Wolters Kluwer

Gross examination of the placenta

AUTHOR: Drucilla J Roberts, MD
SECTION EDITOR: Amy McKenney, MD
DEPUTY EDITOR: Alana Chakrabarti, MD

All topics are updated as new evidence becomes available and our peer review process is complete.

Literature review current through: **Oct 2023.**
This topic last updated: **Apr 28, 2023.**

INTRODUCTION

The placenta is a fetal organ consisting of an umbilical cord, membranes (chorion and amnion), and parenchyma (picture 1). Maternal or fetal disorders may have placental sequelae since the mother and fetus interface at this site. Conversely, primary placental abnormalities can affect or reflect both maternal and fetal health. Thus, examination of the placenta may yield information on the impact of maternal disorders on the fetus or the cause of preterm delivery, fetal growth restriction, or neurodevelopmental impairment. Placental examination is an essential component of the autopsy in cases of fetal or neonatal death. (See "Stillbirth: Maternal and fetal evaluation".)

Placental examination also aids in a number of difficult situations, such as [1-4]:

- Legal issues regarding the presence of acute versus chronic perinatal stresses and insults, and the timing of these insults.
- Diagnosis of the specific etiologies of adverse pregnancy outcomes.
- Identification of zygosity in multiple gestations, as well as pathologies unique to these pregnancies.
- Identification of potentially recurrent disorders, potentially leading to changes in management and improved outcome of subsequent pregnancies.

The gross examination of the placenta is discussed here. Histopathology is reviewed separately. (See "The placental pathology report".)

BASIC EXAMINATION

There is a general consensus that all placentas should be examined grossly in the delivery room [2,4,5].

At a minimum, the clinician at delivery should note some basic characteristics of the placenta including:

- **Umbilical cord** – Number of vessels, length, insertion site, gross abnormalities (eg, knots)
- **Parenchyma** – Gross abnormalities (eg, discoloration, multiple lobes, focal lesions)
- **Membranes** – Color, placental insertion site, membranous vessels

The cord length measurement may be more accurate when measured by the clinician since the portion of the cord attached to the infant is not available to the pathologist and other portions of umbilical cord may be discarded prior to the placenta arriving in the pathology laboratory. The clinician should record this information in the medical record. The pathologist usually measures placental weight since the placenta is weighed trimmed without the cord and membranes. (See 'Length' below and 'Weight' below.)

The following form is an example of a worksheet that pathologists may use to record their findings (form 1).

INDICATIONS FOR A FULL EXAMINATION

The delivering provider is usually responsible for determining when further examination by a pathologist is necessary, and some institutions provide guidelines for their staff.

The author believes histologic examination of the placenta is indicated in the absence of a normal term delivery (vaginal or cesarean) of a healthy term infant from an uncomplicated pregnancy. A list of potential indications for full histopathologic examination is provided below and in the table (table 1); the list is not all-inclusive and should be guided by clinical judgment [4]:

- Death: stillbirth (current or history of previous stillbirth), maternal death, or neonatal death.
- Neonatal resuscitation or neonatal intensive care unit (NICU) admission.
- Preterm or postterm birth (<37 weeks or ≥42 weeks of gestational age).
- Multiple gestation.

- Any Apgar score <7, cord blood pH <7, resuscitation >10 minutes, persistent hypoglycemia, need for ventilatory assistance, neurologic compromise.
- Obstetric complications/disorders (eg, chorioamnionitis, preeclampsia, acute abruption, nonreassuring fetal heart rate pattern requiring prompt delivery, antepartum bleeding or postpartum hemorrhage, thick meconium, oligohydramnios/polyhydramnios, retained placenta).
- Abnormal gross examination of the placenta (eg, abnormal color, masses, long or short umbilical cord, abnormal membranes).
- Relevant maternal disorders (eg, diabetes [pregestational or poorly controlled], severe hypertension, autoimmune disease, tobacco/alcohol/illegal drug use, metastatic malignancy, infection associated with congenital infection, uterine anomalies or scars).
- History of a placenta with pathology known to recur (eg, abruption, chronic histiocytic intervillositis, massive perivillous fibrin deposition/maternal floor infarct, villitis of unknown etiology).
- Hydrops fetalis.
- Severe fetal/newborn anomaly.
- Severe neonatal anemia.
- Suspected neonatal sepsis.
- Fetal growth restriction, small for gestational age or large for gestational age newborn.

The institutional cost of pathologic evaluation of the placenta is minimal (around USD $290 in 2021 [6]). Pathology assistants and technicians can be trained to perform placental gross examination. Most placentas have just a few and quite characteristic lesions that are easily recognized without special studies or procedures. Those that fall out of this realm should be referred to a pathologist for evaluation. The reimbursement for pathologic examination is usually at a level 5 (88307) and is typically not contested by any of the major insurers.

HANDLING

Information regarding the gestational age and pregnancy history should be provided by the delivering clinician, typically on a pathology requisition sheet. As discussed above, clinicians should document any gross abnormalities that they identified, the length of the umbilical cord (sum of the lengths attached to the placenta and the newborn), and the number of vessels in the cord.

Abnormalities, such as true knots, should not be disturbed by the clinician so the gross abnormality can be viewed by a pathologist.

Labeling multiple gestations — The placentas of multiple gestations should be labeled to correctly pair each infant with its umbilical cord/placenta (usually by cord clamps: one clamp for the firstborn, two for the second, etc).

STORING

The author believes that the safest procedure for placental storage is to have all placentas sent to the pathology department, so the pathologist can triage them based on the clinical history as provided by the clinician or the medical record.

Placentas with indications for full pathologic examination are accessioned and examined that day. All others are kept refrigerated for a short, specified period of time. Fresh placentas should be refrigerated (not frozen) and may be stored this way for several days without significant autolytic changes. As an example, Massachusetts General Hospital stores placentas in a refrigerator for at least seven days. During this time, the neonatal intensive care unit (NICU) admission list is checked daily for infants transferred from the regular nursery, and the maternal readmission list is checked. After seven days, if the infant has not been admitted to the NICU and the mother has not been readmitted, the placenta is discarded. This practice helps identify those infants with onset of complications after the immediate delivery period and those placentas that should be examined to evaluate postdelivery maternal complications (eg, retained placenta, postpartum hemorrhage, or sepsis).

Fresh versus fixed — A fresh placenta is preferable to a fixed specimen for appreciating placental surface changes, color, making membrane rolls, and palpating for solid lesions. Fixation alters the placental weight and color but improves the identification of some parenchymal lesions, such as perivillous fibrin.

Cultures, cytogenetic studies, and injection of twin placentas to detect anastomoses can **only** be performed on fresh tissue. Only after these tests are performed can the placenta then be placed in fixative (eg, formalin) and kept indefinitely.

CULTURES AND GENETIC STUDIES

Obtaining cultures — Bacterial cultures are rarely needed but, when necessary, can be performed with reliable results on a fresh (not fixed) placenta. Fresh tissue is also required for viral cultures (including severe acute respiratory syndrome coronavirus 2 [SARS-CoV-2], the virus that causes coronavirus disease 2019 [COVID-19]) or polymerase chain reaction

(PCR) testing; by contrast, immunohistochemistry or in situ hybridization may be performed on formalin-fixed paraffin-embedded tissue [7,8].

In cases of live births or stillbirths in which an autopsy examination has been permitted, culturing the infant, fetal/neonatal blood, or fetal lung (at autopsy) is best. For placental microbiologic cultures, the author believes the best procedure is to peel the amnion off the chorion for a significant amount of the fetal surface, and then swab the exposed (and untouched) surface with a sterile swab several times before using the swab for culture inoculation. Alternatively, once the amnion has been peeled, a small portion of chorion can be snipped off with sterile scissors and sent to the microbiology laboratory for culture. The author has performed this technique many times when indicated, such as in cases in which the fetus was stillborn and no autopsy permission was obtained, and has had good results. This technique is especially useful in detecting group B *Streptococcus*, which can cause lethal fetal/neonatal sepsis in the setting of intact membranes and mild chorioamnionitis.

Obtaining genetic studies — The best placental source of tissue for genetic studies is the umbilical cord since it is free of maternal "contaminant." A small portion of chorionic plate can also be used, with minimal maternal contamination. Microarray is more likely to be successful than conventional G-banded karyotype for genetic studies after a fetal demise. (See "Prenatal diagnosis of chromosomal imbalance: Chromosomal microarray".)

UMBILICAL CORD

The umbilical cord is normally composed of two umbilical arteries and an umbilical vein supported by loose gelatinous tissue called Wharton's Jelly.

General appearance — The umbilical cord should have a smooth, white, opaque, shiny appearance with spiraling, on average two complete coils in 10 cm of cord (picture 2). (See 'Coiling' below.)

- **Color**
  - Yellow coloring suggests inflammation (picture 3).
  - Yellow/green coloring suggests meconium staining or umbilical vasculitis.
  - Brown/red coloring is consistent with fetal demise of at least six hours duration and is likely due to red blood cell lysis and hematoidin (picture 4). Longer retentions of fetal demise result in a clay gray discoloration.

(See "The placental pathology report", section on 'Umbilical cord'.)

- **Nodules –** Nodules on the cord suggest abscesses and may be a sign of *Candida* infection (picture 5).

- **Edema –** Mild edema of the umbilical cord is common and of no clinical significance (picture 6). Massive edema (resulting in regional or diffuse cord diameters of greater than 3 cm) can cause vascular compromise and is often associated with acute changes in the fetal heart rate pattern. Massive cord edema may be evidence of an "angiomyxoma" [9,10]. Massive edema is also seen with patent urachus and reverse micturition. (See 'Cysts' below.)

- **Strictures –** A stricture may be an artifact or the result of torsion or amniotic bands. Strictures can be important findings, as they have been implicated in the etiology of fetal demise or compromise. They should be carefully described, and cord samples should be taken on both sides of and through the stricture.

Coiling — The umbilical cord has a characteristic coil. Twists of the umbilical cord are also common, and a twist can be "untwisted" but coils cannot. Coiling is usually to the left (like a barber pole), but the direction of coil (left versus right) is not an important factor in pregnancy outcome [11,12]. The average number of coils is 2 per 10 cm, or a coiling index of 0.2 (number of coils divided by the length of cord examined). To minimize the effect of coiling variability, ideally, the cord coiling index is measured over the entire length of cord. The genesis of cord twist is unclear but likely reflects fetal movement or differences in the growth rates of the fetal vessels [13,14].

The pattern of cord coiling may be important and should be noted (figure 1) [15-19]. The segmented and linked patterns may be more highly associated with cord occlusions due to torsion (figure 1) [17].

Coiling of the umbilical cord is thought to protect it from compression, kinking, stretch, and torsion, thus preventing disruption of the blood supply to the fetus. Coiling may also facilitate umbilical venous blood flow [20]. Cords with no coiling are associated with poor fetal growth and decreased activity and may result from severe neural and musculoskeletal anomalies [21,22]. Several studies have reported an increased frequency of adverse pregnancy outcome, including congenital anomalies, growth restriction, fetal heart rate abnormalities, preterm birth, and intrauterine death in pregnancies with an uncoiled umbilical cord [23-26]. Both hypocoiled and hypercoiled (picture 7) umbilical cords are associated with an increased frequency of small for gestational age neonates (15 to 16 percent versus 5 percent with normocoiled cords for both) and nonreassuring fetal heart rate patterns (22 to 29 percent versus 11 percent) [27,28]. (See "The placental pathology report", section on 'Cord coiling'.)

Placental insertion site — The umbilical cord normally inserts centrally or slightly eccentrically and directly into the placental disk. Fewer than 10 percent of insertions occur at the margin of the placenta (battledore placenta) (picture 8), and even fewer insert into the membranes (velamentous insertion) (picture 9).

Abnormal cord insertions should be noted, and histologic examination of any membranous vessels should be undertaken to look for thrombi and vascular pathology, which are associated with these abnormalities [29]. Abnormalities of umbilical cord insertion are more common in pregnancies resulting from in vitro fertilization [30,31] and in multiple gestations.

- **Velamentous cord insertion –** A velamentous cord insertion refers to a cord that inserts into the membranes rather than the placental disk (picture 9). The velamentous vessels are surrounded only by fetal membranes, with no Wharton's jelly, thus they are prone to compression or disruption. Velamentous vessels can also occur between lobes of a bilobed placenta or between the main placenta and accessory lobes.

  Velamentous umbilical cord has been associated with several obstetric complications, including fetal growth restriction, prematurity, congenital anomalies, and low Apgar score. (See "Velamentous umbilical cord insertion and vasa previa".)

- **Furcate insertion –** A furcate (forked) cord is a rare variation of the velamentous cord. The fetal vessels are also unsupported by Wharton's jelly, but they separate from each other and fan out to form a vascular "tent" just above the disk (picture 10). Although the appearance is worrisome, furcate cords are rarely associated with an adverse pregnancy outcome but have been associated with an increased risk for preterm delivery.

- **Marginal insertion –** A marginal cord insertion refers to an otherwise normal umbilical cord located within 2 cm of the edge of the placenta. This occurs in approximately 6 percent of pregnancies. It has a normal amount of Wharton's jelly, in contrast to a velamentous insertion, which is also often at the placental margin [32].

Length — Cord length increases with advancing gestational age; other variables have a modest effect on length [33,34]. The average length at term is approximately 55 cm, with a wide normal range (35 to 77 cm) [21,33,35]. The length should be noted and compared with published standards; an example is provided in the table (table 2). The length measurement should include the portion of cord on the infant after cord transection at delivery, as well as the part remaining with the placenta. Thus, it is best determined in the delivery room. If the length of the cord attached to the infant is not provided, the pathologist cannot determine whether the total length of cord (placental portion plus infant portion) is shorter than normal and will not make the diagnosis of "short umbilical cord" (table 2). We

diagnose a "long umbilical cord" when more than 70 cm of cord is received at term [11,36,37].

The length of the umbilical cord can be of critical importance when evaluating a case of perinatal morbidity or mortality. Cord length is determined, in part, by hereditary factors, but also, it is thought, by the tension the fetus places on the cord when it moves. For this reason, short cords are associated with fetal inactivity often related to fetal malformations, myopathic and neuropathic diseases, oligohydramnios, and some syndromes [22,38]. Long cords may be caused by a hyperactive fetus and have been associated with cord accidents, such as entanglement, knotting, and prolapse [21,34]. Long cords are also associated with placental lesions indicative of intrauterine hypoxia, such as delayed villous maturation, as well as fetal death, fetal growth restriction, and long-term adverse neurologic outcome [39]. In addition, the longer route that blood must travel to and from the fetal heart when the cord is very long may result in fetal heart failure of vascular thromboses (fetal vascular malperfusion).

Knots

- **False knots** – False knots are tortuosities of the umbilical vessels that form bulges; they are not associated with any adverse outcome.

- **True knots** – True knots occur in 1 percent of births and are generally single, simple, and loose (picture 11) [40]. However, tight (picture 12), complex, or multiple true knots increase the risk of intrauterine demise, particularly if the cord is long and during the second trimester when the fetus has a lot of room to move.

  The medical record should document the presence of a true knot, the tightness or laxity of the knot, the presence of unilateral edema of the cord relative to the knot, and whether thrombi are in the vessels. A pathologist should obtain a section through the knot for histologic examination to diagnose inflammation, necrosis, or thrombosis in the vessels. (See "Umbilical cord abnormalities: Prenatal diagnosis and management", section on 'Knots'.)

Vessels — A single umbilical vein conducts blood from the placenta to the fetus and is essential for fetal survival. The two umbilical arteries pump blood from the fetus to the placenta. Their redundancy is evident, given that the presence of a single umbilical artery or hypoplastic second umbilical artery is not lethal.

The number of umbilical vessels is best counted by cutting the cord in a relatively uniform region (away from bulges of false knots and away from the small perivascular hematomas often associated with cord traction or clamping at delivery) at least 5 cm from the placental insertion since the two arteries sometimes fuse near the insertion (ie, Hyrtl's anastomosis) [29]. Two vessel cords should be documented in the medical record and confirmed by

histologic examination. (See "Single umbilical artery" and "The placental pathology report", section on 'Single umbilical artery'.)

Vessels that are expanded and contain laminated thrombi should be investigated since this abnormal finding is evidence of fetal vascular malperfusion (see "The placental pathology report", section on 'Fetal vascular malperfusion'). Possible etiologies include cord compression from a tight true knot, cord prolapse, or head compression; marginal or membranous cord insertion; a fetal hypercoagulable state (eg, sepsis, hereditary thrombophilia); and maternal diabetes mellitus [41].

True hemangiomas of the umbilical cord are rare and usually associated with fetal death. These present as mass-like lesions of considerable length and diameter (picture 13) [42-44].

Hematomas — Hematomas due to trauma/traction at delivery are commonly observed near the cord clamp and at the insertion of the cord into the placental disk. True spontaneous hematomas of the umbilical cord do occur but are rare and often lethal.

Spontaneous hematomas present as large umbilical cord masses and can compress the umbilical vein, resulting in fetal death. In the author's opinion the cord vessel that ruptured and caused the hematoma likely was weakened by damage due to either inflammatory, toxic (meconium), or pressure necrosis.

Neoplasms — Rare anomalies of the cord sometimes noted macroscopically include teratomas, cysts, and angiomyxomas [45-48]. These unusual findings should be documented by a full histopathologic examination.

Cysts — Umbilical cord cysts are either true epithelial cysts, arising from embryonic remnants of the vitelline duct and urachus, or pseudocysts, usually from marked edema of Wharton's jelly. They can occur anywhere along the length of the cord. First-trimester umbilical cord cysts noted on ultrasound are often transient and have no clinical significance [49-52]. Persistent cysts have been associated with a variety of fetal anomalies (particularly patent urachus and omphalocele), but the true prevalence of fetal anomalies in these patients cannot be determined from the small retrospective case reports and series that have been published [53]. If a cyst or pseudocyst is associated with massive umbilical cord edema, the possibility of a patent urachus and reverse micturition should be considered.

Preparation for histologic examination — After the gross examination described above has been performed, the cord is transected at the disk, and at minimum, two sections are isolated and placed in formalin for histologic examination. These specimens should incorporate the distal and proximal regions of the cord, as well as any areas that appear abnormal. Cords that are abnormally colored, especially due to meconium, should be extensively sectioned (at least three cassettes). The sections should be labeled so they can be

oriented by the pathologist. Sections through true knots should be obtained to examine for vascular damage (necrosis, inflammation, thrombosis).

MEMBRANES

The placental membranes are composed of two layers: the layer nearest the fetus (facing the amniotic cavity) is the amnion and the outer layer is the chorion (figure 2). The amnion and chorion are derived from extraembryonic tissues of the zygote. The exocoelomic cavity, which is a remnant of the early gestational sac, separates the two layers until approximately 14 weeks of gestation, at which time the amnion and chorion adhere to one another.

The tensile strength and elastic properties of fetal membranes allow them to accommodate to the changing needs of the fetus across gestation.

Color — Fetal membranes are translucent, slightly blue/gray, and glistening (picture 14). Abnormal membrane color or clarity suggests a pathologic condition. As discussed above, meconium stains the membrane yellow/green, hemosiderin stains it brown/green, and infection stains it yellow/green (picture 15); a tan membrane may represent a combination of these problems (picture 16). Mild infection might only increase membrane opacity and dullness.

Surface — The membranes should be smooth. Small, flat, pearly white lesions that adhere to the amnion near the cord insertion site may represent squamous metaplasia, a benign finding (picture 17). Subchorionic nodules of waxy white/tan/gray fibrin deposits are also a common, normal finding.

By comparison, nodules **on** the fetal surface of the amnion are unusual and suggest a disease process such as abscesses or amnion nodosum. These nodules should be evaluated histologically. (See "The placental pathology report", section on 'Solid and cystic abnormalities'.)

Insertion — The insertion of the membranes at the placental disk should be noted, although the significance of various types of insertions is controversial. The membranes usually emerge from the disk smoothly and flush with the margin; this is referred to as a marginal insertion. There may be an increased risk of fetal morbidity when the membranes originate from just inside the margin of the disk (circummarginate) or from deep inside the margin (circumvallate) due to abnormally deep implantation in the disk or early marginal hemorrhage at implantation [54]. These extrachorial insertions of the membranes are identified by the presence of either a rim of fibrin internal to the placental margin (circummarginate insertion) or a fold of membranes internal to the placental margin

(circumvallate insertion). (See 'Circumvallate placenta' below and 'Circummarginate placenta' below.)

Preparation for histologic examination — After inspection of the membranes, the pathologist should obtain two membrane rolls: one at the shortest distance to the disk from the rupture site and another randomly taken. An approximate 2 cm wide strip of membranes should be cut from the area most distal from the disk to the disk, including a small portion of disk margin. Any membranous vessels (either due to velamentous insertion of the umbilical cord or a "wandering" membranous vessel) should be sampled in this strip. The membranes can be wrapped like a "jelly roll" around a pair of forceps (or similar instrument) while holding the marginal piece of placenta and placed in formalin. One transverse section from each roll is used for histologic examination (picture 18). The remaining membranes can be trimmed off the disk and returned to the bucket.

PARENCHYMA

The placental parenchyma is composed of a branching, highly vascularized stromal compartment covered by trophoblast: the chorionic villi. Parenchyma is composed of maternal tissues (decidua and its components and the intervillous blood), villi (placental components), and circulating fetal blood. The stroma is mesoderm composed of fibroblasts with admixed macrophages (Hofbauer cells) and filled with vascular channels.

Weight — The disk (without membranes and umbilical cord) should be weighed and the weight compared with standards; one example is provided in the table (table 3). The amount of blood in the placenta comprises a significant proportion of total weight and is affected by iatrogenic factors, such as delayed cord clamping at delivery.

Placental weight correlates with birth weight; normal values of the fetal-to-placental weight ratio change during the course of gestation (approximately 1:4 at 27 weeks, increasing to approximately 1:7 at term). Placental weights significantly deviating from the normal range should prompt consideration for special studies, such as viral cultures, flow cytometry, or genetic studies.

Maternal diabetes mellitus (especially gestational or poorly controlled type 2 diabetes mellitus), fetal or maternal anemia, fetal hydrops, placental mesenchymal dysplasia, and congenital syphilis are associated with heavy placental weights. Preeclampsia, fetal growth restriction, some aneuploidies (not trisomy 21), fetal alcohol exposure [55], and some infections are associated with light placental weight.

Dimensions and appearance — The normal term placental disk is a single, relatively symmetrical discoid organ that occupies approximately one-fifth of the surface of the

chorionic sac. The dimensions of the placenta should be recorded. A normal term placenta is 2 to 4 cm thick and approximately 20 cm in diameter. Thickness is measured after cutting, and size is represented by the major and minor diameters of the usually ovoid/round disk.

- **Maternal surface –** The maternal surface is maroon and divided into lobules or cotyledons, which should be inspected to ensure that all are present (ie, the placenta is complete). Disrupted placentas may be associated with placenta accreta spectrum and require focused sampling around the disrupted area. Blood clot (fresh or organized) adherent to the maternal surface, especially if it distorts the cotyledons, may represent an abruption and should be described.

- **Parenchyma –** The maternal surface of the placental disk should be cut into 1 to 2 cm wide strips just to, but not through, the chorionic plate. The examination, then, is a combination of palpation and visual inspection. The parenchyma should be a spongy, soft, red/maroon tissue. Mottled colors and firm areas are abnormal and should be noted (measured and sampled). (See 'Preparation for histologic examination' below.)

  Firm areas (related fibrin deposition or other pathologies [eg, chronic villitis, fetal vascular malperfusion]) and mass lesions (consisting of infarcts, intervillous thrombi (picture 19), cysts, tumors, or abscesses) are abnormal and should be examined histologically. (See "The placental pathology report".)

  Infarcts are relatively common findings at the placental periphery, as it is a poorly maternally perfused region of the placenta. Recent infarcts are red, while older infarcts and fibrin deposits are gray (picture 20). Infarcts are typically firm and gritty whereas fibrin is firm and smooth/waxy. In a normal-sized placenta, peripherally located infarcts or fibrin occupying less than 5 percent of the placental mass is not clinically important, but larger and/or central lesions are associated with fetal growth restriction, stillbirth, and neurologic sequelae (see "The placental pathology report", section on 'Maternal vascular malperfusion' and "Placental pathology: Findings potentially associated with neurologic impairment in children"). Another common placental mass is an intervillous thrombus (picture 19 and picture 21). These lesions are laminated (like a thrombus, with lines of Zahn) and are usually vaguely rectangular and soft. They can be red (if fresh) or white (if remote). Intervillous thrombi are not usually associated with perinatal morbidity but should be sampled to ensure that the gross diagnosis is not in error.

  An orange rind-like band of fibrin deposition along the maternal floor is suspicious for a maternal floor infarction, a lesion that carries significant morbidity and recurrence risk [56,57]. Increased fibrin can present as a plaque (isolated nodule) or diffusely throughout the placenta. This should be noted and well sampled. Diffuse fibrin deposition is a diagnostic feature of massive perivillous fibrin deposition (MPFD) and

can be present in other diseases, such as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) placentitis [58]. MPFD has significant associated morbidity and mortality as well as a high recurrence risk. If the placenta has at least 25 percent of the disk occupied by fibrin, it should be photographed and well sampled in order to make this clinically important diagnosis. (See "The placental pathology report", section on 'Maternal floor infarction/massive perivillous fibrin deposition'.)

Severe fetal anemia may cause the placenta to appear pale and edematous (picture 22). One should consider all of the etiologies of fetal anemia, including maternal-fetal hemorrhage and alloimmunization, and if the clinical situation supports it, consideration of maternal antibody and Kleihauer-Betke analysis might be suggested based on the placental examination alone.

Calcification (calcium speckling) may be observed and palpated usually over the basal surface of the cotyledons or in lesions such as fibrin plaques or infarcts. It is a normal part of placental maturation in the third trimester [59,60] and occurs earlier in smokers [61]. Placental calcification in the second trimester is an abnormal finding; most studies have found an association with adverse pregnancy outcomes (eg, fetal growth restriction, fetal distress) [62]. Second-trimester calcification is often associated with placental infarction or perivillous fibrin deposition, which are signs of placental insufficiency.

- **Fetal surface** – The fetal surface of the placenta is shiny, gray/blue/purple, and translucent. The chorionic vessels along the fetal surface of the placenta can be examined easily through the transparent amnion and chorion covering them. The fetal surface is inspected to look for large vessels coursing to the edge, which suggest a placental lobe (succenturiate or accessory lobe) may have torn away and been left behind in the uterus (picture 23). It is also important to note the presence of cysts, subchorionic hematomas, squamous metaplasia, amnion nodosum, and thrombi or calcification of vessels.

  Hemorrhage between the amnion and chorion is usually an artifact related to traction on the cord during delivery of the placenta.

Placental variants

Placenta succenturiata — Placenta succenturiata refers to a placenta with an additional/accessory lobe or lobes of placental tissue located a few centimeters away. A placental artery and vein extend from and within the membrane of the main placental mass to each lobe and then divide into smaller vessels supplying individual cotyledons (picture 24). The ancillary lobes function normally, but can be associated with complications such as placenta previa or vasa previa. In addition, the succenturiate lobe(s)

may be retained after the main placental disk has been delivered. This can result in postpartum hemorrhage or infection days or weeks after delivery.

Placenta membranacea — Placenta membranacea refers to a rare type of placenta in which the chorionic sac is covered by functional placental tissue without free membranes. These placentas are very thin and deeply implanted (with occasional placenta accreta), thus requiring manual removal. They are associated with slightly increased risks of second-trimester miscarriage, preterm birth, and antepartum and postpartum bleeding [63].

Duplex, bipartite, and tripartite placentas — Duplex placenta (ie, bilobed or bilobate placenta (picture 25)) refers to complete separation of the placenta into two more or less equal sized lobes with separate umbilical arteries and veins that unite in a single umbilical cord. Bipartite (bidiscoidal) and tripartite (tridiscoidal) placentas do not have as complete a separation between lobes as the duplex placenta. The incomplete parenchymal separation typically occurs in the area of the cord insertion site, which may be very thin or absent (fenestrate placenta).

The umbilical cord of such placentas may insert into a chorionic bridge between lobes. More commonly, membranous vessels extend between lobes or from either lobe to the cord. When the vessels are membranous, vasa previa, compression, and thrombosis are major concerns.

Circumvallate placenta — Circumvallate placenta refers to a placenta with an unusually small chorionic plate, but with growth of extrachorial placental tissue (picture 26). A double layer of amnion and chorion, as well as necrotic villi and fibrin, form a raised white ring around the surface of the placental disk at a variable distance from the umbilical cord insertion site; the fetal vessels do not extend beyond this ring. The extrachorionic tissue constitutes a small proportion of the placenta and usually does not compromise fetomaternal exchange. However, these placentas are more prone to premature separation and second trimester bleeding is common. There is often evidence of remote hemorrhage if sections are taken through this region with thrombus and hemosiderin. Circumvallate placenta has also been associated with fetal growth restriction, preterm prelabor rupture of membranes, and preterm birth [64-66].

Circummarginate placenta — The ring of fibrin is flat and near the edge of the disk (picture 27). In the author's experience, it is not associated with increased adverse outcomes.

Preparation for histologic examination — In addition to specimens from all of the abnormal areas, histologic sampling of the parenchyma should include at least three regions (full thickness) that are grossly normal [56,57]. Thus, a total of three sections of normal parenchyma, two membrane rolls, and two sections of umbilical cord are the minimum

number recommended for histologic examination [57]. Additional sections should be obtained based on abnormal gross findings. If examining the placenta fresh, these sections should then be stored in formalin. They should fix at least four hours before trimming into cassettes for processing.

GROSS PLACENTAL FINDINGS OF CLINICAL SIGNIFICANCE

Most important placental pathologic findings are detected on histologic examination. These abnormalities include infections; predictors of neurologic compromise in the infant; and predictors of reproductive/obstetric pathology in future pregnancies, such as massive chronic intervillositis.

Examples of gross lesions that are important include:

- Abscesses, which are almost always due to listerial placentitis and may be a sign that the infant is infected. (See "Clinical manifestations and diagnosis of Listeria monocytogenes infection".)
- Green discoloration, especially of the umbilical cord, may be meconium myonecrosis, which may be associated with perinatal morbidity. Additional sections of the umbilical cord, especially through the most discolored regions, are helpful to make this diagnosis.
- Yellow-green discoloration of umbilical cord may be umbilical "vasculitis," which may be associated with perinatal sepsis and/or neurologic compromise.
- Organized thrombi in umbilical vessels and/or chorionic plate vessels may be associated with congenital stroke, visceral infarcts, and/or neurologic compromise.
- Orange rind-like change of maternal floor may be maternal floor infarction; a placenta in which >25 percent is solid and/or firm may be massive perivillous fibrin deposition (MPFD). These pathologies are rare but often recur and are associated with fetal complications. These findings are also seen in patients with severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) placentitis.
- Masses in the placenta usually appear as firm, tan/white lesions. Although most masses are relatively benign (small peripheral infarcts or intervillous thrombi), all masses should be sampled since they can phenocopy other lesions of particular clinical relevance such as metastatic tumor or placental neoplasms (chorioangioma or choriocarcinoma) [15-19].
- Incomplete maternal floor may indicate retained placenta. The basal plate area around the disruption should be generously sampled to look for adherent myometrial fibers

(evidence of noninvasive placenta accreta spectrum) [67]. (See "Placenta accreta spectrum: Clinical features, diagnosis, and potential consequences".)

- Cysts may be simple cysts (chorionic, septal, or amniotic) or related to a complete or partial mole (including a twin gestation with a normal twin and a complete hydatidiform mole) or placental mesenchymal dysplasia (PMD). PMD is a unique placental developmental anomaly usually associated with many grossly identifiable cysts and rope like chorionic vasculature in a large placenta. It has been linked with fetal growth restriction, stillbirth, Beckwith-Wiedemann syndrome, and some chromosomal abnormalities [68].

(See "Placental pathology: Findings potentially associated with neurologic impairment in children" and "The placental pathology report".)

PATHOLOGY REPORT

(See "The placental pathology report".)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Delivery".)

SUMMARY AND RECOMMENDATIONS

- **Overview** – The placenta is a fetal organ consisting of an umbilical cord, membranes (chorion and amnion), and parenchyma (picture 1). Maternal or fetal disorders may have placental sequelae since the mother and fetus interface at this site. Conversely, primary placental abnormalities can affect both maternal and fetal health. (See 'Introduction' above.)

- **Basic examination** – The umbilical cord, membranes, and placental disk should be examined for abnormalities. A minimum examination should include the number of cord vessels, length of the umbilical cord, color of the placental membranes, and placental weight (trimmed of umbilical cord and membranes). Abnormalities, such as true knots (picture 12), should not be disturbed by the clinician so the gross abnormality can be viewed by a pathologist. (See 'Basic examination' above and 'Handling' above.)

- **Full examination** – We suggest histologic examination of the placenta in the absence of a normal delivery of a healthy term singleton infant from an uncomplicated pregnancy. A list of potential indications for full histopathologic examination is provided in the table (table 1). (See 'Indications for a full examination' above.)

- **Storage** – A fresh placenta can be refrigerated for several days. (See 'Storing' above.)

- **Cultures and genetic testing**

  - Bacterial cultures are rarely needed but, when necessary, can be performed with reliable results on a fresh (not fixed) placenta; fresh tissue is also required for viral cultures or polymerase chain reaction (PCR) testing. By contrast, immunohistochemistry or in situ hybridization may be performed on formalin-fixed paraffin-embedded tissue. (See 'Obtaining cultures' above.)

  - The best placental source of tissue for genetic studies is the umbilical cord. (See 'Obtaining genetic studies' above.)

- **Components**

  - The umbilical cord is normally composed of two umbilical arteries and an umbilical vein supported by loose gelatinous tissue called Wharton Jelly. It should have a smooth, white, opaque, shiny appearance with spiraling, on average two complete coils in 10 cm of cord (picture 2). (See 'Umbilical cord' above.)

  - The placental membranes are composed of two layers: the layer nearest the fetus (facing the amniotic cavity) is the amnion and the outer layer is the chorion (figure 2). (See 'Membranes' above.)

  - The placental parenchyma is composed of maternal tissues (decidua and its components and the intervillous blood), villi (placental components), and circulating fetal blood. (See 'Parenchyma' above.)

REFERENCES

1. Stoeckmann A. Placental examination as a risk management tool. J Healthc Risk Manag 1994; 14:9.
2. Salafia CM, Vintzileos AM. Why all placentas should be examined by a pathologist in 1990. Am J Obstet Gynecol 1990; 163:1282.
3. Salafia CM. Recurrent obstetric complications: how placental pathology can contribute to cost-effective clinical evaluation and a rational clinical care plan. Curr Womens Health

Rep 2002; 2:65.

4. Roberts DJ, Baergen RN, Boyd TK, et al. Criteria for placental examination for obstetrical and neonatal providers. Am J Obstet Gynecol 2023; 228:497.
5. Hargitai B, Marton T, Cox PM. Best practice no 178. Examination of the human placenta. J Clin Pathol 2004; 57:785.
6. Polnaszek BE, Clark SL, Rouse DJ. Pathologic Assessment of the Placenta: Evidence Compared With Tradition. Obstet Gynecol 2022; 139:660.
7. Roberts DJ, Edlow AG, Romero RJ, et al. A standardized definition of placental infection by SARS-CoV-2, a consensus statement from the National Institutes of Health/Eunice Kennedy Shriver National Institute of Child Health and Human Development SARS-CoV-2 Placental Infection Workshop. Am J Obstet Gynecol 2021; 225:593.e1.
8. Watkins JC, Torous VF, Roberts DJ. Defining Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) Placentitis. Arch Pathol Lab Med 2021; 145:1341.
9. Kiran H, Kiran G, Kanber Y. Pseudocyst of the umbilical cord with mucoid degeneration of Wharton's jelly. Eur J Obstet Gynecol Reprod Biol 2003; 111:91.
10. Yavner DL, Redline RW. Angiomyxoma of the umbilical cord with massive cystic degeneration of Wharton's jelly. Arch Pathol Lab Med 1989; 113:935.
11. Lacro RV, Jones KL, Benirschke K. The umbilical cord twist: origin, direction, and relevance. Am J Obstet Gynecol 1987; 157:833.
12. Kalish RB, Hunter T, Sharma G, Baergen RN. Clinical significance of the umbilical cord twist. Am J Obstet Gynecol 2003; 189:736.
13. Georgiou HM, Rice GE, Walker SP, et al. The effect of vascular coiling on venous perfusion during experimental umbilical cord encirclement. Am J Obstet Gynecol 2001; 184:673.
14. Dado GM, Dobrin PB, Mrkvicka RS. Venous flow through coiled and noncoiled umbilical cords. Effects of external compression, twisting and longitudinal stretching. J Reprod Med 1997; 42:576.
15. Jessop FA, Lees CC, Pathak S, et al. Umbilical cord coiling: clinical outcomes in an unselected population and systematic review. Virchows Arch 2014; 464:105.
16. Patil NS, Kulkarni SR, Lohitashwa R. Umbilical cord coiling index and perinatal outcome. J Clin Diagn Res 2013; 7:1675.
17. Ernst LM, Minturn L, Huang MH, et al. Gross patterns of umbilical cord coiling: correlations with placental histology and stillbirth. Placenta 2013; 34:583.
18. Machin GA, Ackerman J, Gilbert-Barness E. Abnormal umbilical cord coiling is associated with adverse perinatal outcomes. Pediatr Dev Pathol 2000; 3:462.

19. Peng HQ, Levitin-Smith M, Rochelson B, Kahn E. Umbilical cord stricture and overcoiling are common causes of fetal demise. Pediatr Dev Pathol 2006; 9:14.
20. Reynolds SR. Mechanisms of placentofetal blood flow. Obstet Gynecol 1978; 51:245.
21. Rayburn WF, Beynen A, Brinkman DL. Umbilical cord length and intrapartum complications. Obstet Gynecol 1981; 57:450.
22. Naeye RL. Umbilical cord length: clinical significance. J Pediatr 1985; 107:278.
23. Strong TH Jr, Elliott JP, Radin TG. Non-coiled umbilical blood vessels: a new marker for the fetus at risk. Obstet Gynecol 1993; 81:409.
24. Rana J, Ebert GA, Kappy KA. Adverse perinatal outcome in patients with an abnormal umbilical coiling index. Obstet Gynecol 1995; 85:573.
25. Degani S, Leibovich Z, Shapiro I, et al. Early second-trimester low umbilical coiling index predicts small-for-gestational-age fetuses. J Ultrasound Med 2001; 20:1183.
26. de Laat MW, Franx A, Bots ML, et al. Umbilical coiling index in normal and complicated pregnancies. Obstet Gynecol 2006; 107:1049.
27. Predanic M, Perni SC, Chasen ST, et al. Ultrasound evaluation of abnormal umbilical cord coiling in second trimester of gestation in association with adverse pregnancy outcome. Am J Obstet Gynecol 2005; 193:387.
28. Rogers MS, Ip YW, Qin Y, et al. Relationship between umbilical cord morphology and nuchal cord entanglement. Acta Obstet Gynecol Scand 2003; 82:32.
29. Fujikura T. Fused umbilical arteries near placental cord insertion. Am J Obstet Gynecol 2003; 188:765.
30. Schachter M, Tovbin Y, Arieli S, et al. In vitro fertilization is a risk factor for vasa previa. Fertil Steril 2002; 78:642.
31. Englert Y, Imbert MC, Van Rosendael E, et al. Morphological anomalies in the placentae of IVF pregnancies: preliminary report of a multicentric study. Hum Reprod 1987; 2:155.
32. Ebbing C, Kiserud T, Johnsen SL, et al. Prevalence, risk factors and outcomes of velamentous and marginal cord insertions: a population-based study of 634,741 pregnancies. PLoS One 2013; 8:e70380.
33. Georgiadis L, Keski-Nisula L, Harju M, et al. Umbilical cord length in singleton gestations: a Finnish population-based retrospective register study. Placenta 2014; 35:275.
34. Linde LE, Rasmussen S, Kessler J, Ebbing C. Extreme umbilical cord lengths, cord knot and entanglement: Risk factors and risk of adverse outcomes, a population-based study. PLoS One 2018; 13:e0194814.
35. Mills JL, Harley EE, Moessinger AC. Standards for measuring umbilical cord length. Placenta 1983; 4:423.

36. Heifetz SA. The umbilical cord: obstetrically important lesions. Clin Obstet Gynecol 1996; 39:571.
37. Stefos T, Sotiriadis A, Vasilios D, et al. Umbilical cord length and parity--the Greek experience. Eur J Obstet Gynecol Reprod Biol 2003; 107:41.
38. Krakowiak P, Smith EN, de Bruyn G, Lydon-Rochelle MT. Risk factors and outcomes associated with a short umbilical cord. Obstet Gynecol 2004; 103:119.
39. Baergen RN, Malicki D, Behling C, Benirschke K. Morbidity, mortality, and placental pathology in excessively long umbilical cords: retrospective study. Pediatr Dev Pathol 2001; 4:144.
40. Spellacy WN, Gravem H, Fisch RO. The umbilical cord complications of true knots, nuchal coils, and cords around the body. Report from the collaborative study of cerebral palsy. Am J Obstet Gynecol 1966; 94:1136.
41. Vern TZ, Alles AJ, Kowal-Vern A, et al. Frequency of factor V(Leiden) and prothrombin G20210A in placentas and their relationship with placental lesions. Hum Pathol 2000; 31:1036.
42. Caldarella A, Buccoliero AM, Taddei A, et al. Hemangioma of the umbilical cord: report of a case. Pathol Res Pract 2003; 199:51.
43. Ghidini A, Romero R, Eisen RN, et al. Umbilical cord hemangioma. Prenatal identification and review of the literature. J Ultrasound Med 1990; 9:297.
44. Kamitomo M, Sueyoshi K, Matsukita S, et al. Hemangioma of the umbilical cord: stenotic change of the umbilical vessels. Fetal Diagn Ther 1999; 14:328.
45. Clausen I. Umbilical cord anomalies and antenatal fetal deaths. Obstet Gynecol Surv 1989; 44:841.
46. Carvounis EE, Dimmick JE, Wright VJ. Angiomyxoma of umbilical cord. Arch Pathol Lab Med 1978; 102:178.
47. Kreczy A, Alge A, Menardi G, et al. Teratoma of the umbilical cord. Case report with review of the literature. Arch Pathol Lab Med 1994; 118:934.
48. Lee MC, Aterman K. An intestinal polyp of the umbilical cord. Am J Dis Child 1968; 116:320.
49. Sepulveda W, Leible S, Ulloa A, et al. Clinical significance of first trimester umbilical cord cysts. J Ultrasound Med 1999; 18:95.
50. Rempen A. Sonographic first-trimester diagnosis of umbilical cord cyst. J Clin Ultrasound 1989; 17:53.
51. Skibo LK, Lyons EA, Levi CS. First-trimester umbilical cord cysts. Radiology 1992; 182:719.
52. Ross JA, Jurkovic D, Zosmer N, et al. Umbilical cord cysts in early pregnancy. Obstet Gynecol 1997; 89:442.

53. Zangen R, Boldes R, Yaffe H, et al. Umbilical cord cysts in the second and third trimesters: significance and prenatal approach. Ultrasound Obstet Gynecol 2010; 36:296.
54. Naftolin F, Khudr G, Benirschke K, Hutchinson DL. The syndrome of chronic abruptio placentae, hydrorrhea, and circumvallate placenta. Am J Obstet Gynecol 1973; 116:347.
55. Steane SE, Young SL, Clifton VL, et al. Prenatal alcohol consumption and placental outcomes: a systematic review and meta-analysis of clinical studies. Am J Obstet Gynecol 2021; 225:607.e1.
56. Chen A, Roberts DJ. Placental pathologic lesions with a significant recurrence risk - what not to miss! APMIS 2018; 126:589.
57. Khong TY, Mooney EE, Ariel I, et al. Sampling and Definitions of Placental Lesions: Amsterdam Placental Workshop Group Consensus Statement. Arch Pathol Lab Med 2016; 140:698.
58. Schwartz DA, Mulkey SB, Roberts DJ. SARS-CoV-2 placentitis, stillbirth, and maternal COVID-19 vaccination: clinical-pathologic correlations. Am J Obstet Gynecol 2023; 228:261.
59. TINDALL VR, SCOTT JS. PLACENTAL CALCIFICATION A STUDY OF 3,025 SINGLETON AND MULTIPLE PREGNANCIES. J Obstet Gynaecol Br Commonw 1965; 72:356.
60. JEACOCK MK. CALCIUM CONTENT OF THE HUMAN PLACENTA. Am J Obstet Gynecol 1963; 87:34.
61. Brown HL, Miller JM Jr, Khawli O, Gabert HA. Premature placental calcification in maternal cigarette smokers. Obstet Gynecol 1988; 71:914.
62. Chen KH, Chen LR, Lee YH. Exploring the relationship between preterm placental calcification and adverse maternal and fetal outcome. Ultrasound Obstet Gynecol 2011; 37:328.
63. Ekoukou D, Ng Wing Tin L, Nere MB, et al. [Placenta membranacea. Review of the literature, a case report]. J Gynecol Obstet Biol Reprod (Paris) 1995; 24:189.
64. Celik OY, Obut M, Keles A, et al. Outcomes of pregnancies diagnosed with circumvallate placenta, and use of uterine artery pulsatility index and maternal serum alpha-fetoprotein for prediction of adverse outcomes. Eur J Obstet Gynecol Reprod Biol 2021; 262:57.
65. Suzuki S. Clinical significance of pregnancies with circumvallate placenta. J Obstet Gynaecol Res 2008; 34:51.
66. Taniguchi H, Aoki S, Sakamaki K, et al. Circumvallate placenta: associated clinical manifestations and complications-a retrospective study. Obstet Gynecol Int 2014; 2014:986230.

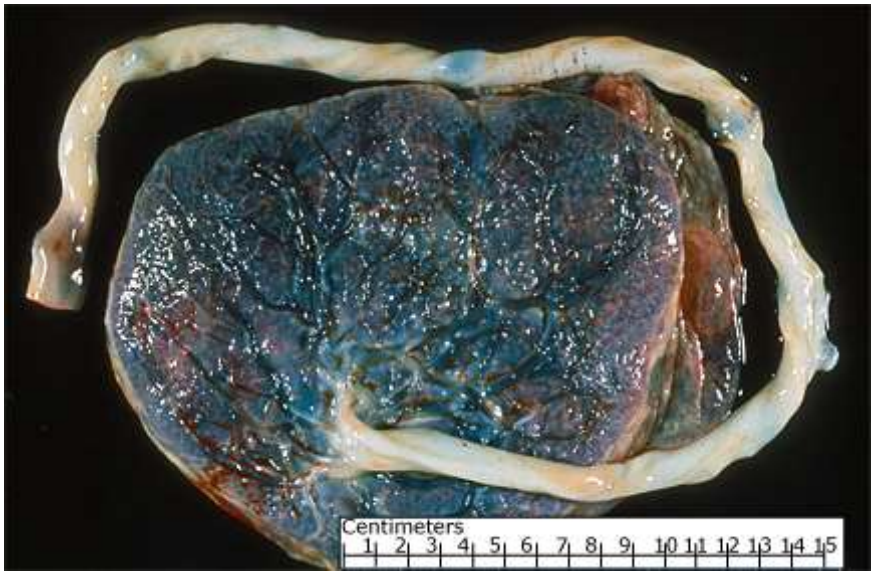
67. Hecht JL, Baergen R, Ernst LM, et al. Classification and reporting guidelines for the pathology diagnosis of placenta accreta spectrum (PAS) disorders: recommendations from an expert panel. Mod Pathol 2020; 33:2382.

68. Pawoo N, Heller DS. Placental mesenchymal dysplasia. Arch Pathol Lab Med 2014; 138:1247.

Topic 5393 Version 44.0

GRAPHICS

Normal placenta



Placenta, umbilical cord, and some of the membranes.

Courtesy of Drucilla J Roberts, MD.

Graphic 62366 Version 3.0

Placental worksheet: Template for dictation

Sample pathologist's dictation for the pathology report of a singleton placenta:

The specimen was received fresh, labeled with the patient's name and unit number, and consists of a singleton placenta.

Cord insertion _____ cm from margin (or _____ cm in membranes from margin).

Cord length _____ cm, cord diameter _____ cm, total number of coils _____, direction of coils _____ (left or right).

Number of vessels _____.

Other cord findings _____ (torsion, abnormal color, nodules, masses, etc).

Cord color _____ (white, yellow, green, brown, etc).

Membranes inserted _____ % (marginally, circummarginately, circumvallate).

Membrane color _____.

Other membrane findings _____ (nodules, hemorrhage, membranous vessels, etc).

The trimmed placental weight is _____ grams.

Disk measurement _____ cm in greatest diameter and _____ cm thick.

Fetal surface findings _____ (nodules, masses, chorionic vascular thromboses, etc).

Maternal surface _____ (complete, disrupted, masses, calcification, fibrin, hematomas, indentations, etc).

Parenchyma _____ (normal = beefy, spongy, red; lesions = number, size, percent of mass involved, location).

Gross summary:

Placenta _____ grams, approximately _____ percentile for _____ weeks gestational age with _____.

Cassette key:

- **A1:** Cord and membranes
- **A2 to 4:** Parenchyma (put lesions in last cassette, add more if needed)

Sample pathologist's dictation for the pathology report of a twin placenta:

*The specimen was received fresh, labeled with the patient's name and unit number, is a **fused/separate** twin placenta, **oriented/not oriented** to birth order by cord clamps. (If fused – the vascular equator divides the placenta mass _____ % to twin A and _____ % to twin B. There **are/are not** surface vascular anastomoses [review with staff and describe if anastomoses are present – A-A, V-V, A-V direction]. The dividing membrane is **thin/thick** and **clear/cloudy**.)*

If separate placentas, describe each separately using singleton template. If fused, use:

Twin A:

One cord clamp, _____ % of the fused disk mass.

Cord insertion _____ cm from margin (or _____ cm in membranes from margin).
Cord length _____ cm, cord diameter _____ cm, total number of coils _____, direction of coils _____ (left or right).
Number of vessels _____.
Other cord findings _____ (torsion, abnormal color, nodules, masses, etc).
Cord color _____ (white, yellow, green, brown, etc).
Membranes inserted _____ % (marginally, circummarginately, circumvallate).
Membrane color _____.
Other membrane findings _____ (nodules, hemorrhage, membranous vessels, etc).
Fetal surface findings _____ (nodules, masses, chorionic vascular thromboses, etc).
Maternal surface _____ (complete, disrupted, masses, calcification, fibrin, hematomas, indentations, etc).
Parenchyma _____ (normal = beefy, spongy, red; lesions = number, size, percent of mass involved, location).
Twin B:
Two cord clamps, _____ % of the fused disk mass.
Cord insertion _____ cm from margin (or _____ cm in membranes from margin).
Cord length _____ cm, cord diameter _____ cm, total number of coils _____, direction of coils _____ (left or right).
Number of vessels _____.
Other cord findings _____ (torsion, abnormal color, nodules, masses, etc).
Cord color _____ (white, yellow, green, brown, etc).
Membranes inserted _____ % (marginally, circummarginately, circumvallate).
Membrane color _____.
Other membrane findings _____ (nodules, hemorrhage, membranous vessels, etc).
Fetal surface findings _____ (nodules, masses, chorionic vascular thromboses, etc).
Maternal surface _____ (complete, disrupted, masses, calcification, fibrin, hematomas, indentations, etc).
Parenchyma _____ (normal = beefy, spongy, red; lesions = number, size, percent of mass involved, location).
Disk:
The trimmed fused placental weight is _____ grams.

Disk measurement _____ cm in greatest diameter and _____ cm thick.

Gross summary:

Fused/separate twin placentas _____ grams, approximately _____ percentile for _____ weeks gestational age with _____.

Cassette key:

- **A1:** Cord and membranes twin A
- **A2 to 4:** Parenchyma twin A
- **A5:** Cord and membranes twin B
- **A6 to 8:** Parenchyma twin B
- **A9:** Dividing membrane (if present)

Graphic 52233 Version 6.0

Indications for full histopathologic examination of the placenta

	Recommended criteria based on the expert opinion of 16 placental pathologists and a pathologists' assistant, formulated using a modified Delphi approach[1]	Other suggested criteria*
Antepartum indications		
Maternal indications	▪ Maternal death ▪ Systemic disease/disorders exclusive to: • Diabetes, pregestational or poorly controlled gestational • Infection potentially associated with fetal infection (eg, cytomegalovirus, syphilis, Zika virus, COVID-19) • Metastatic cancer • Severe hypertensive disorder (eg, chronic hypertension, gestational hypertension, preeclampsia with severe features, eclampsia, HELLP syndrome) • Systemic autoimmune disease (eg, antiphospholipid antibody syndrome)	▪ Alcohol use disorder, tobacco smoking, and substance use disorder (including use of medication for opioid use disorder [eg, methadone]) ▪ Assisted reproductive therapies including gestational carrier pregnancies, gamete donor pregnancies, and in vitro fertilization ▪ Body mass index ≥40 kg/m^2 ▪ IUD in place ▪ Prior uterine surgery involving breach of the endometrium (eg, submucosal myomectomy, endometrial ablation) other than cesarean birth ▪ Systemic disease/disorders, including any of the following: • Anemia requiring transfusions or with symptoms • Cancer, any • Cardiovascular compromise (any) • Diabetes, any • Hemoglobinopathy (eg, sickle cell disease or trait) • Hypertension, any • Thrombophilia ▪ Younger or older age (eg, <16 or >40 years)
Obstetric indications	▪ History of a previous placental pathology known to recur (eg, abruption, chronic histiocytic intervillositis, massive perivillous	▪ Cervical insufficiency treated with cerclage ▪ Multiple gestation, any

	fibrin deposition/maternal floor infarct, villitis of unknown etiology) ▪ Pregnancy loss ▪ Second- or third-trimester antenatal bleeding	▪ No prenatal care ▪ Oligohydramnios ▪ Postdates delivery (>42 0/7 weeks) ▪ Prolonged rupture of membranes (>24 hours) ▪ Vanishing twin
Fetal indications	▪ Complications associated with multiple gestation (eg, twin-to-twin transfusion, selective growth restriction, twin anemia-polycythemia sequence, twin reversed arterial perfusion sequence, single fetal demise, conjoined twins) ▪ Fetal demise ▪ Fetal growth restriction ▪ Hydrops fetalis ▪ Severe fetal anomaly (eg, sirenomelia, congenital heart disease, renal agenesis, congenital pulmonary airway malformation, encephalocele, omphalocele, gastroschisis)	▪ Abnormal genetic test results ▪ Anomalies, major; especially deformations and disruptions
Intrapartum indications		
	▪ Acute abruption ▪ Thick meconium in amniotic fluid ▪ Nonreassuring fetal heart rate tracing requiring urgent or emergency delivery ▪ Postpartum hemorrhage ▪ Preterm delivery (<37 weeks) ▪ Suspected chorioamnionitis	▪ Extramural birth (eg, birth at home) ▪ Meconium-stained amniotic fluid, any ▪ Nonreassuring fetal heart rate tracing (eg, NICHD category II or III) ▪ Peripartum hysterectomy ▪ Precipitous birth ▪ Shoulder dystocia ▪ Umbilical cord prolapse ▪ Uterine rupture
Neonatal indications		
	▪ Anatomic anomaly, not detected prenatally ▪ Compromised clinical condition of the neonate at birth defined as any of the following: • Apgar score of <7 at 5 minutes • Cord blood pH <7	▪ Small or large for gestational age

	• Need for ventilatory assistance • Neurological compromise (suspected hypoxic-ischemic encephalopathy) • Persistent hypoglycemia (eg, requiring intravenous dextrose) • Resuscitation >10 minutes • Severe anemia (eg, hemoglobin <10 or 11 g/dL) ▪ Neonatal death ▪ Suspected malignancy ▪ Suspected meconium aspiration ▪ Suspected sepsis	
Placental indications		
	▪ Retained placenta (to rule out placental accreta spectrum) ▪ Severe placental anomaly (eg, mass, massive fibrin deposition, discolored membranes, placenta membranacea)	▪ Placental disorders (eg, placenta accreta spectrum, placenta previa, vasa previa) ▪ Unusual findings in any aspect of the placenta gross examination by an experienced examiner
Other		
	▪ Pregnancy termination for obstetric or maternal indications	▪ Pregnancy termination for fetal indications

NICHD: National Institute of Child Health and Human Development; IUD: intrauterine device; HELLP: hemolysis, elevated liver enzymes, low platelets.

* Additional suggested criteria provided by Drucilla J Roberts, MD, a co-author on the referenced publication below.

Adapted from:

1. *Roberts DJ, Baergen RN, Boyd TK, et al. Criteria for placental examination for obstetrical and neonatal providers. Am J Obstet Gynecol 2022.*

Graphic 141228 Version 2.0

Umbilical cord spiraling

Cord spirals normally 2 coils per 10 cm.

Courtesy of Drucilla J Roberts, MD.

Graphic 69238 Version 2.0

Necrotizing funisitis - "barber pole" umbilical cord

Courtesy of Drucilla J Roberts, MD.

Graphic 57888 Version 1.0

Fetal demise with brown umbilical cord

A brown-/red-discolored umbilical cord is consistent with fetal demise of at least six hours' duration and is likely due to red blood cell lysis and hematoidin.

Courtesy of Drucilla J Roberts, MD.

Graphic 117359 Version 2.0

Candidal funisitis

Candidal funisitis. Notice the faint white nodules (arrow) on the cord surface.

Courtesy of Drucilla J Roberts, MD.

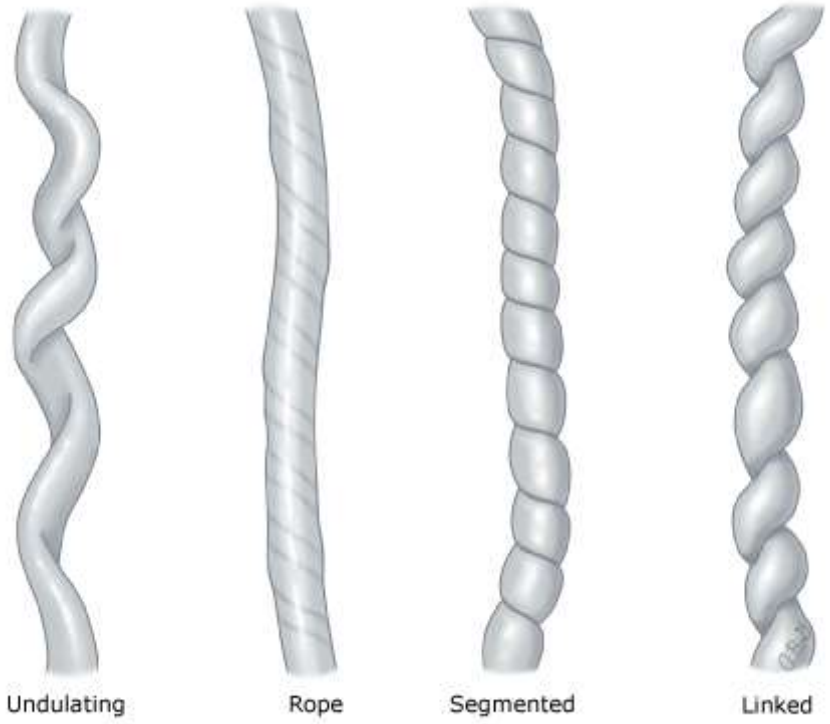
Graphic 121043 Version 1.0

Cord edema

Courtesy of Drucilla J Roberts, MD.

Graphic 74326 Version 1.0

Umbilical cord coiling patterns



Schematic representation of the four gross umbilical cord coiling patterns.

Modified from: Ernst LM, Minturn L, Huang MH, et al. Gross patterns of umbilical cord coiling: correlations with placental histology and stillbirth. Placenta 2013; 34:583.

Graphic 93966 Version 2.0

Fetal demise with hypercoiled umbilical cord

This umbilical cord is hypercoiled. The normal umbilical cord twists/coils a full 360 degrees over approximately every 5 cm of length, resulting in a cord coiling index (coils per centimeter) of approximately 0.2 or 2 coils per 10 cm.

Courtesy of Drucilla J Roberts, MD.

Graphic 117362 Version 3.0

Marginal cord insertion

Courtesy of Drucilla J Roberts, MD.

Graphic 81936 Version 2.0

Velamentous insertion of the umbilical cord

The placental end of the umbilical cord consists of divergent umbilical vessels surrounded only by fetal membranes, with no Wharton's jelly.

Courtesy of Drucilla J Roberts, MD.

Graphic 121044 Version 1.0

Furcate insertion of the umbilical cord

Furcate insertion of the umbilical cord is a rare variation of the velamentous insertion. The fetal vessels a unsupported by Wharton's jelly, but they separate from each other and fan out to form a vascular "tent" just above the disk.

Courtesy of Drucilla J Roberts, MD.

Graphic 121045 Version 1.0

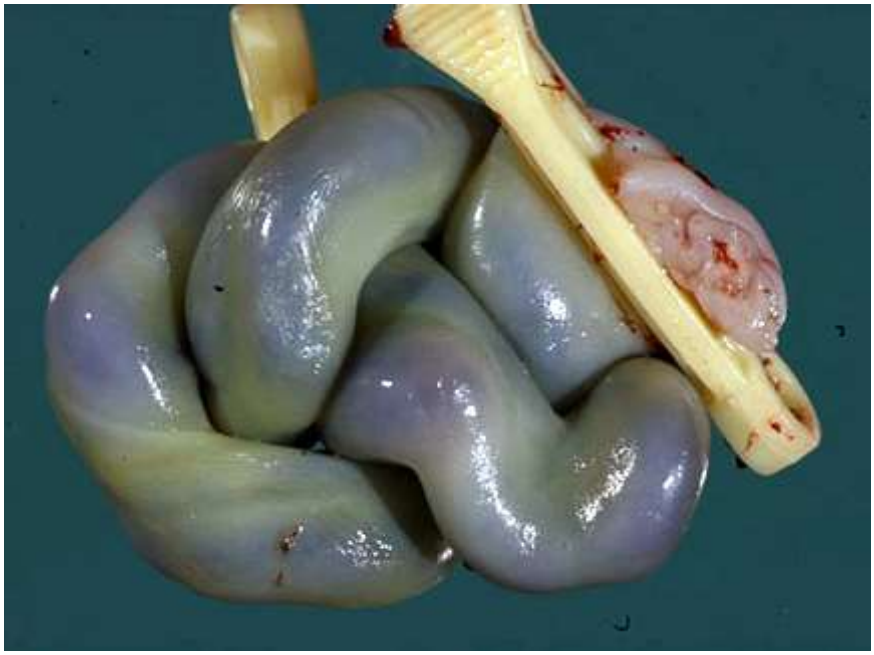
Normal umbilical cord length

Gestation, weeks	Length, cm
20	32±9
24	40±10
28	45±10
32	50±12
36	56±13
38	57±13
40	60±13

Data from: Naeye RL. Umbilical cord length: clinical significance. J Pediatr 1985; 107:278.

Graphic 76589 Version 3.0

Umbilical cord knot



Complex loose true umbilical cord knot.

Courtesy of Drucilla J Roberts, MD.

Graphic 62702 Version 2.0

Tight true knot

Courtesy of Drucilla J Roberts, MD.

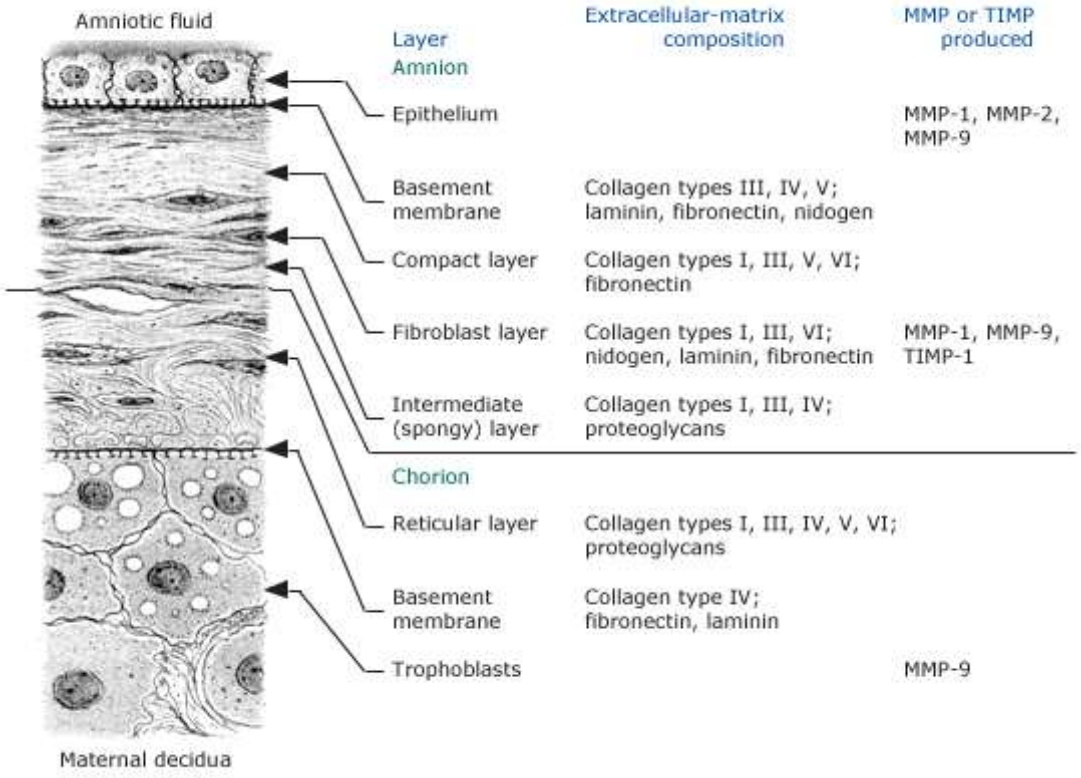
Graphic 71345 Version 2.0

Cord hemangioma

Courtesy of Drucilla J Roberts, MD.

Graphic 53360 Version 2.0

Layers within the amnion and the chorion



Graphic 75084 Version 4.0

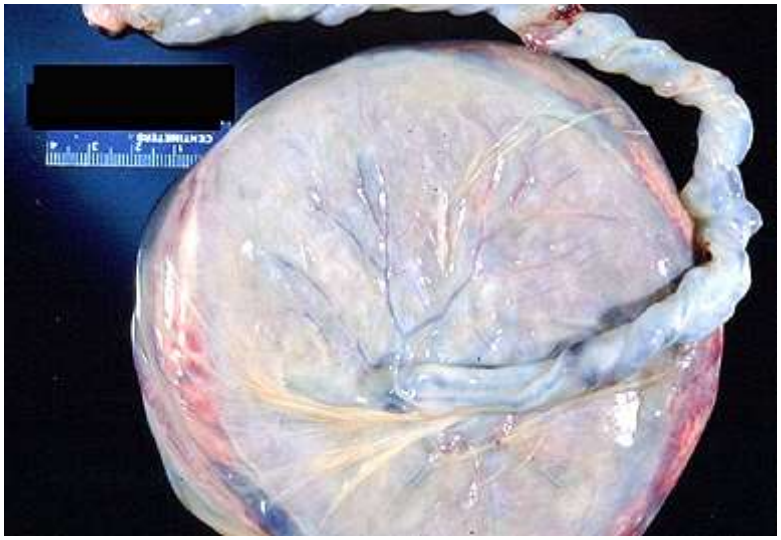
Fetal membranes

Gross photograph of a normal placenta with clear membranes. The amnion has been peeled off of the chorion focally to demonstrate the thinness and clarity of the amnion.

Courtesy of Drucilla J Roberts, MD.

Graphic 65014 Version 2.0

Chorioamnionitis



Gross photograph of the umbilical cord and fetal surface of the placenta illustrating opaque membranous surfaces characteristic of inflammation from chorioamnionitis.

Courtesy of Drucilla J Roberts, MD.

Graphic 56454 Version 2.0

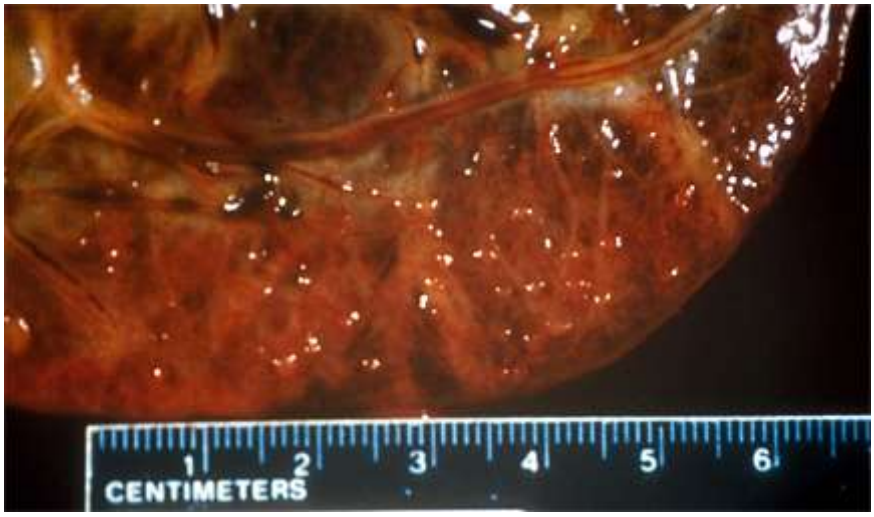
Discordant twin placenta

Chorioamnionitis and meconium in twin 1, normal twin 2.

Courtesy of Drucilla J Roberts, MD.

Graphic 66875 Version 1.0

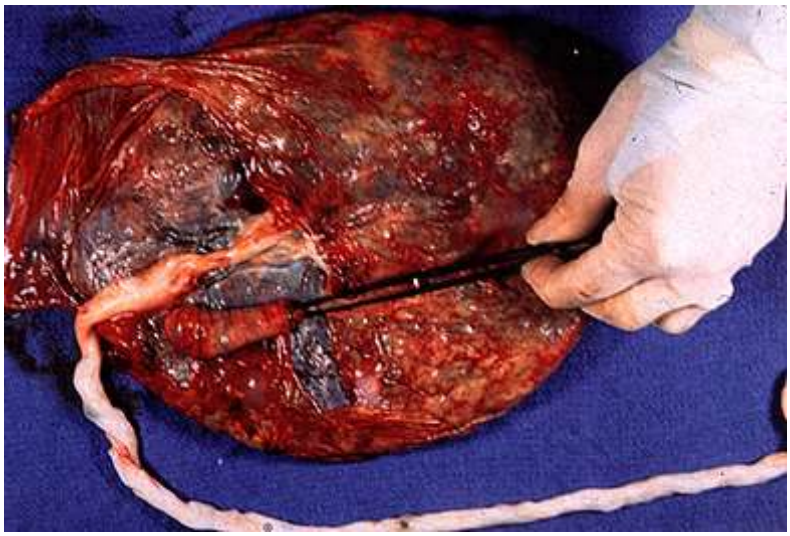
Amniotic squamous metaplasia



Courtesy of Drucilla J Roberts, MD.

Graphic 68291 Version 1.0

The fetal membranes are rolled around the tips of a pair of forceps



Courtesy of Drucilla J Roberts, MD.

Graphic 76740 Version 2.0

Placental weight standards

Gestational age	Singleton					Twin				
	Percentiles					Percentiles				
	10	25	50	75	90	10	25	50	75	90
12			56							
14			83							
16			110							
18			137.8							
20			145			166	190	218	245	270
22	122	138	157	176	191	191	219	251	282	310
24	145	166	189	212	233	232	267	307	346	382
26	175	200	227	255	280	284	330	380	430	475
28	210	238	270	302	331	345	401	464	527	584
30	249	281	316	352	384	409	478	554	631	700
32	290	325	364	403	438	472	554	644	734	815
34	331	369	411	453	491	531	624	727	830	923
36	372	412	457	501	542	582	684	798	912	1014
38	409	452	499	547	589	619	728	850	972	1082
40	442	487	537	587	632	638	753	879	1005	1118

Data from: Pinar H, Sung J, Oyer CE, Singer DB. Reference values for singleton and twin placental weights. Pediatr Pathol Lab Med 1996; 16:901.

Graphic 72177 Version 2.0

Intervillous thrombus

Cut section of placental parenchyma with a dark red, laminated, space-occupying lesion grossly consistent with an intervillous thrombus.

Courtesy of Drucilla J Roberts, MD.

Graphic 53438 Version 2.0

Placental infarct (maternal origin)

Courtesy of Drucilla J Roberts, MD.

Graphic 82377 Version 1.0

Placental thrombus: Laminated white soft parenchymal lesion

The laminated white soft parenchymal lesion represents a remote intervillous thrombus. By comparison, the red lesions represent recent intervillous thrombus.

Courtesy of Drucilla J Roberts, MD.

Graphic 130490 Version 1.0

Hydropic placenta

Courtesy of Drucilla J Roberts, MD.

Graphic 50241 Version 1.0

Accessory lobe of the placenta connected with membranous vessels

An accessory lobe (arrow) of the placenta connected with membranous vessels.

Courtesy of Drucilla J Roberts, MD.

Graphic 121046 Version 1.0

Placenta with accessory lobe

Placenta succenturiata refers to a placenta with an additional lobe (arrow) of placental tissue located a few centimeters away. A placental artery and vein are within the membrane and extend from the main placental mass to the accessory lobe.

Courtesy of Drucilla J Roberts, MD.

Graphic 117363 Version 2.0

Bilobate placenta

Bilobate placenta (also called bilobed or duplex placenta) with umbilical cord inserting marginally betwe
the two lobes.

Courtesy of Drucilla J Roberts, MD.

Graphic 121047 Version 1.0

Circumvallate placenta and cord

A double layer of amnion and chorion, as well as necrotic villi and fibrin, form a raised white ring around the surface of the placental disk at a variable distance from the umbilical cord insertion site; the fetal vessels do not extend beyond this ring. The extrachorionic tissue constitutes a small proportion of the placenta and usually does not compromise fetomaternal exchange.

Courtesy of Drucilla J Roberts, MD.

Graphic 121048 Version 1.0

Circummarginate placenta

Note that the point of membrane insertion is inside the edge of the fetal side of the disc, and the amnior chorion "double back" around this edge. Vessels radiate from the cord insertion site to the ring and then disappear from view.

Contributor Disclosures

Drucilla J Roberts, MD Other Financial Interest: American Registry of Pathology [Placental pathology]; Cambridge University Press [Placental pathology]. All of the relevant financial relationships listed have been mitigated. **Amy McKenney, MD** No relevant financial relationship(s) with ineligible companies to disclose. **Alana Chakrabarti, MD** No relevant financial relationship(s) with ineligible companies to disclose.

Contributor disclosures are reviewed for conflicts of interest by the editorial group. When found, these are addressed by vetting through a multi-level review process, and through requirements for references to be provided to support the content. Appropriately referenced content is required of all authors and must conform to UpToDate standards of evidence.

Conflict of interest policy

→