INTRODUCTION
Pregnancy is accompanied by profound hormonal alterations and these endocrinial changes during gestation have been proved to be the causative factor altering the lipid metabolism.

In early weeks of pregnancy the corpus luteum serves as a source of hormones soon the action is taken over by placenta which serves as the main endocrine organ bringing about continued and higher production of steroid and other hormones. Besides the anterior pituitary, adrenal cortex also have an important role to play in the extrapolation of hormones, which support pregnancy.

The oestrogen level during pregnancy increases progressively to reach maximum at term then values of oestrogen falls within 3 days and reaches at the basal level by 7th post partum day. Oestrogen causes increase in HDL cholesterol while LDL decreases. Biosynthesis of VLDL is enhanced while triglyceride lipase activity reduced. Progesterone brings about a decrease in HDL-c and increase in LDL-c, it reduces hepatic triglyceride lipase activity.

The hyperlipidemia of pregnancy is potentially significant from several stand points. (1) The rise in plasma triglyceride may enhance the availability of essential and non-essential triglyceride fatty acids for placental transfer to fetus. (2) The cholesterol rise
may increase the supply of cholesterol needed for placental progesterone synthesis and transplacental cholesterol transfer to the fetus. (3) The plasma triglyceride elevation may be the barometer of a general metabolic adaptation by the mother to augment nutrient flow to the fetus. (4) The hyperlipidemia may stress maternal lipid homeostasis to an extent that subclinical or mild hyperlipidemia becomes clinically detectable analogous to the prediabetes recognised in a women when she develops gestational diabetes. (5) The hyperlipidemia of pregnancy could itself function as an atherosclerosis risk factor.

The pattern of change in level of serum cholesterol and triglyceride during pregnancy is as follows. The serum cholesterol and triglyceride levels steadily increase starting from second trimester up to term and attain a peak just prior to the onset of labour and then abruptly falls with the expulsion of placenta, but not reaching the pregestational levels. In post partum period these fall gradually.

The abrupt fall in lipoprotein levels that occurs immediately after delivery also raises some queries. How this raised lipoprotein disappears all of sudden? Where does this lipoprotein go? Is it shifted to fetal circulation during labour? or is it shifted from maternal circulation to extravascular/subendothelial compartment?
Although studies are available for the antepartum and postpartum phase, not much has been done during intrapartum phase. Hence a study had to made regarding the changes in lipoprotein profile during actual process of labour. Thus in this study it was observed whether there is any relation of serum lipoprotein profile to the process of labour whether preterm, term pregnancy - induced or spontaneous; normal or abnormal, emergency or elective caesarean section.

AIMS AND OBJECTIVES

1. To study the lipoprotein profile changes during various stages of labour and within 24 hours of postpartum period.

2. To study the lipoprotein profile changes, in relation to mode of delivery e.g. vaginal delivery, induced vaginal delivery, elective or emergency caesarean section.

3. To study the variation in lipoprotein fractions in umbilical cord blood of newborn with respect to mode of delivery of mother.

4. To study the effect of parity on lipoprotein fractions during various stages of labour and postpartum period.

5. To study the role of diet (vegetarian Vs nonvegetarian) on lipoprotein fractions during intrapartum and postpartum phase of mother and umbilical cord blood of newborn baby.