INTRODUCTION
Hypertension is considered to be a disorder of multifactorial etiology and hyperlipoproteinemia is one of the constituent of this multifactorial etiology. Hypertension is a major risk factor for coronary, cerebral and renal vascular diseases, which cause over half of all the deaths in the western country. Hyperlipoproteinemias are the disturbances of lipid metabolism that result from accelerated synthesis, or retarded degradation of lipoprotein that transport cholesterol and triglycerides through plasma. The clinical importance of plasma lipoprotein is to cause two life threatening diseases - atherosclerosis and its related diseases and pancreatitis. Some hyperlipoproteinemias are the direct result of primary defect in the metabolism of lipoprotein particles. Other forms of lipoproteinemias are secondary, that is, the elevated plasma lipoprotein level occurs as a part of a constenation of abnormalities caused by an underlying disorder in a related metabolic system, such as thyroid hormone deficiency or insulin deficiency. Despite much emphasis being placed on the link between an elevated fasting lipoprotein levels and atherosclerosis, it has also been observed that a large number of normocholesterolemic subjects are equally affected by the atherosclerotic process. Thus it is evident that fasting lipid levels do not reflect the true risk of an individual.
Hypertension may enhance atherogenesis by directly producing injury via mechanical stress on the endothelial cells at the specific high pressure sites in the arterial tree. This would allow the sequence of events in the chronic injury hypothesis of atherogenesis to take place.

In addition, hypertension might allow more lipoprotein to be transported through the intact endothelial lining cells by altering the permeability. Hypertension markedly increases lysosomal enzyme activity presumably owing to stimulation of the cellular disposal system by the internalization of increased amount of plasma substances.

In the Framingham Cohort, the risk of developing coronary disease rose progressively with increasing systolic or diastolic pressure, both in the middle aged and the elderly. The greater awareness of the dangers of elevated blood pressure along with the availability of safer and more effective antihypertensive agent has led to a therapeutic explosion.

Since there is no dividing line between normal and high blood pressure, arbitrary levels has been established to define those who have an increased risk of developing or morbid event. Blood pressure varies widely throughout the day and night, whether the person usually has normal or unusually high levels. Since every one exhibits such swings, the designation, labile hypertension is neither useful nor meaningful.
The joint National Committee on Detection, Evaluation and Treatment of high blood pressure defines hypertension when systolic blood pressure $\geq 140$ mm Hg and D.B.P. $\geq 90$ mm Hg in adults aged 18 years or older. In adults, when D.B.P. below 85 mm Hg is considered to be normal between 85 and 89 is high normal, 90 to 104 is mild hypertension, 105 and 114 moderate hypertension and $\geq 115$ is severe hypertension. When the systolic blood pressure below 140 mm Hg, it is considered normal, between 140 and 159 mm Hg is border line isolated systolic hypertension, $\geq 160$ is the isolated systolic hypertension. Arterial blood pressure should be taking on the average of two or more readings on two or more occasions after patient has been supine for 5 minutes.

The incidence of hypertension among blacks is greater at every age beyond adolescence, and a given level of hypertension tends to induce more vascular damage in blacks than in whites.

Although our understanding of the pathophysiology of an elevated arterial pressure has increased in 90 to 95 percent of cases, the etiology is still largely unknown. As a consequence in the most of the cases, the hypertension is treated non specifically, resulting in a large number of minor side effects and a relative high (50 percent) non compliance rate.

Rudmick et al (1926) showed that in about 50% of cases, the causes of hypertension was obvious, known
as secondary hypertension, in about 95% of all hypertensive persons, there was no recognizable cause i.e. they have essential hypertension.

Individual risk, age, race, sex, smoking, serum cholesterol, glucose intolerance, weight, heredity may alter the prognosis of the disease.

The Framingham study and other epidemiological surveys more clearly defined certain risk factors for premature cardiovascular disease, in addition to hypertension. For varying levels of blood pressure, the Framingham's data show the increasing likelihood of a vascular complication over the next eight years for both men and women at various ages as more and more risk factors are added. Obviously the higher the overall risk, the more intensive the intervention should be.

An interesting and disturbing connection between untreated hypertension and hypercholesterolemia has been noted in multiple populations. This connection may be mediated through insulin resistance and obesity but also found in non-obese hypertensive. Clearly, through this association of hypertensives are often burdened with an even greater risk than imposed by their blood pressure alone.

In general vascular complication of hypertension can be considered on either hypertensive or atherosclerotic. The former are more directly caused by
the increased blood pressure per se and can be prevented by lowering this level, the latter have more multiple causations.

Genetic alteration may initiate the cascade to permanent hypertension. Clearly, heredity plays a role although no discriminatory gene markers are currently available.

Cigarette smoking raises blood pressure probably through the nicotine induced release of nor-epinephrine from adrenergic nerve endings.

Much is still uncertain about their exact relationship to the development of hypertension. However, an abnormal lipid level is common to several of these risk factors.

A reduction in plasma lipoprotein cholesterol level, achieved by diet and drugs reduce the risk of myocardial ischemia in subjects of hyperlipoproteinemia (Peto et al, 1985). So modification of diet has been shown to result in the progression or regression of atherosclerotic level in several experiments (John et al, 1972).

Considerable epidemiological data are now available which established a direct relationship between increased total serum cholesterol concentration and the development of atherosclerosis.
On the contrary, a strong inverse relationship has been established between HDL cholesterol and the risk of coronary artery disease (Gordon et al., 1981, Golbert et al., 1985). Diet plays a vital role in the causation of atherosclerosis. Transient post prandial rise of beta lipoprotein, VLDL, chylomicron, many cause repeated cholesterol deposition in cells in the arterial wall over the year, while fasting cholesterol value may remain within normal range over the same duration. Thus, it seems logical that post prandial response of an individual to high cholesterol fat load may more appropriately be related to the risk of developing atherosclerosis in future.

I has already been mentioned that various trial have confirmed an association between lipid levels and coronary and other cardiovascular disease. Here we are trying to study the behaviour of lipoprotein profile in the hypertensive individual and their first degree relatives and finding out the high risk person among the first degree relatives and subsequently the chances of developing the disease in future.

Importance of decreased level of high density lipoprotein and apo-lipoprotein A in the development of coronary and other cardiovascular atherosclerotic process has been repeatedly stressed (Gstein et al., 1977, Brady et al., 1978 and Miller et al., 1979).
Understanding of cholesterol turnover has been augmented after the identification of LDL receptors by Sir Goldstein. LDL receptors explain the high dynamic equilibrium of cholesterol molecules in the vascular compartment because radioactive cholesterol disappear within 20 minutes of its ingestion.

Most of the normal subject showed a fall in the STC and LDL level at one hour while among the first degree relatives of IHD, there was rise of STC and LDL levels at one hour (Arora et al, 1989).

The aim of this study is to evaluate the changes in lipoprotein profile induced after ingestion of single high cholesterol diet in hypertensive individual and their first degree relatives, and to find out the basal lipoprotein profiles in hypertensive patients and their first degree relatives.