INTRODUCTION
Essential hypertension is a major public health problem of the century. It is a complicated disorder and heterogeneous in origin. Hypertension is also considered as a psychosomatic disease as various physical, psychological and visceral factors play an important role in its etiology (Shukla and Tripathi, 1984).

Normal adult blood pressure is arbitrarily defined as a systolic pressure equal to or below 140 mm Hg together with a diastolic (fifth Korotkoff Phase) equal to or below 90 mm Hg. Hypertension in adults is arbitrarily defined as systolic pressure equal to or greater than 160 mm Hg and/or diastolic (fifth phase) equal to or greater than 95 mm Hg. The term borderline hypertension is used to denote blood pressure values between the normal and hypertensive ranges (WHO, 1978).

Blood pressure is the force exerted by the blood pumped from the heart against the walls of the arteries. Or, it is the amount of force required to circulate blood round the body. In hypertensive disease, the pressure rise is ultimately achieved through increased tone in the vascular smooth muscles of the arterioles, thereby increasing
the vascular resistance to the forward flow of blood (Peart, 1967).

Patients with arterial hypertension and no definable cause are said to have primary, essential or idiopathic hypertension, secondary hypertension is defined as one with identifiable cause. It can also occur due to administration of certain drugs (WHO, 1978).

Essential hypertension is a global problem. The prevalence of essential hypertension was reported to be maximum in developed countries. North America has the highest incidence with a range of 12.6 to 33.5 per cent in different parts of United States of America and 23.5 per cent in Canada, whereas it was low in Mexico of the same continent with only 5.9 per cent (Intersalt Study, 1988). A prevalence of 24 per cent has been reported in Zimbabwe of the African continent. In the European countries, an average of 20 per cent in the United Kingdom, 18 per cent in Germany and Italy and 16 per cent in Spain have been recorded (Intersalt Study, 1988).

Studies in Asian countries reveal a lower percentage of prevalence, the incidence being 15.5 per cent in Russia, 13 per cent in people's Republic of China and only 10 per
cent in Japan (Intersalt Study, 1988). In India the prevalence of hypertension in adult population varies from 3 to 10 per cent (Wasir, 1989) and the average figure is 4.8 per cent (Sapru, 1984). In New Delhi the prevalence was 13.6 per cent and in Ladakh 10 per cent (Intersalt Study, 1988). Previously, Malhotra (1971) found blood pressure to be higher in South Indians (15.2%) when compared to North Indians (6.2 per cent).

Hypertension constitutes a syndrome of risks of which high blood pressure is but one part. Large scale epidemiologic studies have established that high blood pressures is associated with an increased risk of Cardio Vascular events (Australian Therapeutic Trial Management Committee, 1980; Hypertension detection and follow-up Programme Cooperative Group, 1982; Medical Research Council working party, 1985; The International Prospective Primary Prevention Study in Hypertension Collaborative Group, 1985). But clinical trials of antihypertensive therapy have shown an inconsistent reduction in major cardiovascular end points suggesting that factors beyond high blood pressure are important in the genesis of atherosclerotic disease in hypertensive patients. Patients with hypertension have an exaggerated vulnerability to the consequences of lipid abnormalities (Weber, 1989).
It has recently been established that hypertension is characterized by insulin resistance and altered glucose tolerance (Modan et al., 1985). As a result, high plasma concentration of insulin produces proliferative effects on vascular smooth muscles and connective tissue and may adversely affect the lipid profile (Weber et al., 1991). Altered cation transport is one of the several mechanisms by which insulin resistance might raise blood pressure. The \( \text{Na}^+ \text{K}^+-\text{ATPase} \) and \( \text{Ca}^{2+}-\text{ATPase} \) pumps are insulin sensitive. Thus, when insulin resistance is present, the activity of these pumps in the smooth muscle of the arterial wall might be reduced. This would lead to an intracellular accumulation of sodium and calcium, thereby sensitizing the vascular wall to pressor substances. Moreover, secondary hyperinsulinemia will occur and insulin has been shown to stimulate sympathetic nervous system activity and to increase renal tubular absorption of sodium (Flack and Sowers, 1991).

The left ventricle is also involved in hypertension independent of blood pressure. Cardiac adaptation to a prolonged increase in arterial pressure results in left ventricular hypertrophy (LVH). Data from the Framingham cohort and later studies have clearly documented that LVH is an important risk factor for the development of
subsequent cardiovascular morbidity and mortality (Messerli et al, 1989). There is growing evidence that the onset of the increases in the muscle mass of the left ventricle and changes in the diastolic filling characteristics are at the very early stages of hypertension (Weber et al, 1991).

Hypertension is characterized by structural changes in the arterial circulation. Hypertension and hyperplasia of arterial walls, linked to increased laying down of connective tissue elements are common findings. These changes are reflected by diminished arterial compliances and can be demonstrated prior to the appearance of clinical hypertension (Weber et al, 1991).

Liver function tests were abnormal in 15.8 per cent of men and 6.2 per cent of women in hypertensives attending the Glasgow blood pressure clinic. Liver dysfunction was related to alcohol consumption, heavy body weight, male sex, young age and higher diastolic blood pressure (Ramsay, 1977).

Stroke is a major complication of hypertension. Cerebral, cerebellar and brain stem haemorrhage are associated more with hypertension. Hypertensive encephalopathy
is often associated with an extreme elevation of arterial pressure and is characterized by variable disturbances of consciousness ranging from transient confusion to coma, often with convulsions. Severe headache, nausea, vomiting are common accompaniments (WHO, 1978).

Renal complications of hypertension include premature or accelerated atherosclerosis and nephrosclerosis which slowly produce renal impairment (WHO, 1978). Retinal haemorrhages, exudates and papilloedema are also common (WHO, 1978).

The availability of effective drugs has revolutionized the treatment of high blood pressure but each drug has unwanted side-effects which manifest in certain individuals. Diuretics cause hypokalaemia, beta blockers, though rare, cause wheezing, depression and sleep disturbances. Vasodilators like dihydralazine expose the patients to head aches and oedema. Drugs acting on central nervous system like methyldopa were observed to produce depression, and day time drowsiness and less commonly liver damage (O'Brienn and O'Malley, 1982; Baujon, 1989). Bendrofluazide and Propranolol were found to cause impotence (Medical Research Council Working Party, 1985) and calcium channel blockers cause oedema and provocation of congestive heart failure (Davidson, 1989).
Large and complex trials are not needed to show that treatment is beneficial in severe hypertension. But when the focus is switched to milder hypertension, much larger populations are exposed to life-long therapy. In the United States about 58 million people are hypertensive and about 35 million have blood pressure which would qualify them for drug treatment (O'Brien and O'Malley, 1988). Even in a developing country like India around 4.8 per cent or 15.8 million people (adults above 20 years) were diagnosed as hypertensives and 70 per cent or 11.06 millions of these are borderline hypertensive (Wasir and Mohan, 1985). The magnitude of the problem today might be much higher as these figures are based on 1981 census.

Thus, diagnosis of mild to moderate hypertension becomes very important before putting a patient on life-long drug therapy. Cheung et al (1989) found normal blood pressure in a sub-group of their subjects who were previously found hypertensive on whole day blood pressure monitoring.

In view of the life-long drug treatment and their deleterious side effects, Swales et al (1989) stress on repeated measurements of blood pressure over a prolonged period and recommend that borderline hypertensives should
be monitored without pharmacological treatment. Hence, non-pharmacologic measures to reduce blood pressure are very essential to patients with mild hypertension with no other cardio-vascular risk factors and no evidence of target organ damage.

Association of obesity with high blood pressure is well documented. Intersalt Study (1968) observed that Body Mass Index and alcohol intake were strongly, positively and independently associated with blood pressure. Regular exercise was recommended to reduce hypertension (Wilcox et al, 1982).

Similarly, significant interaction between smoking and cardiac events was reported (International Prospective Primary Prevention Study in Hypertension, 1985; Medical Research Council Working Party, 1985).

A considerable amount of direct evidence accumulated over the past 50 years has established that excessive intake of sodium can be associated with an increase in blood pressure (Kempner, 1944; Murphy, 1950; Cruz-Coke et al, 1964; Prior et al, 1968; Jobian, 1979; Mac Gregor, 1982; Dodson et al, 1989). Despite the established relation,
the disease continues to manifest itself in forms that are unresponsive to sodium manipulations. Alternative lines of inquiry into the role of dietary factors have led to the investigation of the role of potassium and calcium in regulation of blood pressure.

It was observed that potassium supplements decrease blood pressure (Parfrey et al., 1981; Mac Gregor et al., 1982; Khaw and Conner, 1984). Potassium excretion was negatively correlated with blood pressure and significant positive relation between urinary sodium to potassium ratio was noted (Intersalt Study, 1988).

The hypotensive mechanisms of potassium have been attributed to its diuretic and natriuretic action (Addison, 1928; Morino et al., 1978; Skrabal et al., 1981), altered activity of renin-angiotensin system (Ophir et al., 1983; Tannen, 1983), alteration of peripheral resistance (Chen, 1972), and through its effect on central or peripheral nervous system (Battarbee et al., 1979; Parfrey et al., 1981).

Calcium intake and serum calcium levels were found to be lower in hypertensives (Mc Carron, 1982). There appears to be a urinary calcium leak in hypertensives (Kasteloot and Geboers, 1982; Strazzula et al., 1983).
Calcium supplements were found to cause significant fall in blood pressure in a section of subjects (Resnick et al., 1984; McCarron et al., 1984), while Aalberts et al. (1988) found no such effect.

Calcium is necessary for many physiological functions in the body. It is essential for the function of two cardio-vascular events namely cardiac out-put and peripheral resistance. It is required for the contraction as well as relaxation of the cells (McCarron, 1985).

The hypotensive activity of calcium regulating hormones like parathyroid hormone is under investigation (Bukoski and Kramer, 1991). Calcitonin gene related peptide has also been found to be an extremely potent vasodilator (Nelson et al., 1988).

In view of the magnitude of the problem of hypertension, with over 0.504 million people being added to the list of hypertensives every year in India (Wasir and Mohan, 1985), the management of hypertension requires new dimensions specifically in the non-drug therapy. The present investigation is an endeavour in the same direction.
Information regarding the effects of potassium and calcium on blood pressure and their characteristic hypotensive nature on supplementation to hypertensive in the Indian environment is scanty. Hence this study was taken up with the following objectives.

1. To record blood pressure values in male subjects of 41 - 60 years of age.

2. To assess the relationship between blood pressure values and certain specific hypertensive risk factors including family history of hypertension.

3. To conduct a diet survey and estimate the nutrient intake of each subject.

4. To study the effect of intervention with Potassium and Calcium supplementation on the levels of Blood pressure, Sodium, Potassium and Calcium in serum and urine, and

5. To study the associations between certain biochemical parameters, Body Mass Index, Family history, dietary factors and Blood Pressure values in the sample.