INTRODUCTION
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Ischemic heart disease consists of major cause of mortality in present stress age and is a global problem involving both developing as well as developed countries (WHO, 1982; Hiroyasu et al, 1989; Gordon, 1977). The most important risk factor for ischemic heart disease are atherosclerosis and hyperlipidemia leading to deposition of lipids on the intima of arteries causing narrowing of blood vessels.

A direct relationship exists between STC and LDL levels and rate of CHD (Atherosclerosis study group, 1984, Grundy, 1986; Hulley Rhodes, 1982). Also the 26 years follow up of Framingham study has shown that the incidence of CHD increases as serum cholesterol value increases.

There is an increased risk of coronary heart disease (CHD) with a high serum total cholesterol concentration (Goldbourt, 1985, Grundy, 1986; Thomas, 1990), a high low density lipoprotein (LDL) level (Steinberg et al 1989; Brown et al 1986; Keys et al, 1972, Kannel et al, 1971) and a low high density lipoprotein (HDL) level. (castelli 1986 a; Goldbourt, 1985; Kannel et al, 1979).

Increased levels of triglycerides, total cholesterol, low density lipoprotein, very low density lipoprotein and decreased level of HDL-cholesterol are the major risk factors in causing atherosclerosis and ischemic heart disease (Bhatia, 1980).
Of particular significance is the evidence that certain plasma lipoprotein abnormalities are casually related to atherosclerosis and atherosclerotic heart disease and others are predictive of high risk of this disorder (Lewis, 1988). Elevation of serum cholesterol level or more specifically a LDL cholesterol level is widely accepted as a major risk factor for development of ischemic heart disease (Key, 1972; Kannel et al., 1971).

Various clinical and experimental studies of various kinds have firmly established that elevated plasma concentration of LDL are associated with accelerated atherogenesis (Tyroler, 1987; Goldstein et al., 1977; Steinberg, 1983; 1989).

Perhaps the most pertinent question is whether fasting cholesterol levels does really reflect an individuals risk for CHD as more than 40% of young patients of CHD do not reveal raised fasting cholesterol level (Gregory et al., 1983), yet they have rampant, atherogenous vascular involvement.

Diet plays a vital role in the causation of atherosclerosis and CHD. Modification of diet has led to progression or regression of atherosclerotic lesions in several experimental models (John et al., 1982).

Zilversmit (1973) postulated that atherogenesis may be a post prandial phenomenon. Transient rise of beta-VLDL chylomicrons and formation of several species of unusual lipoproteins may cause repeated cholesterol deposition in cells in arterial wall over the years, therefore, the post prandial response of an individual to high cholesterol fat load may be more appropriately related to his/her risk of developing atherosclerosis.
It is now stated that presence of LDL receptors and unidentified hormonal or neurogenic reflexes affecting these receptors could be responsible for bringing a dynamic equilibrium between blood and tissue cholesterol.

Medical Scientists are of the opinion that Antilipidemic, antidiabetic and antihypertensive drugs and other measures that can decrease catecholamine levels are considered to be remedy for myocardial infarction (Raab, 1971) It is now a well established fact that reduction in blood cholesterol level reduces the risk of myocardial ischemia. 25% reduction of blood cholesterol levels reduces the risk of myocardial ischemia by 50% (Lowering blood cholesterol 1985, Tyroler, 1987).

Vigorous global research is going on to search the agents to control hyperlipidemia. It is proved beyond doubt that chronic use of various hypolipidemic agents i.e. Gemfibrozil, alter the lipid-lipoprotein profile favourably (Frick et al, 1987; Helsinki heart study). But it is not yet established that these hypolipidemic drugs (Gemfibrozil) have any acute effect over lipid lipoprotein profile or not.

Further, work has not been done on effect of Gemfibrozil on postprandial behavior after high cholesterol fat diet knowledge of short term and acute effect of the Gemfibrozil on fasting and post-prandial lipid-lipoprotein profile may be a good indicator for screening the subjects to be benefited by the drug.

So we planned to study the acute effect of Gemfibrozil on fasting and post-prandial lipid-lipoprotein profile in healthy subjects.