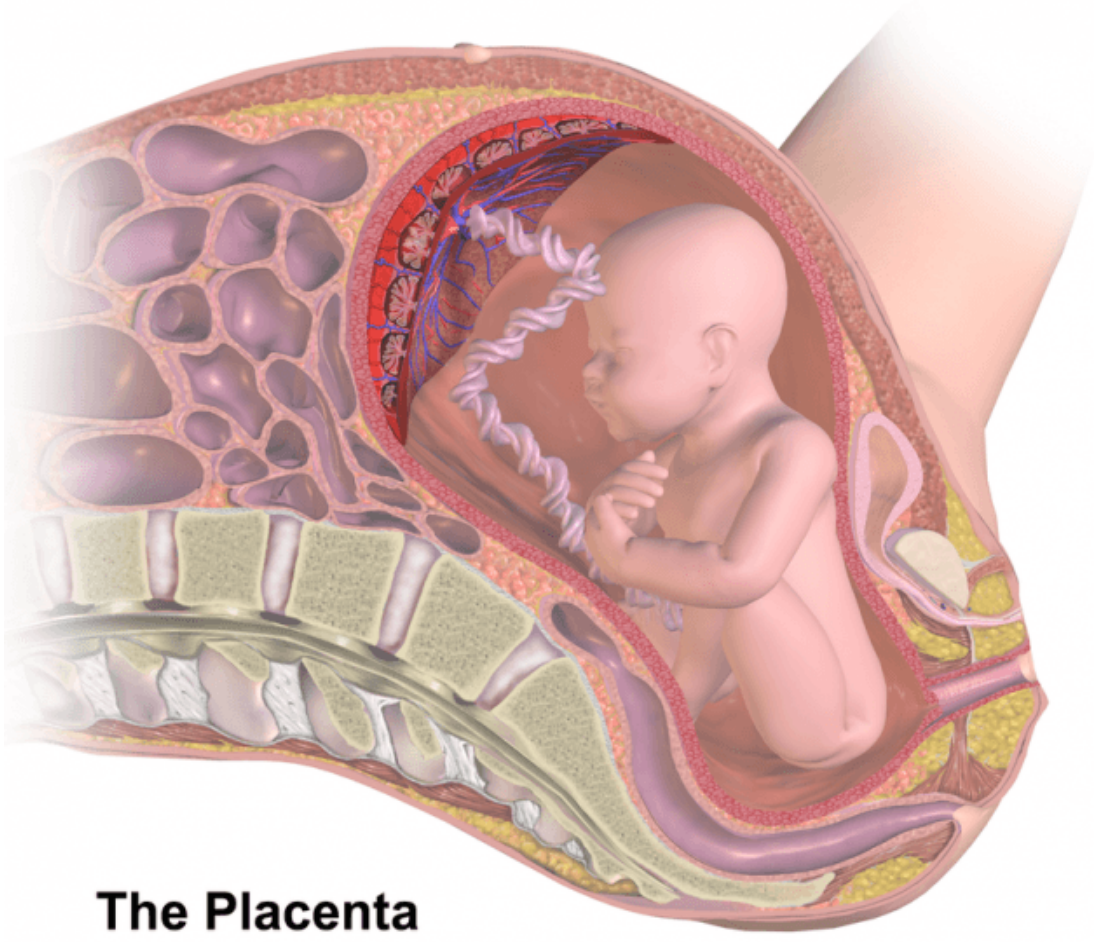
Placenta – Functions and Insufficiency

See online here

During pregnancy, the placenta develops and adapts to the needs of the child. The placenta fulfils numerous tasks, but mainly serves as the source of the child's supply of oxygen and nutrients. Due to various causes, a malfunction is possible, whereby the child is under-supplied and can no longer grow properly. Early diagnosis along with prompt therapeutic measures are therefore important.



The Placental Circulation

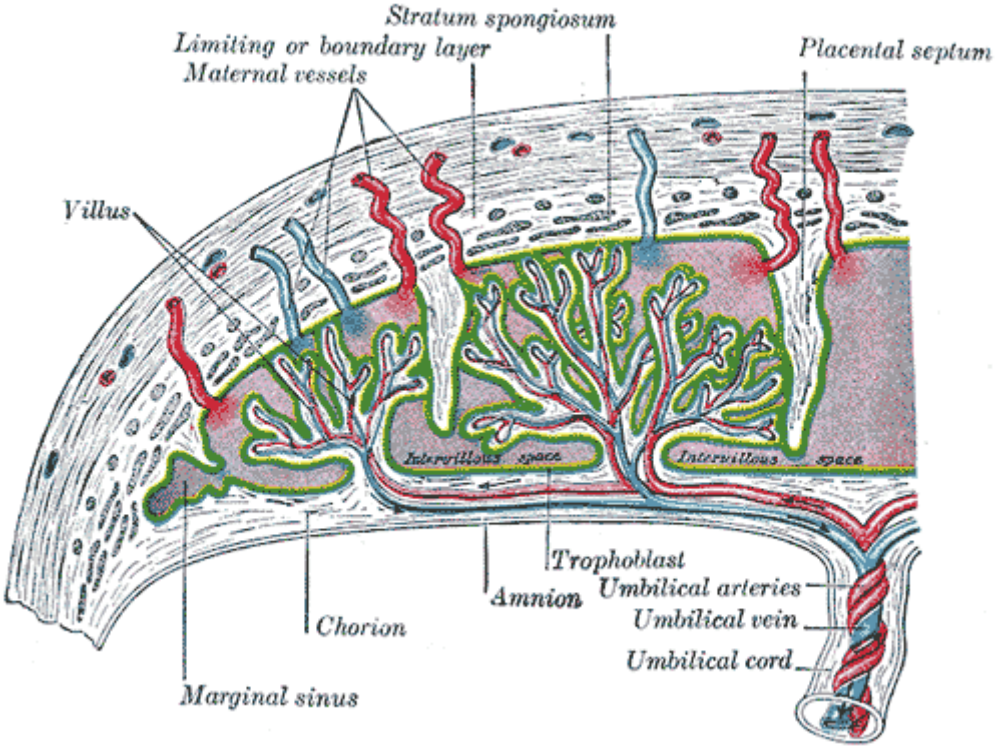


Image: Maternal blood fills the intervillous space. Nutrients, water, and gases are actively and passively exchanged, then deoxygenated blood is displaced by the next maternal pulse. By Henry Gray, License: Public Domain

The placenta is a connection between mother and child. The exchange of gas and substances takes place via the so-called **placental barrier**. This is created from the end of the 3rd gestation week from the endothelium of the fetal blood vessels, the mesoderm, and the cyto and syncytiotrophoblast. This barrier ensures that there is, at no time, a direct link between the blood circulation of mother and child. The exchange surface of the placenta is the surface of the chorionic tree. Maternal blood flows around this surface and facilitates the **exchange of substances** with it.

The maternal placenta is then expelled after the termination of pregnancy or after birth.

Placental Function

The placenta has, in addition to the **metabolic** function, an **endocrine** and **immunological** function.

A major task is the **exchange of substances** through the placental barrier. Most substances are transported in accordance with their concentration gradient. Other molecules, such as water, lactate, and glucose, are transported actively.

During pregnancy, the placenta represents another major, endocrine gland. It is related to the mother’s pituitary gland and the childish adrenal glands. As the placenta is not hormonally active at the beginning of early pregnancy, hormone production is maintained 1st by the ovaries and the pituitary. They produce steroids (**estrogen and progesterone**), human chorionic gonadotropin (**hCG**), and human placental lactogen (**hPL**).

The **estrogen concentration** increases steadily in the course of pregnancy. The hormone causes the **uterus growth** and supports at the end of pregnancy the **birth preparedness**. **Progesterone** is an important hormone during early pregnancy. In the

early weeks of pregnancy, progesterone is produced by the *corpus luteum graviditis*; this function is then usually taken over before the 12th week of pregnancy by the syncytiotrophoblast. The progesterone **lowers** the **uterine muscle tonus**.

The **hCG** is produced by **syncytiotrophoblasts**. The hormone concentration rises continuously until the end of the 1st trimester. In the earliest weeks of pregnancy, the hCG concentrations should double every 2 days.

The **hPL** is also formed by the trophoblast. It has structural similarity to the somatotropin and thus has growth hormone-like effects. Besides, it has an **insulin-stimulating effect**. On the 1 hand, this promotes the supply of glucose and amino acids – on the other hand, this is often the cause of **gestational diabetes**.

The placenta also has an immunological function. It favors the supply of the child with **maternal immunoglobulin G (IgG)**. Even after the child's birth, the immunoglobins exist in the blood of the child.

Image: Maternal side of a whole human placenta, just after birth. By תמרה דהן ⍰ דולה, License: CC BY-SA 3.0

The Placental Insufficiency

The placental insufficiency corresponds to an **insufficient supply of nutrients to the child**. The placental insufficiency can be classified into **acute and chronic forms**.

The acute form occurs after a few minutes or hours and the chronic placental insufficiency is noted after weeks to months; the chronic form corresponds with **intrauterine growth retardation** or lack of development.

Pathogenesis of placental insufficiency

The causes of a malfunction of the placenta in the acute form may be: **a vena cava compression syndrome** or intrapartum an **inferior placental abruption**.

The chronic placental insufficiency arises in **maternal systemic diseases** such as anemia, diabetes mellitus or the presence of uterus myomatosus. In addition, **pregnancy-related disorders** play a role, including the multi-parity, rhesus incompatibility, and abortions.

Clinic of placental insufficiency

Symptoms of acute placental insufficiency **depend on the cause**. In placental abruption, severe pain right up to board hard uterus is possible. Typically, it occurs in acute placental insufficiency to a change of the cardiotocography (**CTG)**.

The chronic placental insufficiency is less associated with maternal symptoms but more with leading the child to a **growth restriction** and **intrauterine asphyxia**. The symptoms may develop up to the sudden death of the child.

Diagnosis of placental insufficiency

The diagnosis of acute placental insufficiency is provided by the CTG. Typical findings are **late decelerations**, a **silent type of oscillation** or persistent bradycardia. Frequently, sonography is performed to confirm the diagnosis.

The chronic placental insufficiency is confirmed by sonography. During sonography, particular attention is given to the amount of amniotic fluid present. A **small amount of amniotic fluid** would be a typical indicator.

Therapy of placental insufficiency

Treatment is initially **causally** and causes of the insufficiency should be eliminated. The choice of treatment also depends on the week of pregnancy.

Acute causes can be eliminated, e.g., by a left lateral position (vena cava compression syndrome). In chronic forms, the risk and benefit should be weighed. Among other things, this involves the risk of prematurity against the risk of a chronic shortage supply. Accordingly, it is then decided whether a section is recommended or not.

References

Berven, E., & Freberg, A. (2010). *Human placenta: Structure and development, circulation, and functions*. New York: Nova Biomedical Books.

Costa, S., Proctor, L., Dodd, J., Toal, M., Okun, N., Johnson, J., ... Kingdom, J. (2008). Screening for Placental Insufficiency in High-risk Pregnancies: Is Earlier Better? *Placenta*, *29*(12), 1034-1040. doi:10.1016/j.placenta.2008.09.004

Giardi, G. (2017). Placental insufficiency and the fetal brain. *Placenta*, *57*, 230. doi:10.1016/j.placenta.2017.07.033

Gruenwald, P. (1975). *The Placenta and its maternal supply line: Effects of insufficiency on the fetus*. Lancaster, England: MTP Press.

Saito, S. (1994). Placental functions and cytokines. *Placenta*, *15*(7), A87. doi:10.1016/0143-4004(94)90213-5

Notes