INTRODUCTION
INTRODUCTION

Hyperlipidemia, hypertension and smoking are potent and modifyable risk factors in the causation of atherosclerosis and coronary heart disease (CHD). 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.

Perhaps the most pertinent question is whether fasting cholesterol (lipid levels) level does really reflect an individual's risk for CHD. How does one explain the fact that more than 40% of young patients of documented 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). Also CHD mortality in U.S.A. has decreased by 30% (from 1963 to 1983) due to decreased consumption of animal fats and cholesterol (National Centre for Health Statistics, Washington).
The individual response to high cholesterol fat diet varies enormously but remains constant for an individual over a long period of time (Kingsbury, 1960). 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 risk of developing atherosclerosis.

The correlation between post prandial responses of an individual and risk of atherogenesis has not been studied in details. Proper definition and correct interpretation of post prandial response is necessary in the formulation of cholesterol fat tolerance test. Such a test should be of immense use in identifying persons at risk of developing atherosclerosis and CHD.

The earlier misconception was that cholesterol is a slowly metabolised substance and that it cannot alter blood cholesterol level before 2 hours. That's why the previous efforts of several workers (Albrink and Man, 1956; Pomeranz, 1954) did not yield any useful results because they calculated the cholesterol and other lipid subfractions 2-6 hours after test load. 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.

A few studies in this direction have already been done in our department. Feeding high cholesterol fat breakfast for 7 days in young and old subjects resulted in increased level of STC, with rise of HDL in younger subjects and rise of LDL in older subjects (Arora et al, 1984; 1985). Since prolonged feeding is not practicable on a mass scale for screening purposes, a pilot study was conducted by Arora et al (1989) to study acute changes in serum lipid profile after high cholesterol test feeding. Majority of healthy subjects showed a fall in STC and LDL at 1 hour while diabetics first degree relatives of IHMD and minority of healthy population showed a rise in STC and LDL at 1 hour.

Studies by Arora et al (1990) showed similar trends when tests were done in diabetes, IHMD and hypertensive subjects.

These findings prompted us to assess the content of cholesterol fat tolerance test in healthy and disease subjects again.