Chapter 13

Placental Shape Aberrations

General Considerations

Round or oval placentas are the predominant human placental form but many other shapes exist (Fig. 13.1). When the placenta is irregular, its shape is presumably determined by *location, atrophy, and perhaps the manner of original implantation*. Anomalies may develop from abnormal fetal genes expressed by the placenta, an abnormal maternal environment, or an abnormal fetal-maternal interaction. Interestingly, for each aberrant placental shape in humans, there is a counterpart in animals. Thus, the placenta membranacea is similar to the normally diffuse placenta in equines; the zonary placenta is typical of carnivores and so on.

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Abnormally shaped placentas are important to document as they can be associated with adverse outcome.

The Multilobate Placenta

The Bilobed Placenta

One of the most striking abnormalities is the **bilobed placenta** (placenta bilobata), in which *two roughly equal sized lobes are separated by a segment of membranes* (Figs. 13.1 and 13.2). It is present in 2–8% of placentas. The umbilical cord may insert in either of the lobes or in a velamentous fashion (see Chap. 15), in between the lobes, the latter being the most common arrangement. Even if the cord insertion is not velamentous, there *are always membranous vessels connecting the two lobes*. If one lobe is much smaller than the other, the placenta is said to have a **succenturiate** or **accessory lobe** (see below).

Succenturiate Lobes (Accessory Lobes, Placenta Succenturiata)

Succenturiate lobes have an incidence of approximately 5–6%. They may be single or multiple and differ from bilobed placentas only in the size and number of accessory lobes (Figs. 13.1 and 13.3). *Approximately one half are associated with infarction or atrophy of the succenturiate lobes*, much higher than the overall incidence of infarction, which is estimated to be 13%. As with bilobed placentas, membranous vessels are always present, connecting each lobe, and thus may be susceptible to damage. The umbilical cord most commonly inserts into the dominant lobe.

Pathogenesis

The pathogenesis of abnormally shaped placentas is thought to be due to **"dynamic placentation,"** in which the original implantation of the blastocyst is modified by placental expansion overlying poorly decidualized or vascularized regions of the uterus. This can result in local atrophy as growth occurs in other directions, leaving sections of the placenta "behind." A classic example is lateral implantation in between the anterior and posterior walls of the uterus with one lobe on the anterior and one on the posterior wall and atrophy in between. Other local factors leading to multilobation are implantation over *leiomyomas, in areas of previous surgery, in the cornu, or over the cervical os*. After implantation, there is preferential growth in areas of superior perfusion and atrophy in areas of poor perfusion. This process has also been called **trophotropism**. Indeed, intermediate forms exist in which there are two lobes, with partial or complete infarction of residual villous tissue between the lobes with thinning of these areas. This suggests that normal discoidal placentation was originally present, but local factors led to atrophy or infarction, resulting in multilobation.

The designation of multilobate placenta should only be invoked when there is only membranous tissue between lobes.

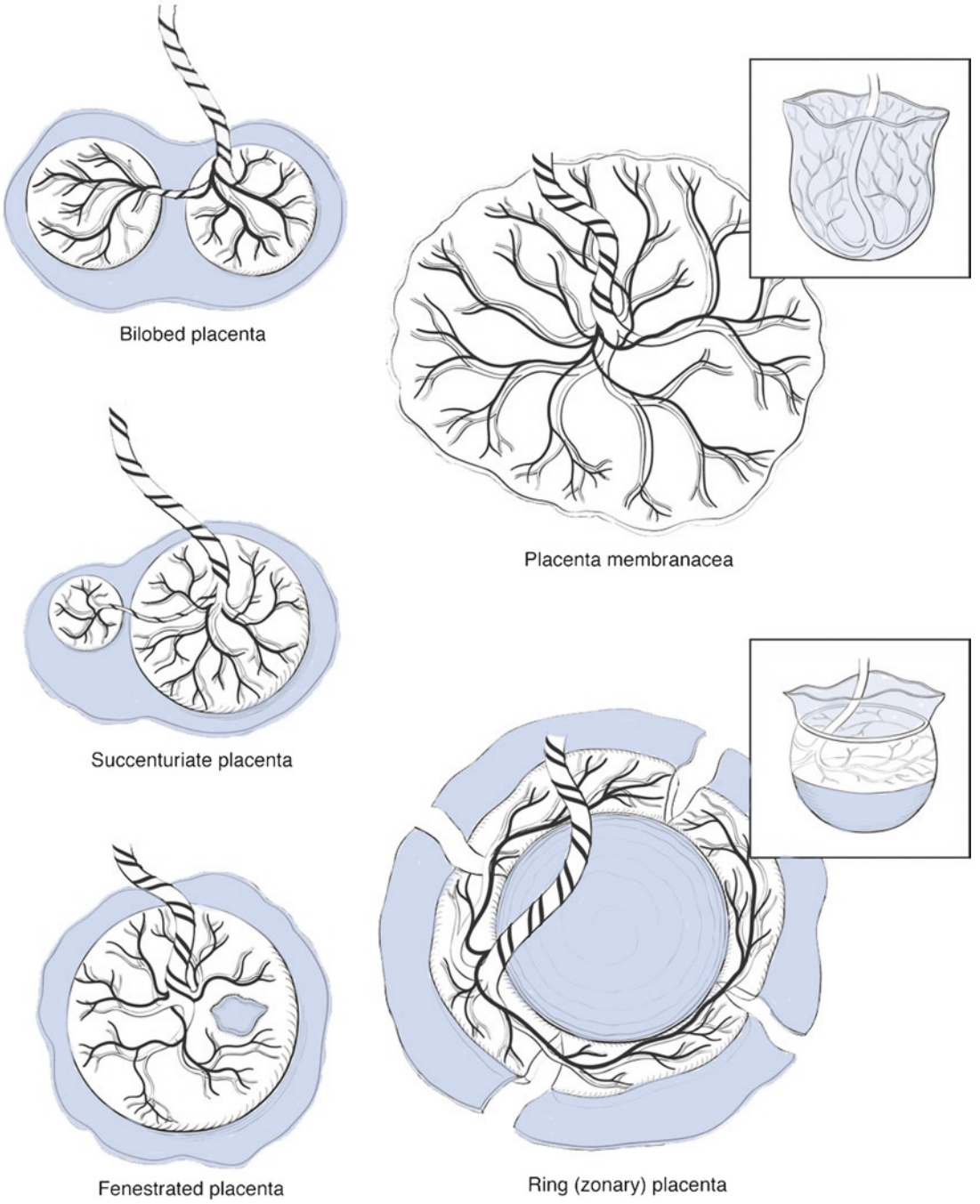

Figure 13.1. Diagram depicting placental shape abnormalities including multilobation (bilobed placenta and succenturiate placenta), placenta fenestrata, placenta membranacea, and ring or zonary placenta.

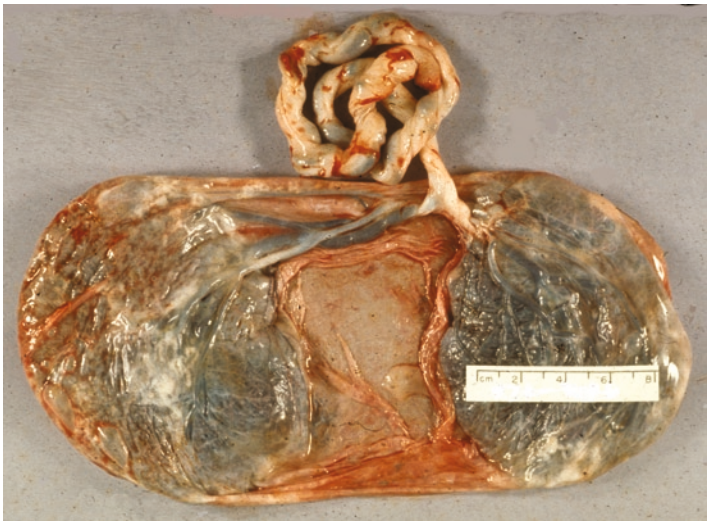

Figure 13.2. Bilobed placenta. Membranous vessels course from the velamentous cord insertion in between the two lobes.

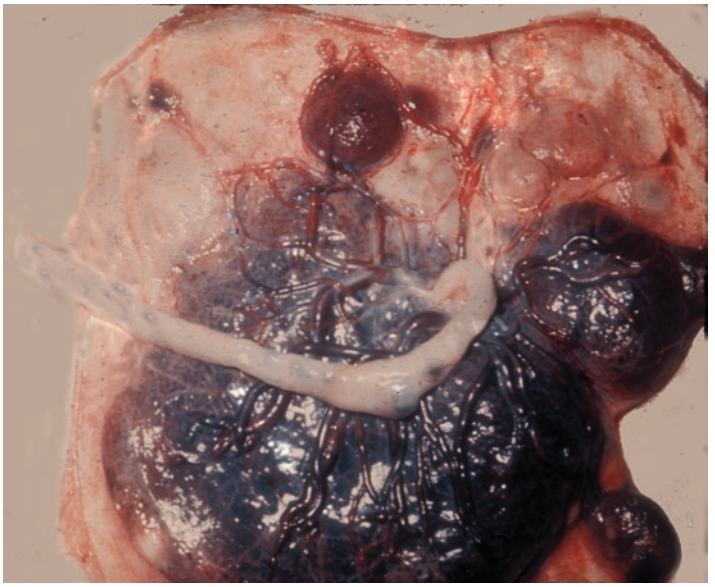

Figure 13.3. Succenturiate lobes in an immature placenta, with infarction of some of the lobes.

Clinical Features and Implications

There are **always** membranous vessels connecting the villous tissue between lobes. These membranous vessels, being devoid of the protection of Wharton's jelly, are susceptible to damage from *compression, rupture, or thrombosis*. They may occasionally present clinically as *vasa previa* with bleeding. Vasa previa occurs when the membranous vessels present "previous" to the delivering part of the baby (see Chap. 15).

Both bilobed placentas and succenturiate lobes are more common in twins and multiparas women and in placentas conceived via assisted reproductive technology. Complications associated with multilobed placentas include *non-reassuring fetal status, antenatal bleeding, postpartum hemorrhage, placenta previa, and retained placental tissue*.

Suggestions for Examination and Report
(Bilobed placenta and placenta with succenturiate lobes)

Gross Examination: Each lobe should be measured and weighed individually unless there are many small accessory lobes. The integrity of the membranous vessels connecting the lobes should be evaluated and it is recommended that sections be taken of the vessels. This is best accomplished by rolling the membranes containing the vessels creating a membrane roll with cross sections of these vessels. If the vessels are ruptured, show hemorrhage into the adjacent membranes or show gross thrombosis, photographs should also be taken.

Comment: It should be noted that accessory lobes are present and in the gross part of the part or a comment, that the membranous vessels are present and whether there is hemorrhage, disruption or thrombosis. If any of these are present, the possibility of fetal hemorrhage should be considered.

Circumvallate and Circummarginate Placentas

Pathologic Features

In **circumvallate** placentas, the membranes of the chorion laeve do not insert at the edge of the placenta but rather at some inward distance from the margin, toward the umbilical cord (Fig. 13.4). At the margin, one usually finds variable amounts of fibrin, recent clot, and old blood. In **complete circumvallates**, there is a complete circumferential ring that restricts the total surface of the chorion frondosum (Figs. 13.5 and 13.6). At the periphery, "naked" placental tissue protrudes. The fibrin that is present at the insertion of the membranes causes plication of the membranes, which is characteristic of circumvallates (Fig. 13.7). The amnion may follow the chorion into this plica, or most commonly it flatly covers the plica without infolding. When no plication of the membranes occurs, it is called a **circummarginate placenta** (Figs. 13.4 and 13.8). These two forms blend into each other, and partial forms are common.

Gross examination of the placenta *shows yellow-brown marginal fibrin* peripheral to the fibrin present at the membrane insertion. In cases where there is midtrimester hemorrhage and premature delivery, there may be substantial blood at the margin. Hemorrhage may undermine the margin of the placenta, thus imitating abruptio placentae. On microscopic examination, sections taken from the margin of the

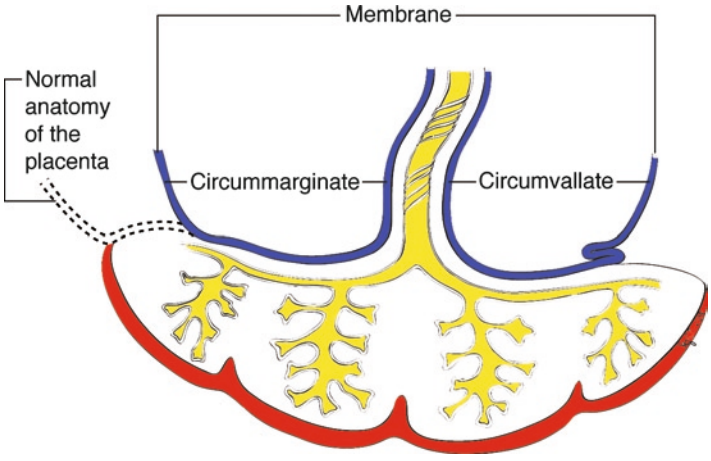

Figure 13.4. Diagram of circummarginate and circumvallate membrane insertion. In both, the fetal membranes do not insert at the edge of the placenta but rather at some point inwards. In circumvallation there is a plication of the membranes evident as a fibrin ridge on gross examination. In circummargination, the membrane insertion is flat and a ridge is not present.

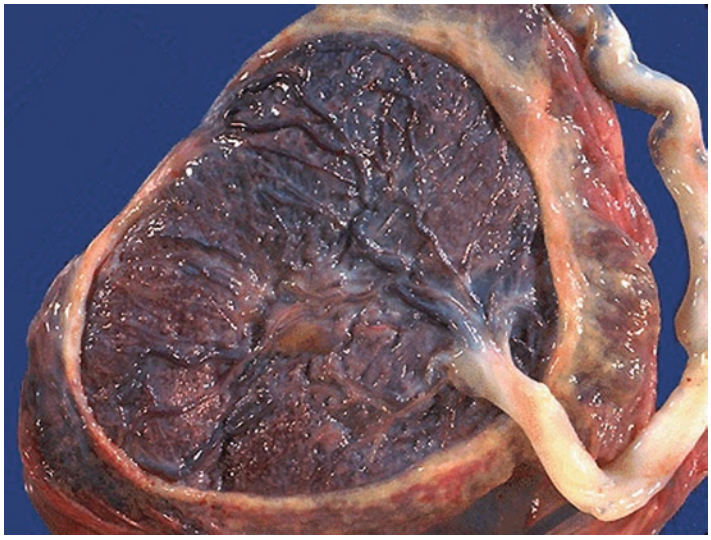

Figure. 13.5. Typical circumvallate placenta with prominent ridge of fibrin at the periphery.

placenta will show *absence of membranous covering peripherally with hemosiderin deposition and fibrin* (Fig. 13.7).

Clinical Features and Implications

The incidence of circumvallation is from 1.0 to 6.5% and the incidence of circummargination is up to 25% of placentas. They are rarely found in the first trimester. The most common complications of circumvallation are *antenatal bleeding and premature delivery*.

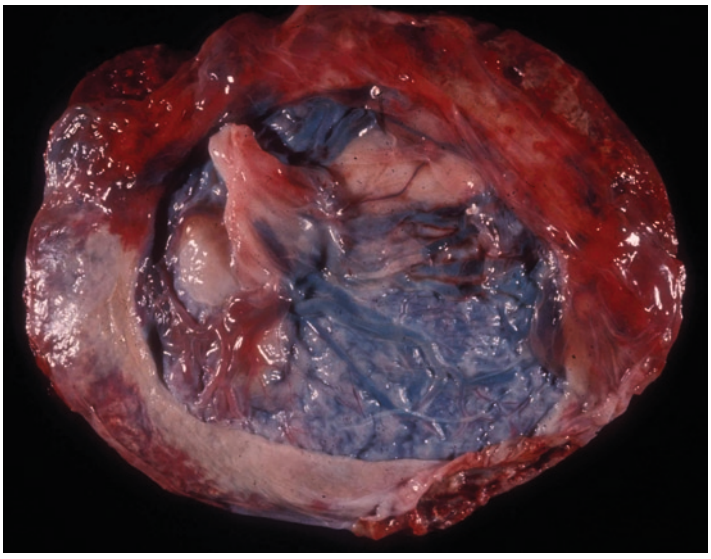

Figure 13.6. More extreme circumvallation with prominent subchorionic fibrin.

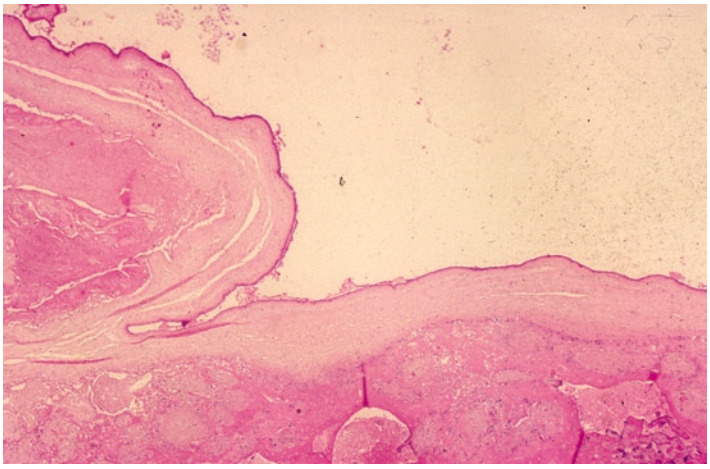

Figure 13.7. Margin of circumvallate placenta. The homogeneous material under the plica represents fibrin, atrophies villi and degenerated blood. H&E ×2.

Additional, uncommon associations include premature membrane rupture, oligohydramnios, non-reassuring fetal status, abruption, perinatal or intrauterine death, congenital anomalies, single umbilical artery, and intrauterine growth restriction. Cases with extensive hemorrhage and **marginal hematomas** may lead to significant clinical bleeding. Large hematomas may elevate the chorion laeve from its insertion site and cause disruption of the fetal vessels. Thus, the vaginal bleeding in these cases is frequently a mixture of

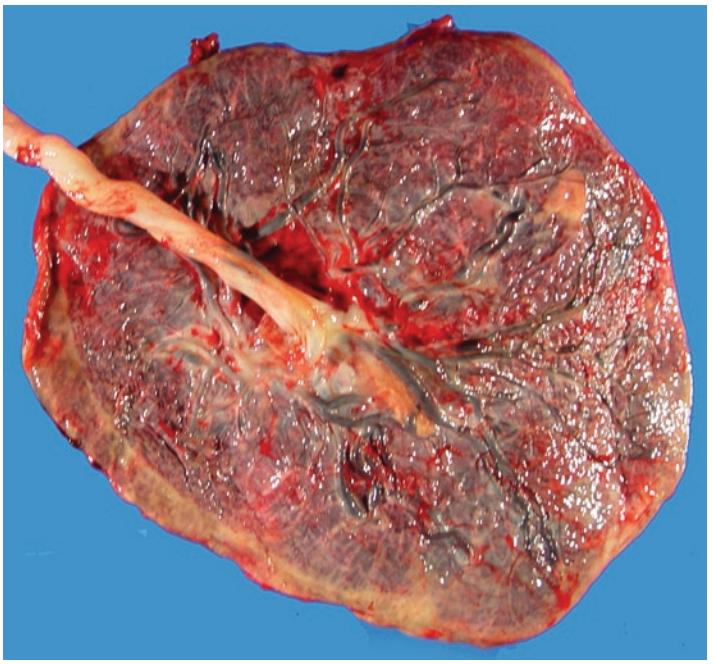

Figure 13.8. Sickle-shaped circummarginate placenta (*lower left*).

maternal and fetal blood. If there is bleeding from placental vessels, significant neonatal anemia may result. It has been suggested that circummargination has little clinical impact, while circumvallation has adverse clinical associations. Many studies have supported this view, but essentially these two entities are really aspects of the same process.

Pathogenesis

A number of theories have been presented as to the origin of circumvallation and circummargination. One theory is that circumvallation occurs due to marginal hemorrhage, which undermines the membranes and pushes them over the chorionic plate. Another theory is that circumvallates develop because the embryo implants too superficially and grows outward in a "polypoid" fashion, and yet another theory is that the embryo implants too deeply. It has also been suggested that there is a deficiency of amniotic fluid pressure necessary for growth resulting in a disconnection between growth of the amniotic cavity and fetus. Others have suggested that the etiology is poor development of the chorion frondosum and increased lateral growth of the placenta. The most recently favored theory is that marginal hemorrhage undermines the membrane attachment. The latter theory is supported by the fact that many of these cases show old hemorrhage around the periphery. However, it is likely that there may be different types and origins of circumvallation, and this issue is yet to be completely resolved.

Extramembranous Pregnancy

Pathogenesis

In **extramembranous** or **extrachorial pregnancy**, there is early rupture of both and the amnion and chorion, leading to escape of the fetus into the uterine cavity (Fig. 13.9). Evidence that the fetus must have escaped out of the membranes much earlier is manifested by the diminutive hole through the membranes, which may barely admit the umbilical cord, let alone the fetus (Fig. 13.9). It is a rare condition, and as with other conditions associated with early membrane rupture, such as amnionic bands (see Chap. 14), the etiology remains obscure.

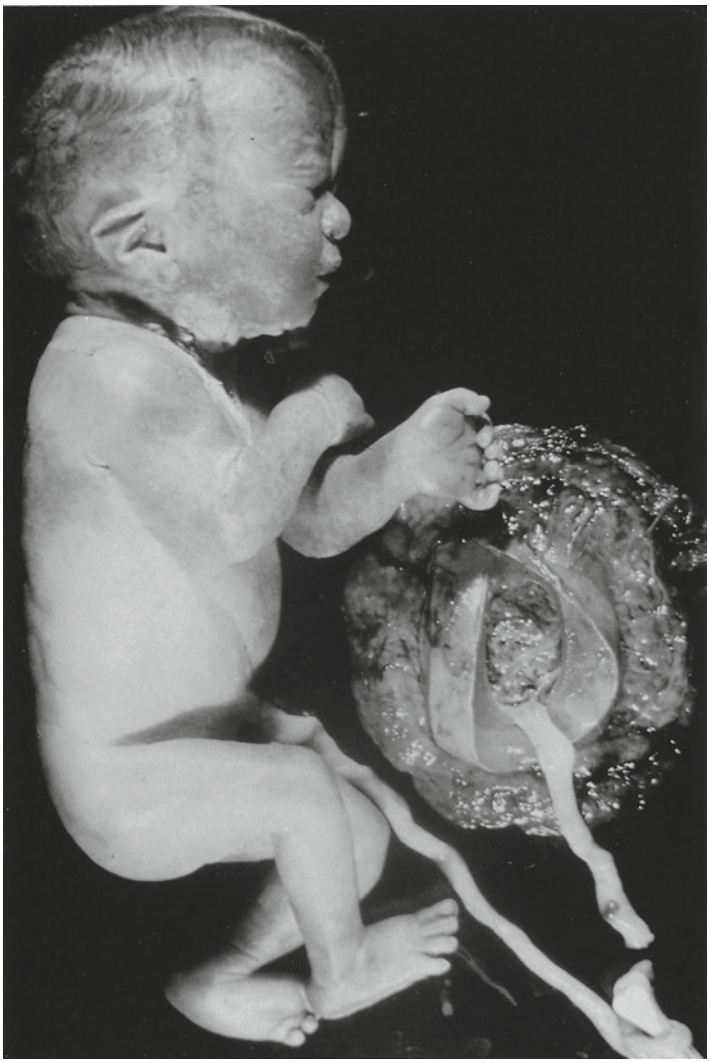

Figure 13.9. Extramembranous pregnancy with fetus. Note the size of fetus and the membrane opening. The placenta shows marked circumvallation.

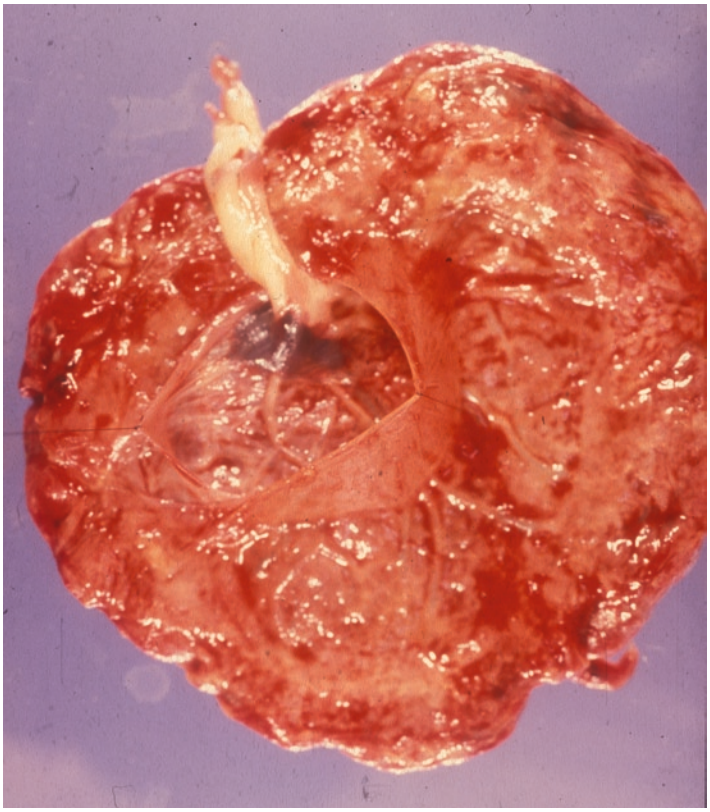

Figure 13.10. Extramembranous pregnancy with circumvallation and diminutive opening.

Pathologic Features

The placenta shows marked circumvallation (Fig. 13.10), the cord is usually short, and the fetal surface shows lack of membranous covering, being covered instead with fibrin and a brownish discoloration consistent with hemosiderin (Fig. 13.11). The remaining membranes are relatively normal. Microscopically, one finds substantial quantities of posthemorrhagic hemosiderin in the membranes. There also may be sparse amnion nodosum (see Chap. 14) over the placental tissue, but this is usually not striking.

Clinical Features and Implications

Due to membrane rupture and loss of amniotic fluid, extramembranous pregnancies are usually associated with prolonged amniorrhea due to periodic fetal urination. In addition, there are severe positional deformities of the fetus and pulmonary hypoplasia due to the oligohydramnios. Ascending infection is occasionally associated with extramembranous pregnancy, but this is not a constant finding. The majority of these pregnancies aborts or terminates prematurely and such fetuses only occasionally survive.

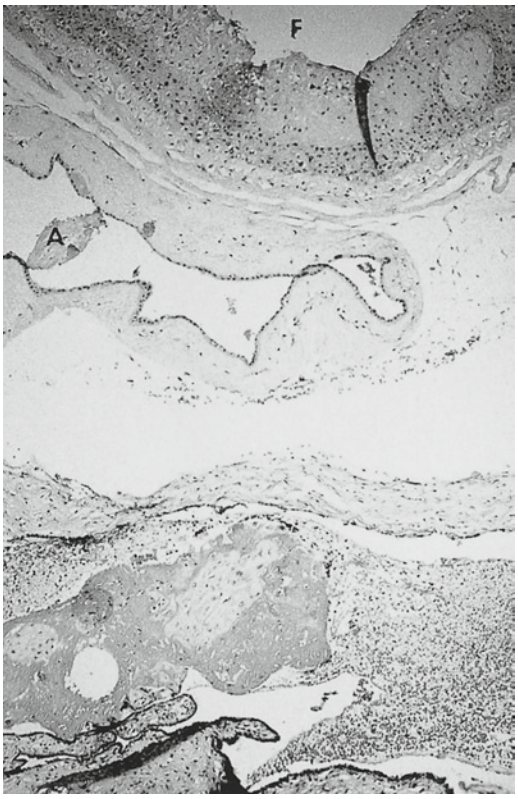

Figure 13.11. Membrane insertion in the extramembranous pregnancy shown in Fig. 13.9. *Below* is the villous tissue, membranes folded at *top right* and the fetus lay in space F. *A* amnion nodosum.

Suggestions for Examination and Report
(Circumvallate placenta, circummarginate placentas and extramembranous pregnancy)

Gross Examination: Extent of the circumvallation should be noted as well as a lack of amnion over the fetal surface and the presence of hemosiderin (brown discoloration) and fibrin. It should also be noted whether there is plication or the membranes (circumvallate) or not (circummarginate). The presence of extensive marginal hemorrhage (marginal hematoma) and disruption of fetal vessels should also be noted.

Comment: A comment is not necessary except when disrupted fetal vessels are present. In this case, the possible role of circumvallation in the etiology may be suggested. If extreme circumvallation and an extramembranous pregnancy are present, a comment may be made on the association with a history of chronic leakage of amniotic fluid prior to delivery, oligohydramnios, fetal positional deformities and pulmonary hypoplasia.

Placenta Membranacea (Placenta Diffusa)

Placenta membranacea is a rare abnormality of placental form in which all, or nearly all, of the circumference of the fetal sac is covered by villous tissue. The placental tissue is generally quite thin (approximately 1 cm), and is often disrupted (Figs. 13.1 and 13.12). Partial placenta membranacea can also occur. The etiology is not fully understood, but it seems obvious that those villi destined to atrophy and become the chorion laeve are retained while there is lack of growth of the villi destined to become the chorion frondosum. Underlying reasons postulated for lack of villous growth relate mostly to abnormalities of the endometrium, such as endometrial hypoplasia, poor vascular supply of the decidua basalis, endometritis, multiple curettages, adenomyosis, or atrophy of the endometrium. Placenta membranacea may manifest clinically as early bleeding and placenta previa. Affected pregnancies often terminate in premature delivery and placenta accreta is relatively common (see Chap. 12). Spontaneous abortion and second trimester fetal demise have also been reported.

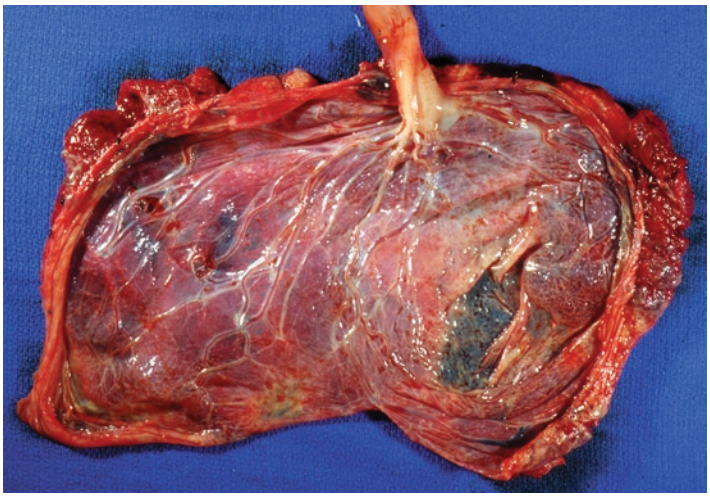

Figure 13.12. Placenta membranacea with virtually free membranes are seen; the placenta is very thin.

Suggestions for Examination and Report
(Placenta membranacea)

Gross Examination: Examination should include the usual measurements of placental dimension such that the increased diameter and excessive thinness of the placenta can be appreciated. As lack of chorion laeve or extraplacental membranes should also be noted.

Comment: Placenta membranacea is of unknown etiology and the clinical associations are vaginal bleeding, placenta previa, premature delivery and placenta accreta.

Miscellaneous Shape Abnormalities

In **placenta fenestrata,** a central area of the placenta is atrophied sufficiently to leave only membranes (Figs. 13.1 and 13.13). One must be careful to rule out the possibility of a missing cotyledon. The etiology is unknown, but it seems reasonable that it may often occur secondary to implantation over a leiomyoma or cornual tube orifice. **Zonary (annular) placentas** have a ring shape (Figs. 13.1 and 13.14). They are likely derived from a placenta previa with focal atrophy of the low-lying villous tissue covering the internal os, leaving a ring shape. Noting the shape and the presence of any membranous or velamentous vessels should be part of the examination of these specimens.

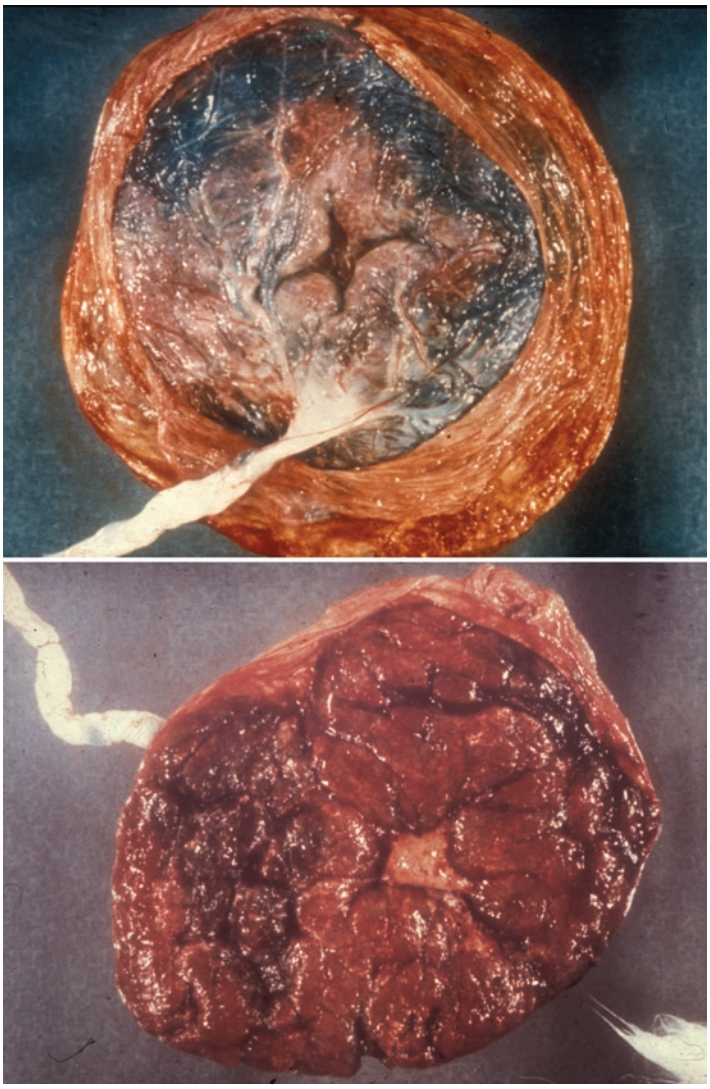

Figure 13.13. Placenta fenestrata. The central area of the placenta has a distinct defect, only chorionic membranes being present. (Courtesy Dr. L.F. Moreno, Caracas, Venezuela).

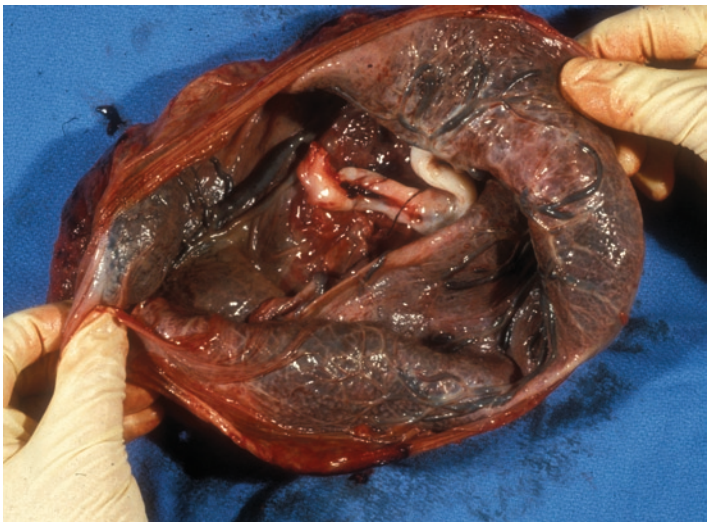

Figure 13.14. Zonary placenta showing a ring like shape. The placenta has been placed on its edge to demonstrate the unusual shape. The membranes ruptured and the infant delivered through the opening. Membranes were intact on the other surface and the cord inserted to the side (not seen in this picture).

Placenta Previa

Pathogenesis

The term **placenta previa** refers to the location of the placenta over the internal os. The placenta is thus "previous" to the delivering part of the baby. The overall incidence is somewhere between 0.3 and 1%. When, during early pregnancy, a placenta previa is unmistakably diagnosed, there is often conversion to a "marginal" or higher-lying placenta. The incidence at midtrimester is 5%, but over 90% of these "convert" to a nonprevia by term. Serial ultrasound examinations throughout pregnancy have shown that the placenta actually "wanders," a phenomenon referred to as **"dynamic placentation"** as referred to above. This placental movement is not accomplished by the placenta unseating and relocating itself, but rather through marginal atrophy on one side and growth and expansion on the other, a process called **trophotropism**.

Pathologic Features

It has become customary to subdivide placenta previa into several categories, such as **"central"** (total) and **"partial"** (lateral or marginal) placenta previa. The former generally poses the greater threat and requires early diagnosis. Vaginal delivery is usually permitted only in a marginal previa. In the vaginally delivered marginal placenta previa, the membranes will have no free margin, and the edge of the placenta will frequently be disrupted and hemorrhagic. There are often old clots at this site, varying from firm, laminated, and brown, to friable loose clots or partly necrotic material that is sometimes green or brown. The fetal

vessels of the chorionic surface, when at the edge, may be also disrupted. Portions of placenta are occasionally either atrophied or infarcted.

Clinical Features and Implications

Placenta previa is more common in older women, and is also associated with multiparity, previous abortion, previous cesarean section, and male infants. Placenta previa is associated with a higher risk for abruptio, fetal malpresentation, postpartum hemorrhage, fetal and perinatal mortality, fetal growth restriction, fetal anomalies, prolapsed umbilical cord, and cesarean section. It is one of the principal causes of third trimester bleeding and often necessitates an emergency cesarean section, as both mother and fetus may experience life-threatening hemorrhage. Maternal hemorrhage may originate from the placental margin or from the disrupted intervillous space. Significant neonatal anemia may result from bleeding that occurs from disrupted placental villous vessels or fetomaternal hemorrhage (see Chap. 20).

Placenta previa is often associated with **placenta accreta** and then is called **placenta previa accreta** (see Chap. 12). In placenta previa there is implantation in the lower uterine segment and cervix where there is no normal endometrium and the mucosa does not respond well to the normal hormonal signals for decidualization. Thus, decidualization is deficient, which is the underlying mechanism in placenta accreta. Although placenta previa accreta and cervical pregnancy are relatively rare, the clinical consequences may be dire due to massive hemorrhage and other complications of blood loss.

Suggestions for Examination and Report
(Placenta previa)

Gross Examination: In a vaginal delivery, membrane rupture site **at** the placental margin is consistent with a marginal placental previa and should be recorded. However, since placenta previa is essentially a clinical term, it cannot be diagnosed by placental examination.

Comment: None.

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PHP5, pages 452–472 (Placental Shape Aberrations).

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