tourinary system. The cercariae of S. *mansoni,* which emerge from snails, penetrate the skin to cause an itchy eruption in previously exposed individuals. About 8 weeks after infection Katayama fever with chills, pyrexia, sweating, headache, cough, hepatosplenomegaly and lymphadenopathy occurs. The worms may cause pseudotuberculous granulomas to develop in the liver but ova are mainly deposited in the colon and rectum. Complications from S. *mansoni* infection include: hepatic fibrosis leading to portal hypertension, and portal, mesenteric and vertebral vascular inflammation and fibrosis with subsequent involvement of the pulmonary vascular bed leading to cor pulmonale or neurological deficit and membrane-proliferative glomerulonephritis from circulating immune complexes (19).

Cestode infestations are less common in children than in young adults. The commonly occurring tapeworms are Taenia *soilum* (pork tapeworm) and the Echinococcus species. Cysticercosis due to *T sollum* affects diverse tissues but especially the brain, heart and muscles when autointection occurs from ova being passed in the feces. In *Echinococcus* infestation (hydatid disease) the embryos from ingested eggs very quickly pass through the gastrointestinal tract to the portal circulation to be filtered out in the liver, lungs and other sites where classical hydatid cysts containing larvae appear.

Infestation by the dwarf tapeworm *Hymenolepis nana* is commonly seen by microbiologists examining wet preparations of stool but the worms are rarely recognized by anatomical pathologists because of their small size. Heavy infestation may cause diarrhea and abdominal pain and constitutional symptoms associated with moderate eosinophilia. The cestode requires a single host but also infects mice, fleas and beetles (19).

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Examination of the placenta in late intrauterine death: What can we tell about cause and recurrence risk?

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During the past decades, there has been considerable advancement in the care of a fetus and its mother and, later on, of the newborn. With the great reduction of overall perinatal mortality in highly developed countries we have come to a stage where 50% of all perinatal losses are constituted by intrauterine death affecting a viable fetus older than 26 weeks of gestation. Severe fetal malformations are already excluded from this number, as are the few deaths that are caused by obvious bacterial or viral infections. Thus, the majority of fetal demise in the third trimenon is due to some kind of placental insufficiency that occurs in two quite different clinical settings and which can be described as "chronic" or "acute" placental insufficiency.

"Chronic" placental insufficiency

Chronic insufficiency comprises all conditions in which reduced placental function is reflected by fetal growth retardation. Obviously there are cases where the growth of a child for genetic reasons is below the 5th percentile and the placenta is small simply because the corresponding child is small. Growth retardation proper is characterized by a declining curve of fetal growth caused by *in* utero starving: it is thus necessarily "chronic". Starvation itself can be compatible with fetal survival for periods up to 6 and even 8 weeks. In general, compromise of the maternal circulation is better tolerated, especially when it occurs in "succession" as opposed to the compromise of fetal circulation, which is also much more ominous in regard to the underlying disease condition. *Post mortem* findings in the fetus will reveal severe reduction in the weight of internal organs, especially the liver, thymus and spleen with some growth parameters remaining around the 50th percentile for a long time, especially brain weight and foot length. The placenta of a growth-retarded child is usually characterized by areas that have ceased to function. These usually constitute gross morphological findings of a focal nature, the most typical being infarcts of different age, size and location. It is important to estimate the volume of placental tissue lost and to denote whether all infarcts have the same appearance (thus having occurred eventually all at the same time) or whether they exhibit different color and Consistency (giving the impression that they have occurred in succession). The most typical accompanying microscopic finding is an increased vascularization of the chorionic villi which have remained viable and which thus try to compensate for the losses. At the time when chronic placental insufficiency leads to fetal malnutrition, many of the mothers are also unhealthy; they have symptoms of preeclampsia, are

found to smoke or to be addicted to other drugs or are found to suffer from acquired or hereditary thrombophilic conditions.

"Acute" placental insufficiency

Acute insufficiency is the term used when fetal death is the first noted symptom of a placental disturbance. Thus, neither the intrauterine growth curve nor the *post mortem* examination of the fetus indicate *in utero* starvation. Fetuses that have succumbed to acute placental insufficiency are mostly around the 50th percentile when their age-related weights and measurements are taken. Many of them will show petechial bleedings at the surface of the lungs, heart and thymus as well as congestion of the meninges. Although the pathophysiology of these findings is not quite understood, they are interpreted as signs and symptoms of hypoxia. The corresponding placenta is mostly perfect in size and shape and does not exhibit focal changes. However, the color of the cut surface is paler (resembling in this regard a placenta of 20 weeks or less) and does not exhibit the dark red appearance that is so characteristic of a placenta during the last 4-6 weeks of pregnancy. Microscopic examination will reveal the cause of this reduction in color to be a largely reduced vascularization of the chorionic villi. Mothers are usually completely healthy with the exception of a few who suffer from diabetes mellitus type I.

Details of "acute insufficiency" morphology and pathophysiology

A fetus exhibiting signs of hypoxia dies from suffocation, which is almost always an "acute" condition. However, initiating events leading to reduced vascularity of chorionic villi must date back quite some time. The morphological hallmark of reduced vascularity is found in the terminal villi which during the last 6 weeks of pregnancy are characterized by an increasing number of dilated capillaries seeking close contact with the maternal blood. This sinusoidal transformation leads to thin cytoplasmic membranes, which comprise the only remaining barrier between fetal and maternal blood. It has been postulated, but not proven, that these membranes are the site of oxygen exchange. Assuming that this is the case, placentae with underdeveloped "syncytio-capillary membranes" have not fully developed their respiratory capacity. Nutrition of the fetus is not disturbed as the transport of many nutrients requires active transport by carriers and does not occur at the syncytio-capillary membranes. The disturbance of vascular development presents with two different morphological patterns. One is a concomitant increase of villous stroma and enlarged placental weight, often considered the typical finding in cases of maternal diabetes mellitus, The other features a pure lack of terminal villi. The examination of placentae from normal pregnancies reveals on average three well-developed sinusoids within terminal villi that have formed syncytiocapillary membranes covering about 35% of the circumference of a terminal villus. In contrast, terminal villi of fetuses that have succumbed to *in utero* hypoxia, on average present with less than one transformed capillary and the syncytiocapillary membranes covers at the most 10% of the villous circumference. These differences are highly significant, however, when placentae from children who had shown severe alteration of their cardiotocogram before and during birth were examined again: only one transformed capillary with membrane was found on average at the terminal villi. This indicates that the reduction in vascular maturation is a prerequisite of hypoxia but that it does not necessarily lead to fetal demise. Currently, most fetuses survive this developmental abnormality of the placenta because they are rescued by birth.

Epidemiology and cause of vascular maturation defect

It has been known for a long time that maternal diabetes mellitus can lead to fetal and placental overgrowth endangered by sudden intrauterine death and increased fetal mortality. Invariably, in nearly all cases the placentae exhibit strongly increased diameters of stroma-rich terminal villi that do not exhibit sinusoids and membranes. We retrospectively studied the obstetric history of 54 women who had experienced an *in utero* death. In 32, placental insufficiency had been "acute" due to maturation defect of the chorionic villi. Only three of these mothers were diabetic during pregnancy (<10%). Although the event dated back to between 5-20 years, none of the other 29 women with placental morphology "typical of diabetes mellitus" developed diabetes later on. Furthermore, it transpired that women with this type of placental abnormality did not experience another in *utero* death, in contrast to mothers who lost their child due to a compromise of maternal or fetal circulation (Table I).

Perspectives

Chronic placental insufficiency, sometimes termed "nutritional insufficiency", can be detected by careful monitoring of the fetal growth curve, offering the chance to rescue a baby from the adverse uterine environment by preterm delivery. In many cases, mothers have underlying disease and there is a risk of recurrence in the eventual case of another pregnancy. Acute insufficiency, sometimes termed "respiratory insufficiency", is very rarely detected when CTG examination is performed during a hypoxic period and which is sometimes provoked by so-called stress tests. With the exception of maternal diabetes mellitus there seems to be no predisposing maternal disease and the condition is thus termed "idiopathic". An increased risk of recurrence regarding *in utero* death has not been found. However, the morphological finding of striking immaturity of chorionic villi can occur in consecutive pregnancies. Overall, it seems that most children do not succumb to this developmental abnormality as they are rescued by birth. Nevertheless, the lack of a method to detect the latent stage of a defect that can manifest acutely with fetal hypoxia and death is disappointing.

Table 1. Obstetric history of women experiencing intrauterine death of a fetus grouped by the pathology-pattern circulatory compromise ("chronic") *versus* maturation defect ("acute").

	Number of women included	Mothers with children	Liveborn after	Fetal death in a previous pregnancy	Women remaining without children	Average number of live children
Chronic insufficiency	22/54	5	16	3 (13.6%)	3 (13.6%)	1.8
Acute insufficiency	32/54	16	22	0	4(18.2%)	1.9

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