The study of lipid profile from autopsy hearts showed accumulation of triglyceride and cholesterol ester rich in erucic and docosahexanoic acid residues in different portions of atherosclerotic hearts. Presence of erucic acid in the human heart has suggested a definite habit of consumption of mustard oil by them. Erucic acid is not found or occurs in body fat. It only could be detected in body on consumption of oils containing it. In the present investigation, formation of atherosclerotic plaques was maximum in the left ventricular sites of atherosclerotic hearts. This could may be due to the effect of dietary oils. The left ventricular working capacity and coronary flow rate of rat heart are intimately related with dietary fat (de Deckere and ten Hoor, 1979).

Significantly elevated levels of phosphatidylcholine and sphingomyelin and decreased content of cardiolipin in different portions of atherosclerotic hearts indicated alteration in phospholipid profile. This may lead to alteration of the rigidity of biomembranes. Gas liquid chromatographic analysis of total phospholipid also showed significantly higher erucic acid residues in age groups of 31-40 and 41-50 years. Thus, it is reasonable to suggest that erucic acid could be responsible for fatty changes in human heart. It is true, induction of fatty changes, if not solely due to erucic acid, it could be an etiologic factor. Fibrotic changes as observed in human atherosclerotic hearts could be consequent to altered lipid profile,
mainly the phospholipids, since phospholipids along with cholesterol maintains the rigidity of biomembranes.

Histological findings indicated the fatty changes among the muscle fibres of atherosclerotic hearts do occur primarily. It could be followed by necrosis, histiocytic infiltration and finally fibrosis. It is quite relevant to mention that in the present feeding experiment with rats, fibrosis was observed only on prolonged feeding with mustard oil diet (vide chapter-1, part-B). In heart block patients significantly elevated levels of free fatty acid in sera was observed as compared to that of normal subjects. This was also observed by Bhattacharyya, et al (1977) in acute myocardial infarction.

Elevated levels of total cholesterol and triglyceride in heart block patients were observed. Erucic and behenic acid residues were present in lipid fractions of both normal and heart block patients. The content of erucic or behenic acid in sera of heart block patients was higher in the age groups of 46-60 and 61-75 years. Alteration in other fatty acid residues along with elevated erucic or behenic acid content of heart patients in comparison to normal subjects could be an etiologic factor for heart block syndrome.

Experimental studies with feeding of mustard oil to rats showed fatty accumulation in heart homogenate, mitochondria and serum. Changes in the cardiac phospholipids in boars (Kramer and Hulan, 1977a), cardiac lipid in chicks (Kramer and Hulan, 1977b) with different levels of rapeseed oil or erucic acid have been reported.
Accumulation of triglyceride in heart homogenate and mitochondria with very high incorporated erucic acid residue was observed in mustard oil group. In cholesterol ester fraction erucic acid was also incorporated with change in other fatty acids. This altered lipid profile could be responsible for fibrotic changes in the cardiac muscle of rats following feeding of mustard oil diet. Further gross change in phospholipid profile, in both heart homogenate and mitochondria, especially sphingomyelin and cardiolipin fractions were altered. In both the fraction preferential incorporation of erucic acid residue was observed, with two fold increase in mitochondrial sphingomyelin in 20% mustard oil group.

Impaired heart mitochondrial function with decreased production of ATP have been reported (Houtsmuller et al., 1970, Christopherson and Bremer, 1972a, Swarttouw, 1974, Clandinin, 1978, Renner et al., 1979) following feeding of diets containing erucic acids. In the present study, change in heart mitochondrial lipid and fatty acid profile of rats on mustard oil diet could explain the impaired mitochondrial capabilities for oxidation towards various substrates. It has been found that cardiolipin is tightly bound to cytochrome oxidase (Awasthi et al., 1971) which indicates the importance of this phospholipid as a structural component of the respiratory chain. The specific effect of erucic acid on the cardiolipin might indicate its specific inhibitory effect on the
mitochondrial fatty acid catabolism as well as on the mitochondrial respiration and energy supply to the heart. The low values of cardiolipin in the present study could be attributed to the damage of myocardial cell by erucic acid.

Serum analysis from rats on mustard oil diet showed decreased level of triglyceride and increased content of cholesterol ester in it. Low level of triglyceride is associated with reduction of very low density lipoprotein. Increase in low density lipoprotein was also observed. Lipoprotein analysis of patients with heart block syndrome revealed significant increase in lipoprotein types IIa, IIb, and IV. These changes in lipoprotein types are in conformity with the level of triglyceride, cholesterol and phospholipid in the serum. Further, considerable erucic acid residues was observed in these lipid classes. This cardiac malfunction could be reasonably related to the lipoprotein types.

Enzyme analysis from the sera of heart block patients and from rats on mustard oil diet showed significant elevation from those of the control groups. Thus, release of enzyme into serum could be an indirect effect of erucic acid induced damage to myocardium in both instances.