Lung alveoli are lined by a group of phospholipids known collectively as surfactant that prevents the collapse of small alveoli during expiration by lowering surface tension.

The surfactant is continually produced from type 2 alveolar cell (10% of the lung parenchyma), maximum production will be after 28 wk.

The predominant phospholipid( about 80% of total) is phosphotidylcholine (lecithin); and it’s production is enhanced by cortisol, growth retardation and prolonged rupture of membrane; and is delayed in diabetes.

Phosphoatidylglycerol is another type of potent phospholipid that is present in the amniotic fluid ,and it is more predictive of respiratory distress syndrome especially in diabetic preganant women.
Oligohydramnios and reduced intrathoracic space (daiphragmatic hernia) or chest wall deformities can result in pulmonary hypoplasia, which lead to progressive respiratory failure from birth.

Respiratory distress syndrome (RDS) is specific to babies born prematurely and is associated with surfactant deficiency.

RDS may be complicated by hypoxia, intraventricular haemorrhage and necrotizing enterocolitis.

The incidence and severity of RDS can be reduced by administrating steroids antenatally to mothers at risk of preterm delivery.
Fetal blood

- The first fetal blood cells are formed on the surface of the yolk sac from 14 to 19 days after conception.

- Haemopoiesis from the yolk sac continues until the 3rd post-conceptional month.

- During the 5th wk of embryonic life extramedullary haemopoiesis begins in the liver and to a lesser extent in the spleen.

- The bone marrow starts to produce red blood cells at 7-8 wk and is the predominant source of red cell from 26 wk of gestation.

- Most haemoglobin in the fetus is the fetal haemoglobin (HbF) that has 2 gamma chains (2 alpha, 2 gamma).
While the adult Hb is composed from HbA (2 alpha, 2 beta) chains and HbA2 (2 alpha, 2 delta) chains.

- 90% of fetal Hb is HbF from 10 to 28 wk.
- From 28-34 wk a switch to HbA occurs.
- At term the ratio of HbF to HbA is 80:20, by 6th month of age only 1% of the Hb is HbF.
- The HbF had high affinity for oxygen than HbA.
- This with association with a higher Hb concentration (at birth the mean capillary Hb is 18 g/dL) will enhance transfer of oxygen across the placenta.
- Abnormal Hb production results in thalassaemia.
● Beta major thalassaemia without treatment will results in severe anemia, fetal growth restriction, poor musculoskeletal development and skin pigmentation due to increased iron absorption.

● Severe alpha thalassaemia results in severe fetal anemia with cardiac failure, hepatoseplenomegaly & generalized odema, the infants are stillborn or shortly die after birth.

**Immune system:**

● The fetus requires an effective immune system to resist intrauterine and perinatal infections.

● Lymphocytes appear from 8 wk.

● By the middle of the second trimester all phagocytic cell, T and B cells and the complements are available to mount a response.
Early infections with any of the TORCH organisms will affect the immune system.

Immunoglobulin g (IgG) originates mostly from maternal circulation and crosses the placenta to provide passive immunity.

The fetus normally produces a small amount of IgM and IgA, which don’t cross the placenta.

Detection of IgM & IgA in the newborn without IgG is indicative of fetal infection.

General immunological defences include:
- The amniotic fluid (lysosymes, IgG).
- The placenta (lymphoid cells, phagocytes, barrier).
- Granulocytes from liver and bone marrow.
- Interferon from lymphocytes.
Skin and homeostasis

- Fetal skin protects and facilitates homeostasis.
- The thickness of the skin increases progressively from the 1st month of gestation until birth.
- A stratum corneum forms in the 5th month and after 23 wks, the appearance of the skin approaches that of the adult epidermis.
- Vernix (consisting of desquamated skin cells, cholesterol, and glycogen) is covering the skin of the fetus in the last wks.
- Preterm infants have no vernix and thin skin; this allows a proportionately large amount of insensible water loss.
- Heat may be conserved by peripheral vasoconstriction and can be generated by brown fat catabolism, which is deficient in preterm or growth restricted babies.
Alimentary system and energy stores

- The primitive foregut and hindgut are present by the end of 4\textsuperscript{th} wk as a straight tube suspended by the mesentery from the dorsal body wall.

- The midgut is herniated into the base of the umbilical cord during the 6\textsuperscript{th} wk because the abdominal cavity is too small to accommodate the enlarging liver & intestine.

- By 12\textsuperscript{th} wk the gut will re-enter the abdominal cavity but prior to that the gut undergoes rotation.

- Failure of the re-entre results in the development of omphalocele.

- The inestinal tract is patent from the time when the GIT is fully formed.
The swallowing reflex develops and matures gradually.

The fetus continually and increasingly swallow amniotic fluid up to 20 ml/hr at term.

A failure in the swallowing mechanism as in neurological abnormalities e.g. anencephaly and an obstruction gut e.g. atresia of the oesophagus will result in polyhydramnios.

Peristalsis in the intestine occurs from the 2nd trimester.

The large bowel is filled with the meconium, meconium stained liquor is a sign of post maturity and fetal hypoxia.

Body water content gradually diminishes and the glycogen and fat stores increase about 5 fold in the last trimester.

Preterm infants and growth restricted fetuses have reduced glycogen and fat stores.
The primitive liver appears at about 18\textsuperscript{th} day of embryonic life as a diverticulum arising from the duodenum.

By the 25\textsuperscript{th} day it has developed into a T shaped outgrowth which is invaded by blood vessels.

The large portion of this diverticulum gives rise to the parenchymal cells and the hepatic ducts, while the small portion gives rise to the gallbladder.

The liver plays an important in haemopoiesis starts from 6\textsuperscript{th} wk and peaks at 12-16\textsuperscript{th} wks and continues until 36\textsuperscript{th} wk.
Fetal liver differs from adult in that it has reduced ability to conjugate bilirubin because of relative deficiencies in necessary enzymes like glucuronyl transferase.

The placenta is performing the normal metabolic function of liver.

The glycogen store is small in the 1st trimester but it is maximal at term.

The premature and growth restricted infants are more prone to jaundice and hypoglycemia.
Kidney and urinary tract

- The metanephros forms the renal collecting system (ureters, pelvis, calyces, and the collecting ducts).

- The mesenchyme of the nephrogenic cord forms the renal secretory system (glomeruli, convoluted tubes, loops of Henle).

- Nephrogenesis is complete by 36 wk.

- The maturation and the concentrating ability of the fetal kidneys is gradual, it is immature in preterm infant that may lead to abnormal water, glucose, sodium, or acid-base homeostasis.

- Fetal urine forms much of the amniotic fluid.

- Renal agenesis result in severe oligohydramnios.
Fetal movement (quickening) can be first perceived by the mother by 18-20 wk in primigravida, and several wks earlier in multigravida.

Self monitoring of fetal movement is an important method for fetal well-being.

Diminished fetal movement may indicate chronic hypoxia and growth restriction, this will need further investigation.

With maturation of the central nervous system, the fetus develops more complex and well defined behavioral states named 1F-4F:
Amniotic fluid

- By 12 wk the amnion comes into contact to the inner surface of the chorion and obliterates the extra-embryonic space.

- The two membranes didn’t contain blood vessels or nerves but do contain significant quantity of phospholipids and enzymes.

- Choriodicedual function play a pivotal role in initiation of labour through production of prostoglandin E2 and F2a.

- The amniotic fluid is initially secreted by the amnion.

- By 10th wk it is mainly a transudate of the fetal serum via the skin and umbilical cord.

- From 16 wks the skin become impermeable to water, so the increase in the amniotic fluid is through a contribution of kidney and lung fluids, and removed by fetal swallowing.
- state 1F is similar to quiet (non REM) sleep, absence of eye and body movements.

- state 2F periodic body and eye movement are present (REM sleep).

- state 3F is like quiet wakefulness when there are eye but no body movements.

- state 4F body in active ongoing body and eye movement.

  > 80% of time the fetus will alternate between 1F and 2F state.
Amniotic fluid volume increases progressively:

- 10 wk: 30ml
- 20 wk: 300ml
- 30 wk: 600ml
- 38 wk: 1000ml

From term there is rapid fall in the volume (40 wk: 800ml, 42 wk: 350 ml).

The function of amniotic fluid:

- Protect the fetus from mechanical injury.
- Permit fetal movement and preventing limbs contracture.
- Prevent adhesions between the fetus and amnion.
- Permit fetal lung development, if there is absence of the fluid especially in the 2nd trimester this will lead to pulmonary hypoplasia.