The Placenta Diaries

typical umbilical cord is about as long as the baby from heel to crown, which is roughly 60 cm (24 in). A cord is considered long if it is greater than 70 cm (28 in). Long cords may be hereditary or may develop when active babies pull against them and cause them to elongate (Roberts, 2017). Multiparous women, older women, women with diabetes, and women with higher pregravid BMI are more likely to grow babies with long cords (Georgiadis, et al. 2014). Male fetuses, large fetuses, and fetuses with large placentas tend to have longer cords. Mothers taking selective serotonin reuptake inhibitors (SSRIs) during pregnancy tend to have more active fetuses with longer cords. (Kivistö, et al, 2016). Although long cords usually indicate a well-oxygenated and neurologically advanced fetus, they are more susceptible to compression, knotting, twisting, or prolapse, any of which can cause hypoxia, neurological damage, or death. The fetus with a long cord is also at risk for growth restriction. The heart must work harder to circulate blood though an unusually long cord, putting the fetus at risk for heart failure. Conversely, CNS-impaired fetuses move less vigorously with less tension on the cord, thereby growing an abnormally short cord, less than 32 cm (13 in). Short cords are associated with decreased fetal activity, which may be related to fetal malformations, diseases that affect muscle or the nervous system, oligohydramnios, and some syndromes (Roberts, 2017). Knots True knots occur in 1% of births and are usually loose (Roberts, 2017). Loose knots do not impair circulation and are benign. Risk increases with tight or multiple true knots, especially with hypo-coiling or hyper-coiling of the cord. Sometimes a knot that is loose antenatally will tighten as the fetus descends in labor, causing variable heart rate decelerations that tend to worsen as the fetus moves through the pelvis. If the cord is enough to compromise blood flow, you will often see cord edema on one side of the knot. There is no standard of care for managing the fetus with a cord knot discovered on ultrasound, though it is logical to follow with close monitoring. FALSE KNOTS are bulges of tortuous umbilical vessels and are not associated with any adverse outcome. UMBILICAL VASCULAR THROMBOSIS occurs in 1/1300 to 1/1500 deliveries and carries high risk of mortality and morbidity. Thrombosis is more likely to develop in the umbilical vein (71-85%) than in an umbilical artery (11-15%), but a clot in the umbilical artery is more likely to be lethal for the fetus. More than 90% of cord thromboses are associated with other cord abnormalities (knots, compression, stretching of short cord during labor) obstetrical complications (infection, preeclampsia, phlebitis), or fetomaternal hemorrhage (Dussaux, et al, 2014). CORD HEMORRHAGE OR HEMATOMA Umbilical cord hematoma carries a perinatal loss rate greater than 50%. (Abraham et al, 2015). Cord hemorrhage may be related to infection, cord twisting and

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water is forced out of the umbilical vessels and into Wharton's jelly by an increase in intravascular pressure. Umbilical edema is sometimes seen near tight cord knots and in association with intra-amniotic infection and maternal diabetes. Massive edema, resulting in a cord diameter greater than 3 cm, can cause vascular compromise and

The

Placenta Diaries

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THE PLACENTA DIARIES

The placenta is like a gestational diary, containing all the juicy details about the baby's previous 9 months. Armed with a little knowledge and an inquisitive mind, the health care provider is often able to connect the dots between placental pathology and neonatal disease. You can spot problems that require immediate clinical intervention; uncover the reason for fetal or newborn distress; determine whether an insult occurred hours, days, or weeks ago; identify unsuspected maternal conditions such as lupus or vascular disease; and even speculate on the likelihood that the newborn will develop adult disease. Placental examination can also help pinpoint the cause of death in the case of stillbirth.

THE CORD AND MEMBRANES

Length

The typical umbilical cord is about as long as the baby from heel to crown, which is roughly 60 cm (24 in). A cord is considered long if it is greater than 70 cm (28 in). Long cords may be hereditary or may develop when active babies pull and stretch them (Roberts, 2017). Multiparous women, older women, women with diabetes, and women with higher pre-gravid body-mass indexes are more likely to grow babies with long cords (Georgiadis, et al., 2014). Male fetuses, large fetuses, and fetuses with large placentas tend to have longer cords. Mothers taking selective serotonin reuptake inhibitors (SSRIs) during pregnancy tend to have more active fetuses with longer cords. (Kivistö, Lehto, Halonen, Georgiadis, & Heinonen, 2016).

Short cords correlate with
Delay in second-stage labor
Abruptions
Uterine inversion
Cord rupture
Decreased fetal movement
CNS dysfunction
Psychomotor impairments
Congenital neuromuscular disease

Long cords correlate with
Exceptionally active fetus
Cord knots
Fetal entanglement
Cord prolapse

Although long cords usually indicate a well-oxygenated and neurologically advanced fetus, they are more susceptible to compression, knotting, twisting, and prolapse, any of which can cause hypoxia, neurological damage, or death. The fetus with a long cord is also at risk for growth restriction. The heart must work harder to circulate blood through an unusually long cord, putting the fetus at risk for heart failure.

Conversely, CNS-impaired fetuses move less vigorously and put less tension on the cord, thereby growing abnormally short cords, less than 32 cm (13 in). Short cords are associated with decreased fetal activity, which may be related to fetal malformations, diseases that affect muscle or the nervous system, oligohydramnios, and some conditions such as Down Syndrome (Roberts, 2017).

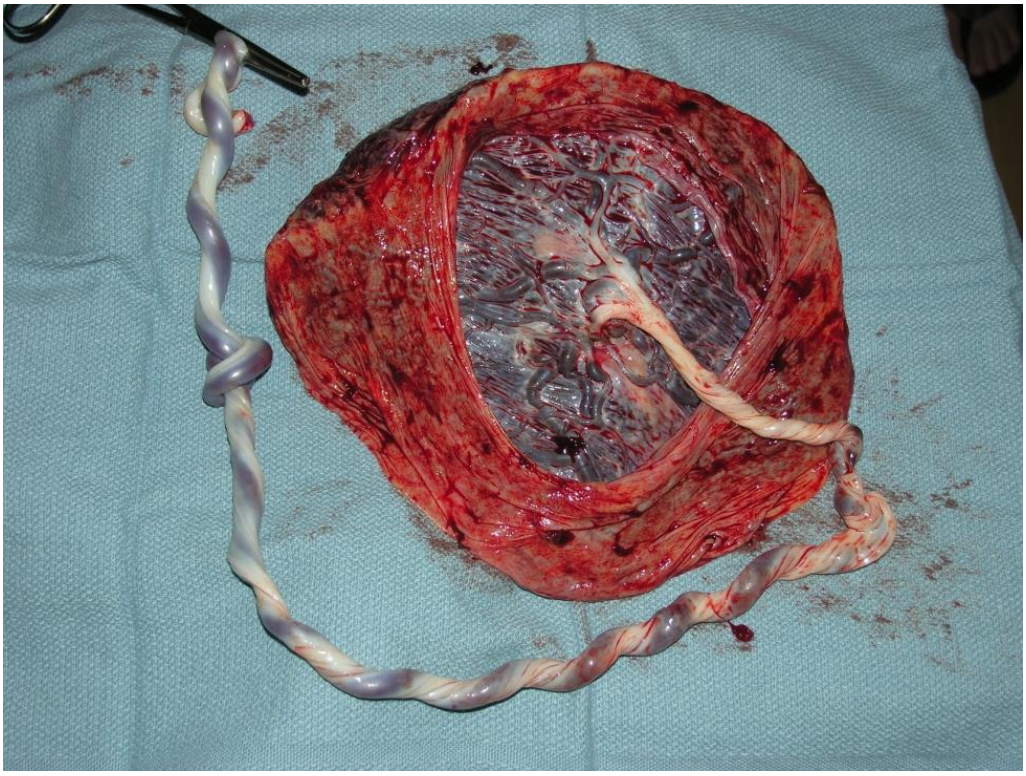

Figure 1. Long cord with a loose knot, which was wrapped around the baby's body and legs. The baby had 9/9 Apgars.

Knots

True knots occur in 1% of births and are usually loose (Roberts, 2017). Loose knots do not impair circulation and are benign. Risk increases with tight or multiple true knots, especially with hypocoiling or hypercoiling of the cord. Sometimes a knot that is loose antenatally will tighten as the fetus descends in labor, causing variable heart-rate decelerations that tend to worsen as the fetus moves through the pelvis. If the cord is constricted enough to compromise blood flow, you will often see cord edema on one side of the knot. There is no standard of care for managing the fetus with a cord knot discovered on ultrasound, though it is prudent to follow with close monitoring.

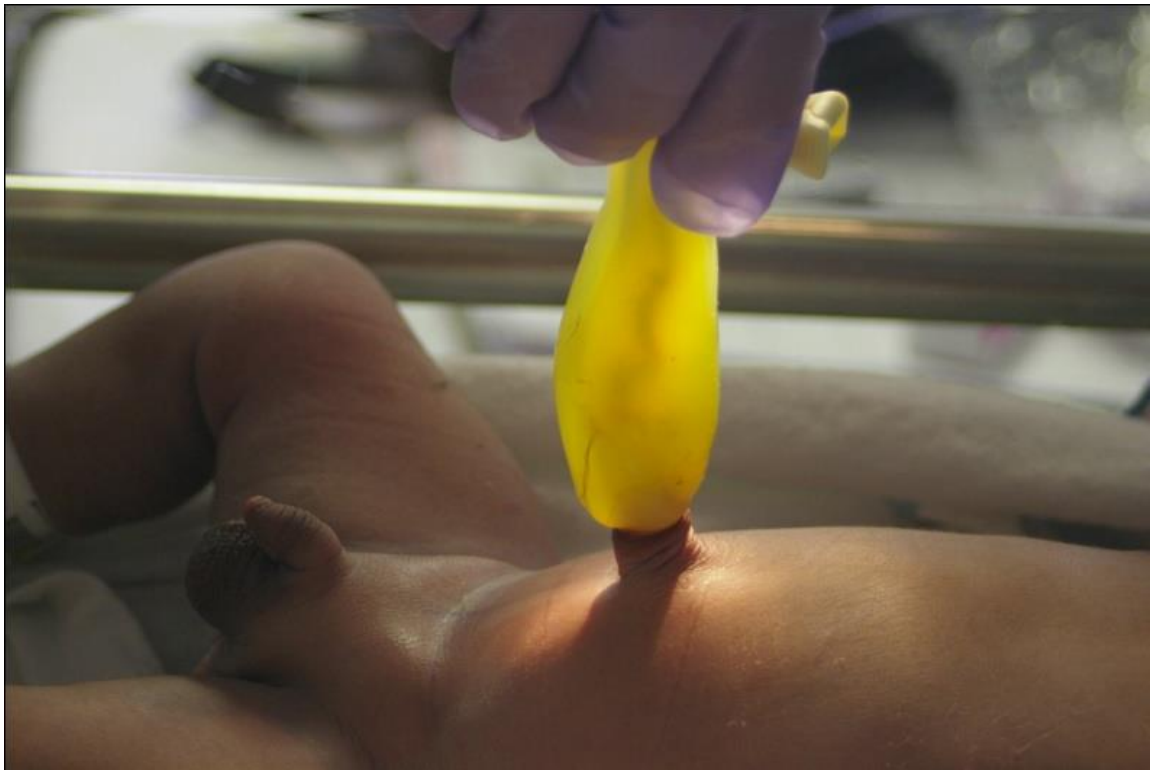

Figure 2. Cord edema. This child's mother had hypertension. She underwent cesarean section for fetal distress.

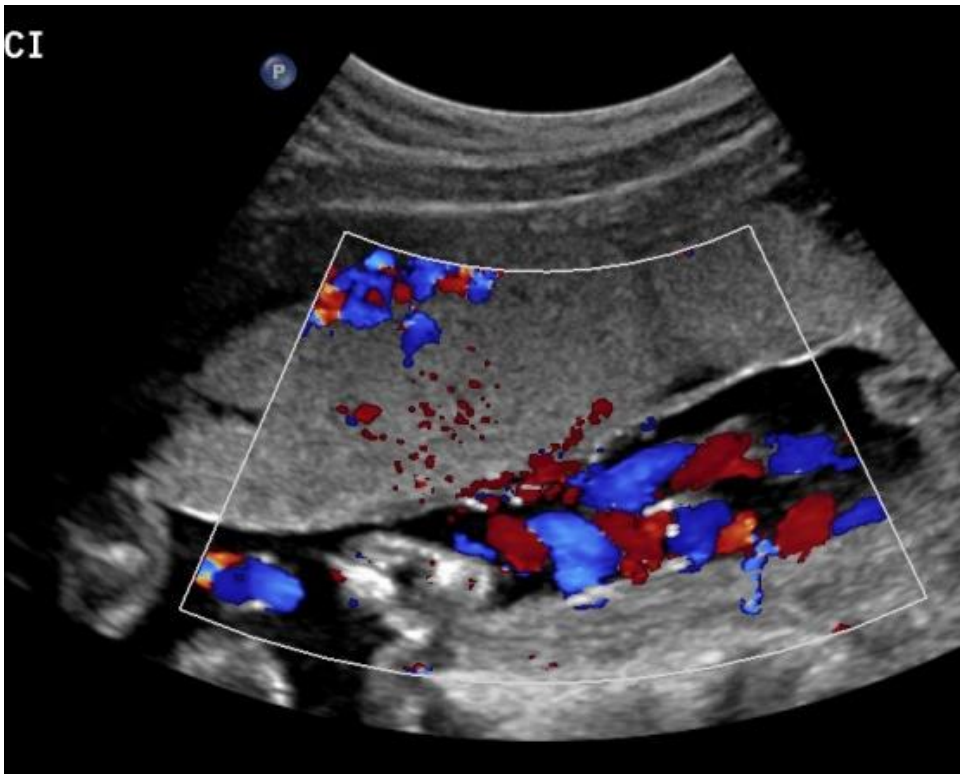

Figure 3. Two-vessel cord. Twenty percent of infants with a single umbilical artery are found to have associated conditions including cardiovascular, GI, and renal abnormalities; esophageal atresia; and multiple-anomaly syndromes.

False knots are bulges of tortuous umbilical vessels and are not associated with any adverse outcome.

Umbilical vascular thrombosis occurs in 1/1,300 to 1/1,500 deliveries and carries high risk of mortality and morbidity. Thrombosis is more likely to develop in the umbilical vein than in an umbilical artery, but a clot in the umbilical artery is more likely to be lethal for the fetus (Dussaux, et al., 2014). More than 90% of cord thromboses are associated with other cord abnormalities (knots, compression, stretching of short cord during labor), obstetrical complications (infection, preeclampsia, phlebitis), or fetomaternal hemorrhage (Dussaux, et al., 2014).

Cord hemorrhage or hematoma. Umbilical-cord hematoma carries a perinatal loss rate greater than 50% (Abraham Rathore, Gupta, & Benjamin, 2015). Cord hemorrhage may be related to infection, cord twisting and traction, true knots, abnormal vessel walls, umbilical cord cysts, trauma, or postmaturity; but often there is no obvious cause (Abraham et al., 2015). The hematoma is usually located at the fetal cord insertion site. The hematoma can cause fetal death through blood loss or by forming a tense pocket of blood that puts pressure on adjacent vessels and blocks circulation.

Cord edema is associated with hypertensive and preeclamptic pregnancies. Wharton's jelly— a gelatinous substance inside the umbilical cord largely made up of long chains of sugar molecules, along with fibroblasts and macrophages—protects and insulates the blood vessels. (Interestingly, the same substance is present in the vitreous humor of the eyeball.) Cord edema develops when water is forced out of the umbilical vessels and into Wharton's jelly by an increase in intravascular pressure. Cord edema is sometimes seen near tight cord knots and in association with intra-amniotic infection and maternal diabetes. Massive edema, resulting in a cord diameter greater than 3 cm, can cause vascular compromise and abnormal fetal heart-rate patterns (Roberts, 2017).

Umbilical cord stricture is a narrowing that reduces or stops the circulation of blood. It ismost likely to occur when a section of cord devoid of Wharton's jelly becomes twisted (Gurusamy, Thangavelu, & Venkataswamy, 2017). Stricture can also occur when an amniotic band (formed when strands of the amniotic sac separate and entangle parts of the fetus). wraps around the cord.

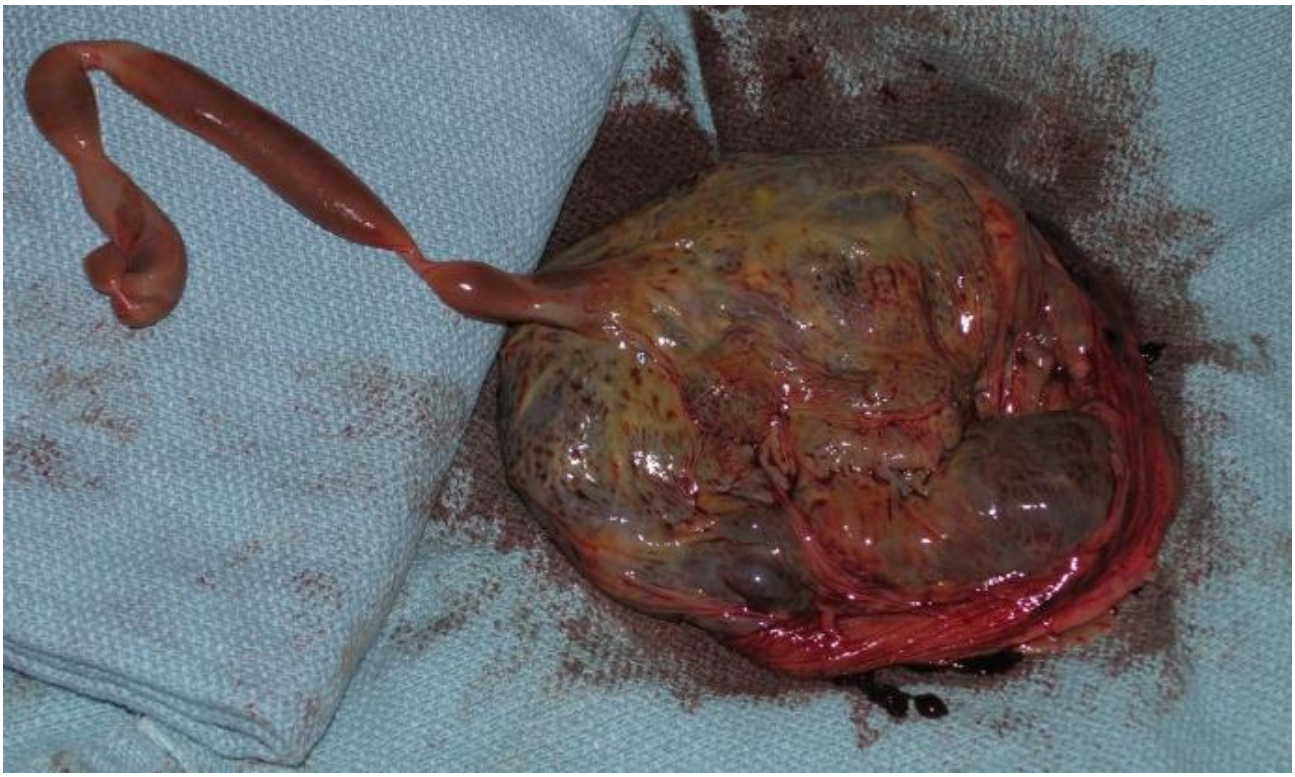

Figure 4. Cord stricture. The cord was twisted in an area of deficient Wharton's jelly, causing fetal demise at 21 weeks.

Hypercoiled/Hypocoiled Umbilical Cords

Coiling of the umbilical cord seems to protect it from compression, kinking, and torsion and gives it greater capacity for stretch during delivery. Most cords twist like a barber pole from the 9th week of gestation. The direction of twist isn't clinically significant, and it may be related to the size difference in umbilical arteries (the right artery is slightly larger than the left). Genetics and fetal activity may play a role as well.

The average umbilical cord has about 1 coil per 5 cm of length (Bhojwani, Sharma, Bhojwani, & Bhatnagar, 2016). Cords with little or no coiling (hypocoiled) and cords with excessive coiling (hypercoiled) are associated with poor fetal growth, abnormal fetal heart-rate patterns, and preterm birth. Hypocoiling of the cord is associated with severe anomalies, particularly neural and musculoskeletal, and with intrauterine death (Roberts, 2017).

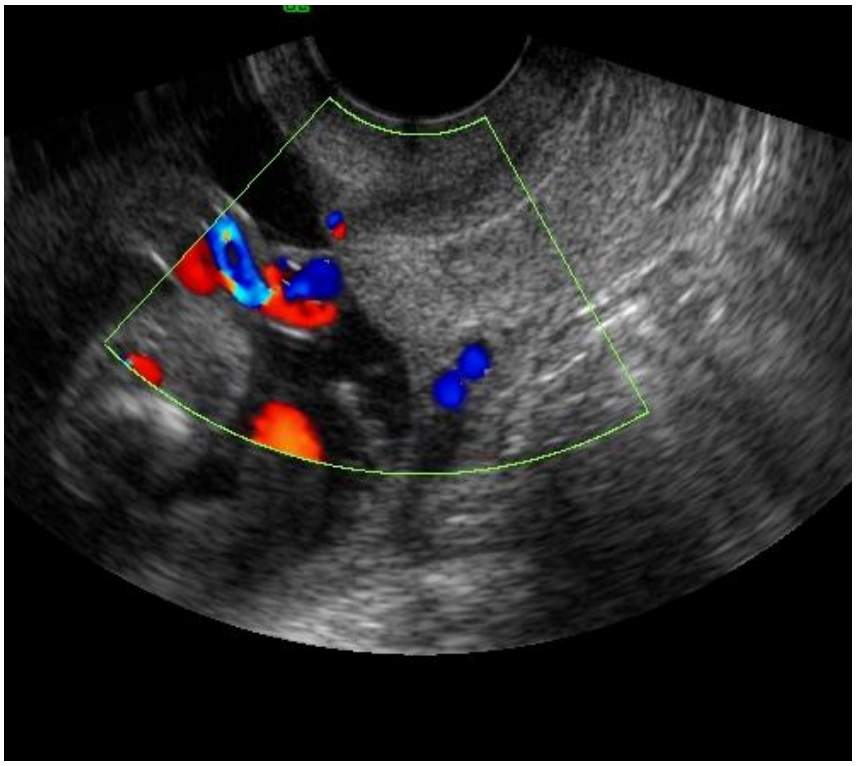

Figure 5. This 28-week ultrasound scan was reported to the provider as vasa previa. It was, however, a funic presentation with a fundal placenta. By the time of delivery, the cord had moved and was no longer an issue.

Velamentous umbilical cord insertion

Velamentous umbilical cord insertion involves a cord that inserts into the membranes rather than into the placental disc (Roberts, 2017). Velamentous vessels run through the fetal membranes without the protection of Wharton's jelly, making them vulnerable to breakage and disruption. Velamentous vessels may run between lobes of a bilobed placenta or between the main placenta and accessory lobes.

Although velamentous cord insertion is sometimes identified on ultrasound, diagnosis is often not made until the placenta is delivered. The condition may be associated with intrauterine growth restriction, preterm delivery, congenital anomalies, low Apgar scores, retained placenta, or antepartum/intrapartum bleeding. Velamentous vessels can also rupture, causing fetal hemorrhage and often death.

Vasa Previa

Vasa previa is a rare condition in which the velamentous vessels cross the membranes of the lower uterine segment in front of the fetal presenting part. If these vessels rupture, the fetus can exsanguinate. Rupture of the vessels can occur with or without rupture of the membranes, resulting in a vaginal hemorrhage consisting of fetal blood. These vessels are also vulnerable to compression by the fetal presenting part.

Vasa previa is more common in pregnancies with a second-trimester low-lying placenta or placenta previa (even if it has resolved), in pregnancies resulting from assisted reproductive technologies, and in women with bi-lobed or succenturiate-lobe placentas in the lower uterine segment. On ultrasound examination, vasa previa can be differentiated from a funic presentation (cord is presenting part) by repositioning the mother into theTrendelenberg position. A cord will shift position; vasa previa will not. Rarely, vessels are palpable on vaginal examination across the presenting membranes.

Consider vasa previa when vaginal bleeding occurs with rupture of membranes and a non-reassuring fetal heart pattern, such as sinusoidal. In the case of vasa previa, only an emergent cesarean section can save the fetus. The hemorrhage consists of fetal blood, and a term fetus contains only about 250 ml. Suggested management for an antenatally diagnosed vasa previa is twice-weekly nonstress testing at 28–30 weeks' gestation, possible hospital admission with continuous monitoring after 32 weeks, and preparation for emergency delivery if fetal distress or hemorrhage occurs. The goal is delivery by cesarean once fetal lung maturity has been established.

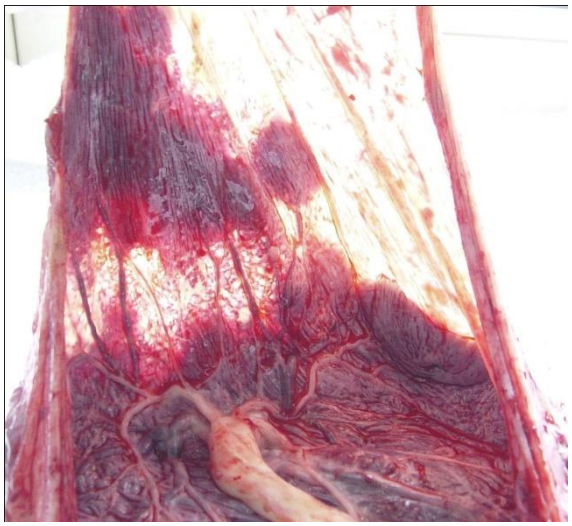

Figure 6. Velamentous vessels running through membranes to a succenturiate lobe.

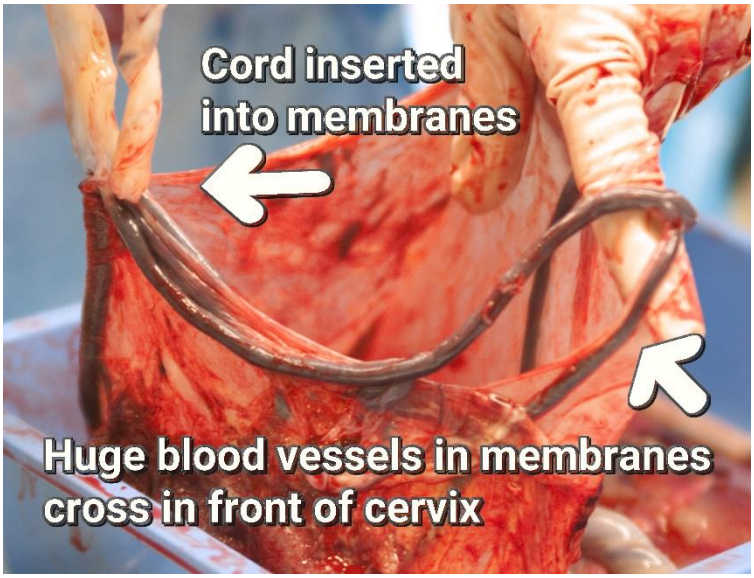

Figure 7. Vasa Previa with velamentous cord insertion on twin placenta. The babies were delivered by cesarean and both were healthy.

GROSS EXAMINATION OF THE PLACENTA

Size

At 24 weeks, the placenta is about one quarter the weight of the baby, and at term it is about one seventh. Abnormally large placentas usually reflect venous congestion from processes such as maternal diabetes, intervillous thrombi, severe maternal anemia, syphilis, chronic intrauterine infection, open neural-tube defects, fetomaternal hemorrhage, fetal cardiac problems, fetal hydrops, placental chorioangiomas, and other conditions. They are associated with a slightly increased risk of neurological abnormalities.

Low placental weight is associated with preeclampsia, infection, fetal growth restriction, and some trisomies. Bilobed or succenturiate placentas may result from the placental adaptation to intrauterine conditions, for example, a bicornuate uterus or uterine fibroids.

Color

Placentas should be deep red. Both abnormally dark and abnormally light-colored placentas are associated with fetal pathology. Fetal polycythemia is associated with a darker, enlarged placenta. A pale placenta reflects fetal anemia or congenital infection, as with parvovirus. Fetal anemia can result from a large fetomaternal hemorrhage, abruption, or other causes of fetal bleeding.

Maturation

Calcification (calcium speckling) is a normal part of placental maturation. Accelerated placental maturation occurs with hypertension and tobacco use and seems to be a response to chronic hypoxemia. Maternal diabetes and fetal anemia are associated with a placenta that remains abnormally immature, at term resembling a second-trimester placenta.

Meconium

Meconium staining of the amniotic fluid occurs when the bowels of a mature fetus begin the normal process of peristalsis, or secondary to fetal distress. Meconium contains bile salts, enzymes, and other compounds. If thick meconium remains in the fluid long enough, it damages vasculature and causes vasoconstriction. Therefore, meconium passage can be either the cause or the result of fetal distress.

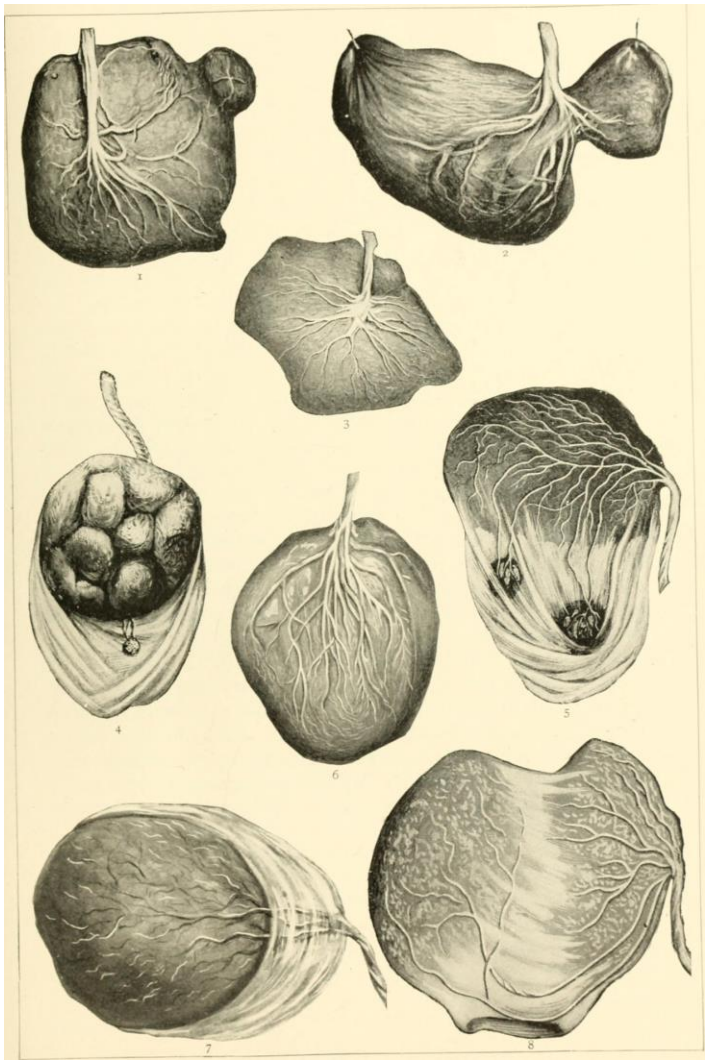

Figure 8. Placentas of all shapes and sizes. I and 2 are placentas with accessory lobes. Placenta 3 shows a normal eccentric cord insertion. 4 and 5 show placentas with velamentous vessels connecting accessory lobes. 6 is a Battledore placenta. 7 is a velamentous cord insertion. A is a bi-lobed singleton placenta with halves connected by velamentous vessels. (Hirst, 1918, plate 3).

Clinically, meconium passage means different things at different gestational ages. At term, meconium passage is common in otherwise uncomplicated deliveries. Whereas the term fetus has meconium in the lower bowel waiting for release, a preterm fetus must move the meconium over a longer distance before it is expelled, reflecting a greater severity or duration of stress. Passage of meconium in the preterm infant is also associated with ascending infections or *Listeria* infection.

Infarctions and Fibrin

Rigid areas in the placenta may be related to fibrin deposition, usually due to infarction or to small hemorrhages under the chorion. A fresh infarction appears red, granular, and firm and may show associated venous congestion. An older infarction also feels granular, but is white, gray, or

yellowish (Roberts, 2017). Placental infarcts are areas of ischemic necrosis following local obstruction of uteroplacental circulation, as in the case of a retroplacental hematoma or a thrombosed maternal blood vessel. In a normal-size placenta, infarcts and fibrin deposits involving greater than 5% of the placenta are associated with fetal growth restriction, stillbirth, and neurological damage (Roberts, 2017).

An orange rind-like band of fibrin deposition along the maternal floor may be a maternal floor infarction, a lesion that carries significant morbidity and recurrence risk. In this case, the placental villi are not dead, but embedded in fibrin. The etiology is unclear, but it is associated with IUGR and a high stillbirth rate.

Chorioangiomas

Chorioangiomas are vascular tumors of the placenta consisting of masses of small capillaries. They seem to be more common in women who live at high altitudes. Small lesions are benign, but large chorioangiomas are associated with polyhydramnios, cutaneous hemangiomas, , and premature delivery. The vascular network of large lesions can place great demands on the fetal heart and even result in fetal congestive heart failure. There is also an association with hemolytic anemia.

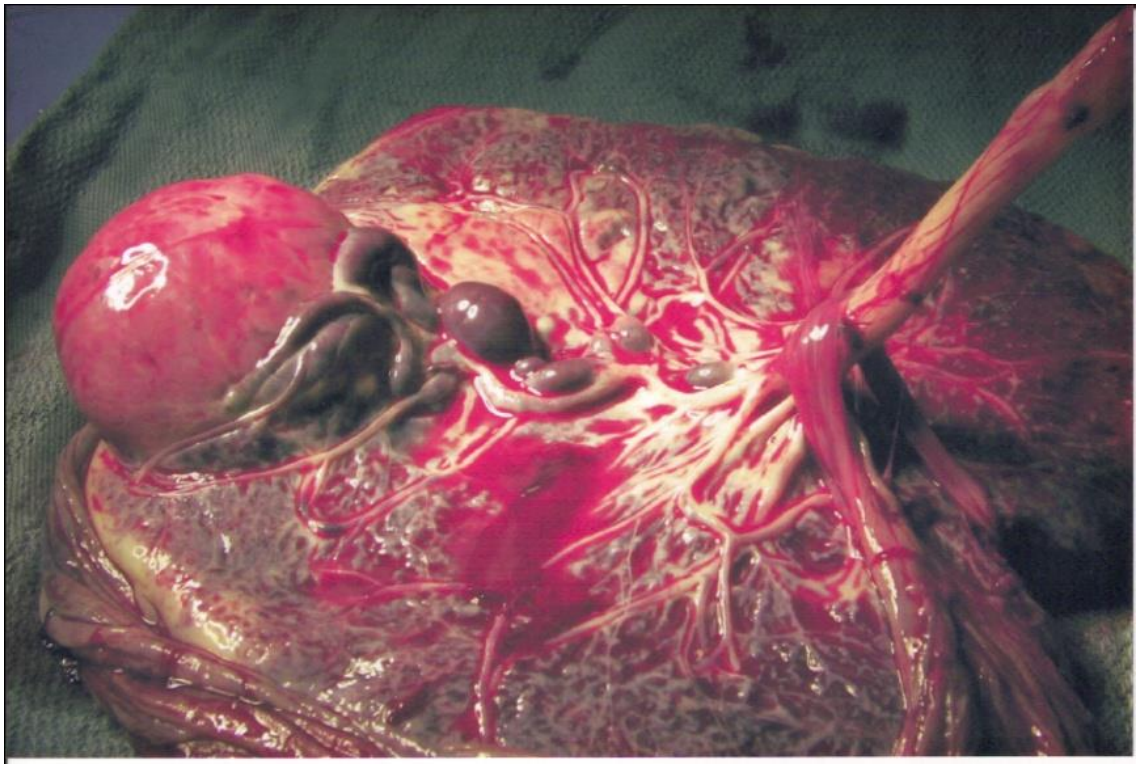

Figure 9. Chorioangiomas are associated with polyhydramnios, premature delivery, fetal anemia, fetal congestive heart failure, intrauterine growth restriction, and placenta previa. In this case, the baby was healthy at birth.

Intervillous Thrombus

An intervillous thrombus is a disruption in the villous vasculature, similar in appearance to an infarction, that allows the fetus to bleed into the maternal system. Fetomaternal hemorrhage (FMH) is a common event, usually involving small amounts of blood transferred across the placenta around the time of delivery. Massive FMH cannot be predicted by clinical risk factors and can occur without any clear-cut clinical signs or predisposing events. More than half occur antepartally, and many are probably unrecognized. FMH is often associated with placental abnormalities such as placental abruption, vasa previa, velamentous cord insertion, chorioangiomas, or choriocarcinomas.

FMH >20–30 mL at delivery is fairly common, occurring in about 1 in 200–300 deliveries. FMH >80 mL and >150 mL appears to occur in 1 in 1,000 deliveries and 1 in 5,000 deliveries, respectively (Moise, 2017). If symptoms are present at all, common warning signs of FMH include decreased fetal movements, hydrops fetalis, sinusoidal pattern on the fetal monitor, fetal or neonatal distress, low Apgar scores, severe neonatal anemia, early neonatal death, and stillbirth. Some infants show multiorgan failure with respiratory distress, enlarged liver, DIC, renal failure, pulmonary hemorrhage, and cardiomegaly. Fetal cells normally persist in the maternal circulation for several weeks, so a Kleihauer-Betke test may be performed on the mother days after the delivery of an anemic infant.

INTRAPARTUM COMPLICATIONS

Abruption

Abruption causes complete loss of local blood flow to the placental tissue. Inspection of the placenta within an hour of the abruption reveals a retroplacental clot that is not tightly adherent to the placenta. Hours later, the clot begins to indent the villous tissue, and the chorionic villi first become ischemic, then infarct. Over the course of days, the infarct will become a firm, yellow/white plaque. Even acute abruptions develop and change over time. Rarely is abruption one brief event. If the abruption involves more than 50% of the placenta, the fetus will usually die if birth does not occur within minutes. Small abruptions are not usually visible on ultrasound.

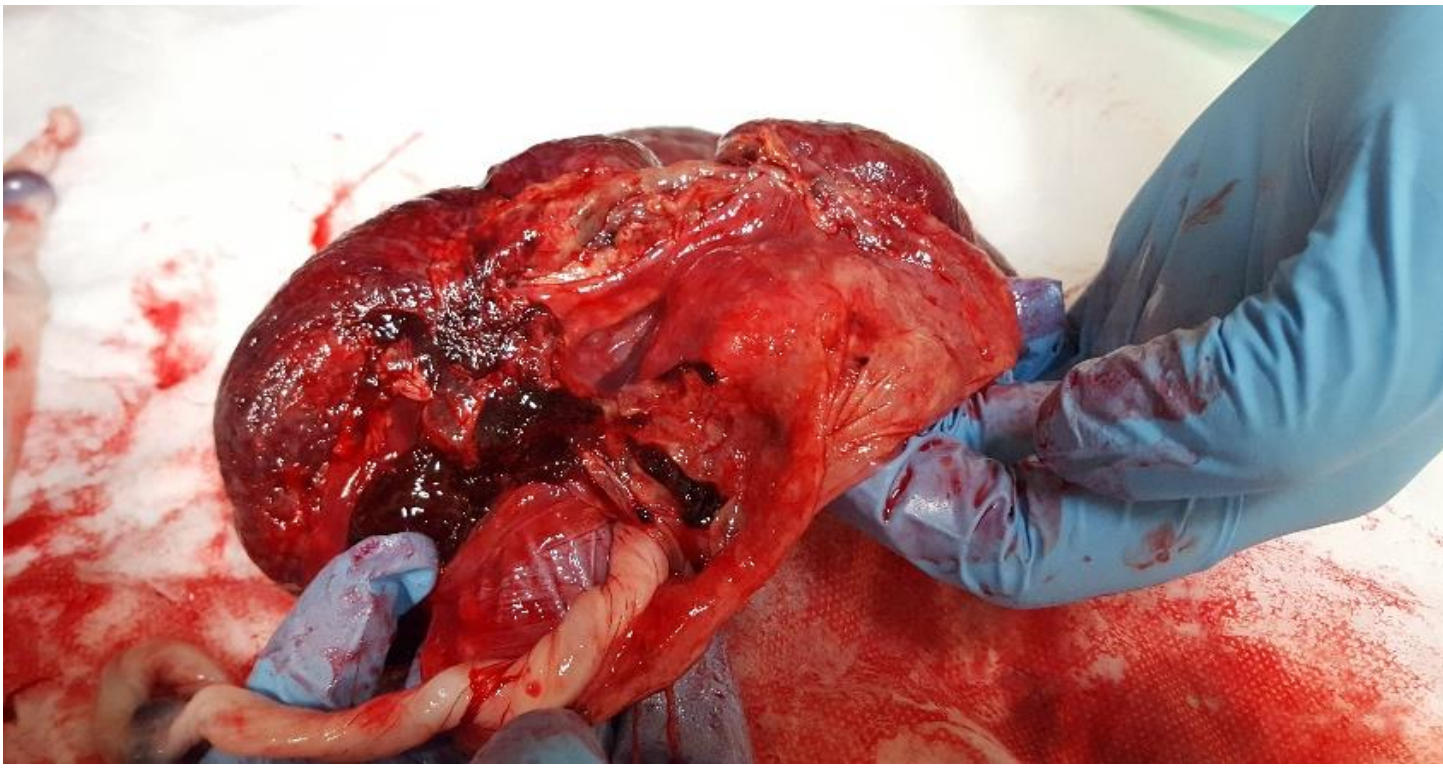

Figure 10. Small, fresh abruption on a circumvallate placenta with a marginal insertion. The membranes ruptured at the onset of second stage, and the fluid was grossly bloody. The baby emerged minutes later covered in blood but with 9/9 Apgars. Evidently, the abruption occurred shortly before delivery and did not compromise fetal oxygenation.

Chorioamnionitis

Chorioamnionitis is strongly associated with premature rupture of the membranes and premature delivery. Infective organisms can ascend from the vagina or—as in the case of TORCH infections, syphilis, and other diseases lurking in the maternal blood—can cross the placenta. Signs include fever (100%); maternal leukocytosis (70–90%); maternal tachycardia >100/min (50–80%); fetal tachycardia >160/min (40–70%), uterine tenderness (4–25%); bacteremia (5–10%); or purulent or malodorous amniotic fluid (Tita, 2017).

The placenta with chorioamnionitis may show congestion of chorion and amnion, with opaque gray-yellow to gray-blue membranes if severe or chronic (Jaiman, 2015). Acute chorioamnionitis may appear normal on gross examination, but is apparent on histological exam.

Intrauterine Origins for Adult Disease

The word term *epigenetic* refers to any process that alters gene activity without changing the DNA sequence, causing modifications that can transmitted to daughter cells (Weinhold, 2006). The intrauterine environment influences the development of adult disease to an extent that we are just beginning to realize. Maternal health and environment can modify the expression of certain genes of the fetus and cause changes that perpetuate over generations, especially in the cases of maternal poor nutrition, obesity, gestational diabetes mellitus; and IUGR (Carolan-Olah, Duarte-

Gardea, & Lechuga, 2015). Severely growth-restricted infants are predisposed to type 2 diabetes and adult coronary heart disease and show decreased scores on childhood on neurological developmental tests (Visentin, et al., 2014). There may also be a direct correlation between the high rate of low-birth-weight babies born to African American women and the high rate of hypertension, diabetes, and end-stage renal disease in this population.

We can reduce the incidence of obesity, diabetes, cardiovascular morbidity, and neuropsychiatric diseases with improved nutrition and reduced exposures to environmental chemicals during development (Heindel & Vandenberg, 2015). Through greater understanding of the mechanisms by which prenatal exposures influence future health, we can improve the lifetime health of generations to come.

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Abigail Eaves, CNM, MSN • Full Circle Midwifery Birth & Health Center

Bonnie Urquhart Gruenberg, CNM, MSN, CRNP, is the award-winning author of *Birth Emergency Skills Training; Manual for Out-of-Hospital Midwives* (Birth Guru /Birth Muse Press, 2008) and co-developer of the workshop bearing the same name. She was trained in a busy home-birth practice, caught her next 1,500 babies at tertiary-care hospitals, and now attends births in a free-standing birth center, at home, and in the hospital with Birth Care and Family Health Services in Lancaster County, PA. Besides *BEST*, Bonnie has written and illustrated 12 books on topics ranging from midwifery and maternity care to the wild horses of the Atlantic coast. She is currently completing the second edition of *Birth Emergency Skills Training* and designing an online CME course to complement it. She enjoys painting, photography, nature, horses, and raising rare-breed chickens.