INTRODUCTION
Atherogenesis results due to interaction of multiple factors viz. age, smoking, hypertension and hyperlipidemia. Diet also plays a vital role in its causation. Modification of diet has led to progression or regression of atherosclerotic lesions in several experimental models (John et al. 1982). However individual response to high cholesterol fat challenge varies enormously but remains constant for an individual over a long period of time (Kingsbury K.J. 1960).

Importance of basal fasting cholesterol level in calculating an individual risk for coronary artery disease has perhaps been over emphasised. Over more than forty percent of young patients of documented CAD do not reveal raised fasting cholesterol level (Gregory S. et al. 1983), yet they have rampant atherogenous vascular involvement. What is the mechanism of atherosclerosis in such cases?

Zilversmit in 1973 postulated that atherogenesis may be a post prandial phenomenon. Transient post prandial
rise of Beta VLDL, chylomicron and formation of several species of unusual lipoproteins, may cause repeated cholesterol deposition in cells in arterial wall over the years. While fasting cholesterol value may remain well 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 his risk of developing atherosclerosis in future. Little has been done in establishing a correlation between post prandial response of an individual and risk of atherogenesis. Even different post parandial responses after cholesterol fat load have not been studied in depth. Proper defination of these responses and correct interpretation may perhaps be the first major step towards formulation of cholesterol tolerance test.

Previous effort in this directions by several after workers (Albrink and Man 1956, Pomeranz, 1954) all over the world have borne little fruits, primarily because each of them calculated serum total
cholesterol and other lipid subfraction 2-6 hour after
test load, thinking that cholesterol is a slowly
absorbed substance and can not alter blood cholesterol
level before two hours.

The identification of LDL receptors by
Joseph Goldstein, and its role in control of serum
cholesterol metabolism, has changed whole of the
scenario. Cholesterol now no more remains such a
inert substance as thought before, in fact disappearance
of intravenous radio active cholesterol within 20
minutes of injection from the vascular compartment,
reflects its high dynamic state with the tissue
cholesterol. Perhaps this dynamic equilibrium is
achieved by the presence of LDL receptor and yet undefined
hormonal or neurogenic reflexes affecting those
receptors.

As already stated, there is enormous individual
variability in responses after cholesterol fat feeding.
Results of previous studies in our department have
further strengthen this view. Feeding high cholesterol
fat breakfast for seven days in young and old subjects
resulted in increased levels of serum total cholesterol,
with predominant rise of HDL in youngs and that
of LDL in older subjects (Arora R.C., Gupta
This study also brought out the fact, that in
some subjects, prolong cholesterol fat feeding
results in fall in STC level instead of rising.
Present knowledge offers no satisfactory explanation
for the above phenomenon.

Since prolong feeding is not practicable
on a mass scale for screening purpose, so we
decided to study changes in basal lipoprotein
profile after ingestion of single high cholesterol
load in normal and diseased human volunteers.

AIMS OF THE STUDY

1) To assess the changes in basal lipoprotein
profile after giving single high cholesterol
test load.

2) To correlate these changes for quantitative
and qualitative risk of an individual for
atherogenesis.