The trophoblast differentiates into two layers:

1. *an inner* layer the CYTOTROPHOBLAST
2. *an outer* zone the SYNCYTIOTROPHOBLAST
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.
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**
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.
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**.
These vessels, in turn, establish contact with the intraembryonic circulatory system, connecting the placenta and the embryo.

- Capillaries in tertiary villi make contact with capillaries developing in the mesoderm of the chorionic plate and in the connecting stalk.
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
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.
Cytotrophoblastic cells in the villi penetrate progressively into the overlying syncytium until they reach the maternal endometrium. Here they establish contact with similar extensions of neighboring villous stems forming a thin outer cytotrophoblast shell.

This shell gradually surrounds the trophoblast entirely and attaches the chorionic sac firmly to the maternal endometrial tissue.

Villi that extend from the chorionic plate to the decidua basalis (decidual plate: the part of the endometrium where the placenta will form) are called Stem or anchoring villi. Those that branch from the sides of stem villi are free (terminal) villi, through which exchange of nutrients and other factors will occur.
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.
The placenta is a two-component organ that connects the fetus to the maternal uterine endometrium. The fetal component is derived from the trophoblast and extraembryonic mesoderm, specifically the chorion frondosum. The maternal component is derived from the uterine endometrium, specifically the 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
1-Chorion laeve: adjacent to

2-Chorion frondosum: adjacent to

Chorion laeve

Chorion frondosum
Chorionic plate: extraembryonic mesoderm + trophoblast
most cytотrophoblast 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.

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.
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.
There are actually three types of previa:

1. **Placenta previa centralis (Complete previa)**: Completely covers the internal cervical os.

2. **Placenta previa marginalis**: Covers the internal os partially.

3. **Placenta previa lateralis**: Does not approach the internal os.

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

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 PREGNANCY …..SUSPECT 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.
(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.
(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.
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-Somatotrophin (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.
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**
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.

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).