

# Pregnancy

## Placental

## function and dysfunction

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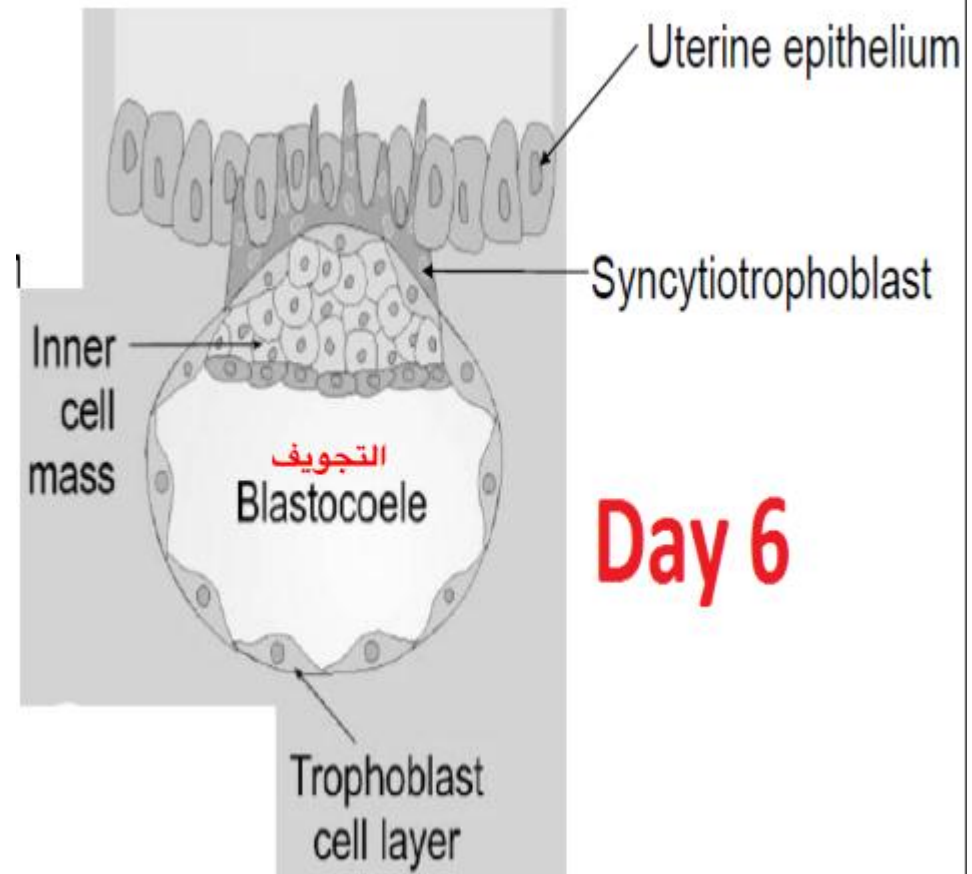
**Module leader: Assist Prof. Salam Abdelameer**

# Objectives:

- Describe process of implantation of conceptus into endometrium
- Describe structure of the placenta & its adaptation for exchange of materials between fetal & maternal blood
- Describe arrangement of fetal blood vessels within placenta
- Describe factors influencing passive diffusion of substances across placenta
- Identify major substances which are actively transported across placenta
- Describe role of placenta as an endocrine organ supporting pregnancy
- Describe the hormonal basis of testing for pregnancy
- Describe function of placenta as a provider of passive maternal immunity to the neonate.

# Implantation

- Blastocyst enters the uterine cavity (4-5 days after fertilization)
- The endometrium is ready to receive it for pregnancy to be established.
- After a day or so in the uterine cavity the blastocyst implants into the endometrium
- Implantation involves interaction between Trophoblast cells & the epithelium of the uterus.

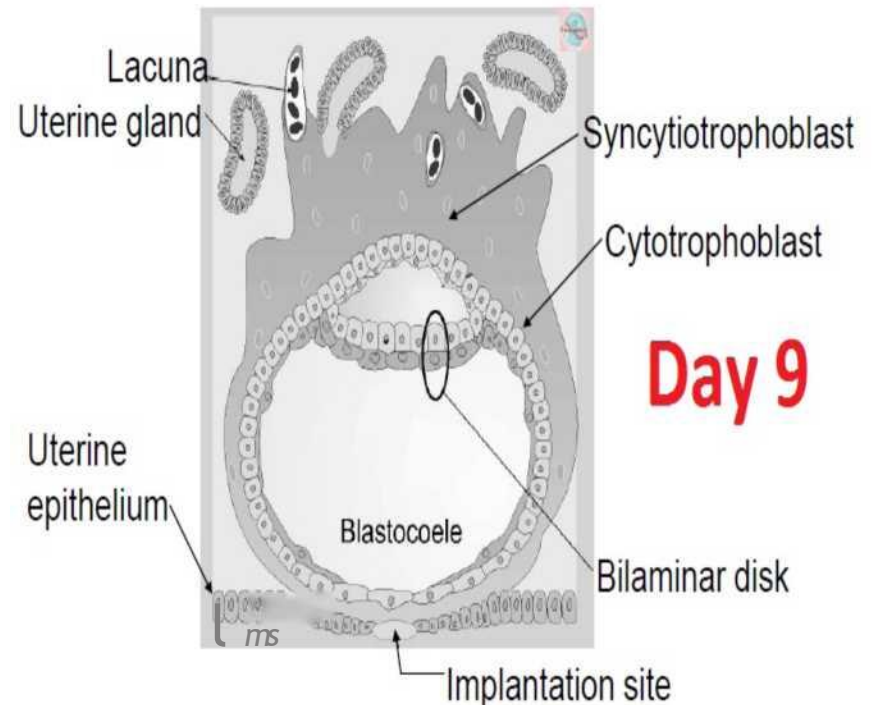
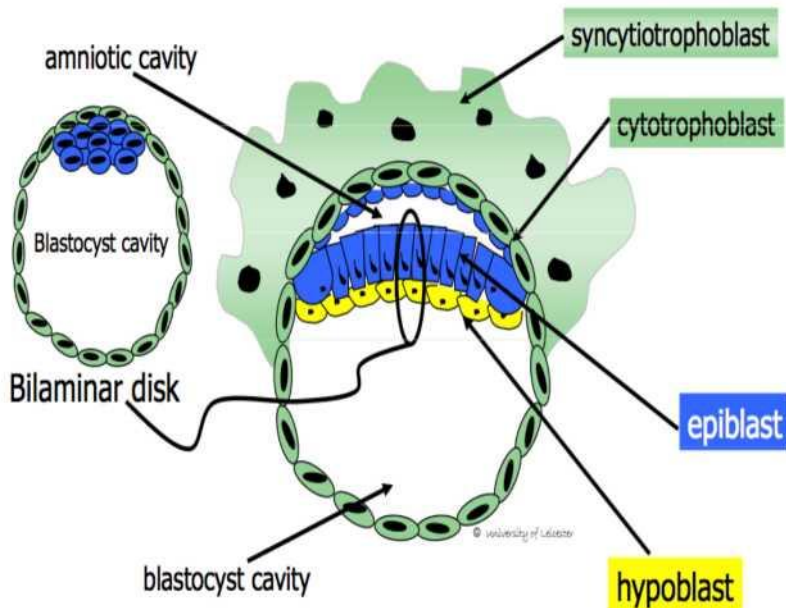


- Further embedding of the blastocyst into the endometrium is dependent upon the invasive property of the trophoblasts

Blastocyst outer layer called the **Syncytiotrophoblast** differentiated from the underlying **Cytotrophoblast**.

- By the **10<sup>th</sup> day after fertilisation**, the blastocyst is fully embedded within the endometrium.

## The bilaminar disc



- Implantation is **Interstitial**
  - Uterine epithelium is breached and conceptus implants within stroma
- **Placental\_membrane\_**becomes **progressively\_thinner\_**as the needs of the fetus increase
- Placenta is **Haemomonochorial**
  - **One\_layer\_of\_trophoblast\_**ultimately separates maternal blood from fetal capillary wall (syncytio-vascular).

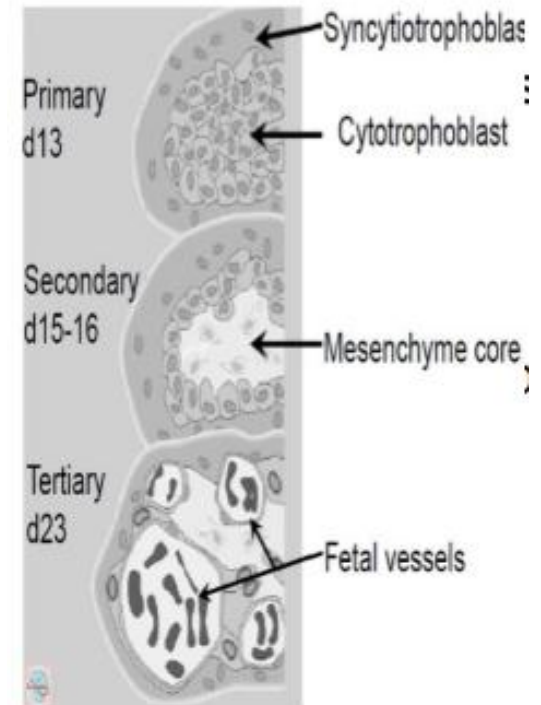
# Aims of Implantation

## 1. Establish the basic unit of exchange( villi)

- **Primary villi**
  - Early, finger-like projections of trophoblast
- **Secondary villi**
  - Invasion of mesenchyme into core
- **Tertiary villi**
  - Invasion of mesenchyme core by fetal vessels

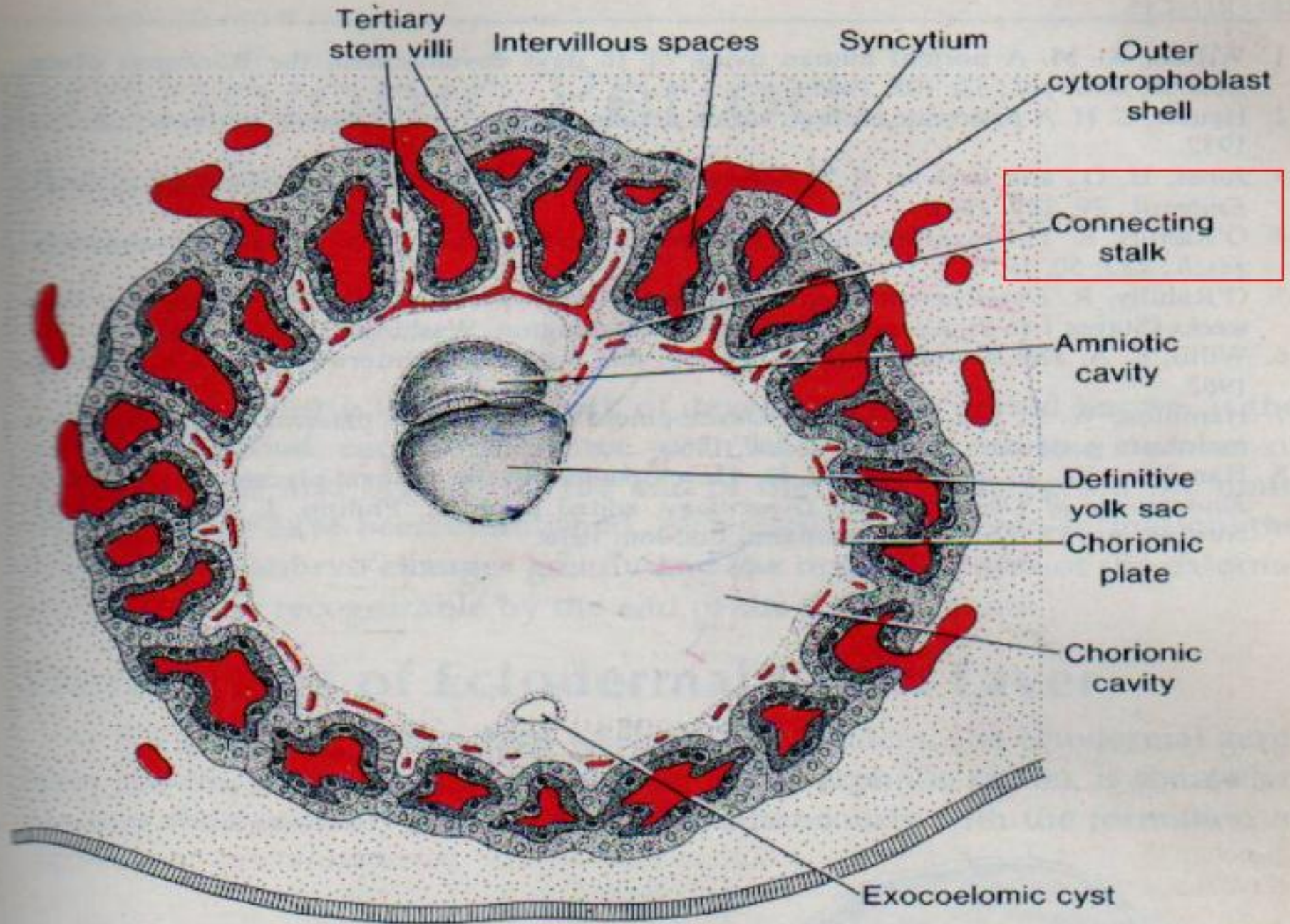
## 2. Anchor the placenta

- Establishment of outermost cytotrophoblast shell



## 3. Establish maternal blood flow within the placenta

Hence ,when the heart start to beat in the 4th week of development, the villous system is ready to supply the embryo with the necessary nutrient & oxygen. By the 20th day the embryo is attached to its trophoblastic shell by a narrow connecting stalk (primitive

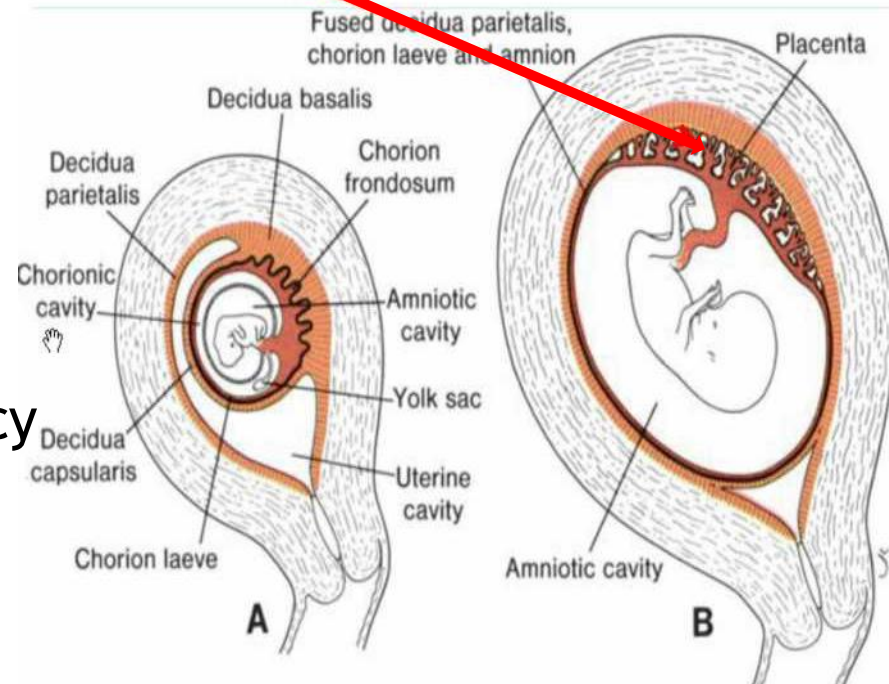


# Histology of Implantation

- The endometrium is prepared for implantation

## 1. Decidualisation

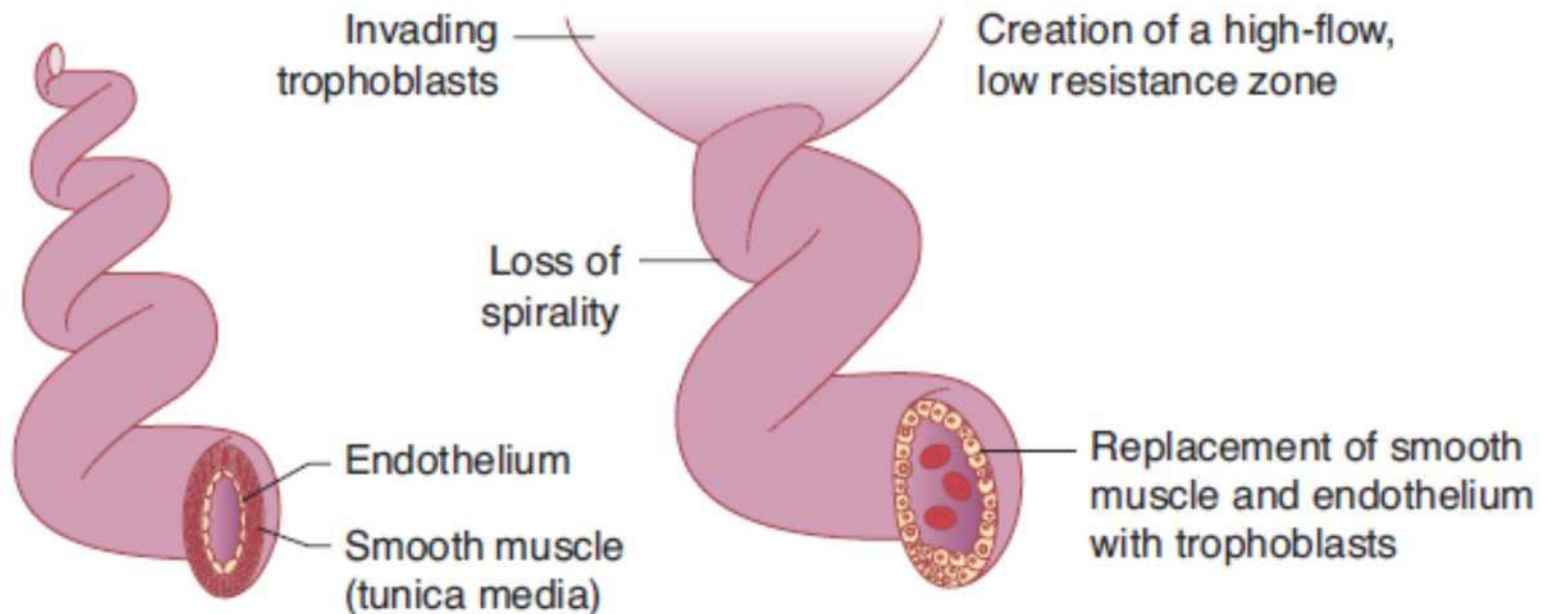
- Pre-Decidual” cells
- The decidual reaction provides the balancing force for the invasive force of the trophoblast
- Stimulated by Progesterone
- Without this balancing force, complications such as haemorrhage can occur, e.g. in ectopic pregnancy





## 2. Remodelling of Spiral Arteries

- Creation of low resistance vascular bed
- Maintains the high flow required to meet fetal demand, particularly late in gestation (The maternal blood flow to the placenta increases throughout pregnancy from 50 mL/min in the first trimester to 500-750 mL/min at terms)



Physiological change of spiral arteries by invading trophoblast

# Implantation Defects

## Ectopic Pregnancy

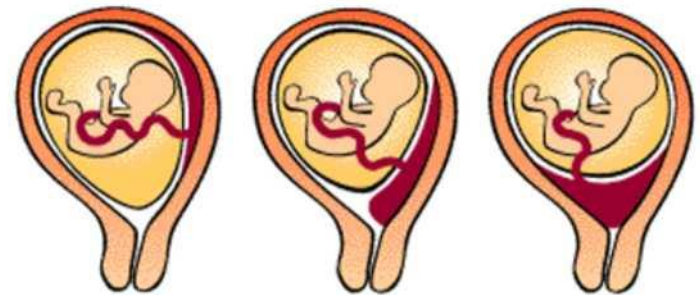
- Implantation at site other than uterine body
- Most commonly fallopian tube
- Can be peritoneal or ovarian
- Can very quickly become a life-threatening emergency

## Placenta Praevia

- Implantation in the lower uterine segment
- Can cause haemorrhage in pregnancy
- Requires C-Section delivery

## Incomplete Invasion

- Placental insufficiency
- Pre-Eclampsia



Normal  
Placenta

Marginal  
Placenta Previa

Complete  
Placenta Previa

# Structure of the Placenta

- The placenta has both maternal and fetal components and a good placenta determines a good pregnancy.
- Development of the placenta begins soon after fertilisation at compaction, with cells of the outer cell mass destined to develop into the structures that support the embryo/fetus during pregnancy.

By the beginning of the 4<sup>th</sup> month, the placenta has 2 components:

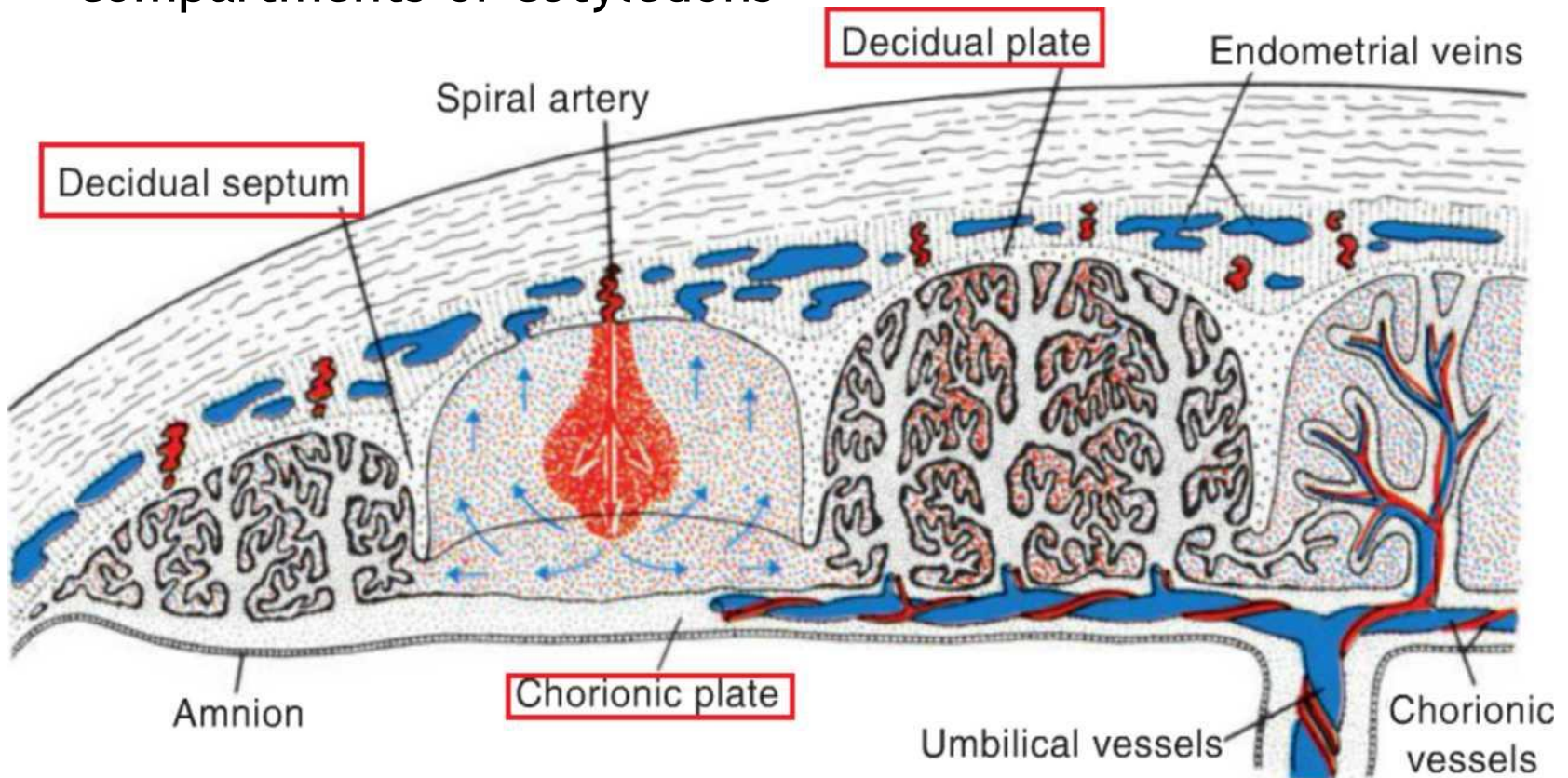
## 1. A fetal portion

- Formed by the **chorion frondosum**
- Bordered by the **chorionic plate**

## 2. A maternal portion

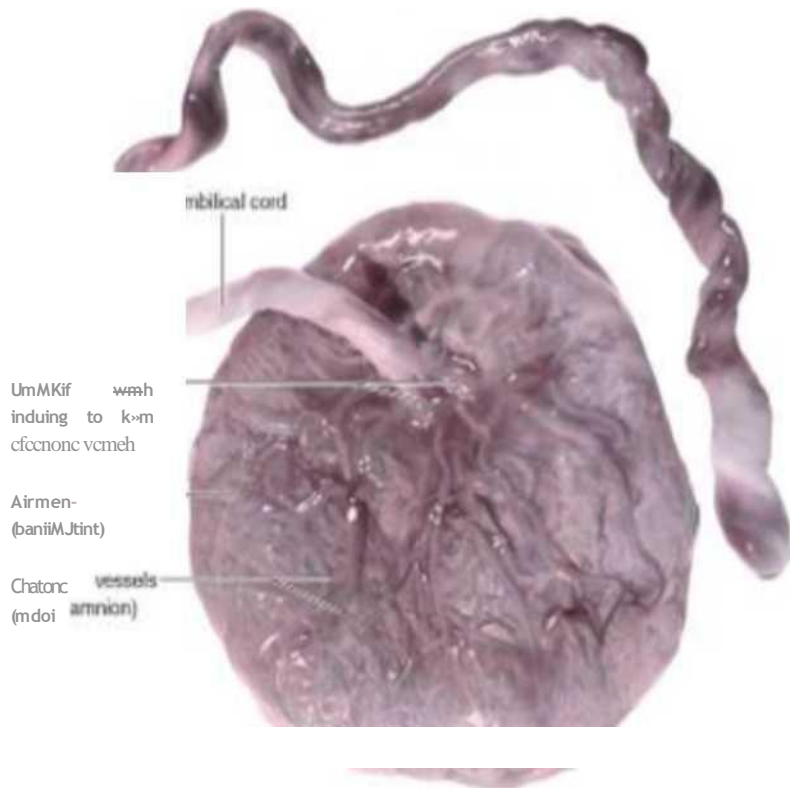
- Formed by the **decidua basalis**
- The **decidual plate** is most intimately incorporated into the placenta
- Between the chorionic and decidual plates are the **Intervillous Spaces**, which are filled with maternal blood.

During the fourth and fifth months, the decidua forms a number of **Decidual Septa**, which project into the intervillous spaces but do not reach the chorionic plate. These septa divide the placenta into a number of compartments or cotyledons

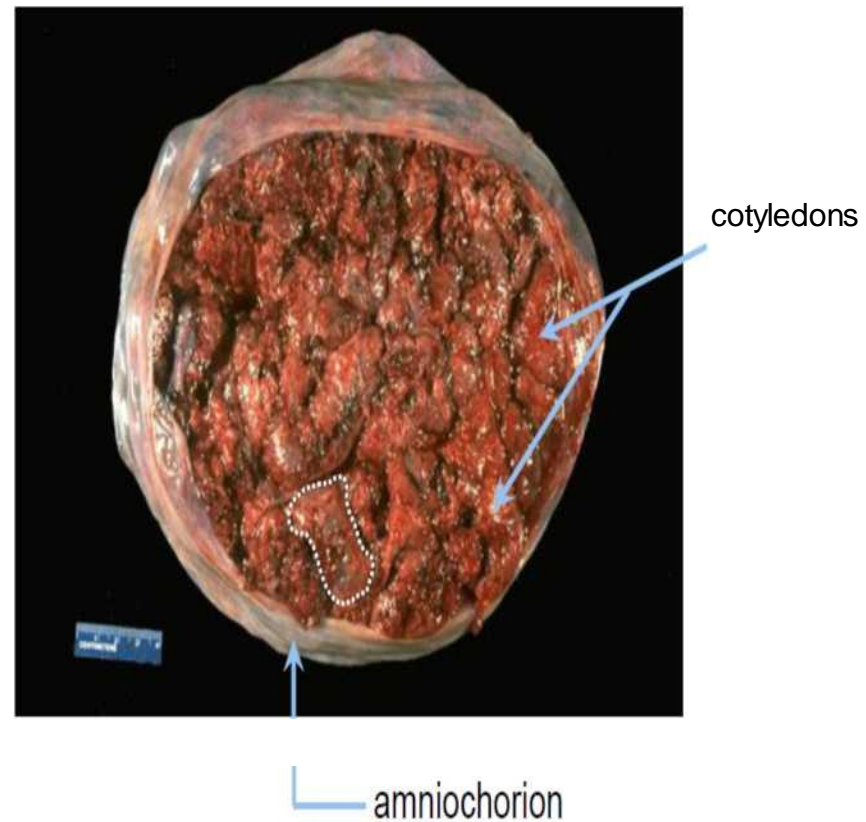


As a result of the continuous growth of the fetus & expansion of the uterus, the placenta also enlarges. Throughout pregnancy it covers approximately 15 - 30% of the internal surface of the uterus.

### Gross morphology - fetal aspect



### Gross morphology - maternal aspect



# Placental adaptation for the exchange of materials between fetal and maternal blood

## First Trimester Placenta

- Placenta established
- Placental 'barrier' to diffusion still relatively thick
- Complete cytotrophoblast layer beneath syncytiotrophoblast

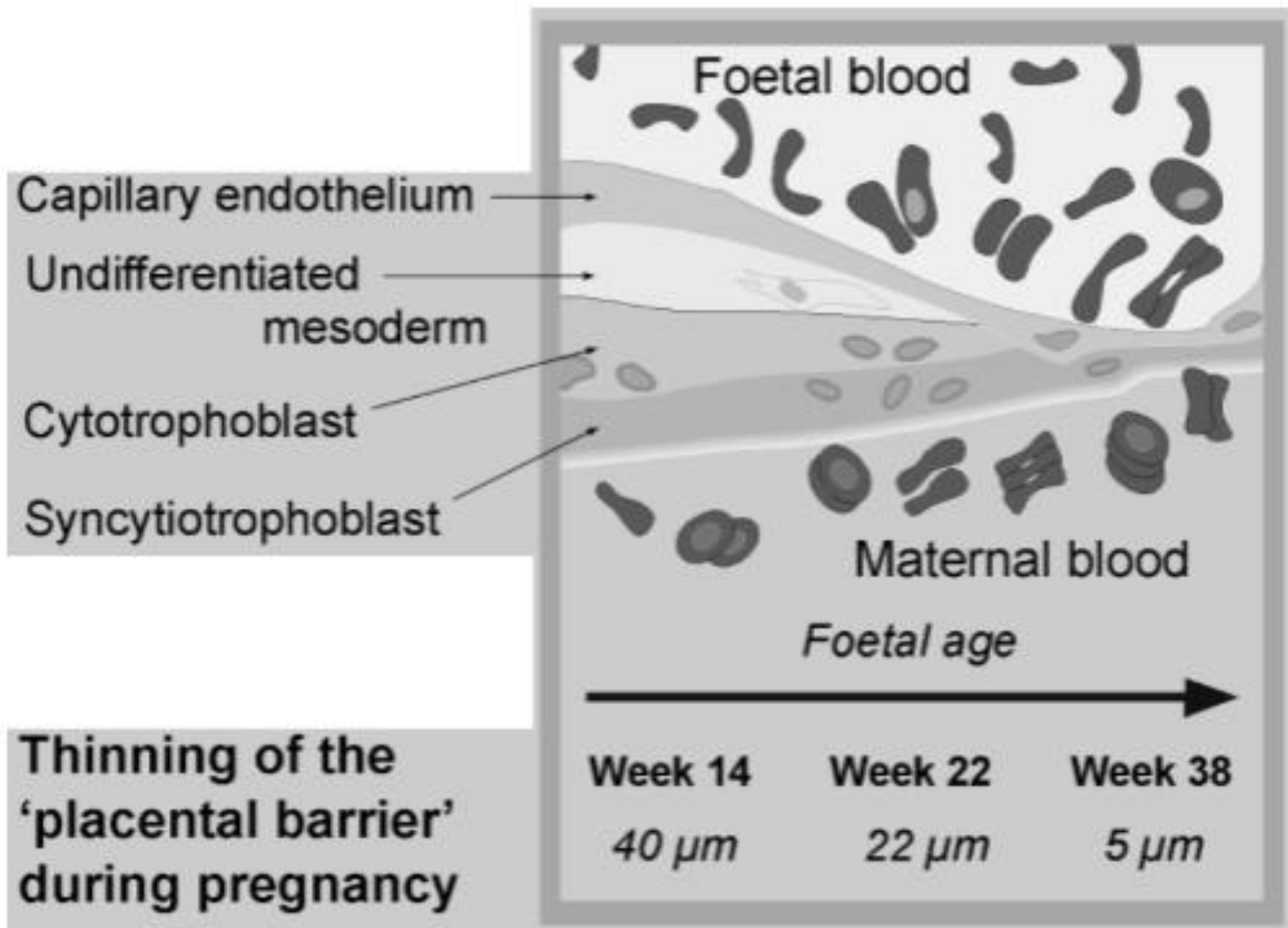
## Term Placenta

- Surface area for exchange dramatically increased

Placental 'barrier' is now thin

Cytotrophoblast layer beneath syncytiotrophoblast lost

The placenta at term is circular forming a spongy disc ~~20 cm~~ in diameter & ~~3cm~~ in thickness, its weight is about 500 gm. But there is direct relation with fetal weight.



# Arrangement of fetal blood vessels within the placenta

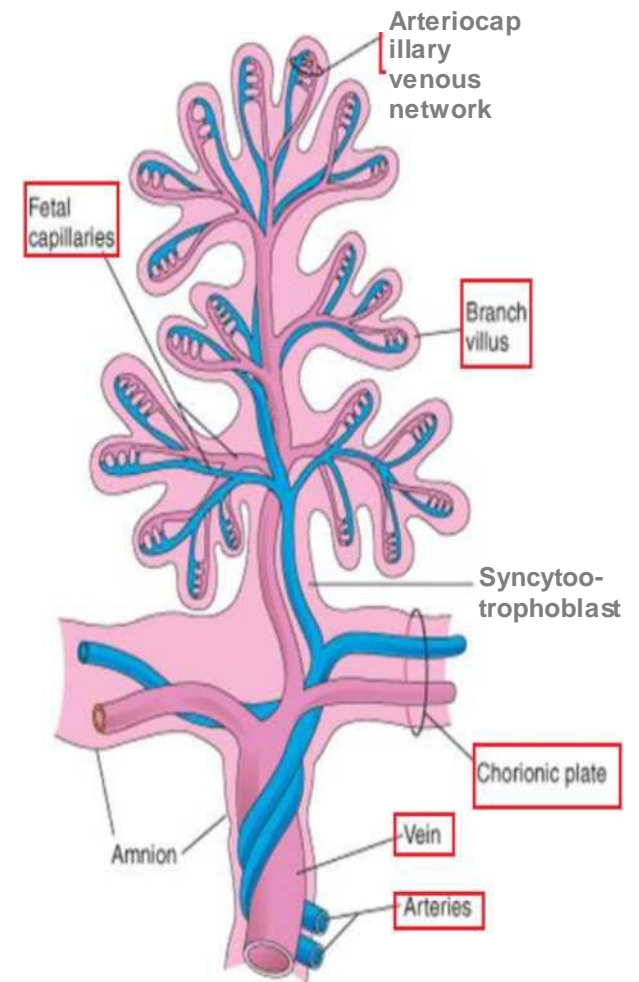
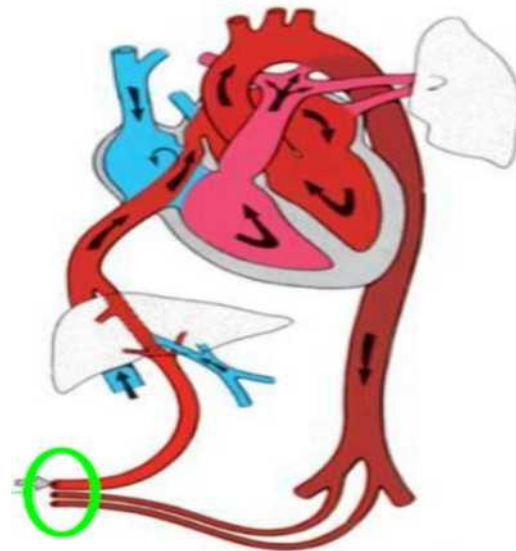
The umbilical arteries and veins project into tertiary villi, which are bathed in oxygenated maternal blood.

## Two Umbilical Arteries

- Deoxygenated blood Fetus -> Placenta

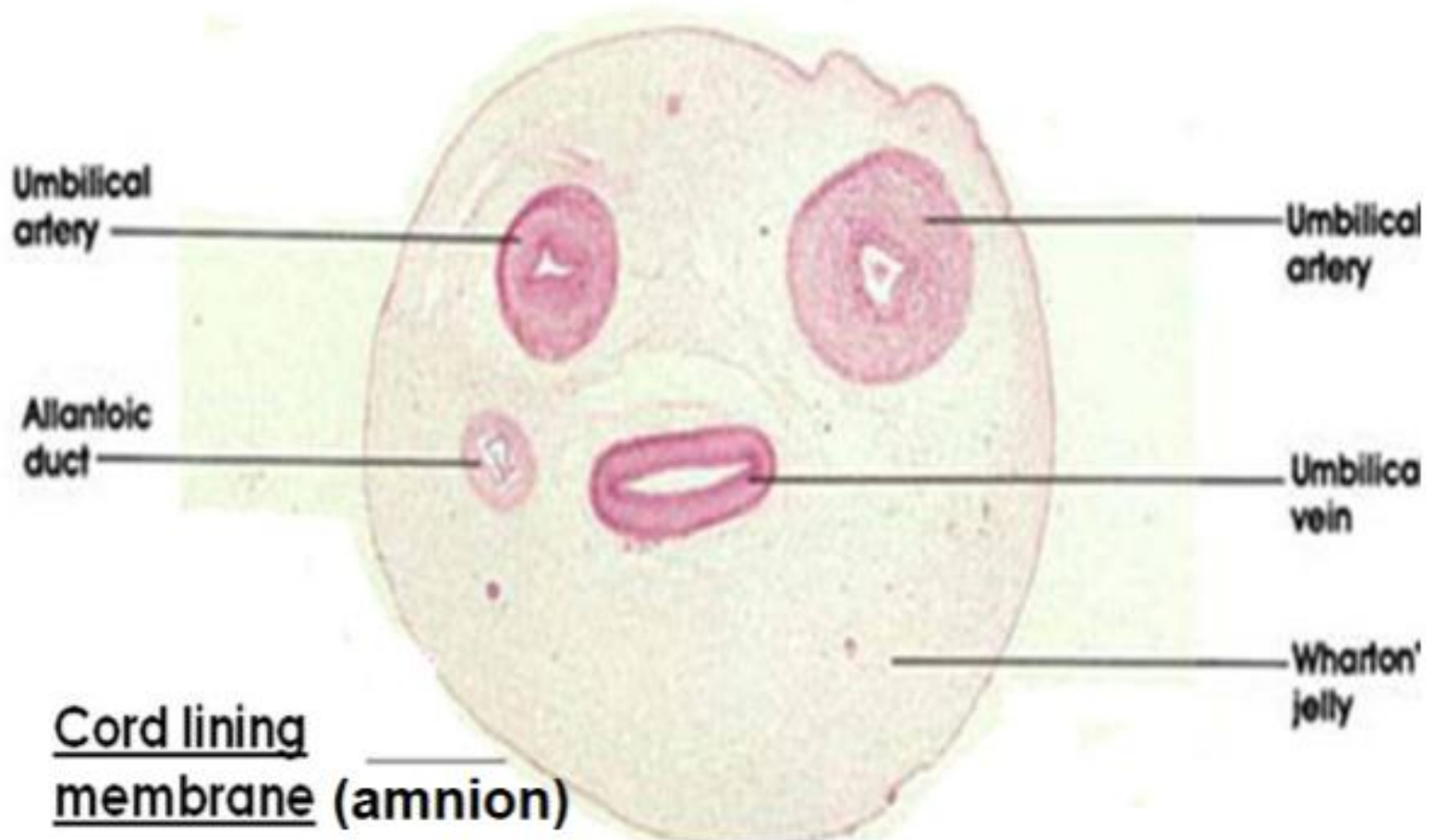
## One Umbilical Vein

Oxygenated blood Placenta ^ Fetus



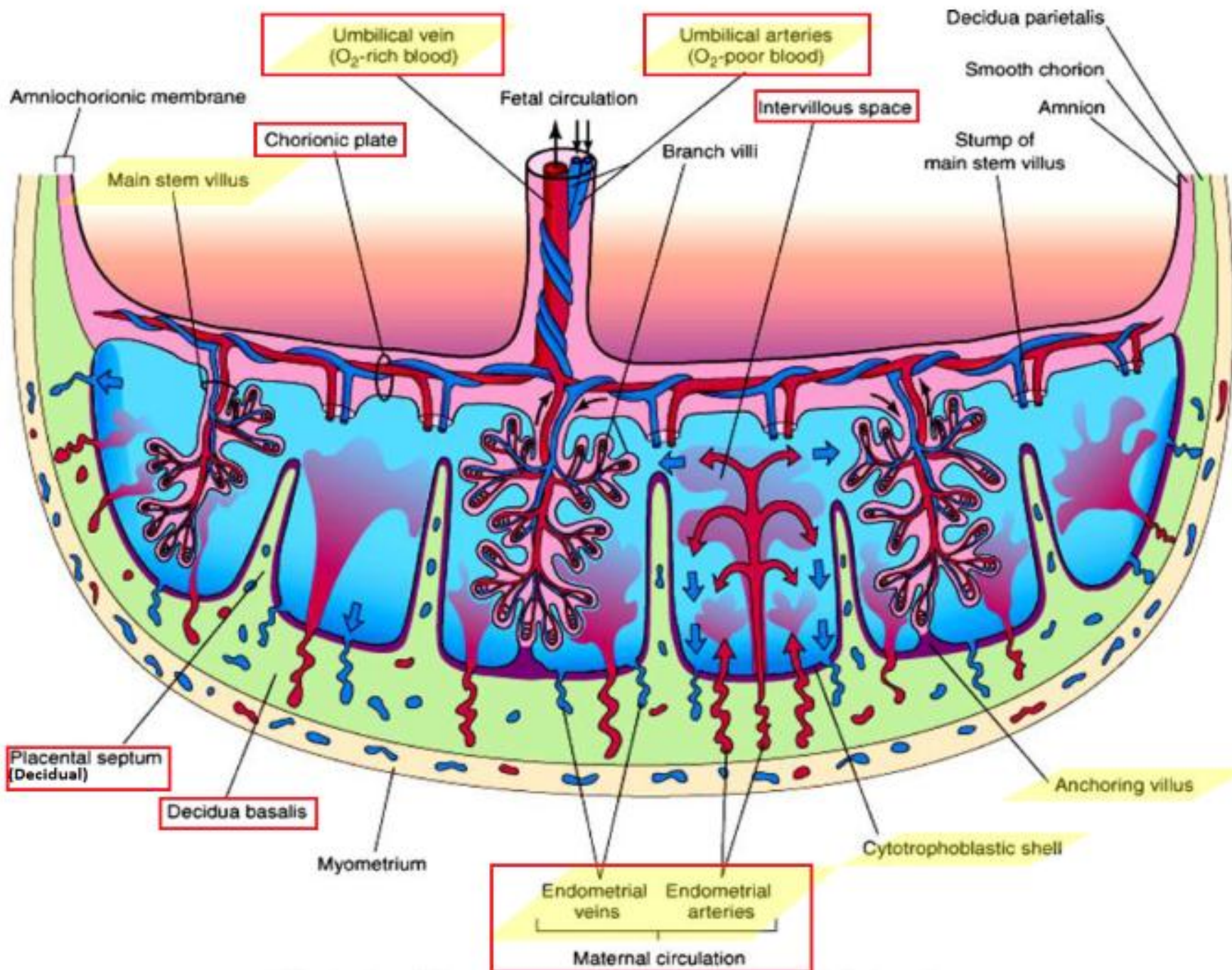


# Cross section of umbilical cord



**Cotyledons** receive their blood through 80 - 100 spiral arteries that pierce the decidual plate. **Pressure in these arteries forces oxygenated blood deep into the intervillous spaces** and bathes the numerous small villi of the villous tree in oxygenated blood.

As the pressure decreases, blood flows back form the chorionic plate towards the decidua, where it enters the endometrial veins.



# Factors influencing the passive diffusion of substances across the placenta

- Concentration Gradient
  - The steeper the gradient, the more diffusion
- Barrier to diffusion
  - Placental membrane gradually thins throughout pregnancy as the demand of the fetus increases
- Diffusion distance
  - Haemomonochorial
- Materno-fetal exchange occurs through a variety of means, and involves both simple and facilitated diffusion, active transport and receptor mediated endocytosis.

## Simple Diffusion

- Water
- Electrolytes
- Urea and uric acid
- Gases
  - Flow limited, not diffusion-limited
  - Fetal O<sub>2</sub> stores are small - maintenance of adequate flow is essential

## Facilitated Diffusion

- Glucose **Active Transport**

Specific transporters are expressed by the syncytiotrophoblast

- Amino acids
- Iron
- Vitamins

The placenta is not a true 'barrier' however.

**Teratogens** can access the fetus via the placenta, giving physiological consequences. Teratogens are particularly damaging during critical stages of development.

- Thalidomide
- Alcohol
- Therapeutic drugs
- Drugs of abuse
- Maternal smoking

Some **pathogens** are also able to cross the placenta:

- Varicella zoster
- Cytomegalovirus
- *Treponema Pallidum*
- *Toxoplasma gondii*
- Rubella • HIV

# The placenta as an endocrine organ supporting pregnancy

Hormones produced by the placenta are :

**Protein**

human chorionic gonadotrophin (hCG)  
human chorionic somatomammotrophin  
human chorionic thyrotrophin  
human chorionic corticotrophin

**Steroid**

progesterone  
oestrogen

# Protein Hormones

- **Human Chorionic Gonadotrophin (hCG)**
  - Produced during the **first two months** of pregnancy
  - **Supports the secretory function of the corpus luteum**
  - Produced by **syncytiotrophoblast**, therefore is pregnancy specific
- **Human Chorionic Somatommotrophin (hCS)**
  - Influences maternal metabolism, increasing the availability of glucose to the fetus
- **Human Chorionic Thyrotrophin**
- **Human Chorionic Cortiotrophin**

## Steroid Hormones

- Steroid hormones from the placenta are responsible for maintaining the pregnant state.
- **Progesterone**
  - Placenta takes over production from the corpus luteum (Week 11)
  - Influences maternal metabolism by increasing appetite
- **Oestrogen**
- The placenta also synthesises **glycogen, cholesterol and fatty acids**.



# Hormonal basis of diagnosis of pregnancy

## Pregnancy test

### Human Chorionic Gonadotrophin (hCG)

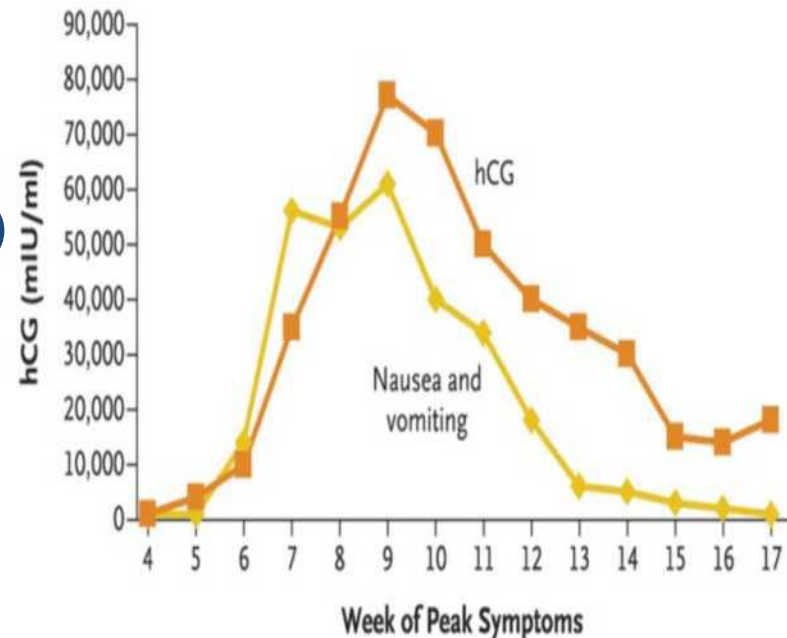
- Produced during the **first two months** of pregnancy
- Supports the secretory function of the corpus luteum**
- Produced by **syncytiotrophoblast**

specific

- Excreted in maternal urine**

there fore is used as the basis for

**Pregnancy Testing** (serum, urine)



# The function of the placenta as a provider of passive maternal immunity to the neonate

## Passive Immunity

- Immunological competence begins to develop late in the first trimester, by which time the fetus makes all of the components of **complement**.
- Fetal immunoglobulins consist almost entirely of **Maternal Immunoglobulin (IgG)**, which begins to be transported from mother to fetus at approximately **14 weeks**. The IgG is transported via **Receptor Mediated Pinocytosis**. Eventually the concentration of IgG in fetal plasma exceeds that of maternal plasma.
- In this manner the fetus gains passive immunity against various infectious diseases. Newborns produce their own IgG, but adult levels are not attained until the age of 3.

## Haemolytic Disease of the Newborn

- Rhesus blood group incompatibility of mother & fetus
- Mother previously sensitised to rhesus antigen (e.g. previous pregnancy)
- IgG against rhesus crosses the placenta and attacks foetal RBCs
- Now uncommon because of prophylactic treatment
  - Rhesus -'ve mothers pregnant with Rhesus +'ve fetus given **Rhesus specific IgG** during pregnancy and shortly after birth, to prevent sensitisation in the event of exposure to the antigen (The given IgG will bind to antigen before the mother's immune system can mount a response)

***THANK YOU***