Introduction
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Hyperlipidemia, Hypertension, and smoking are three potent and modifyable risk factors in the causation of atherosclerosis and coronary artery disease. A direct relationship exist between STC and LDL levels and rate of CHD. Even animal studies revealed the cholesterol feeding could induce atherogenesis in animals that would not normally get this disease. The establishment of serum cholesterol as a high risk factor for atherosclerosis further promoted the notion that high fat western diet may play a role in atherogenesis. Amount or type of fat consumed may have an impact on chances of developing heart disease. However it is still a mystery why some people who consume a lot of fat in their diet not develop CAD. Long going studies in our deptt. states that this may be due to functional state of LDL receptor which is corner stone in lipid metabolism.

Also the 26 years follow up of framingham study was shown that the incidence of CHD increases as serum cholesterol value increases.

Perhaps the most important question is whether fasting cholesterol (lipid levels) level does really reflect an individuals 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 has an important role in both management, prevention and progresion of coronary artery disease because substantial reductions in both stroke and CAD mortality rates have accrued over the last three decades and more importantly there reduction have accrued among men and women, among whites and Africans and in all age groups. Modification of diet has led to progression or regression of atherosclerotic lesions in several experimental modeles (John et al, 1982) Also CHD mortality in USA 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 IHD 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, IHD and hypertensive subjects.

These findings prompted us to assess the concept of cholesterol fat tolerance test in patients of Ischemic heart disease and hypertension.