9 ± 0.1</td>
</tr>
<tr>
<td>Fat (g)</td>
<td>8.9</td>
<td>8.8 ± 0.2</td>
</tr>
<tr>
<td>Carbohydrate (g)</td>
<td>32.4</td>
<td>33.0 ± 0.3</td>
</tr>
<tr>
<td>Calcium (mmol)</td>
<td>3.2</td>
<td>2.9 ± 0.1</td>
</tr>
<tr>
<td>Phosphorus (mmol)</td>
<td>5.6</td>
<td>5.1 ± 0.2</td>
</tr>
<tr>
<td>Sodium (mmol)</td>
<td>8.4</td>
<td>7.6 ± 0.1</td>
</tr>
<tr>
<td>Potassium (mmol)</td>
<td>12.1</td>
<td>10.1 ± 0.2</td>
</tr>
</tbody>
</table>

* Calculated from food composition tables.
† Based on seven day pooling of the daily food intake for each subject.
** 1000 K cal = 4.2 MJ.
The literature on diet survey methodology is vast. Still clearcut and confident advice is not available to those who want to embark on a dietary survey.

Trulson and Mc Comm (1959) suggested that perhaps it could be assumed that an adult who has been living in approximately the same physical and social environment should consume about the same amount of food at two different questioning periods.

Swaminathan and Shantha Madhavan (1966), from data on food consumption collected by the seven day weightment method from 566 families belonging to the low socio-economic group in Coimbatore reported as follows, "It appears that in order to assess the dietary patterns of a population, even a random day dietary survey of a family would be sufficient, provided families were covered to represent all the days of the week. But there would always be the criticism against the one day survey, that the day chosen for a particular family may not be representative of its dietary pattern due to several factors. Therefore, since the three-day survey had been observed to give similar intake value as the seven day method and no additional gain was observed by extending the survey for more than three days, the three day weightment method of diet survey appears to be an equally efficient tool to use in an
economically poor population in which there would be very limited day to day variation in the consumption patterns."

Caggiula et al (1985) compared single vs multiple day food records for estimates of intake for sodium, potassium and calories, and the correspondence was assessed between sodium and potassium intake and 24 hour urinary excretion. The average intake for sodium, potassium and calories obtained from one-day food records proved to be as good an estimate of the six-day average as did values from multiple day records. Similarly, the one day food record proved a good estimate of the mean 24 hour urinary values for sodium and potassium. If properly collected and analysed, a one-day food record is a good estimate of a population's intake of sodium and potassium while multiple days of recording are necessary to characterize individual intake.

In a pilot study, Pietinen (1982) tested the usefulness of food consumption data in estimating sodium intake. A four day food record and three 24 hour urine samples were collected and analysed for sodium intake and excretion respectively. The results indicate that food consumption data collected in nutrition surveys could be used for estimating sodium intake.
A simplified inventory method for quantifying dietary sodium, potassium and energy was developed by Frank et al (1983). The Food Inventory reduced heterogeneity of foods consumed and simplified quantification. It was self-administered and consisted of 10 preselected foods categories, each containing 24 to 40 food items. The subjects had to choose only from the lists provided and enter the quantity consumed. This dietary assessment method can be useful in metabolic or behavioural studies requiring quantification of electrolytes and energy.

Food habits differ due to income of the people, education, size of the family and knowledge in nutrition. Diet surveys would be reliable only when the subjects are sincere, educated and are trained about the methods followed in the survey.

Nutritional correlates of blood pressure are of major concern to the nutritionists of the world. Weight reduction in obese individuals is highly recommended to bring down the blood pressure. A prescription of a low calorie, low fat, high fibre diet to such patients would bring down weight along with mild exercise. Also, switching over to a diet with high P/S ratio is being recommended as polyunsaturated fatty acids were observed to attenuate
hypertension. Inspect of the diverse views of scientists, a moderate reduction in sodium intake is still being advised. An increase in daily intake of potassium and calcium are definitely not deleterious and hence an attempt can be made to include potassium and calcium rich foods into the daily menu. With knowledge of the various risk factors of hypertension and with due care being taken to control each one of them, the prevalence of mild to moderate hypertension can be reduced considerably with only non-pharmacological treatments.