Discussion
DISCUSSION

One of the most startling facts, that has to be faced is that fasting cholesterol (lipid levels) do not sufficiently represent an individual's risk for future atherosclerosis and CAD. How does one explain the fact that more than 40% of young patients of documented CHD do not reveal raised fasting cholesterol (Gregory et al., 1983), yet they have rampant, atherogenous vascular involvement (Gregory et al., 1983).

The concept of estimation of diet induced changes in lipid lipoprotein profile and its relevance in predicting an individual's risk for future atherosclerosis still remains an unchartered terrain. The wide variability in assimilating a cholesterol fat test load is the main reason why till now no reasonable cholesterol tolerance test could be devised.

The manner in which an individual (healthy or diseased) responds to single high cholesterol fat diet (HCFD) load, could possibly help in determining his chances of developing atherogenic process. This has formed the basis of the present work.

This work has been conducted on healthy male volunteers (20-30 years of age), hypertensive, IHD (ischemic heart disease) patients and diabetic patients.
SERUM TOTAL CHOLESTEROL (STC)

The basal serum total cholesterol (STC) was 195.3±36.18, 199.0±29.40, 208.7±32.02 and 219.7±47.73 mg/dl in groups A, B, C and D respectively. While groups A and B had basal STC within normal limits set by lipid research clinics, IHD (Group C) and diabetics (Group D) had 10-20% more basal STC level than normal subjects of the same age. The highest basal STC was found in diabetics.

Various Indian studies support our findings of high basal STC in diabetics. (Mehrotra et al, 1989) found basal STC in 25 cases of diabetes as 233.6±26.6 mg/dl. Pant et al (1985) found 259.38±44.79 mg/dl.

On feeding single high cholesterol test load consisting of 800 mg of egg cholesterol, a fall in STC after 1 hour after was seen in majority of healthy subjects (Group A) and NIDDM subjects. In contrast the IDDM patients, patients of IHD (Group C) and hypertensive (Group B) subjects showed a distinct increase in STC after 1 hour. After 3 hours, the levels of STC started reaching fasting levels but they were below basal levels in group A and NIDDM while in IDDM, group B and Group C they were above fasting values.

Thus on the basis of post prandial response the healthy population can be divided in three groups. The majority of the population shows a fall in serum total cholesterol and LDL levels, 1 hour after feeding, while in the remaining there is no change. In contrast to
these results, majority of the subjects who were predisposed to the risk of CHD showed a rising trend of STC. Similarly all type I diabetics showed a rising trend of STC. In the past Nikkila et al (1962) and Havel (1957) have also reported fall in STC level after feeding. The explanation for this fall could be related to the suppression of LDL receptors after over night fasting (Medical Clinics of North America Vol 66, No. 2, March 1982 page 344). When fat cholesterol load is given LDL receptors are stimulated by as yet some undefined hormonal or neurogenic reflexes, in anticipation of the cholesterol load that will enter the circulation. Large amount of LDL from intra-vascular compartment shifts intracellularly, resulting in an acute fall in serum LDL and STC levels after 1 hour. The Cholesterol levels slowly increase after 3 hours as a result of the absorbed cholesterol and the reverse intravascular movement of LDL that had entered the tissues earlier.

In group B (hypertensives), IHD and diabetics patients the increase in STC and LDL at 1 hour can be explained by some inherent biochemical block in anticipating and assimilating the cholesterol load.

**HDL CHOLESTEROL**

The basal HDL levels were 42.6±7.74, 43.7±8.56, 38.7±7.66 and 41.09±7.40 mg/dl in group A, B, C and D respectively. These levels were all within normal limits
set by lipid research clinics. Group C subjects (IHD patients) had a distinctly lower level of HDL than other groups. The low level of serum HDL in IHD patients correlates with other Indian studies. Shashi Kumar (1988) and Wahal et al. (1985) reported 37.6±6.42 and 36.67±10.13 mg/dl respectively in their studies.

While healthy subjects and diabetics did not show any appreciable change in HDL at 1 hour and 3 hours. The hypertensive and IHD subjects showed a distinct fall in HDL at 1 hour and a subsequent fall at 3 hour.

**SERUM TRIGLYCERIDES (STG)**

The basal STG levels were 126.10±45.36, 109.08±
22.75, 101.50±24.17 and 155.54±35.60 mg/dl in groups A, B, C and D respectively. All groups have STG levels within the limits set by Lipid research clinics. Diabetics (group D had the highest level of basal STG. After feeding HCPD, healthy subjects and diabetics showed an increase in STG levels at both 1 and 3 hours which was significant. Hypertensive subjects showed a significant increase in STG at 1 hour but a fall at 3 hours. IHD patients showed very minimal changes at 1 and 3 hours.

The peak level of STG in our study was observed at the end of 3 hours (especially in healthy and diabetic subjects).
Barritt (1956), Brown et al (1961) and Agarwal (1964) reported peak STG levels 4 hours after feeding in healthy subjects.


**VLDL CHOLESTEROL**

Changes in VLDL reflected similar to those seen in STG.

**LDL CHOLESTEROL**

Basal LDL values were 127.48±30.07, 133.48±27.60, 151.10±32.70 and 146.52±50.92 mg/dl in groups A, B, C and D respectively. These values were on the higher side of the limits set by Lipid research clinics.

Basal LDL values were lowest in healthy subjects and highest in IHD subjects. While healthy and NIDDM patients showed a fall in LDL at 1 hour and 3 hours, IDDM, hypertensives and IHD patients showed a significant increase at 1 hour which did not return to basal levels at 3 hours.

The explanation for the observed phenomenon is the same as that for STC.