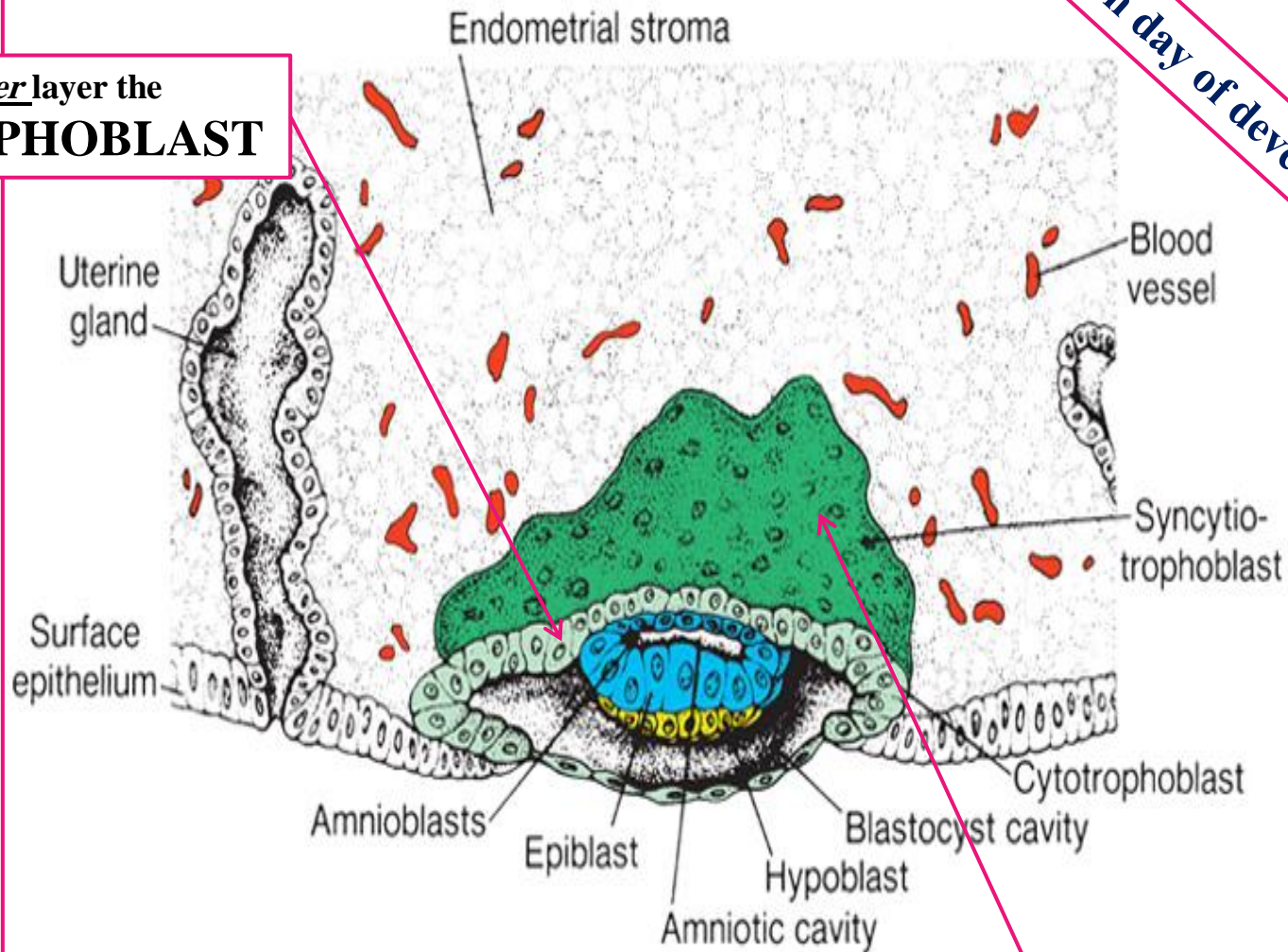


The trophoblast differentiates into two layers:

At the eighth day of development

(1) an inner layer the
CYTOTROPHOBLAST



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(2) an outer zone the
SYNCYTIOTROPHOBLAST

DAY 9

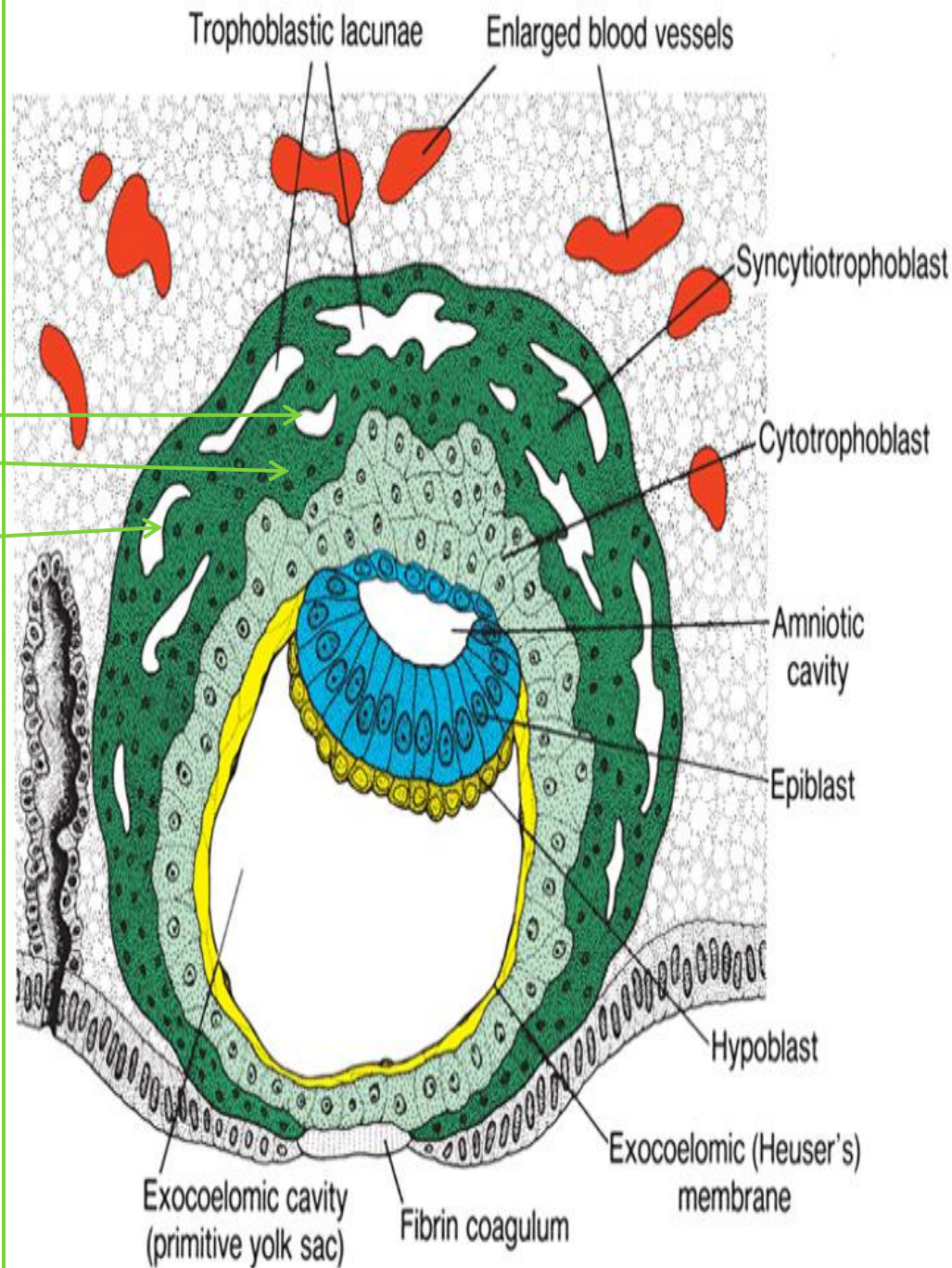
❖ At the trophoblast
vacuoles

appear in the syncytium.

These vacuoles fuse and form large
lacunae

This phase of trophoblast development
is known as the

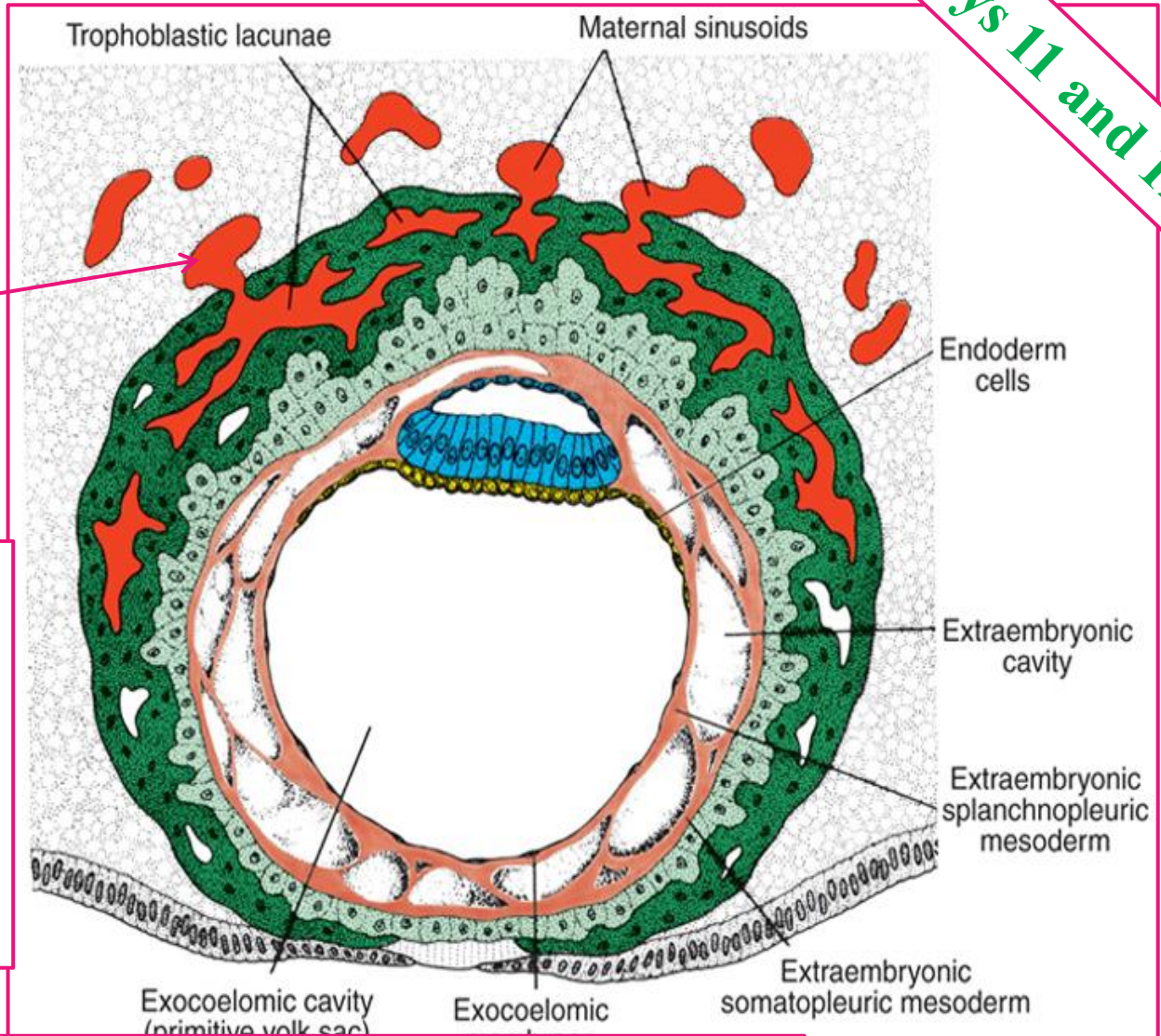
LACUNAR STAGE



Days 11 and 12

➤ The **syncytiotrophoblast** start to penetrate deeper into the stroma and eroding the **maternal capillaries** known as sinusoids.

➤ The syncytial lacunae become continuous with the sinusoids, and maternal blood enters the **lacunar system**

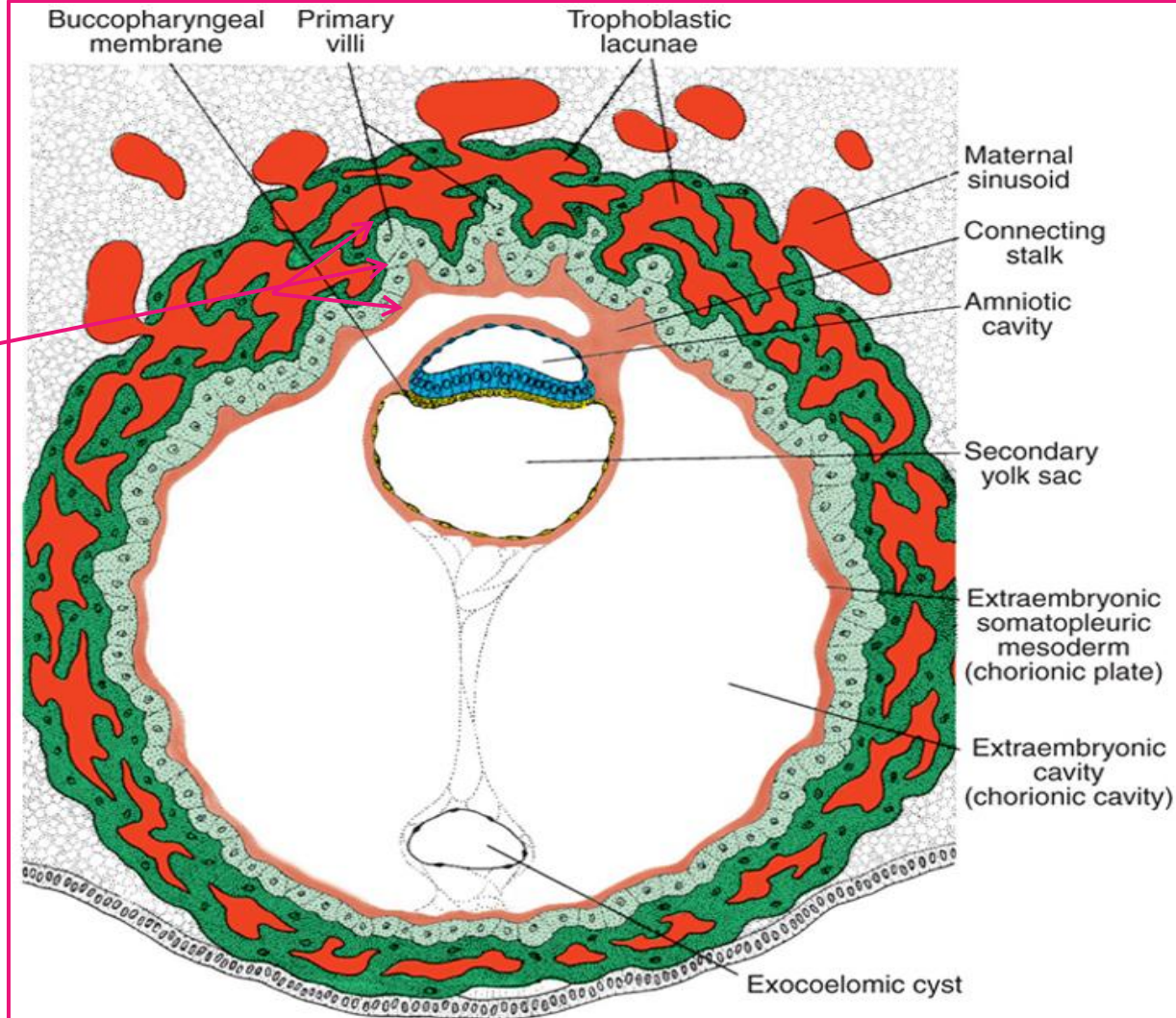


Thus establishing the
**UTEROPLACENTAL
CIRCULATION**

Cells of the cytotrophoblast proliferate locally and penetrate into the syncytiotrophoblast, forming cellular columns surrounded by syncytium.

Cellular columns with the syncytial covering are known as

PRIMARY VILLI

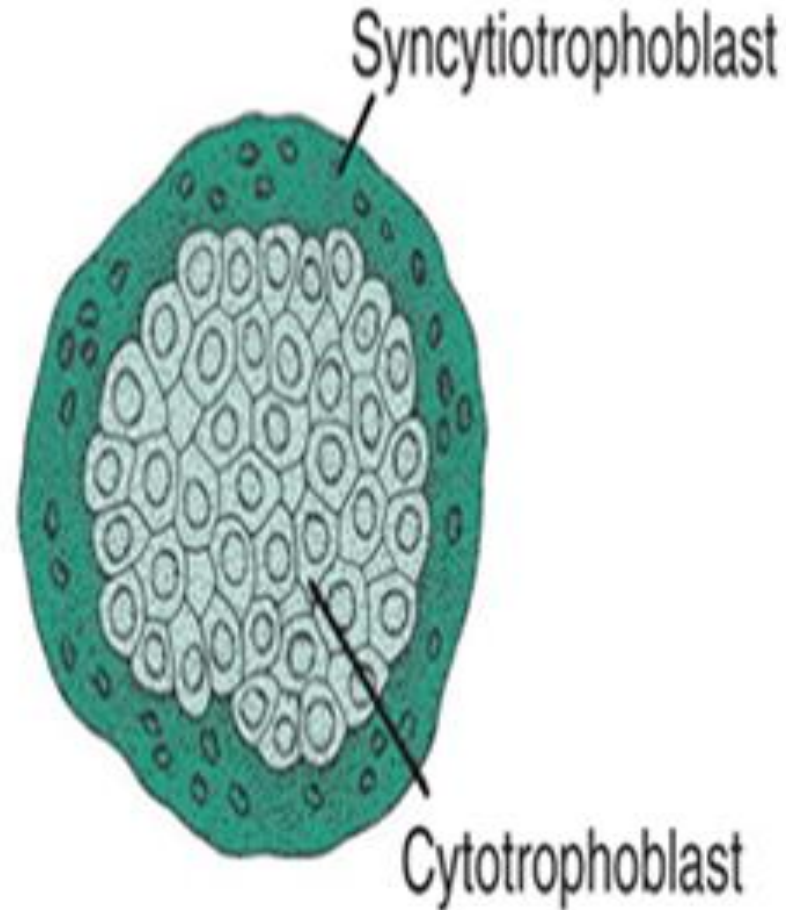


By the beginning of the third
week

The trophoblast is
characterized by

primary villi

that consist of a
cytotrophoblastic core covered
by asyncytial layer

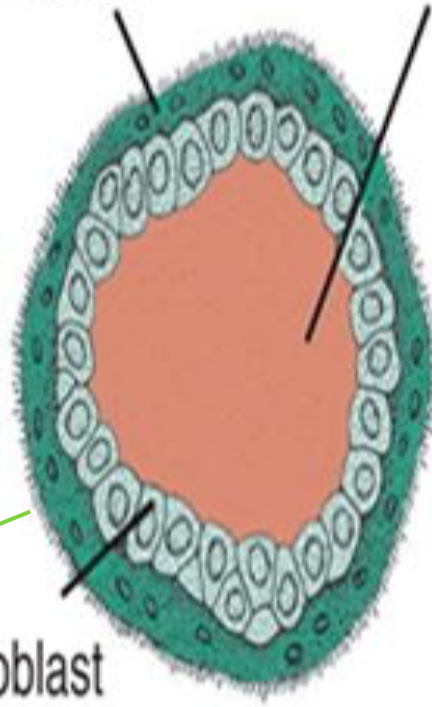


A Primary villus

During further development mesodermal cells penetrate the core of primary villi and grow toward the decidua
The newly formed structure is known as
a secondary villus

Syncytiotrophoblast

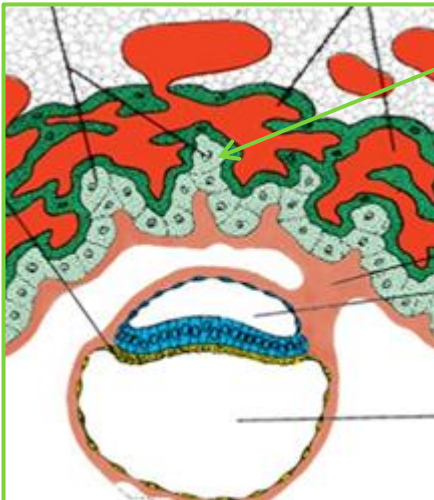
Mesoderm core



Cytotrophoblast

B

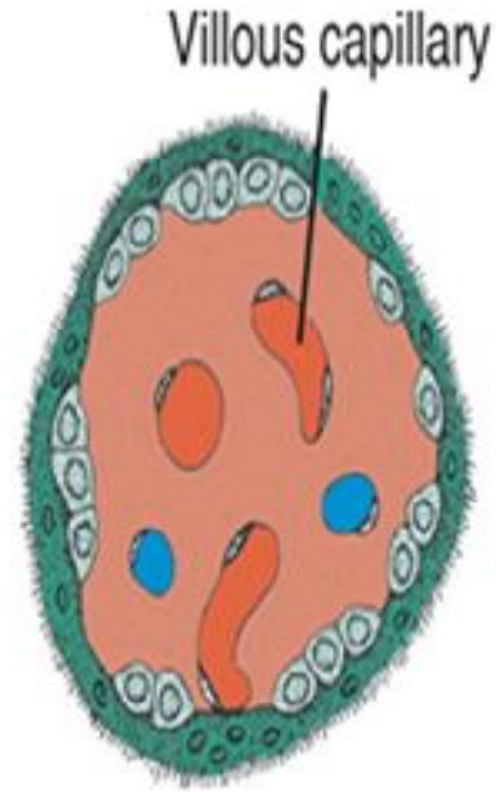
Secondary villus



By the end of the third week, mesodermal cells in the core of the villus begin to differentiate into blood cells and small blood vessels

forming the villous capillary system The villus is now known as a

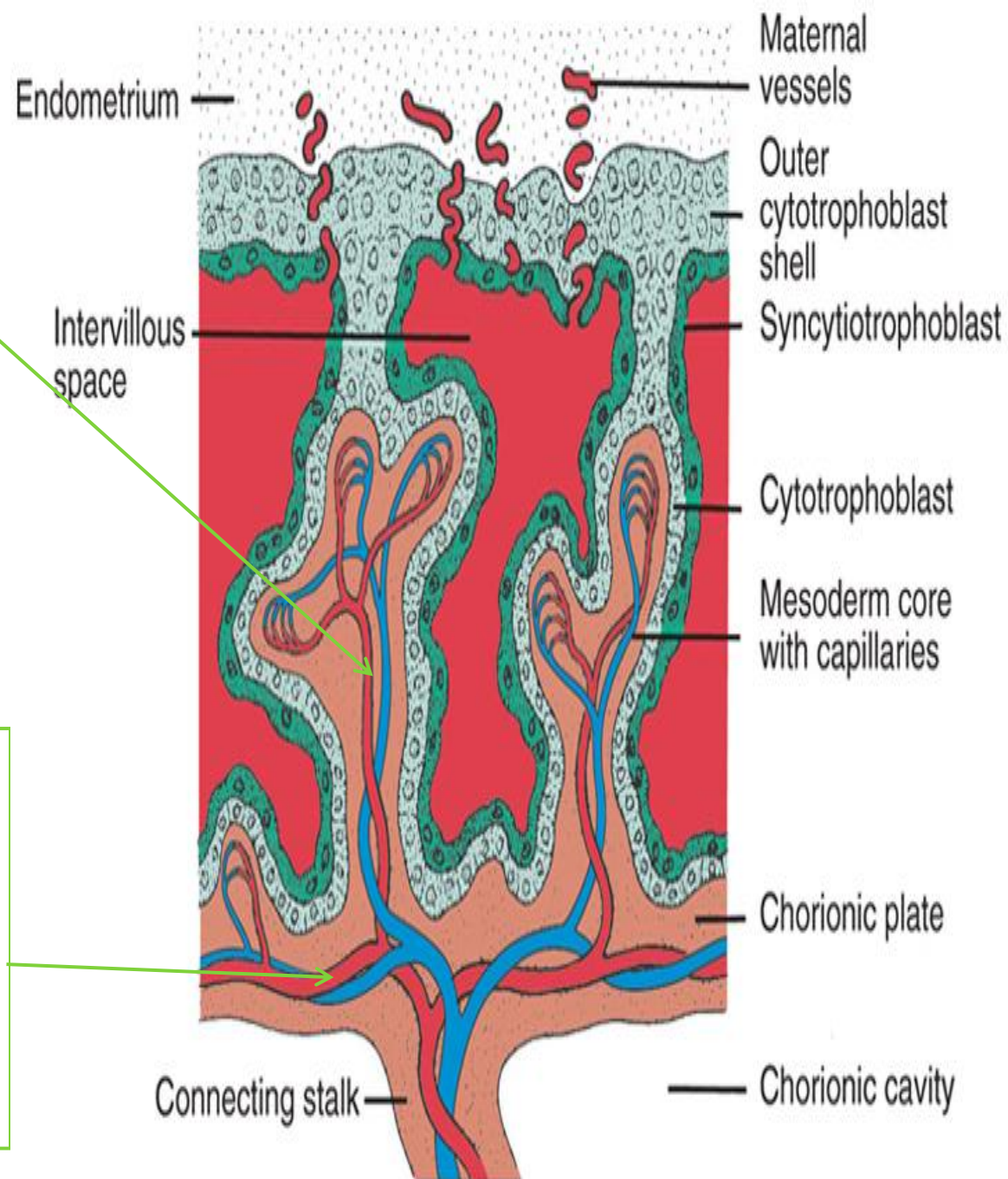
Tertiary villus or definitive placental villus



C Tertiary villus

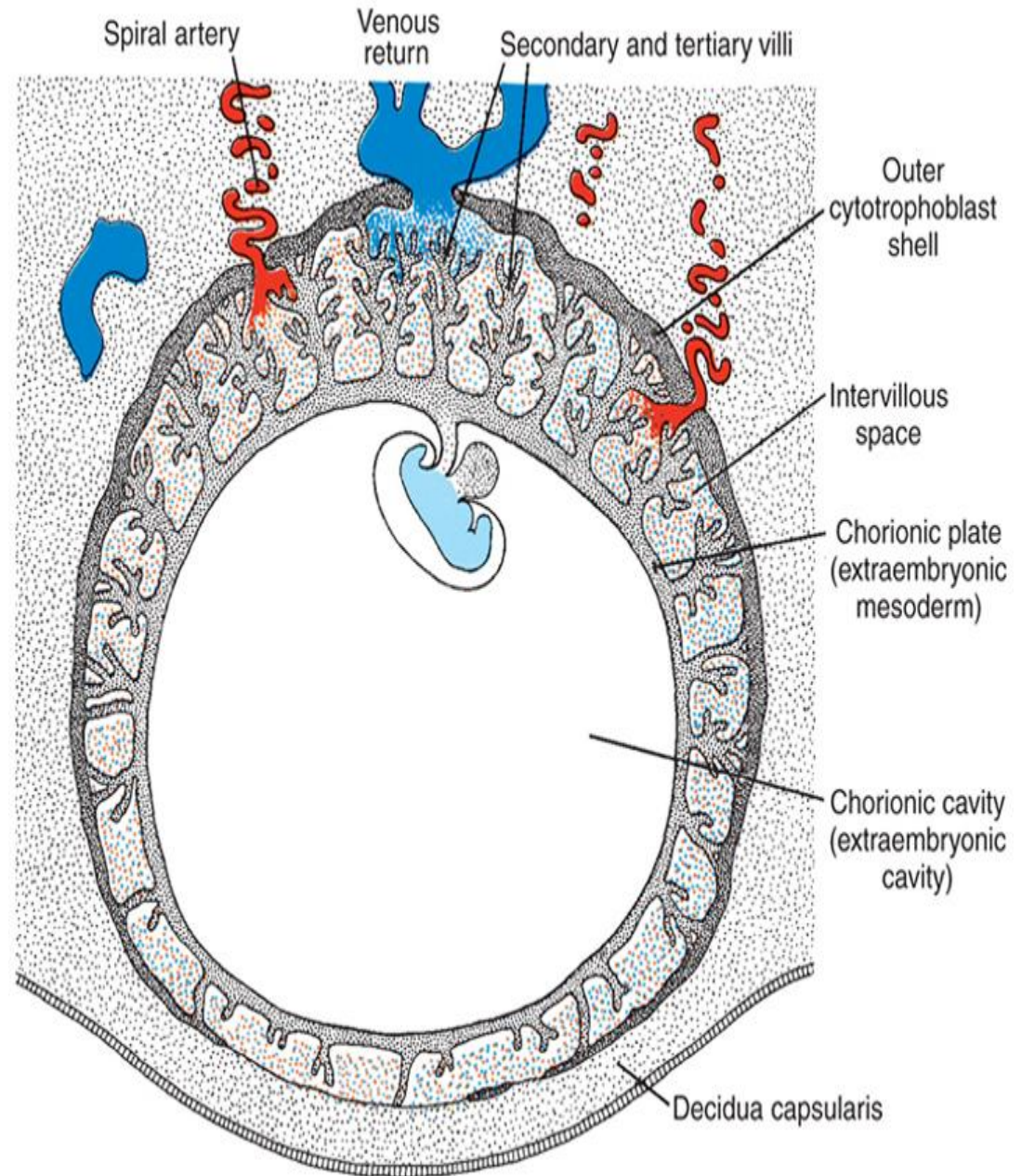
➤ **Capillaries in tertiary villi make contact with capillaries developing in the mesoderm of the chorionic plate and in the connecting stalk**

These vessels, in turn, establish contact with the intraembryonic circulatory system, connecting the placenta and the embryo



Maternal blood is delivered to the placenta by spiral arteries in the uterus

➤ During the following months, numerous small extensions grow out from existing stem villi and extend as free villi into the surrounding lacunar or intervillous spaces.



The placental membrane, which separates maternal and fetal blood, is initially composed of four layers:

- (1) the endothelial lining of fetal vessels
- (2) the connective tissue in the villus core
- (3) the cytotrophoblastic layer
- (4) the syncytium



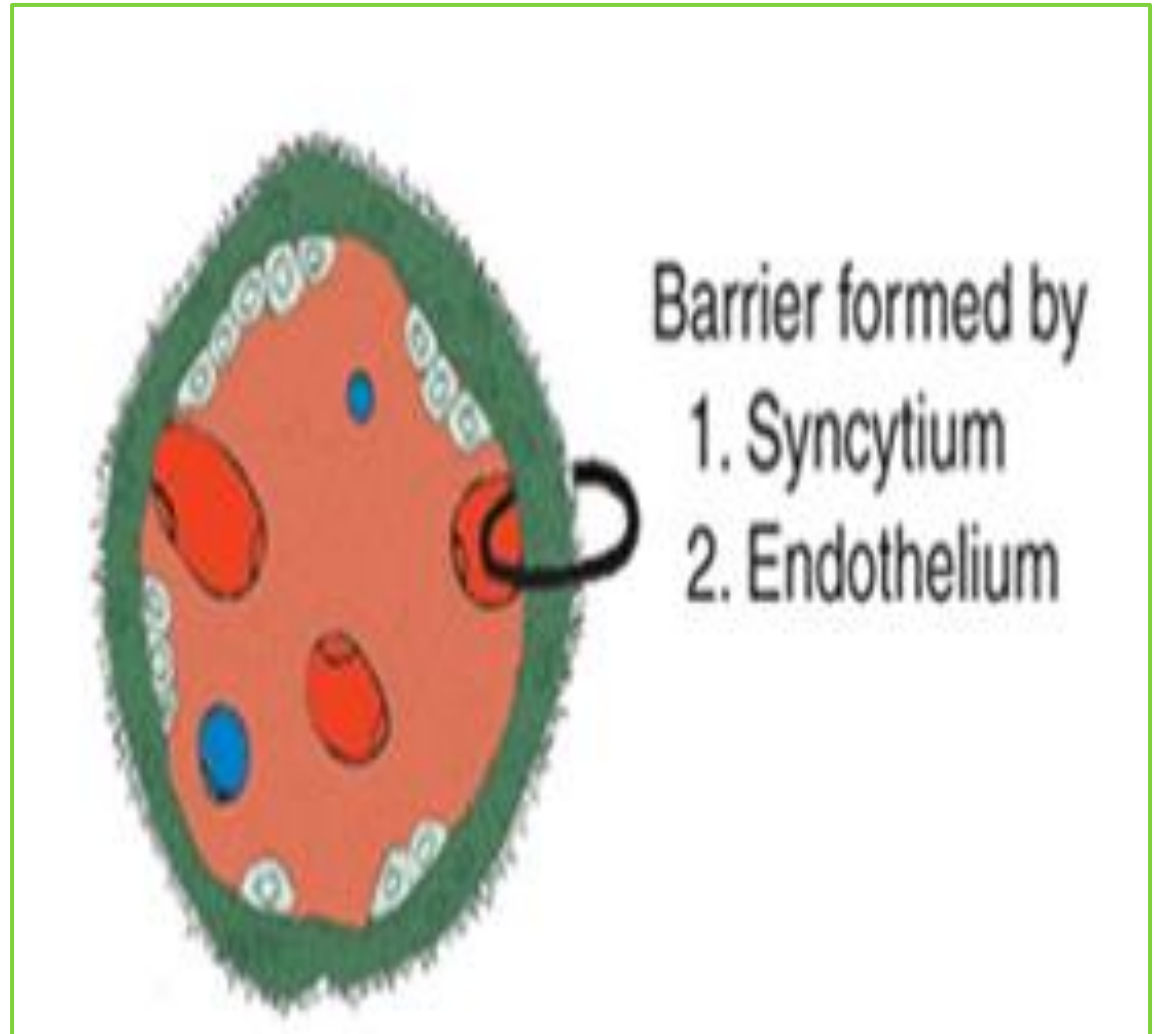
Barrier formed by

1. Syncytium
2. Cytotrophoblast
3. Connective tissue
4. Endothelium

FROM THE FOURTH MONTH ON

The placental membrane **thins** because the **endothelial lining of the vessels comes in intimate contact with the syncytial membrane**, greatly increasing the rate of exchange

Some times called the **placental barrier**, the placental membrane is not a true barrier, as many substances pass through it freely

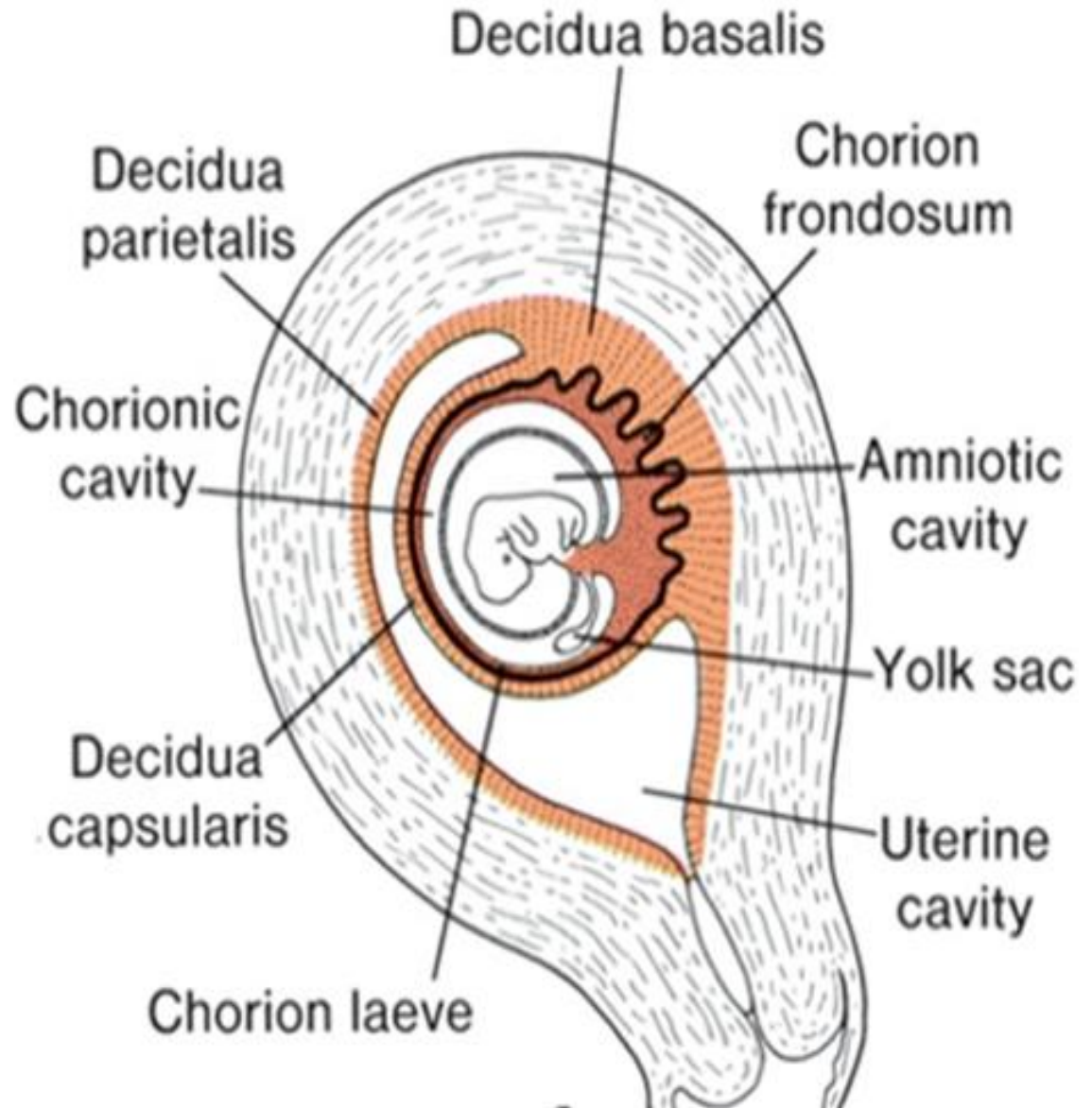


By the beginning of the fourth month, the placenta has two components:

(1) a fetal portion, **formed by the chorion frondosum**

(2) a maternal portion, formed by **the decidua basalis**

In the junctional zone, trophoblast and decidual cells intermingle.



PLACENTA

```
graph TD; A[PLACENTA] --> B[Two component]; B --> C[Fetal]; B --> D[Maternal]; C --> E["is derived from the trophoblast and extraembryonic mesoderm<br/>CHORION<br/>FRONDOSUM"]; D --> F["The maternal component is derived from the<br/>Uterine endometrium<br/>DECIDUA BASALIS"];
```

Two component

Fetal

is derived from the trophoblast and
extraembryonic mesoderm

CHORION
FRONDOSUM

Maternal

The maternal component is derived from the

Uterine endometrium
DECIDUA BASALIS

Decidua

➤ Decidua: (is the structure that will separate)

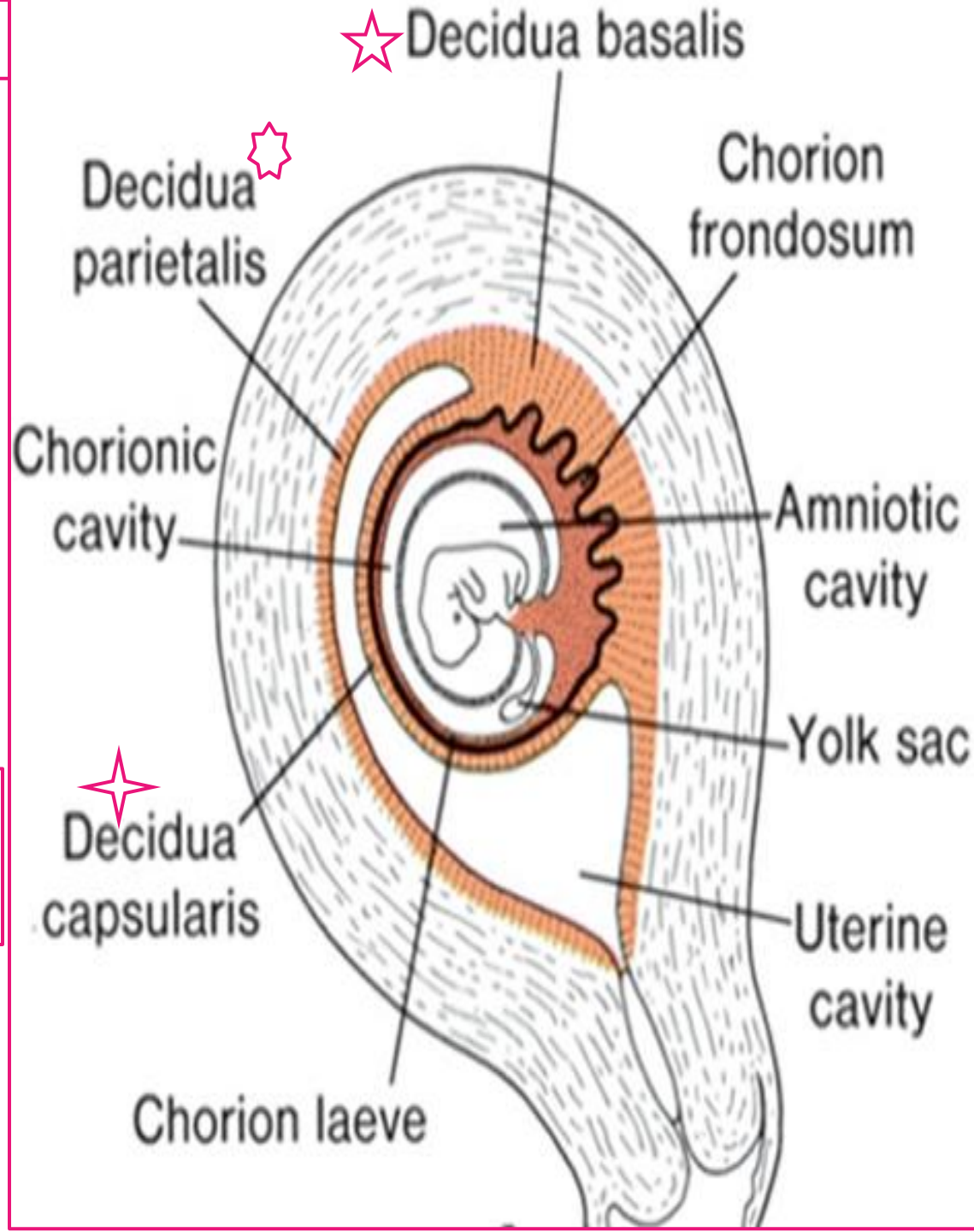
➤ is the endometrium after implantation

Parts:

➤ Decidua basalis: under the implantation site

➤ Decidua capsularis: between the implantation site and the uterine lumen

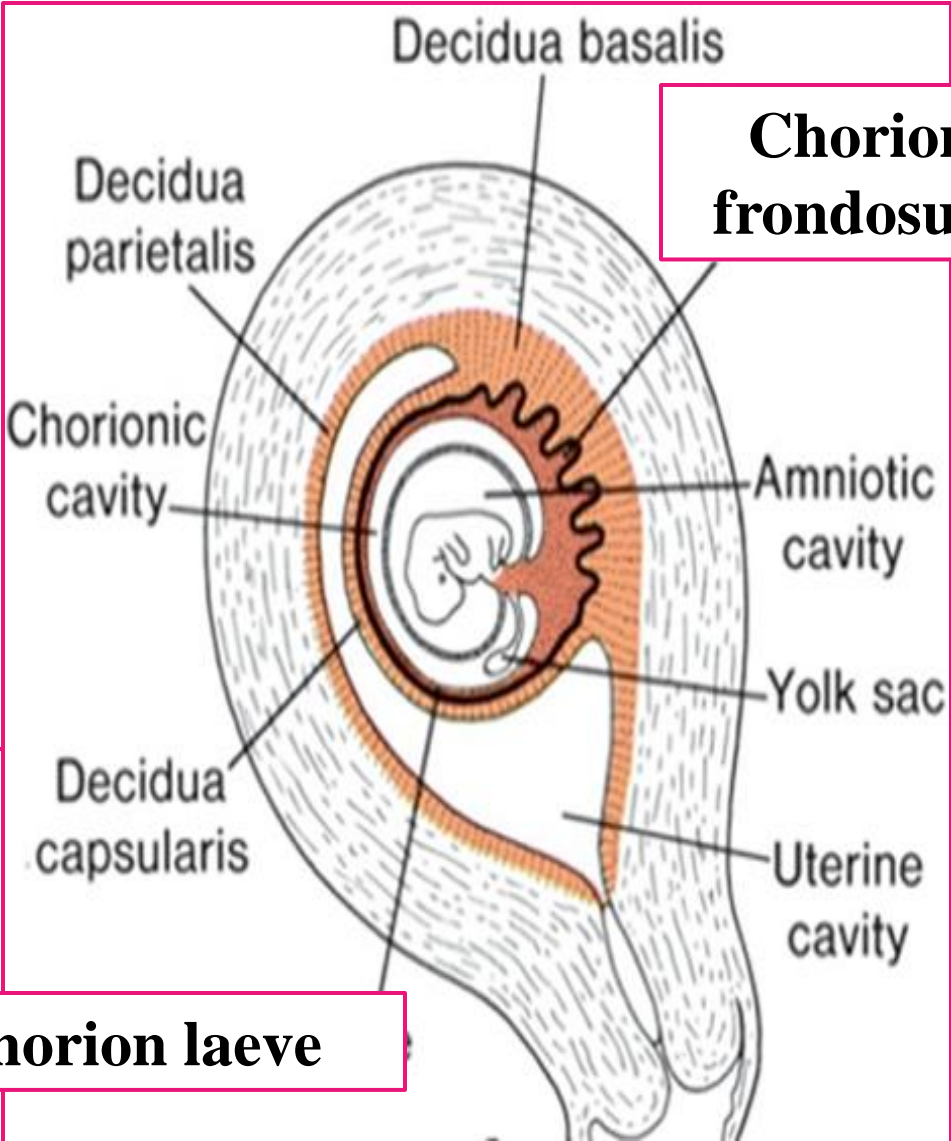
➤ Decidua parietalis: remaining endometrium



PARTS OF CHORION

1-Chorion
laeve:
adjacent to

**2-Chorion
frondosum:
adjacent to**

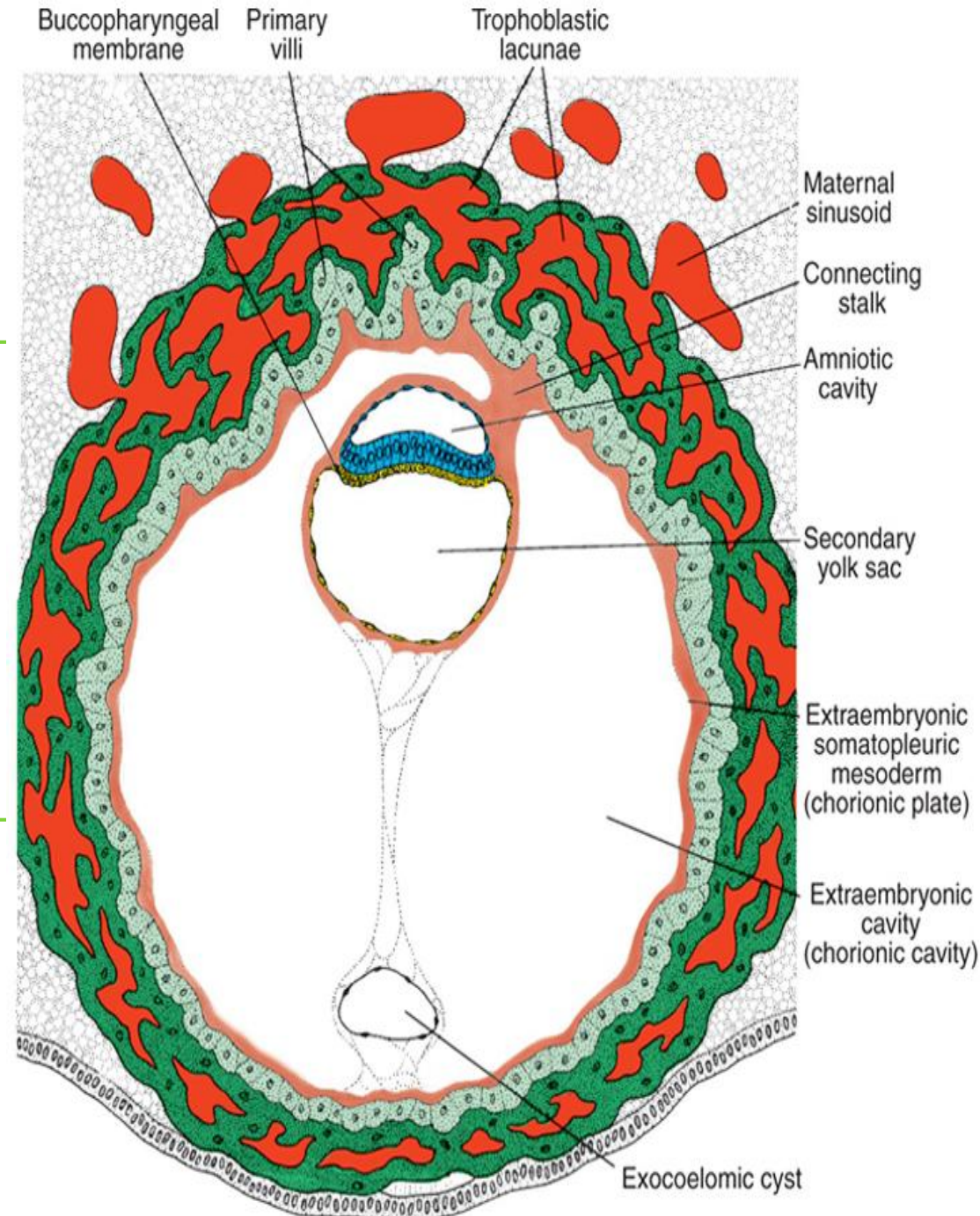


Chorion frondosum

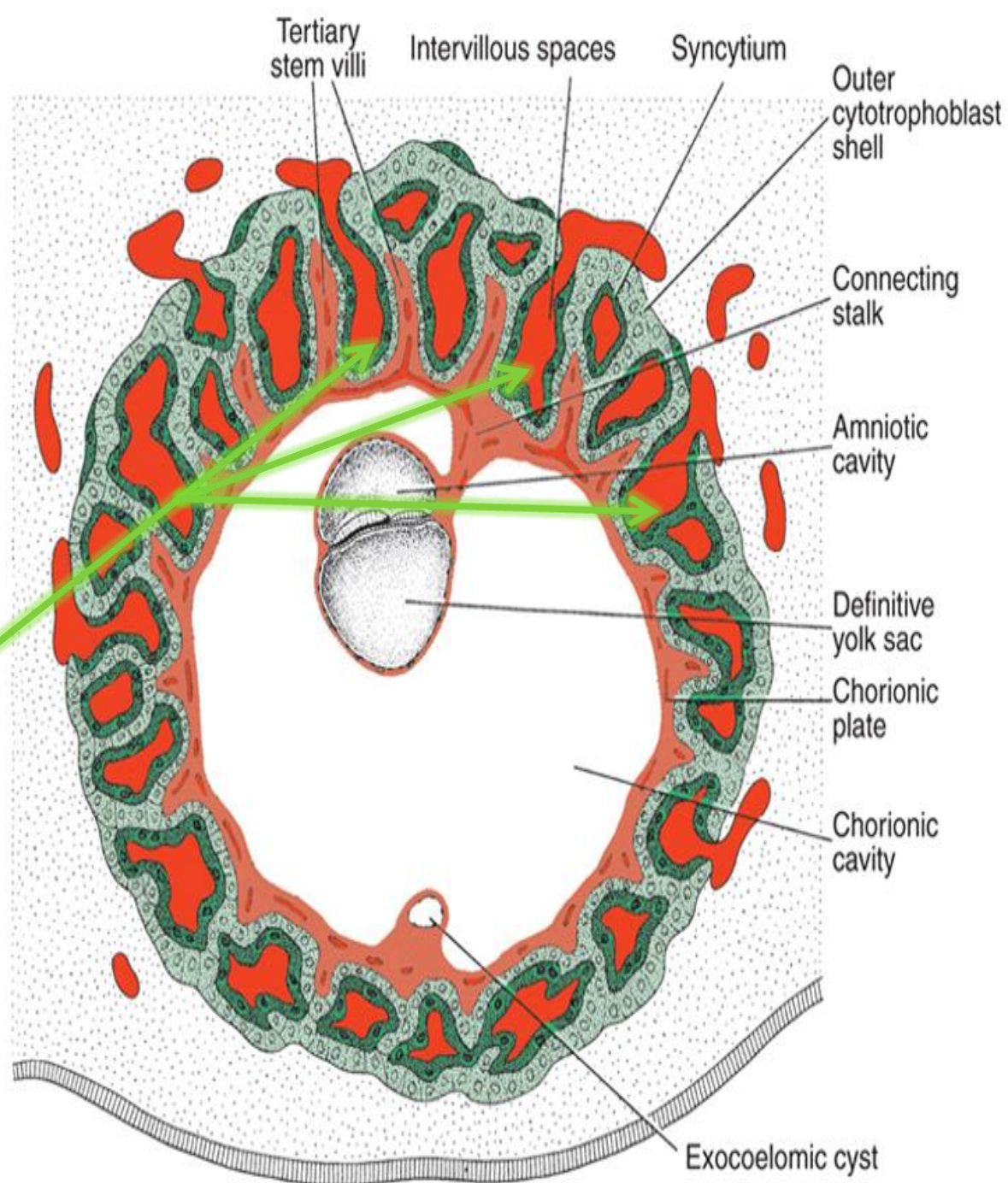
Chorion laeve

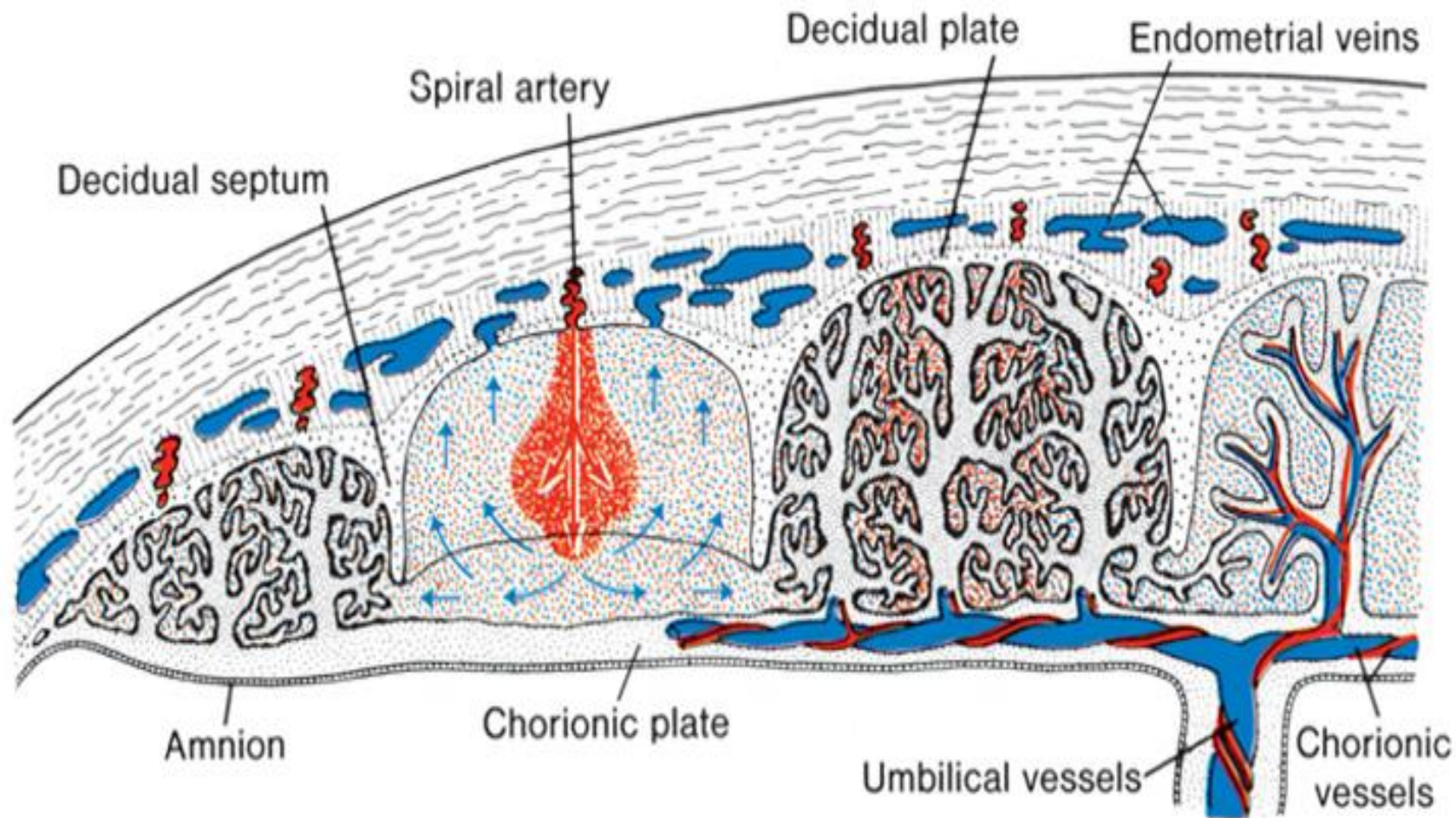
REMEMBER

**Chorionic plate :
extraembryonic
mesoderm
+
trophoblast**

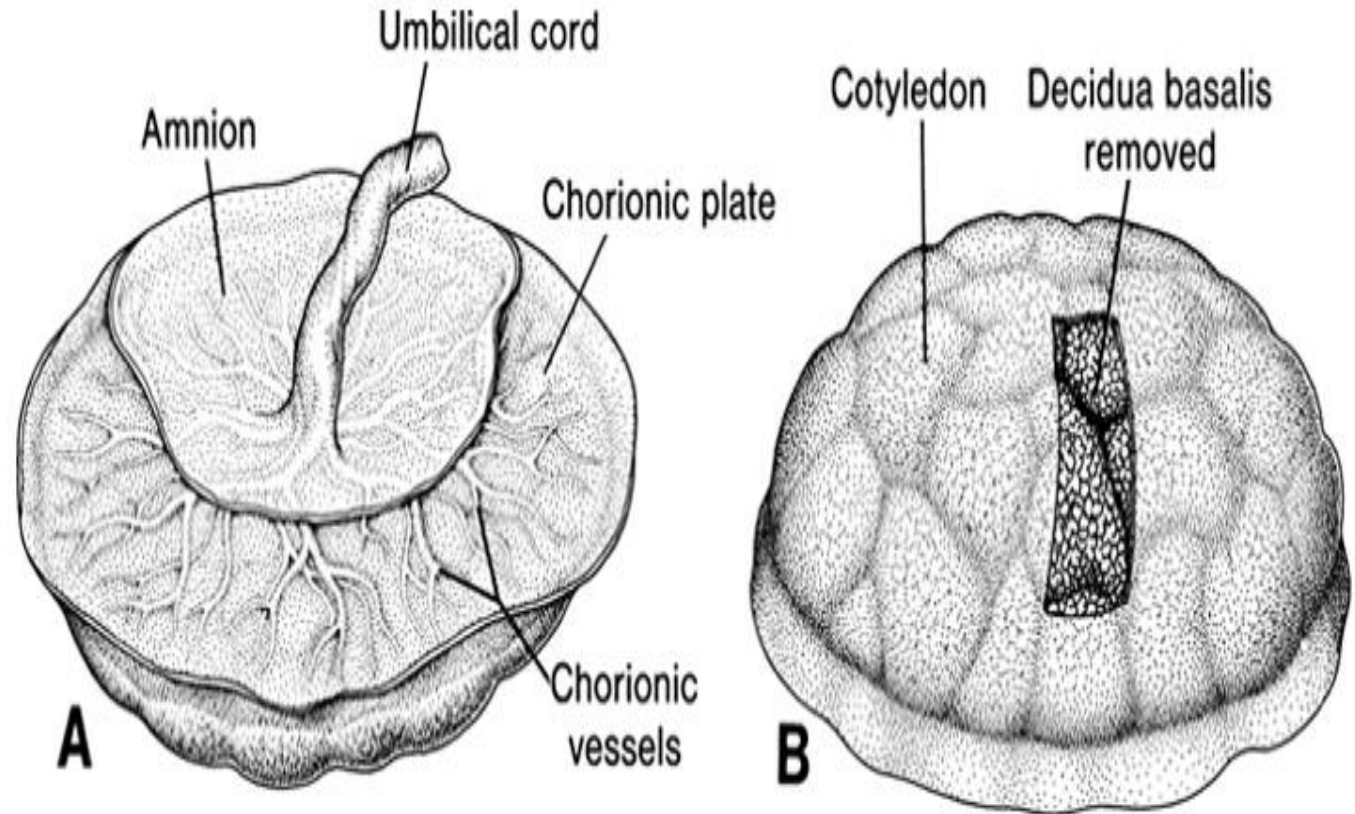


most cytotrophoblast cells have degenerated. Between the chorionic and decidual plates are the **intervillous spaces**, which are filled with maternal blood.





During the fourth and fifth months, the decidua forms a number of decidual septa, which project into intervillous spaces but do not reach the **chorionic plate**



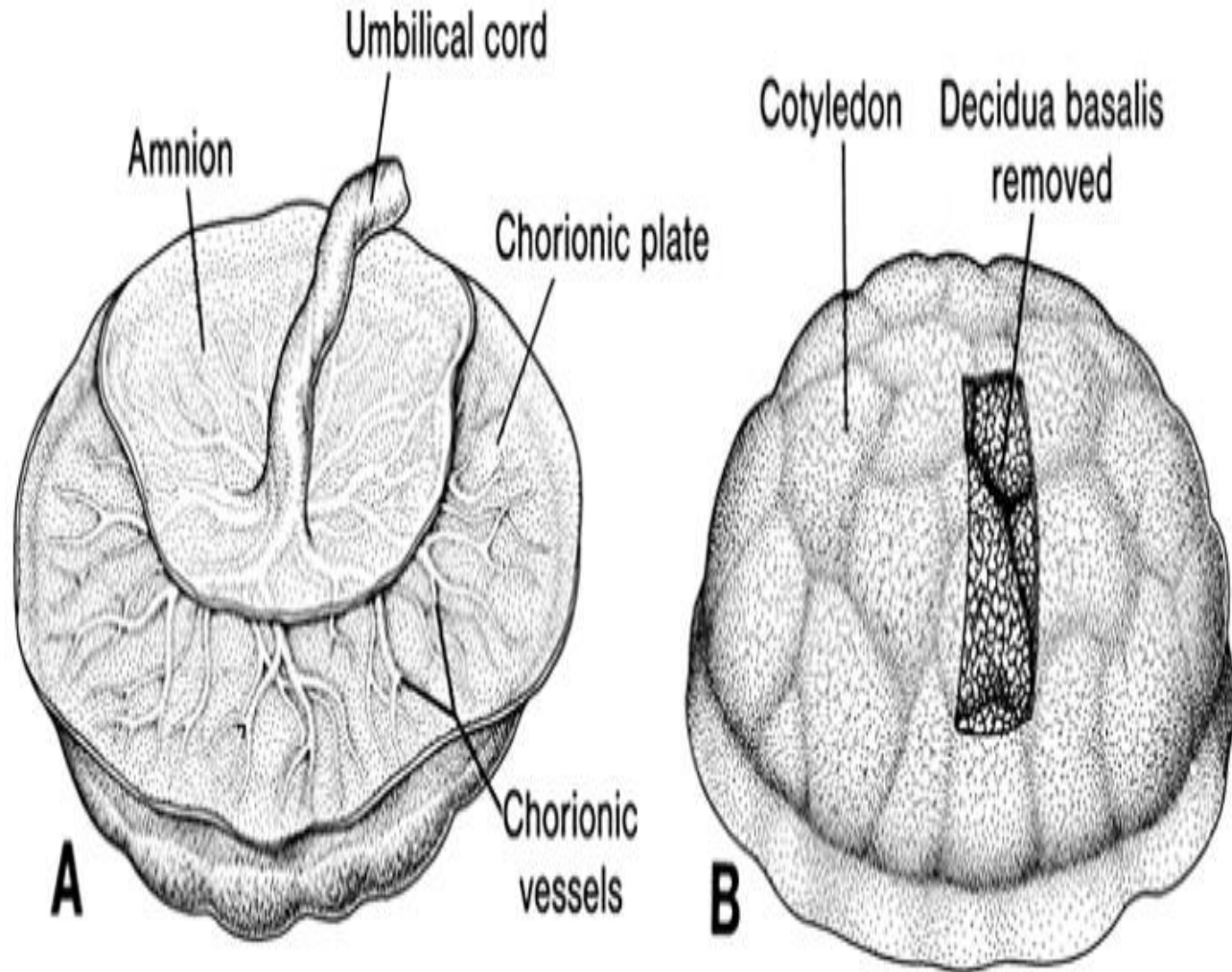
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As a result of this septum formation, the placenta is divided into a number of compartments, or **Cotyledons**

At full term

- the placenta is discoid with a diameter of 15 to 25 cm
- is approximately 3 cm thick, and weighs about 500 to 600 g
- approximately 30 minutes after birth of the child, is expelled from the uterine cavity as the afterbirth.

When the placenta is viewed from the maternal side, 15 to 20 slightly bulging areas, the cotyledons, covered by a thin layer of decidua basalis, are clearly recognizable



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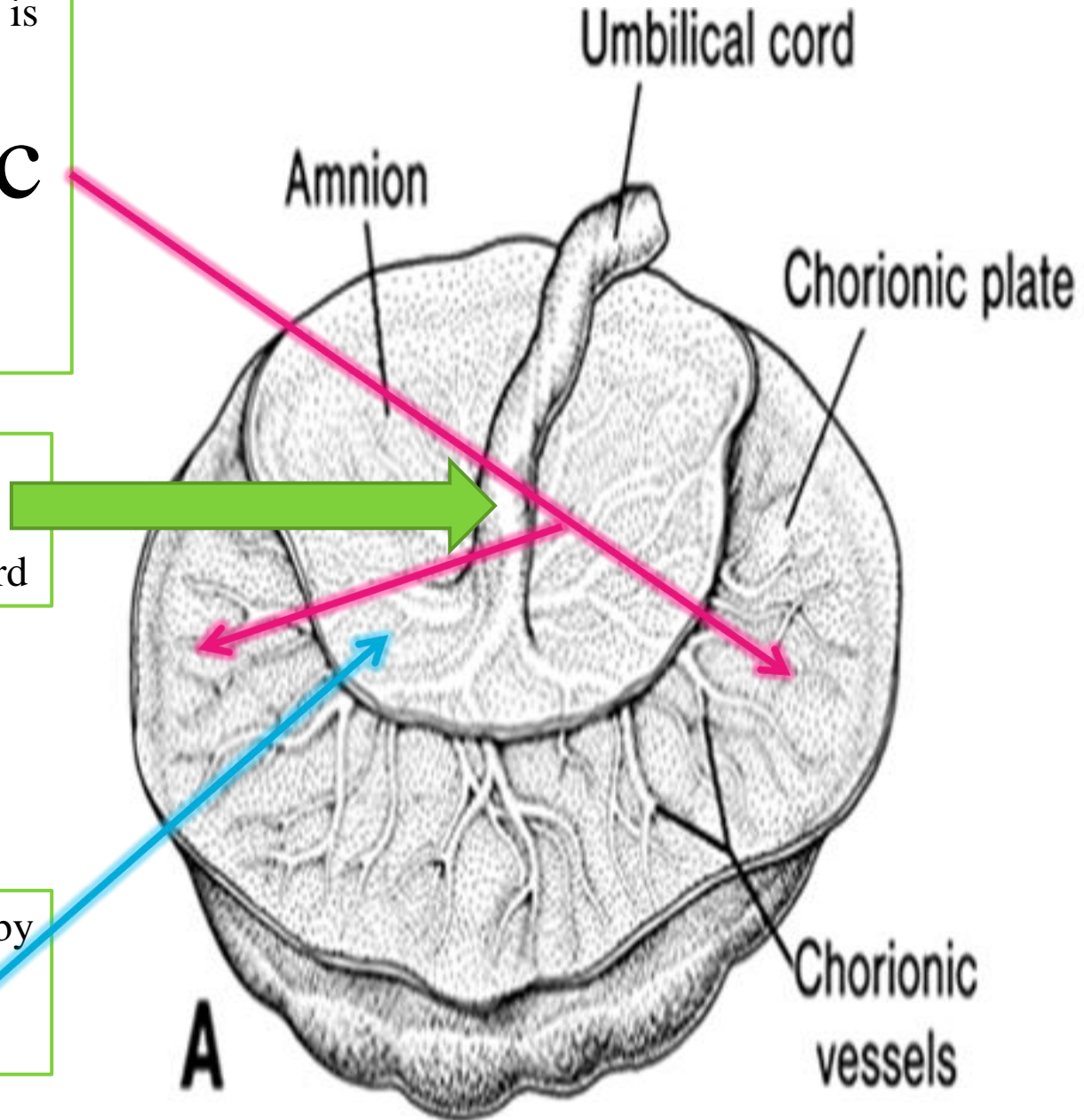
The fetal surface of the placenta is covered entirely by

the chorionic plate

A number of large arteries and veins, the chorionic vessels, converge toward the umbilical cord

The chorion, in turn, is covered by

the amnion



Placenta previa: the implantation of the placenta at lower uterine segment

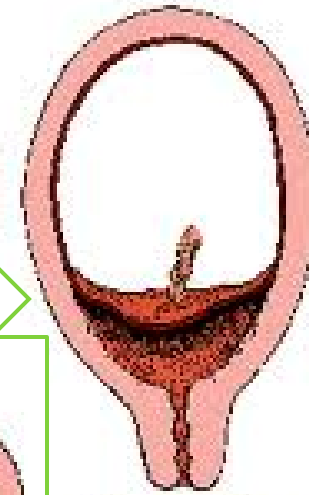
There are actually three types of previa

Placenta previa centralis (Complete previa)
Completely covers the internal cervical os

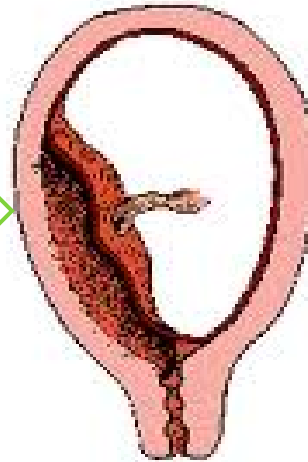
Placenta previa marginalis
covers the internal os
partially

Placenta
previa
lateralis
Does not
approach the
internal os

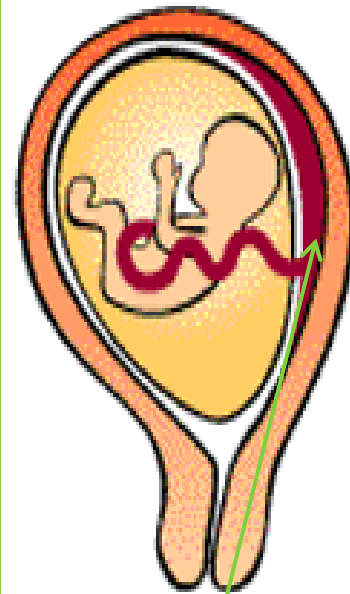
Lateralis



Complete



Marginal



**Normal
Placenta**

vaginal examination can occasionally trigger heavier bleeding that, if severe enough, would mean an emergency Caesarean. That is why the procedure is normally carried out in the **operating theatre**, so that the operation can be performed quite quickly, if needed.

BLEEDING DURING LATE STAGES IN PREGNANCYSUSPECT AND THINK ABOUT Placenta previa..... be careful when thinking of vaginal examination...

Main functions of the placenta are

(a) exchange of gases

(b) exchange of nutrients and electrolytes

(c) transmission of maternal antibodies, providing the fetus with passive immunity

(d) production of hormones, such as progesterone, estradiol, and estrogen
(in addition, it produces hCG and somatomammotropin)

(e) detoxification of some drugs.

(a) exchange of metabolic and gaseous products between maternal and fetal bloodstreams

(b) Transmission of Maternal Antibodies

Immunological competence begins to develop late in the first trimester, by which time the fetus makes all of the components of **complement**.

Immunoglobulins

consist almost entirely of **maternal immunoglobulin G (IgG)**

that begins to be transported from mother to fetus at approximately 14 weeks.

In this manner, the fetus gains passive immunity against various infectious diseases. Newborns begin to produce their own IgG, but adult levels are not attained until the age of 3 years.

Read only

(c) Hormone Production

1-During the first two months of pregnancy, the syncytiotrophoblast produces human chorionic gonadotropin (hCG), which maintains the corpus luteum. This hormone is excreted by the mother in the urine, and in the early stages of gestation, its presence is used as an indicator of pregnancy

2-estrogenic hormones, predominantly estriol, until just before the end of pregnancy, when a maximum level is reached. These high levels of estrogens stimulate uterine growth and development of the mammary glands.

Note:all hormones are synthesized in the syncytial trophoblast.

Read only

3-By the end of the fourth month the placenta produces progesterone in sufficient amounts to maintain pregnancy if the corpus luteum is removed or fails to function properly

4-Somatomammotropin (formerly placental lactogen). It is a growth hormone-like substance that gives the fetus priority on maternal blood glucose and makes the mother somewhat diabetogenic. It also promotes breast development for milk production.

(d)The Placental as a Barrier

Most maternal hormones do not cross the placenta. The hormones that do cross, such as thyroxine, do so only at a slow rate

Although the placental barrier is frequently considered to act as a protective mechanism against damaging factors, many viruses, such as:

Rubella

cytomegalovirus

Coxsackie

Varicella

Measles

poliomyelitis virus



traverse the placenta without difficulty. Once in the fetus, some viruses cause infections, which may result in cell death and birth defects



Teratology

The most sensitive period for inducing birth defects is the third to eighth weeks of gestation, the period of embryogenesis.

Manifestations of abnormal development are
death
malformation
growth retardation
functional disorders

Infectious Agents

Rubella used to be a major problem, but the ability to detect serum antibody titers and development of a vaccine have significantly lowered the incidence of birth defects from this cause. Today approximately 85% of women are immune.

Toxoplasmosis and syphilis cause birth defects. Poorly cooked meat; domestic animals, **especially cats**

Malformations following maternal infection with

Measles

mumps

hepatitis

Poliomyelitis

ECHO virus

Coxsackie virus

influenza virus

is low if not nonexistent

thalidomide, an antinauseant

and sleeping pill. In 1961 it was noted in West Germany that the frequency of **amelia and meromelia (total or partial absence of the extremities)**, a rare hereditary abnormality, had suddenly increased . This observation led to examination of the prenatal histories of affected children and to the discovery that many mothers had taken thalidomide early in pregnancy

Read only

Cocaine has been reported to cause

a number of birth defects, possibly due to its action as a vasoconstrictor that causes hypoxia.

Isotretinoin (13-*cis*-retinoic acid), an analogue of

vitamin A, has been

shown to cause a characteristic pattern of malformations known as the

isotretinoin embryopathy or vitamin A embryopathy

Cigarette smoking has not been linked to major birth defects, but it does contribute to intrauterine growth retardation and premature delivery. There is also evidence that it causes behavioral disturbances.

alcohol ingestion

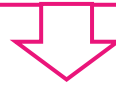
There is a well-documented association between maternal **alcohol ingestion** and **congenital abnormalities**, and these defects, together with mental retardation and growth deficiency, make up the **fetal alcohol syndrome (FAS)**. **Even moderate alcohol** consumption during pregnancy may be detrimental to embryonic development. **The central nervous system** is particularly sensitive to alcohol, and **alcohol-related neurodevelopmental disorder (ARND)** may result from exposure. The incidence of FAS and ARND together is 1 in 100 live births. Furthermore,

alcohol is the leading cause of mental retardation.

Diabetes.

Disturbances in carbohydrate metabolism during pregnancy in diabetic mothers cause a high incidence of

- stillbirths (when the fetus has died in the uterus)
- Abnormally large infants
- congenital malformations



The risk of congenital anomalies in children of diabetic mothers is 3 to 4 times that for the offspring of nondiabetic



The variety of observed malformations includes caudal dysgenesis (sirenomelia).