REVIEW OF LITERATURE
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Lipids are a heterogeneous group of compound related either actually or potentially to fatty acids. They have the property of being relatively water insoluble and soluble in non-polar solvent such as ether, chloroform and benzene.

Lipids are important because of their high energy value, importance of fat soluble vitamins.

Bloor (1969) has classified lipids into simple, compound and derived lipids. Simple lipids are esters of fatty acid with various alcohols. They include fats (cholesterol and triglycerides) and waxes. Compound lipids are esters of fatty acid containing groups in addition to an alcohol and a fatty acid. They include phospholipid, cerebroside and other compound lipid. Derived lipids are substances derived from the above mentioned groups by hydrolysis. They include fatty acids (saturated and unsaturated), glycerols and steroids.

There are dramatic changes in many biochemical, physiological and anatomic function which take place as a result of birth and continue during the first hours, days and even weeks of extrauterine life. Common to all changes are the needs for satisfactory energy supply and lipid used as main energy substrate.
Cross and co-worker (1957) found the respiratory quotient (R.Q.) of the newborn infants at birth to be 1.0, which began to fall shortly after birth and that it fell to a level close to 0.7 by the third day of life.

\[
R.Q. = \frac{\text{Rate of CO}_2 \text{ output}}{\text{Rate of O}_2 \text{ uptake}}
\]

\[
C_6 H_{12} O_6 + 6 O_2 \rightarrow 6CO_2 + 6H_2O = 6/6 = 1.0
\]

\[
C_{57} H_{104} + 80 O_2 \rightarrow 57 CO_2 + 57 H_2O = 57/80 = 0.71
\] (Triolein)

Triolein is the most abundant fat in the body. A fall of this magnitude in R.Q. is usually acceptable evidence of a change from the utilisation of carbohydrate to the utilisation of fat as a source of calories. Although the R.Q. of the newborn must be cautiously interpreted (James, 1964) fat metabolism appears to be an important means of obtaining energy in the fasting newborn (Smith, 1959). Furthermore, a progressive rise in blood ketone occurs within a few hours after birth and reaches a peak in 2 to 3 days indicating increased oxidation of free fatty acid.

**CHOLESTEROL**

Cholesterol is a complex monohydric secondary alcohol, stable white crystalline substance, insoluble in water but readily soluble in non-polar solvents. Chemically
the basic structure consist 27 carbon atom ring referred to as cyclopentenoperhydrophenanthrene ring. It is widely distributed in all cells of body. The total cholesterol content of the body is about 200 mg/kg of body weight (Bell et al., 1972). Two third of cholesterol in blood is found in esterified form, while one third is present as free cholesterol (Fredrickson et al., 1967).

**Cholesterol Metabolism:**

Most of tissue primarily of skin and intestine have the capacity to synthesize cholesterol (Sracek et al., 1950). The rate of cholesterol synthesis is high in liver, intestine and skin and accounts for as much as 90% of total body cholesterol production, while it is low in other body tissue.

Dietary cholesterol is absorbed in intestine in company with other lipids and is incorporated in chylomicrons and very low density lipoprotein (VLDL). 80-90% absorbed cholesterol undergoes esterification, which may take place in intestinal mucosa. Chylomicron deliver thin cholesterol to liver from where it is transported to plasma in form of VLDL which ultimately changes to low density lipoprotein (LDL) (Conner, 1979).
PLASMA TRIGLYCERIDES AND CHOLESTEROL TRANSPORT:

Ultimately LDL is taken up and broken down by extrahepatic parenchymal tissues and cholesterol is delivered to cells and cellular cholesterol synthesis is influenced by the lipid composition of the medium. The main enzyme in this process is "3 hydroxy 3 methyl glutaryl coenzyme-A-reductase" which can be induced by cholesterol deprivation.

EXCRETION:

It occurs mainly by two pathways. One being conversion to bile acid and its excretion while the other being excretion of neutral sterol in faeces. Liver is the main organ for cholesterol disposal. Before elimination, cholesterol must enter in liver and be excreted in bile either as cholesterol or cholic acid. Loss through urine is negligible.

TRIGLYCERIDES:

Triglycerides are esters of fatty acids and glycerol. Human adipose tissue consists chiefly of triacylglycerols regardless of anatomical location. Triglycerides form the largest portion of dietary fat.

Metabolism:- Triglyceride metabolism is said to have two pathways namely exogenous and endogenous pathway.
Exogenous pathway of triglyceride metabolism (Fredrickson et al, 1967):

In adults 1.2 gm/kg of triglycerides are ingested daily (Henry, 1977). From the intestinal mucosa, they are largely absorbed as chylomicrons and reach the blood through thoracic duct. These chylomicrons are taken to adipose tissue and skeletal muscles and then triglycerides are hydrolysed there to release free fatty acids and monoglycerides. Free fatty acids are either re-esterified and stored in the adipose tissue and skeletal muscle or oxidised (Havel, 1961).

Endogenous pathway of triglyceride metabolism:

Endogenous hyperlipidemia perhaps first become a distinctly recognizable phenomenon, when Watkin et al (1950) noted that fat free high carbohydrate diet increased the triglyceride concentration.

Liver is considered as the major site for endogenous triglyceride metabolism (Havel, 1962 and Goldstein, 1961). An important source of fatty acids needed for triglyceride synthesis is the plasma free fatty acid. The flux of the free fatty acids into the liver, heart and skeletal muscles, is governed by their rate of release from adipose tissue. Any factor that increases lipolysis or decreases glycerol esterification in the adipose tissue causes out-pouring of free fatty acid (Steinberg and Vaughan, 1965). Much of
these fatty acid removed by liver and excess beyond what it can use or store is resynthesized into glyceride and resecreted as VLDL (Havel, 1957). VLDL is the chief triglyceride binding lipoprotein present in the plasma. VLDL particles carry 5 - 10 times triglycerides than cholesterol esters.

**Lipoproteins:**

These are globulin particles of high molecular weight and transport non-polar lipid & cholesterol and triglycerides) through plasma. Each lipoprotein particles contains a non-polar core, comprising of triglycerides and cholesterol in varying proportions. Surrounding the core is a polar surface coat of phospholipids that stabilize the lipoprotein particles, so that can remain insoluble in the plasma. Surface coat consist of phospholipids, esterified cholesterol and apoprotein. The proportion of these lipids and proteins however, differ greatly resulting in differences in physiochemical properties which permit their separation (Chait, A, 1978).

Boydd, Nobbe and Schettler (1967) have classified lipoproteins into 4 major classes that are normally present in plasma.

The lipoprotein classes differ in composition of non-polar lipids, in the core, in the composition of
apoproteins and in density, in size and electrophoretic motility. The five classes of lipoproteins are -

1. Chylomicron,
2. VLDL (Very low density lipoproteins),
3. IDL (Intermediate density lipoprotein),
4. LDL (Low density lipoprotein),
5. HDL (High density lipoprotein).

**Apolipoprotein** - These are part of lipoprotein molecule as protein. The apolipoprotein have three general functions -

- They provide structural element to lipoprotein particles and thus are important in maintaining stability.
- Act as ligands for specific receptors,
- Act as activation or inhibition of specific enzyme involved in lipoprotein metabolism.

**Metabolism in Fetus** :-

Before birth, fetus utilizes carbohydrate as the major fuel for energy production. This correlates well with the observation that the respiratory quotient (R.Q.) in fetus and birth is one (James, 1972). In utero transfer of glucose occur through placenta. Free fatty acids have been shown to cross placenta but there is little or no transfer from mother to fetus of cholesterol, triglycerides
or phospholipids (Forfar and Arneil, 1984). The synthesis of lipids in fetus proceeds from glucose and fatty acid precursors in early stage of gestation and lipid content in fetus increase to 300 fold from first month to nine months of gestation (Roux et al, 1974). After birth with cutting off of the nutrients from maternal circulation and before milk feeding is started the newborn has to depend on its own endogenous source of nutrients for survival. As the carbohydrate stores of body are meagre and protein metabolism can account for only a fraction of the total energy requirements, body lipids become a major source for energy for newly born infants. Increased mobilisation of lipid from the stores and increased lipolysis in the immediate post-natal period lead to a rise, the levels of total lipids, cholesterol phospholipids and free fatty acids (Brown et al, 1939; Van Duyne, 1959; Persson, 1956 and Christensen, 1974). The mechanism for the oxidation of fatty acid rapidly increase in activity after birth (Forfar and Arneil, 1984).

However, information on this aspect of lipid metabolism is meagre in the case of premature, term and stressed newborn, as is evident from a brief review of the proceeding so far on this aspect.

It was some 82 years ago, when cord blood lipid measured for the first time by Hirmann and Neumann (1912) when they performed a study on 30 normal delivery and
recorded the maternal and cord cholesterol levels with aim to find out the two value. They observed that the cord blood cholesterol (62 mg/dl) values are considerably lower in comparison to their maternal counterpart (mean value 264 mg/dl).

Gyorgy (1924) observed 6 cases and recorded the cord blood cholesterol and lipid phosphorous value and compared them with maternal serum counterparts. He chose 6 normal full term healthy deliveries which formed his study group. He observed that the mean values of serum cholesterol in cord and maternal blood were 69 mg/dl and 255 mg/dl respectively. These results showed that the value observed of cord blood cholesterol were comparable with those observed earlier and at the same time were less in comparison to material serum values. This finding also supported those by earlier workers. The serum lipid phosphorous values in cord blood were less in comparison to maternal serum lipid phosphorous value.

Gordon and Cohn (1928) conducted a study on 16 full term normally delivered cases to record their cord blood cholesterol levels and lipid phosphorous levels. They observed that the mean cord cholesterol value were 89 mg/dl which were slightly higher than those recorded by earlier workers, while the mean cord lipid phosphorous value were 4.1 mg/dl which were comparable to those observed by other workers. Here the aim was to establish
the normal value and no correlation with maternal values was considered.

Sperry (1936) observed cholesterol value in cord blood of 7 neonates and found that mean values were 61 mg/dl which were in close approximation to those obtained earlier.

Sodowsky et al (1947) performed a study on 14 neonates who were delivered normally and observed the cord and maternal cholesterol values. In their study they found the mean cord blood cholesterol value to be 107 mg/dl which was higher than those observed by earlier workers, while the maternal mean blood cholesterol value were 262 mg/dl which were comparable to earlier obtained value.

Rofstedt et al (1954) studied 82 neonates to observe the cord blood cholesterol and lipid phosphorous values. The mean cord cholesterol value was observed to be 67 mg/dl which was consistent with other observation, while the mean lipid phosphorous value were 4.8 mg/dl which also was in close proximity to those obtained by other workers. In their study Rofstedt et al reported that cord plasma lipid concentration were the same in low birth weight babies as compared to those of normal birth weight but this data was limited to cholesterol and total lipid concentration and the low birth weight babies were not sub-divided into pre-term and small for date groups.
Russ et al (1954) have determined lipoprotein, cholesterol and phospholipid content in mother and their newborn infants. They recorded mean cord blood cholesterol values as 68 mg/dl while the mean maternal values were 282 mg/dl. These values correlated well with those observed in the past.

Brown et al (1959) performed a study on 50 neonates and their mothers to determine normal values of various serum components including proteins, lipoprotein and lipids at birth and their relation with corresponding maternal values. The maternal venous blood sample were collected during 1st stage of labour and cord blood samples were collected just after birth. The mother chosen underwent normal full term labour. The protein levels in maternal blood were 6.32 ± 0.52 gm%, 2.27 ± 0.33 gm% for total and albumin respectively and were 6.13 ± 0.64 gm% and 3.05 ± 0.45 gm% respectively in the cord blood samples for the same. The maternal values were 110.4 ± 17.2 mg%, 257 ± 71 mg%, 847 ± 176 mg% and 273 ± 52 mg% for total lipids, lipoprotein lipids, Beta lipoprotein lipids and cholesterol respectively, while they were 371 ± 75 mg%, 147 ± 40 mg%, 224 ± 41 mg% and 82 ± 17 mg% respectively for the same in the cord blood samples. These findings agreed closely with those of Rofstede et al (1954) and Russ et al (1954).

Desai et al (1977) studied and analysed 113 full term newborns, delivered by normal labour following
uncomplicated pregnancy. The mean birth weight was 2800 gm with a range of 2500 to 3600 gm. The cord cholesterol values ranged between 35 - 128 mg/dl and phospholipids between 58 - 160 mg/dl, while the mean values were 79 ± 17 mg/dl, 62 ± 21 mg/dl and 95 ± 17 mg/dl respectively.

The levels of cholesterol in this study were in agreement with previous recording while the levels of triglyceride observed in this study was higher than those obtained earlier by western authors.

Cress et al (1977) observed 275 neonates for cord blood cholesterol and triglyceride levels and noted cord blood cholesterol level as 70 ± 17 mg/dl with a range of 30 ± 153 mg/dl. The 95th percentile value was 105 mg/dl. Mean cord blood triglyceride level were 33 ± 16 mg/dl with a range of 5 - 192 mg/dl. The 95th percentile value for cord blood triglyceride was 77 mg/dl. They observed that out of 22 neonates whose cord blood lipid values exceeded the 95th percentile values, 9 had hypercholesterolemia and 13 had hypertriglyceridemia. Four neonates had elevated values both cholesterol and triglyceride range 103 - 120 mg/dl and 68 - 117 mg/dl respectively.

None of the 275 neonatal sera had demonstrable amount of IgA antibody, indicating that there was no maternal contribution to the cord blood samples.
A study was carried out on 57 neonates for cord blood lipid profile by Sharma et al (1983). They divided the 57 cases into 3 groups on the basis of gestational age and birth weight. The first group included normal term appropriate for gestational age newborns (31), the second group include full term small for gestational age infants (12) and third group included pre-term appropriate for gestational age infants (14). The classification was done according to Lubschenco et al (1963). The total lipid, cholesterol and free fatty acids in cord blood were estimated.

The cord cholesterol levels in normal full term, small for date and pre-term was 74 ± 17 mg/dl, 64.6 ± 12.3 mg/dl and 64 ± 13 mg/dl respectively while the cord blood phospholipid value were 112 ± 36.3 mg/dl, 101.6 ± 30 mg/dl and 130 ± 11 mg/dl respectively in above mentioned groups. The free fatty acid levels in cord blood were 0.38 ± 0.03 m mol/l, 0.29 ± 0.06 m mol/l and 0.26 ± 0.06 m mol/l in normal full term, small for date and pre-term respectively.

The levels of various lipid fraction in cord blood were seen to be lower in small for gestational age group (second group) and pre-term group (third group) as compared to healthy full term neonates (first group) and the difference was statistically significant only with free fatty acid levels (P < 0.001) and attributed this to lower fat store in small for gestational age infants in comparison to full term healthy
neonates. The lower values in pre-term infants were attributed probably to a possible quantitative or qualitative deficiency of the enzyme lipoprotein lipase (LPL) which was responsible for release of free fatty acid from neutral fats (triglycerides). The lower level of enzyme have also been reported by Sigmura et al (1974) and Prakash et al (1980).

Haridas and Acharya (1984) conducted a study on 180 newborns and their mothers, who belonged to low socio-economic strata. This study consisted of determination of cholesterol and triglyceride value in cord blood and serum on 2nd, 3rd, 4th and 5th day in normal full term, pre-term and low birth weight babies along with maternal blood values.

The mean cord blood triglyceride values were $45 \pm 13.8$ mg/dl (range 20 - 89 mg/dl), $59 \pm 22.3$ mg/dl (range 24 - 119 mg/dl) and $56 \pm 16.1$ mg/dl (range 22 - 95 mg/dl) in normal full term, pre-term infants and small for date babies respectively. The triglyceride values were $157 \pm 48.9$ mg/dl (range 65 - 317 mg/dl), $58.5 \pm 26.2$ mg/dl (range 87 - 190 mg/dl) and $114 \pm 41.2$ mg/dl (range 71-343 mg/dl) respectively in mothers of above mentioned group infants.

The mean cholesterol values were $90 \pm 17.7$ mg/dl (range 55 - 125 mg/dl), $95 \pm 20.2$ mg/dl (range 42-130 mg/dl) and $91 \pm 20.2$ mg/dl (range 42 - 122 mg/dl) in cord blood of normal full term, pre-term and small for date infants, while
they were \(230 \pm 34.9\) mg/dl (range 120 - 330 mg/dl), \(228 \pm 37.6\) mg/dl (range 143 - 292 mg/dl) and \(233 \pm 32.8\) mg/dl (range 167 - 320 mg/dl) respectively in the mothers of the above mentioned infant groups.

The mean triglyceride values were \(65 \pm 13.8\) mg/dl and \(89 \pm 17.1\) mg/dl in normal full term infants on 2nd/3rd and 4th & 5th day respectively. The levels were \(74 \pm 15.3\) mg/dl and \(93 \pm 16.4\) mg/dl in small for date babies on the above mentioned day respectively. The cholesterol levels were \(124 \pm 9.6\) mg/dl, \(156 \pm 9.6\) mg/dl in normal full term, \(119 \pm 11.6\) mg/dl and \(143 \pm 8.7\) mg/dl in pre-term and \(122 \pm 7.2\) mg/dl and \(143 \pm 7.6\) mg/dl in small for date babies on 2nd/3rd and 4th/5th day respectively.

The neonatal cholesterol and triglyceride values were lower than maternal counterparts. The cord blood cholesterol levels were not significantly different in the three groups but by the 4th/5th day, normal full term babies exhibited higher values. The low birth weight and pre-term infants had higher triglyceride value in cord blood than normal full term. The levels continue to be higher in low birth weight babies and pre-term infants but the difference were statistically insignificant.

The difference in cord and maternal lipid values revealed lack of maternal contribution to fetal lipids. The authors stated that low birth weight infants were born
with intrauterine malnutrition which favoured adipose tissue breakdown and liberation of free fatty acids. The portion of free fatty acid escaping oxidation for energy production were synthesized into triglyceride in the cord blood of low birth weight babies. The raised triglyceride levels in post-natal period was due to utilisation of adipose tissue for energy requirements as glucose was conserved for energy requirements of brain and erythrocytes. This liberated free fatty acids which lead to triglyceride synthesis and raised triglyceride levels. The post-natal elevation in cholesterol levels was due to its enhanced synthesis as a result of increased enzyme and substrates required for cholesterol biosynthesis.

Mathur et al (1986) have done a study on 56 newborn for cord cholesterol values. These neonates delivered to healthy mothers, their gestational age was determined by Dobowitz's criteria. In this study out of 56 neonates, 14 were pre-term and 42 were term babies. The cord blood was collected from the placental end just after delivery. They observed mean cord blood cholesterol values to be as 105.27 ± 17.14 mg/dl with a range of 70 - 135 mg/dl. The mean cord blood values in pre-term babies were 92.57 ± 14.94 mg/dl as compared to 112.2 ± 14.58 mg/dl in term babies. In 18 babies weighing less than 2.5 kg the mean value were 93.67 ± 14.64 mg/dl while in 38 neonates weighing more than 2.5 kg the mean value were 110.76 ± 15.46 mg/dl. A positive
correlation was found between birth weight and cord blood cholesterol levels in this study.

Lakhtakia et al (1990) performed a study on 100 neonates to detect the effect of familial hypertension on the cord blood cholesterol and triglyceride levels. They selected 50 cases where there was family history of essential hypertension (in mother, grand parents or in other siblings) and 50 control subjects who were full term delivered neonates, after normal labours, without any adverse feto-maternal factors, born in a family with no history of ischaemic heart disease, hypertension or diabetes mellitus.

The mean ± S.D. cholesterol values in cord blood of neonates with family history of hypertension with the involvement of parents, grand parents and siblings of parents were 123.24 ± 22.32 mg/dl, 93.7 ± 16.96 mg/dl and 88.0 ± 11.95 mg/dl respectively. The cord triglyceride levels in the above mentioned groups were 58.16 ± 17.52 mg/dl, 33.94 ± 16.7 mg/dl and 30.0 ± 13.16 mg/dl respectively.

The mean serum cholesterol levels in the study and control groups were 108.92 ± 26.25 mg/dl and 86.84 ± 26.62 mg/dl respectively. The mean triglyceride levels in the above said group were 45.52 ± 20.89 mg/dl and 28.72 ± 72.81 mg/dl respectively. The differences in the study and control groups regarding the cholesterol and triglyceride levels were highly significant (P < 0.001).
Jagdish Singh et al (1994) conducted a study on pre-term and term newborn to find out the serum cholesterol values in their cord blood. It is also suggested that hypercholesterolemia can be diagnosed at birth by estimation of total cholesterol or low density lipoproteins in umbilical cord blood. The present study was undertaken to determine the normal value of umbilical cord blood cholesterol in the local population and correlation with gestational age, birth weight and sex of the baby was established.

One hundred newborn delivered to healthy mothers with no family history of coronary artery disease, hypertension and diabetes mellitus. The gestational age was determined using the criteria laid down by Dubowitz et al. Cord blood samples were collected from the placental end of cord just after the delivery of baby and cholesterol was estimated.

Of total 100 newborn, term and pre-term were 78 and 22 respectively. 77 babies weighing > 2.5 kg and 23 were of ≤ 2.5 kg. Boys and girls were 56 and 44% respectively.

The mean cord blood cholesterol (± S.D.) level was 90.4 ± 18.2 mg/dl with a range of 51.4 - 126 mg/dl. In term babies the mean level was 96.2 ± 15.1 mg/dl as compared to 69.5 ± 10.9 mg/dl in pre-term (P < 0.001). The mean levels were 96.1 ± 15.0 mg/dl and 66.8 ± 16.0 mg/dl in babies weighing > 2.5 kg and ≤ 2.5 kg respectively (P < 0.001).
Serum cholesterol value shows significant positive correlation with gestational age and birth weight. The mean cord level of cholesterol in boys was $88.7 \pm 19.1$ and in girls was $91.1 \pm 17.2$ mg/dl ($p < 0.05$).

A significant positive correlation was also found between cord blood cholesterol levels and birth weight, the same has been observed by Mathur et al. Sex of baby did not influence the cholesterol values as has been previously observed.

The cholesterol level at birth averages 1.5 m mol/L (60 mg/dl), within a month the average has rises to about 3.0 m mol/L (120 mg/dl) and by the end of first year to 4.5 m mol/L (175 mg/dl), a second rise begins in the IIIrd decade and continue to about age 50 in men and some what later in women (Harrison's, Vol: I, Thirteenth edition, Principles of Internal Medicine, 1994 : 1111). The normal values of serum cholesterol in umbilical cord blood is 45 - 98 mg/dl and serum triglycerides 10 - 98 mg/dl. The serum cholesterol value in full term average is 85 with a range of 45 - 167 mg/dl and infants (3 days - one year) average is 130 mg/dl with a range of 69 - 174 mg/dl (Cloherty Stark, Manual of Neonatology).

LIPID PROFILE IN UMBILICAL CORD BLOOD:

Numerous investigators have shown that in maternal blood the concentration of cholesterol and phospholipids is
greater than normal while in blood from umbilical cord at
time of birth is notably reduced.

Ortega, Gasper et al (1966) in their study on
influence of maternal serum lipids and maternal diet during
the third trimester of pregnancy and umbilical cord blood
lipids in two populations of Spanish newborns noted following
observations. A significant correlation was found to exist
between maternal cholesterol concentration and those of
newborn infants. A correlation was also found between
maternal cholesterol levels and infant HDL–c and LDL–c levels.
Further, a positive correlation was seen between maternal
LDL–c and infant cholesterol and LDL–c. The relationship
between maternal cholesterol and cord blood cholesterol was
independent of participant's dietary anthropometric and
personal data. 3.1% of neonates showed total cord blood
cholesterol concentration of 799.9 mg/dl. The mothers of
these children showed the strongest concentration of
cholesterol and LDL–c in the third trimester of pregnancy,
the shortest pregnancies and the smallest newborns of all
subjects. Negative correlations were found between birth
weight and cord blood cholesterol levels and LDL–c.

Keary and Kilby et al (1994) in their study on
foetal and maternal lipoprotein metabolism in human pregnancy
determined concentration and composition of lipid and apo-
lipoprotein in maternal venous and umbilical arterial and
venous blood. The objective of the study were to establish
whether the placenta has a role in foeto-maternal cholesterol metabolism through either synthesis or transplacental cholesterol flux. Study showed that pregnant women had raised levels of all lipid and lipoprotein fractions as compared with control subjects. In both umbilical venous and arterial blood concentration VLDL, LDL cholesterol ester and triacylglycerols were lower than in maternal blood, but HDL-c levels were similar. There was no umbilical-arterio-venous differences in lipoprotein concentration or composition. This suggests that cholesterol synthesis or free cholesterol diffusions does not occur in the placenta.

Pontis et al (1979) studied antepartum and post-partum lipoprotein levels in parturiting women and in umbilical cord blood of their newborns. The average values reported in umbilical cord blood were far low than that of maternal blood. The differences that exists between mother and baby in this respect varies from case to case and values prevailing in one seems to be entirely independent of those in other i.e. concentration of cholesterol is never same in mother and foetus. The differences have no constant or characteristic pattern.

Arora and Kavita et al (1989) in their study of the changes of lipid lipoprotein profile in normal pregnancy and toxemia of pregnancy during antepartum and post-partum periods and in umbilical cord blood of their newborns, found that STC, HDL-c, STG, VLDL and LDL-c levels in umbilical
cord blood were found to be very low in comparison to intrapartum values of the mothers. No difference in the levels of STC was found in newborns of normal pregnancy or toxemia of pregnancy. HDL-c levels were higher in umbilical cord blood of newborns of normal pregnancy as compared to toxemia of pregnancy while converse was true with STG. However, differences in the levels of HDL and STG in normal pregnancy and toxemia of pregnancy were found to be statistically significant.

Boyd and Wilson (1934) studied exchange of lipid in the umbilical circulation at birth. They took samples of venous blood from the maternal end of the cord with the placenta still attached in utero. This represented the venous blood entering the foetus. The contraction of uterus was assumed to have little effect upon the lipid content of venous blood, an assumption which was substantiated in part by the finding of similar results in cases of caesarean section in which the uterus was not contracted. It was later found that lipid concentration of venous blood slowly increases after the cord is clamped. They concluded that phospholipids, free cholesterol and cholesteryl esters are added to umbilical blood between the time of delivery and the time of placental separation. Neutral fats may be either removed or added to umbilical blood by the placenta.

Konttinen et al (1964) studied serum lipids in normal pregnancy and pre-eclampsia and also umbilical cord blood of both groups. They concluded that cord samples of
both the groups showed low levels of all the lipids studied and no differences were detectable between the two groups. The mean total cholesterol was about 80 mg/dl with a high content carried in alpha fractions. The serum triglycerides values were only about \( \frac{1}{8} \)th of the values seen in their mothers, with no individual correlation between mother and child.

A.V. Karan et al (1996) studied the 67 newborns were investigated. The umbilical cord blood samples were used to determine following indices: Total cholesterol, LDL, HDL-2, HDL-3-cholesterol, total phospholipids, LDL, HDL-phospholipids, triglycerides and apolipoprotein-B. The Chi square test indicated correlation between increased level of triglycerides, phospholipids, LDL and cholesterol HDL-2 and decreased level of cholesterol HDL in the group of newborns with the lowest delivery body mass.