In pregnancy there is re-adjustment of hormones on the part of the mother, almost every endocrine tissue participates in the adaptive changes, that maintains the metabolic state of female during normal pregnancy.

The occurrence of hyperlipidemia during normal pregnancy was known as early as the 1845 (Baqueral and Rodier). Milky appearance of sera of pregnant woman, was due to presence of fat, was showed by Virchow (1847).

The first chemical study was undertaken in 1911 when Chaufford demonstrated an increase of blood cholesterol during normal pregnancy. At the same time Hermann and Neumann studied the lipid particles in whole blood and reported an increase in cholesterol during pregnancy. They concluded that during first 6-7 months, the serum cholesterol might be increased and that during the last two months the increase in serum cholesterol was a rule.

Eminent investigators (Boyd, 1934; Dieckmann and Wegner, 1934; Schwartz et al, 1940; Peters et al, 1947; Russ et al, 1954; and Jacina et al, 1961) observed in their works that normal status is changed and there was an increase in serum cholesterol phospholipids and neutral fat which progressed towards term and decreased after delivery.

Tyler and Underhill (1925) determined the whole blood cholesterol in normal pregnant woman, in each month
of pregnancy and reported that cholesterol increases gradually until term, when it is roughly one third higher than at three months.

LABOUR

Series of events take place in the genital organs in an effort to expel the viable products of conception out of the womb through the vagina into outer world is called labour. Parturition is the process of giving birth. Delivery is the expulsion or extraction of a viable fetus out of the womb. It is not synonymous with labour; delivery can take place without labour as in elective caesarean section. Delivery may be vaginal either spontaneous or aided or it may be abdominal.

Normal Labour

Labour is called normal if it fulfills the following criteria:
1. Spontaneous in onset and at term.
2. With vertex presentation.
3. Without undue prolongation.
4. Natural termination with minimal aids.
5. Without having any complications affecting the health of mother and/or the baby.

Stages of Labour

Conventionally, events of labour are divided into three stages as below:
**First stage**

It starts from the onset of true labour pain and ends with full dilatation of the cervix. It is, in other words, the 'cervical stage' of labour. Its average duration is 12 hours in primigravida and 6 hours in multigravida.

**Second stage**

It starts from the full dilatation of the cervix and ends with expulsion of the foetus from the birth canal. Its average duration is 2 hours in primigravida and 30 minutes in multigravida.

**Third stage**

It begins after expulsion of foetus and ends with expulsion of the placenta and membrane (after births). Its average duration is about 15 minutes in both primigravida and multigravida. The duration is, however, reduced to 5 minutes in the active management.

**Fourth stage**

It is a stage of observation for at least one hour after expulsion after birth. During this period, general condition of the patients and the behaviour of the uterus are to be carefully watched.

**MECHANISM OF ONSET OF LABOUR**

The precise mechanism of initiation of labour is still obscure. Advancement of chemico-hormonal technology and inferences obtained from animal experiment...
put forth the following hypotheses.

1. **Uterine Distension**

   Stretching effect on the myometrium by the growing size of the foetus and liquor amnii can be explain the onset of labour at least in twins or hydroamnios. However, optimal distension theory fails to account for the otherwise causeless preterm labour.

   **Possible mechanism of initiation of labour**

   ![Diagram of possible mechanism of initiation of labour]

<table>
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<tr>
<th>X-factor</th>
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<tr>
<td>Activation of foetal hypothalmo-pituitary - adrenal axis</td>
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<tr>
<td>Cortisol</td>
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<td>Maternal post-pituitary</td>
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<td>Altered balance</td>
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<tr>
<td>Placenta, amnios Chorion decidua</td>
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<td>PGt² , PGF²-alpha</td>
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   **Activation of myometrium contractile system**

2. **Foetoplacental Contribution**

   It has been postulated that due to unknown factors foetal pituitary is stimulated prior to onset of
labour ---- | release of P.C.T.H. ---- Stimulates foetal
adrenals ------ | cortisol secretion ---- accelerated
production of oestrogen and prostaglandins from the
placenta. The probable modes of action of oestrogen are:
1. Increases the release of oxytocin from maternal
pituitary.
2. Promotes the synthesis of receptors for oxytocin
in the myometrium and decidua.
3. Accelerates lysosomal disintegration inside the deci-
dual cells thus increased prostaglandin synthesis.
4. Stimulates the synthesis of myometrial contractile
protein actomyosin through activation of adenosine
triphosphatase.
5. Increases the excitability of the myometrial cell
membranes.

3. **Progesterone**

Increased foetal production of dehydro epiandro-
sterone sulphate (DHEAS) and cortisol may inhibit the
conversion of foetal preg henabine to progesterone, there-
by altering the oestrogen-progesterone ratio. It is
probably the alteration in the oestrogen : progesterone
ratio rather than the fall in the absolute concentration
of progesterone which is linked with prostaglandin
synthesis.

4. **Prostaglandins**

Prostaglandins have attracted much attention in
recent years as a factor to initiate and maintain labour.
The major sites of synthesis of prostaglandins are placenta, membranes, decidua cells and myometrium. Synthesis is triggered by rise in oestrogen level altered oestrogen – progesterone balance, mechanical stretching in late pregnancy, increase in oxytocin receptors, infection, separation or rupture of membranes.

The prostaglandin synthesis reaches a peak during the birth of placenta probably contributing to its expulsion and to the control of postpartum haemorrhage.

5. **Oxytocin**

There is no conclusive proof that oxytocin level is increased prior to labour, there is, however, increase in oxytocin receptors especially in decidua vera which in turn stimulates prostaglandin synthesis. Oxytocin level reaches the maximum at the moment of birth and is caused by a reflex release due to vaginal distension.

6. **Nervous Factor**

Although labour may start in denervated uterus, labour may also be initiated through nerve pathways. Both alpha and beta receptors are present in the myometrium and beta receptors to function predominantly. The contractile response is initiated through the alpha receptors of the post ganglionic nerve fibres in and around the cervix and lower part of uterus. This based on the observations of the onset of labour following stripping or low rupture of the membranes.
7. **Cortisol**

The pituitary adrenal axis of the fetus gives the signal for the initiation of labour. Increase in cortisol levels occurs which raises oestrogen levels. High cortisol levels have been observed in patients of spontaneous labour as compared to caesarean section. These are also increased in foetal and maternal stress during active labour. Cortisol might compete with progesterone receptors in myometrium by binding with the progesterone carrier protein.

An encephaly, postmaturity and adrenal hypoplasia is associated with prolonged gestation due to reduced cortisol levels due to reduced foetal adrenal function.

**Lipoprotein Profile**

The lipids are a heterogenous group of compounds related, either actually, to the fatty acids. They have the common property of being insoluble in water and soluble in non polar solvents. Lipids are important dietary constituents not only because of their high energy value but also because of the fat soluble vitamins and the essential fatty acids contained in the fat of natural foods.

The following classification of lipids is modified from Bloor.

A. Simple lipids: Esters of fatty acids with various alcohols.
   1. Fat: Esters of fatty acids with glycerol, a fat in lipid state, is called oil.
2. Waxes: Esters of fatty acids with higher molecular weight monohydric alcohols.

B. Complex lipids: Esters of fatty acids containing groups in addition to an alcohol and fatty acids.
1. Phospholipids: Lipids containing, in addition to fatty acids and an alcohol, a phosphoric acid residue, they have nitrogen containing bases and other substituents.
2. Glycolipids: Lipids containing a fatty acid, sphingosine and carbohydrates.
3. Other complex lipids – Lipids such as sulpholipids and aminolipids. Lipoproteins may also placed in this category.
4. Precursor and derived lipids – these includes hormones and vitamins.

Cholesterol

It is probably best known steroid because of its association with atherosclerosis. Also this is the precursor of large number of steroids i.e. bile acids, adrenal hormones, sex hormone and Vitamin D etc.

It is widely distributed in all cells of the body. It is the major constituents of the plasma membrane and plasma lipoprotein. It is typically product of animal metabolism and so occurs in food of animal origin e.g. egg, yolk, liver meat etc. The normal level of serum cholesterol ranges from 150-250 mg/dl.
Triglycerides

It forms main bulk of dietary lipids. It is neutral fat. Normal values ranges from 80-240 mg/dl.

Lipoproteins

Lipids are present in plasma in the form of lipoproteins mainly which serve the two important functions - first is transport of triglycerol and another is the transport of cholesterol and its esters. The protein part of lipoprotein is called apoprotein. The lipid content of lipoprotein decreases the density of lipoproteins due to difference in nature and molecular weight of lipid and protein portion of several lipoproteins give them different densities and permit their further separation is centrifuge.

Thus, on the basis of different densities, lipoproteins can be of following types:

a. Chylomicrons

These are formed only by the lymphatic system draining the intestine. The levels fluctuates with the load of triacylglycerol absorbed. They are cleared rapidly from the circulation within one hour.

b. VLDL or Prebeta lipoprotein

These consist mainly of glycerides that are endogenous. It is formed by the hepatic parenchymal cells. Its formation is constant and even occurs in fasting state. It acts as a vehicle of transport of triglycerol from liver to the extrahepatic tissues.
Half life of VLDL is 2 hours and virtually all of the VLDL is converted into LDL metabolically. The level of VLDL between 20-29 years is about 25 mg/dl and between 30-39 years it is 35 mg/dl.

c. Beta-lipoprotein: Low density lipoprotein (LDL)

It is mainly formed by breakdown of VLDL in circulation. They are divided in LDL₁ and LDL₂ types depending upon densities. Cholesterol concentration in plasma is a reflection of the LDL concentration.

Half life of LDL is about 4 days, range of LDL level in circulation is between 170-190 mg/dl.

d. High Density Lipoprotein (HDL)

Also called alpha lipoproteins. These are much smaller than other lipoprotein. These are separated between densities of 1.063 - 1.210 and contain about 50% protein. It helps in destruction of IDL and LDL. It is formed in liver mainly and to a lesser extent also in intestinal epithelium. HDL has a protective role in coronary artery disease. High level of HDL in blood indicates less chances of atherosclerosis.

Normal range of HDL in blood is 75 mg/dl in age group of 20-29 years and 80 mg/dl in the age group of 30 to 39 years.

Plase and Tompkins (1923) have also given figures for the blood and lipids particularly cholesterol during pregnancy. These figures indicate a gradual rise from 4th
month to term.

Gardner and Gainsborough (1929) studied the ratio of cholesterol to cholesterol esters and reported an increase in cholesterol with a decrease in cholesterol ester till the 30th week of pregnancy. In their series reversal of the relationship occurs so that at parturition approximately a normal relationship exists again.

Kaufmann and Mihlbäck (1933) did not notice these fluctuations but they reported little variations from the second month of gestation to term.

According to Dieckman and Weigner (1934) total cholesterol increases to 23% above the first trimester levels and which decreased to 27% at the 8th postpartum week from the values at term.

On the basis of study of primigravida, Oliver and Boyd (1935) stated that there is significant rise in plasma ester and total cholesterol between 31st and 33rd week of pregnancy. By the 20th week of postpartum period values have decreased but still higher than the levels at 12th week of pregnancy.

After a serial estimation of plasma esterified fatty acids on puerperal patients following delivery and through fifth postpartum day, Dannenberg et al (1962) concluded that mean puerperal values for total carbosyl esters were higher than the nonpregnant class. This was statistically significant.
All fractions of esterified fatty acids except cholesterol esters showed a slight increase within 24 hours of delivery and then declined subsequently. These observations were similar with study of Boyd (1935) and Schwarz (1940) but according to Peters (1951), Watson (1957), there is a drop in plasma lipids following delivery.

Two phase study to define and detect changes in total lipids and their fractions during various periods of gestation was done by Mullick and Bagga (1964), according to this study all lipid fractions show a gradual and persistent rise throughout pregnancy.

Bhattacharya (1969) on the basis of their study, concluded that although cholesterol levels were slightly higher in toxaemia group. The cholesterol metabolism seemed to be similar in normal and toxaemia of pregnancy.

Potter and Nistel (1979) studied lipoprotein profile during pregnancy and puerperium in a group of 43 women. The plasma cholesterol concentration was raised by about 50% and major rise was in second trimester. The plasma triglyceride concentration was raised 3 fold and peak was found during third trimester.

Sita Devi and Patrudu et al (1981) longitudinal study of serum lipoprotein in normal pregnancy and puerperium, estimated lipid profile in 23 pregnant women in all three trimesters and in post natal period. Thirty three normal healthy nonpregnant women of the same age group fromed the control. A progressive rise was observed in serum triglyceride and cholesterol reaching the peak in the
3rd trimester and definite fall in post natal period as compared to antenatal period among hyperlipemias, the major one was type IV in the 1st trimester, type IIb and type IV in 2nd trimester and type IIb in 3rd trimester, and type IV in the post natal period. It was also noted that those who had an abnormal lipid profile in the 1st trimester continued to have abnormal lipid profile in the post natal period.

Darmandy and Postle (1982) measured serum lipid concentration in a group of normal women from before conception to throughout gestation and until at least 40 weeks after delivery. The effect of lactation was also studied. The primary change in lipoprotein metabolism during pregnancy, appears to concerned with VLDL which are elevated, the rate of secretion depending upon the lipoprotein lipase activity. After delivery the elevated serum triglyceride decreases rapidly and significant utilisation of serum triglyceride in lactating women could be caused by the tissue specific direction of VLDL towards the mammary gland for milk synthesis.

Fahraeus Lars et al (1985) studied levels of plasma lipoprotein fractions in 19 women at exact gestational ages. The HDL levels elevated in the 14th week and showed a maximum rise of 41% in 28th week of pregnancy. The LDL decreased in early pregnancy but then increased continuously. The VLDL concentration showed an increase from 14 to 36 week. During lactational period, 8 weeks after delivery the LDL concentration remained elevated whereas
the other lipoprotein had returned to pregnancy level.

Erkkola and Viikari et al (1986) investigated serum lipid and lipoprotein fractions one day after delivery, 3 months later in lactating and non-lactating mothers and 12 months later after initiation of menstruation in a group of 62 women. 29 of which formed a truly longitudinal group. STC decreased significantly during the following 9 months. LDL & HDL showed also a significant decrease within the postpartum period. Serum triglycerides decreased with 3 months after delivery but no more significantly lower. In lactating mothers HDL:STC ratio was higher than in non-lactating women. During luteal phase STC and LDL were lower and HDL:STC ratio was higher than earlier during the menstrual cycle. Pregnancy related changes in lipid metabolism did not wane within 3 months after delivery.

Arora and Vinita (1987), in their study, showed STC level of 166.17±49.97 mg/dl in 1st trimester while reached to a peak of of 263.44±39.8 mg/dl during labour. This decreased in the post partum period reaching 190.5± 36.94 mg/dl at one month of post partum period.

Herrera et al (1988) did their work on mechanism involved in maternal hypertriglyceridemia in late normal pregnancy and the physiologic significance reviewed as a model of the effects of sex hormones steroids in lipoprotein metabolism. They concluded that changes in magnitude and even direction of lipoprotein lipase activity in different tissues during gestation contribute not only to be metabolic fate of circulating triglycerides. These
dynamic and metabolic adaptations seen in the mother may directly modify her lipoprotein profile. Under pathological conditions the alterations may be permanently maintained thereby increasing the risk for the development of cardiovascular disease.

Valimaki et al (1990) have studied the serum lipid and lipoproteins in a alcoholic women during pregnancy, and concluded that alcohol abuse clearly reduced the normal increase in total cholesterol and LDL cholesterol during 24 to 40 weeks. HDL-3 was raised and normal increase in VLDL was accentuated in these women.

Arora and Neeta et al (1993) studied the changes in lipoprotein profile in normal pregnancy and artificial termination of pregnancy (Elective/emergency LSCS). They observed that there was rising trend in lipoprotein profile with a peak during labour followed by fall in the post partum period, both in normal as well as artificial termination of pregnancy.

Mazurkiewicz, Walts et al (1994) in new study on serum lipids, lipoproteins and apolipoproteins in pregnant non-diabetic women reported that pregnant women had significant higher concentration of STG, STC, HDL, LDL and apolipoproteins A & B, also that the ratio of STC: HDL, cholesterol was not significantly different.

Udoh and Ham et al (1994) in their study on normal pregnant women observed a progressive increase in the serum total cholesterol and HDL-c throughout pregnancy and decrease in their levels after delivery. In their study on 49 normal
pregnant women STC rose from 154.91±15.39 mg/dl at after one month of delivery. This represents a 39±11 percent increase in STC at 9 months from the 3 months levels. HDL-c showed an increase of 35±10 percent at 9 months level from 3 months level. The most significant months to months increase was recorded between the 6th and 7th month for both STC and HDL-c.

Chaing and Yang et al (1995) in their study of lipid profile in normal pregnancy found that serum total cholesterol, triglycerides, LDL-c were significantly elevated during second and third trimester of pregnancy but dropped sharply after delivery.

LIPID PROFILE IN UMBILICAL CORD BLOOD

So many studies have shown that in maternal blood the concentration of cholesterol and phospholipids is greater than normal while in umbilical cord blood at the time of birth is significantly reduced.

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 cord with the placenta still attached in utero. This represented the venous blood entering the fetus. The concentration of uterus was assumed to have little effect upon the lipid content of venous blood, on assumption which was substantiated in past 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 cholesterol esters are added to umbilical blood between the time of delivery and the time of placenta separation. Neutral fats may be either removed or added to umbilical blood by the placenta.

Sa dowsky et al (1947) observed the cord and maternal cholesterol values in babies delivered normally. Mean cord blood cholesterol values were 107 mg/dl which were higher than observed by earlier workers while the maternal mean blood cholesterol values were 262 mg/dl which were comparable to values obtained earlier.

Brown et al (1959) studied maternal serum during 1st stage of labour and cord blood samples collected just after birth. The mother had normal full term delivery. The maternal values were $1104 \pm 172$ mg/dl, $257 \pm 71$ mg/dl, $847 \pm 176$ mg/dl and $273 \pm 52$ mg/dl for total lipids, lipoprotein lipids, beta lipoprotein and cholesterol respectively. The values in cord blood samples were $371 \pm 75$ mg/dl, $147 \pm 40$ mg/dl, $224 \pm 41$ mg/dl and $82 \pm 17$ mg/dl respectively.

Brady and Carlon (1962) found that the concentration of serum triglycerides is quite low in the cord blood and newborn. This has been confirmed by the Kaplan and Lee (1965) who had the same observations.

Konttinen et al (1964) studied serum lipids in normal pregnancy and pre-eclampsia and also umbilical cord
blood of groups. They concluded that cord blood samples of both the groups showed low levels of all the lipids studied and no difference was 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 1/8th of the values seen in their mothers with no individual correlation between mother and child.

Ortega, Gasper et al (1966) in their study on influence of maternal serum lipids and maternal diet during the third trimester of pregnancy and mubilical cord blood lipids in two population of Spanish newborns noted that a significant correlation was found to exist between maternal cholesterol concentration and those of newborn infant. 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 participants' dietary anthropometric and personal data. 3.1% of neonates showed total cord blood cholesterol concentration of 799.9 mg/dl. The mother of these children showed the strongest concentration of cholesterol and LDL-c in the 3rd 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 at LDL-c.
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 below than that of maternal blood. The difference 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 these in other i.e. concentration of cholesterol is never same in mother and foetus. The difference has no constant or characteristic pattern.

Arora and Kavita et al (1989) in their study of changes in lipoprotein profile in normal pregnancy and toxæmia of pregnancy during antepartum and post partum periods and in umbilical cord blood of their newborns, found that STC, HDLC, STG, VLDL and LDL values of cord blood were very low in comparison to intrapartum values of mothers. Difference in the levels of HDL and STG in normal pregnancy and toxæmia of pregnancy were found to be statistically significant.

Heary and Kilby et al (1994) in their study on foetal and maternal lipoprotein metabolism in human pregnancy determined the concentration and composition of lipid and apolipoprotein in maternal venous and umbilical arterial and venous blood. The objective of the study was to establish whether the placenta has a role in feto-maternal cholesterol metabolism through either synthesis or transplacental cholesterol flux. Study showed that pregnant women had raised
levels of all lipids and lipoprotein fractions as compared with control subjects in both umbilical venous and arterial blood content. Ratio of VLDL, LDL, cholesterol esters and triglycerides were lower than in maternal blood, but HDL-c levels were similar. There was no umbilical arterio-venous difference in lipoprotein concentration or composition. They suggest that cholesterol synthesis or free cholesterol diffusion do not occur in the placenta.

EFFECT OF DIET ON SERUM LIPOPROTEIN PROFILE

A study on cholesterol metabolism was done in 1929 by Gardner and Gainsbrough and concluded that during period of fasting, cholesterol content of plasma varies markedly in different healthy persons but is fairly constant in subjects. A single meal does not cause any changes but prolonged diets high or low in sterol will cause variation in cholesterol, the free cholesterol remains fairly constant but cholesterol esters show greatest change.

According to Mullick and Bagga (1964) in healthy females serum lipids and its fractions vary with the nutritional status which is itself dependent upon the socioeconomic conditions of individual. Values for high income groups are close to those reported by Boyd. In pregnant females the increase in total serum lipids occurring in first 8 weeks of first trimester was more marked in vegetarians than in the nonvegetarians. STC, ester cholesterol and free cholesterol showed the reverse trend. In the 2nd trimester, this difference in the serum total lipids was
narrowed. In 3rd trimester, there was no difference in serum total lipids between vegetarians and non-vegetarians, but there was now a slight increase for the non-vegetarians. Thus diet has no significant influence in lipid synthesis in the later period of pregnancy.

Geen Gun (1966) determined total cholesterol serially in a group of young women before and during pregnancy while they consumed their usual diet or a fat modified diet known to have a hypocholesterolemic effect. During 1st trimester of pregnancy there was a slight but definite fall in serum cholesterol level. After the 1st trimester serum cholesterol level increases gradually to peak at or near term. These changes occur both in normal and hypercholesterolemic females and is not affected by fat modified diets.

Hansen and coworkers studied 80 pregnant women and found no significant correlation between mother's intake of calories, proteins, fat and fatty acids to serum cholesterol and fatty acids levels during 3rd trimester.

Arora and Vinita (1987) studied the influence of dietary fat on STC levels during antepartum, intrapartum and post partum periods of pregnancy and in the cord blood of newborns, and concluded that the levels of STC were higher in subjects taking high fat diet and lower in those taking low and normal fat diet with advancement of pregnancy during labour, after delivery and in late postpartum period values were not significant. However, the cord blood STC values in relation to fat diet of mother in 3rd trimester were highly significant.
HORMONAL CHANGES DURING PREGNANCY, LABOUR AND POSTPARTUM PERIOD AND ROLE OF HORMONES IN LIPROPROTEIN PROFILE CHANGES

The endocrine plays very important role in physiology of reproduction, i.e., following conception transfer of function of pituitary - ovarian axis to placenta which acts temporarily as a new endocrine organ. During pregnancy, there is physiological alteration of endocrine glands, namely the pituitary, thyroid, parathyroid, adrenal and pancreas to maintain the conception, to promote growth and to control labour. Hormonal influence during puerperium is necessary for maintenance of lactation.

HORMONES OF PLACENTA:

At 6 - 8 weeks there is transfer of function of corpus luteum to the placenta which acts temporarily as a new endocrine organ. It produces variety of hormones of which steroids and protein hormones are important
a. Steroid Hormones:
   i. Oestrogens
   ii. Progesterone
b. Protein hormones:
   - Human chorionic gonadotrophin (HCG)
   - Human placental lactogen (HPL).
1. **OESTROGENS**

Oestriol is the main pregnancy oestrogen accounting for 80-90% of the total oestrogen formed. The oestrogen levels during pregnancy increases progressively to reach a level of approximately 150 mg/ml at term (Ronald K. Kalkhoff et al., 1978), a level almost 16 times higher than the values at 8 weeks (Ddesoye et al., 1986).

Oestrogen levels fall significantly within 3 days and reach a basal level by 7th postpartum day, rising again in nonlactating by the 14th day.

Estrogens cause an increase in HDL-c while LDL is decreased. The biosynthesis of VLDL is enhanced but the triglyceride lipase activity reduced.

2. **PROGESTERONE**

After the first trimester, the placenta becomes capable of producing sufficient progesterone to maintain gestation, the levels of which in maternal plasma increases progressively with gestation. Cholesterol derived from maternal blood is the main substrate for the trophoblastic synthesis of progesterone. The levels of the hormone secreted by the placenta approximates 250 mg/day, the levels at term being 7 times the value at 8 weeks.

Progesterone levels fall significantly within 3 days reaching basal levels by 7th day in lactating mothers.
The oestrogen to progesterone ratio is also increased from 0.08 in the first trimester to a value of 0.232 at about 35 weeks. Thereafter it decreased to 0.187 at 38 weeks. No association has been found between lipid and lipoprotein levels to the oestrogen to progesterone ratio, except that the fall of ratio was parallel to the fall in LDL levels immediately before term (Desoye et al., 1986).

Individually progesterone brings about a decrease in HDL cholesterol and an increase in LDL-c. It induces hepatic triglyceride lipase activity. Increased degradation of VLDL and/or IDL resulting in high plasma LDL-c levels.

3. HUMAN PLACENTAL LACTOGEN

A polypeptide hormone secreted by the placenta gradually increases and eventually reaches a maximum of 5-8 ug/ml at term. Maternal concentrations promptly return to undetectable levels within 24 hours.

The levels of HPL clearly parallel the time course of the lipids changes during pregnancy. It has lipolytic activity thus releasing FFA probably by activation of hormone sensitiva lipase. This may occur for foetal requirements in the second half of pregnancy during which the mass of maternal adipocytes are reduced and the foetus gains weight. The portion of free fatty acids not utilised by foetus is incorporated into STG and VLDL in maternal liver (Desoye et al, 1987).
4. **HUMAN CHORTIONIC GONADOTROPIN (HCG)**

Concentration of HCG rises to peak values by 8-12 weeks of gestation. Thereafter there is a decrease in HCG levels to a plateau that is maintained throughout the remainder of pregnancy. It becomes undetectable in urine by 7-10 days postpartum. Free cholesterol was inversely related to HCG levels whereas triglyceride concentration resembled those to insulin.

B. **ANTERIOR PITUITARY HORMONES**

1. **HUMAN GROWTH HORMONE (HGH)**

   Basal levels of growth hormone are low during early pregnancy and do not change remarkably with advancing gestation. Pituitary somatotrophic hormone or growth hormone raises the blood lipid levels. Slow rise in HGH occurs during postpartum phase.

2. **PROLACTIN**

   Concentration of serum prolactin in pregnancy begin to increase approximately 30 days after the mid menstrual cycle peak of lutienising hormone. Rising prolactin levels continue to increase to reach peak levels at term. Serum prolactin declinea rapidly after parturation, if the woman does not breast feed. However, prolactin levels increase sharply with breast feeding episodes, they then decrease to non pregnant values after several months of lactation.
C. **THYROID HORMONES**

1. **THYROXIN**

   Like oestrogens it depresses the blood lipid levels. Patterson, Hund and Nicodeus (1938) believed that hypercholesterolemia of pregnancy is due to subclinical hypothyroidism.

   Lister (1955) and Russell (1956) found that protein bound iodine and serum precipitate iodine are elevated as early as second month of pregnancy. These levels have been found to reach as high as those seen in individual with overt hyperthyroidism.

   Strisower (1958) found that the thyroid hormone depresses serum lipid partition but during pregnancy the tissue become more refractory to the effect of thyroxin.

D. **ADRENAL HORMONES**

**GLUCOCORTICOIDS**

Cortisol metabolism is significantly altered during pregnancy, the maternal plasma levels rising progressively throughout gestation. The plasma levels of transcortin also rises progressively to peak in third trimester. The increased transcortin levels are due to increased oestrogen concentration. The maternal tissues are exposed to an average daily concentration of cortisol that is more than twice normal. Cortisone increases the cholesterol and its ester level and thus is achieved at the cost of neutral fats (Jailer et al, 1957).
E. PANCREATIC HORMONES

INSULIN

The basal levels of insulin tend to become progressively higher as term gestation is approached. Also a much greater amount of insulin is released in response to glucose stimulation. However, during pregnancy a state of insulin resistance exists. Insulin has a pronounced antilipolytic effect and antagonises the lipolytic effect of hormones mainly by inhibiting the hormone sensitive lipase in the adipose tissue. Thus it reduces the release and only of free fatty acids but of glycerol as well.