Pathology of the Placenta
Preface

The placenta is one of the most misunderstood and neglected tissues in anatomical pathology. It is a transient organ lasting only for the duration of a pregnancy and yet is of immense value to clinicians in understanding the success or otherwise of the pregnancy. It is also a unique organ in that it is a composite of two genomic contributions and cells derived from the placenta invade maternal tissue during pregnancy; thus, it is of interest to basic scientists studying immunology and cancer biology.

This book describes the pathology of the human singleton third trimester placenta and builds on the initial effort of a group of pathologists, clinicians and scientists who met in Amsterdam in 2014. It aims to provide agreed nomenclature/nosology and definitions of lesions for pathologists; to define thresholds, where possible, for lesions to enable meaningful clinical correlations; to indicate how strong the evidence is for stated clinical correlations, and, hence, provide management guidance for clinicians; and to acknowledge areas of uncertainty to direct future research.

Internationally recognised experts contributed to this book, the contents and text of which were discussed and workshopped at a three-day meeting – the Dublin Consensus Meeting – to result in this collective text. Accordingly, we hope that this book is accurate and lacks bias and that it represents our best understanding of the pathology of the human placenta at this time.

The book is aimed at the practising pathologist in general and community hospitals as well as in teaching hospitals. It will also provide a source of reference for obstetricians, neonatologists, epidemiologists and researchers. Medico-legal practitioners may find the book useful.

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A historical account of the study of the placenta is provided elegantly in Boyd and Hamilton’s monograph [1]. The placenta is named after its shape, resembling a flat cake, from the Greek word *plakoús*. Mossman described it anatomically as an apposition of fetal and maternal tissues for the purposes of physiological exchange. It has a limited lifespan lasting just the duration of each pregnancy.

1.1 Reasons for Examining the Placenta

Placental pathology provides an autopsy of the pregnancy. There are many reasons for a pathological examination of the placenta, chief of which is to explain pregnancy outcomes. The information from such an examination may have important implications for management of subsequent pregnancies in addition to offering an understanding of the pathophysiology of any adverse outcome from the index pregnancy. It is not practical, however, to perform a pathological examination of every placenta, and, at most institutions, only complicated pregnancies lead to the submission of the placenta for pathology. In fact, a major gap in knowledge is the thorough characterization of “normal” placentas throughout gestation. The indications for pathological examination can broadly be divided into maternal, fetal/neonatal and placental and are discussed further in a later chapter (see Chap. 3).

There are also other reasons to study the placenta. Having paternal and maternal genomic contributions, it is a semi-allogeneic tissue that must partially evade the maternal immune system to support the pregnancy. Ovum donor pregnancies, thus being wholly allogeneic, that result in a healthy offspring point to studies that may have implications for solid organ transplants [2, 3]. The controlled proliferation, migration and infiltration of trophoblastic cells, which are derived...
from the trophoblastic shell and from the tips of anchoring villi, into the maternal decidua and superficial myometrium and especially in the arteries in these structures, are a necessary event for successful placentation; the placenta also spawns syncytiotrophoblast metastases to maternal lungs. These phenomena are akin to those seen in cancer [4, 5].

Placental influences are not restricted to the duration of the pregnancy. Increasingly, epidemiologists are keen to study the placenta and investigate how it affects the long-term well-being of both the mother and her child. The so-called “developmental programming” of fetal organogenesis and maternal cardiovascular function are now thought to lead to a range of adult diseases [6].

There is also a medical-legal imperative for examining the placenta. As the “diary of pregnancy”, it may reveal answers that cannot be recovered otherwise. In cases of unknown cause of adverse outcomes, especially neurological, the placenta may help explain the status of the intrauterine environment and its consequences [7]. The increased prevalence of placental and cord abnormalities in children subsequently diagnosed with cerebral palsy underlines the importance of requesting placental histology in all cases where the baby is delivered in poor condition [8].

### 1.2 Diagnostic Challenge

Until recently, the placenta is one of the most misunderstood and neglected tissues in anatomic pathology. Since placental pathology provides insights into the pregnancy, it may not be a surprise that diagnoses often do not directly affect patient management, but we expect as gaps in knowledge are addressed, the overall significance of accurate and reproducible placental findings will modify our understanding of the underlying pathophysiology for many pregnancy complications, including the most common great obstetric syndromes (preterm labour, preeclampsia, fetal growth restriction and stillbirth). The diagnostic challenges appear to be that clinicians rarely employ the placental pathology report in patient management [9], and pathologists are often insufficiently prepared to recognize clinically significant patterns [10].

The reasons why the placenta is perceived as a difficult organ to examine and to provide a pathologic report are unclear. They may be related to the organ itself, to the clinical contexts and to the pathologist (Table 1.1).

The placenta is an organ that develops over the course of the pregnancy and its gross and microscopic features change over that time period. It is important to recognize these differences so as not to misinterpret findings. The clinical contexts are important also: pregnancies with an adverse outcome may have very normal placentas, and, conversely, abnormal placentas may not be associated with any antenatal or postnatal maternal or neonatal complications. Several features may be seen with one clinical condition, while the same feature may be seen in several clinical conditions or contexts. The clinical significance of identified lesions may depend also on the location of the lesions or their size relative to the size of the placenta.

Inter-observer reproducibility between perinatal/placental pathologists and between them and other anatomic pathologists can be poor. While lack of standardisation of diagnostic criteria has been a problem [10], recent developments [11] can be expected to improve the quality of diagnoses in this area.

### Table 1.1 Possible reasons for difficulty in the examination of the placenta

<table>
<thead>
<tr>
<th>Reason</th>
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<th>Solution</th>
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<td>Artefact hampers analysis</td>
<td>Ice crystal, poor fixation</td>
<td>Large fridge; adequate fixative</td>
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<td>Insufficient or irrelevant clinical information</td>
<td>Gestational age and/or birth weight omitted Clinician unaware of significance of findings</td>
<td>Design of request/triage form Education of clinical and midwifery staff</td>
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<td>Pathologist’s lack of confidence in diagnosis</td>
<td>Insufficient numbers to maintain interest and expertise</td>
<td>Service planning</td>
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1.3 An Approach to Examining the Placenta

No approach to a pathological examination of the placenta is any more or less valid than another, although results and comparability of studies rely on a standardised approach to sampling and examination. Unlike other tissues and organs where their examination will usually result in a single diagnosis, placental examination often does not result in a single diagnosis. More often, a synthesis of the various findings in the different parts of the placenta, namely, umbilical cord, membranes, chorionic plate, placental parenchyma and maternal surface/basal plate, is necessary to reach an opinion or diagnosis. Thus, the approach that is advocated in the layout of this book is to allow for a systematic examination of all the parts of the placenta (Figs. 1.1 and 1.2).

**Fig. 1.1** Features or lesions in the extraplacental membranes and umbilical cord. *MVM* maternal vascular malperfusion; *Amsterdam [11]*

**Fig. 1.2** Features or lesions in the placental disc. *FVM* fetal vascular malperfusion, *MVM* maternal vascular malperfusion, *NOS* not otherwise specified, *VUE* villitis of unknown aetiology; *Amsterdam [11]*
This topographic approach is used for the gross and histological examination. Knowing what features or lesions should be sought in the different compartments of the placenta should reasonably eliminate omissions. Some pathognomonic lesions, such as perivillous fibrin deposition, may be seen in only one compartment. Others, such as amniotic fluid infection, may have one feature that may be distributed over different compartments, while other lesions, such as maternal vascular malperfusion, may have different features distributed over different compartments.

Clinical information is absolutely needed to interpret the abnormalities in the placenta. It may provide clues to look for lesions as lesions cluster with some clinical conditions [12], but such clinical information is often lacking in request forms that accompany the placenta for placental examination [13]. A thorough and methodical evaluation of the placenta would ensure that such clustered lesions are not missed but equally ensure that lesions unrelated to the clinical information provided are not missed either.

1.4 Clinical Pathological Significance

Merely cataloguing the various placental findings is not in itself helpful to clinicians. What is important to the clinician and to the patient is what do the lesions mean to them and what do they need to do about them [14]. It is essential that the placental findings be correlated with the clinical data.

Standardisation of diagnostic criteria of placental lesions is an important first step towards effective communication of the findings of placental examinations to clinicians. The lack of internationally accepted nomenclature has led to identical lesions being called differently, while variations or departures from the prototypical lesion features have led to the use of more descriptive labels. These diagnostic labels can be confusing for clinicians who find the placental reports unwieldy and difficult to comprehend. Even where the lesion is described clearly enough to be understood, the threshold where the lesion becomes critical is often unknown. It must be remembered though that clinico-pathological correlations are not static but are moveable, and clinical developments may change the threshold of diagnostic importance. As an example, chronic villitis was associated with basal ganglia and thalami injury in term infants with hypoxic-ischaemic neonatal encephalopathy but not following the introduction of therapeutic hypothermia (Table 1.2) [15, 16].

Evaluation of placental pathology is useful only if it can be communicated to the care providers in a form that is both comprehensible and easy to use for them. The quality of placental pathology reporting can be variable [17]. Synoptic reporting or structured reports may improve the quality of reports by ensuring that essential data are not omitted (explored further in Chap. 57). Obstetricians found reports that classified placental disease into broad pathological processes [18] would improve interpretation [19].

Clinical implications of placental examination findings are discussed in the individual chapters, but their synthesis into a clinical report is critical (discussed in Chap. 56). As alluded to earlier, gross and microscopic placental findings are contextual and need to be placed in the context of the pregnancy and of the placenta. Standardisation of nomenclature will be an invaluable contribution.

<table>
<thead>
<tr>
<th>Period</th>
<th>Clinico-pathologic correlations</th>
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<tr>
<td>Antenatal</td>
<td>Ultrasound findings, e.g., lucency</td>
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<td>Biochemistry, e.g., PAPP-A, AFP Hypertensive disease, e.g., haemorrhage</td>
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<tr>
<td>Natal and immediate postnatal</td>
<td>Obtunded infant—e.g., cord accidents</td>
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<td>Growth-restricted infant—extensive villous damage</td>
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<td>Stillborn—maternal vascular malperfusion</td>
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<tr>
<td>Future pregnancy</td>
<td>Basal plate myometrial fibres and possible placenta accreta</td>
</tr>
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<td></td>
<td>Maternal uteroplacental vascular thrombosis</td>
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<tr>
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<td>Maternal vascular malperfusion and abruption</td>
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Table 1.2 Examples of clinico-pathological correlation
in that respect. It will also allow prevalence studies to be compared between populations and the benchmarking of placental contribution to various pregnancy complications. Parenthetically, the significance of placental findings may be biased because almost all studies have been on convenience populations and not on large unselected populations [20], apart from the large Collaborative Perinatal Study which since when newer placental lesions have been described. There are no studies correlating placental disease with long-term follow-up and lack of cohort studies to truly determine likelihood of recurrence of lesions and their associations.

1.5 The Future

There have been no studies examining the positive or negative predictive values of individual placental histopathological entities. Much of the current literature in placental pathology is based on expert opinion and case or cohort studies [21]. The challenge now is using standardised nomenclature and whole-of-population studies to provide higher-level evidence to validate the predictive and prognostic value of placental examination.

It is essential to accept that there are significant gaps in knowledge between the snapshot in time examined at delivery and underlying pathophysiology. An explanted cirrhotic liver does not explain the cause of the cirrhosis any more than end-stage heart, or kidney pathology does not explain the lifelong effect of systemic risk factors. The same is true for the placenta and the maternal blood vessels that feed the placenta. Currently, most of what we know is from the end-stage disease process or early pregnancy termination samples that are not linked to pregnancy outcomes. What are needed are new approaches to monitor uteroplacental health throughout gestation and relate these metrics to pregnancy outcomes. Novel approaches using magnetic resonance imaging can provide insight into placental anatomy and function [22], while biochemistry profiles and calculated ratios act as surrogates for placental function [23, 24].

Shotgun next-generation sequencing has already challenged the concept of the microbiome of the placenta as being sterile [25], and, while it may be speculative at this stage, application of this technique may identify genomic, metabolic, transcription and protein profiles in different subsets of pregnancy complications. New technologies like quantitative uteroplacental blood flow assessment [26] and liquid biopsies of the placenta may also yield new insights. Moreover, despite differences between humans and animal models of pregnancy, these types of studies may be valuable to generate hypotheses that may then be turned back into human placental research.

References


2.1 Introduction

The placenta originates with the outer cell layer of the blastocyst, the trophectoderm. The inner cell mass gives rise to the embryo. Differentiated subpopulations of trophoblast drive placentation and, together with the extra-embryonic mesenchyme, form most placental tissues [1]. The umbilical cord and amnion are derived from the inner cell mass (embryoblast). Fetal blood derives oxygen, nutrition, waste management and hormonal influence from the placenta. Fetal blood from the umbilical cord disperses into chorionic plate vessels, penetrates through the chorionic plate in artery-vein pairs into the proximal stem villi, travels further to branches of stem villi and, in the mature placenta, enters multiple series of coiled capillaries in the terminal villi via intermediate villi, eventually returning to the fetus along the same circuits via the umbilical vein (Fig. 2.1). Our understanding of how the placenta performs multiple functions is still evolving. The villous vasculosyncytial membranes of the mature placenta resemble the alveolo-capillary membranes of the lung and presumably optimise gas exchange. The histologic structure supporting many placental metabolic and endocrine functions remains undescribed. The pathologist usually handles tissues clinically characterized by the gestational age calculated since the date of last menses. Unless otherwise stated, all gestational ages described below are the menstrual age.

2.2 Umbilical Cord

2.2.1 Umbilical Cord Development

The umbilical cord develops from structures that exit the early embryo at the umbilical ring, the point where amnion meets the embryonic...
ectoderm. These early components are connected in a mesenchymal bridge to the implantation site termed the body stalk, containing paired allantoic arteries arising from the internal iliac arteries, the allantois connecting with the bladder and paired veins. A second mesenchymal bridge contains the vitelline duct connecting the primitive intestinal loop of the embryo to the yolk sac and accompanying paired vitelline vessels. As the amniotic cavity enlarges, it gathers these structures into one umbilical cord lined by amnion. The yolk sac remains separate and distal to the cord in the chorionic cavity between amnion and chorion. The right umbilical vein regresses in the second month of pregnancy. The vitelline (omphalomesenteric) duct, vitelline vessels and allantoic duct also usually regress but small remnants of one or more of these structures may persist. The normal mature cord has two arteries and one vein. Coiling of the umbilical cord is noted as early as 8-week gestation. It is unknown what exactly causes coiling but it appears related to fetal activity.

2.2.2 Gross Appearance of the Umbilical Cord

The normal cord is white, with increasing opacity as gestation progresses. The cord may be discoloured from prolonged meconium exposure, inflammation or maceration after fetal demise. The length and diameter of the cord increase with fetal growth, with increasing size of the umbilical vessels and increasing amounts of Wharton’s jelly. Most cords have a left twist, with an average of 2 coils per 10 cm. The normal range of coiling is 1–3 twists per 10 cm [2]. In some cases a web of amnion tethers the cord to the surface of the disc. On cut section, three vessels (two arteries and one vein) are usually present (Fig. 2.2). A single umbilical artery is present in about 1% of cases. The umbilical arteries commonly anastomose and divide again near the insertion of the cord on the chorionic plate (Hyrtl anastomosis).

2.2.3 Normal Histology of the Umbilical Cord

Microscopic examination shows a variable amount of gelatinous fluid-filled spaces rich in hyaluronic acids in the cord stroma, with scattered fibroblasts, myofibroblasts, rare mononuclear cells and mast cells. The umbilical vein may be larger in diameter than the paired umbilical arteries. The vein typically has a thinner muscular wall (Fig. 2.3). Allantoic or omphalomesenteric duct remnants are fairly common, as are remnants of the vitelline vessels which may show an accompanying small vessel proliferation.

2.3 Extraplacental Membranes

2.3.1 Development of the Extraplacental Membranes

2.3.1.1 Chorion and Decidua

The fetal membranes consist of three layers of diverse origin: amnion from the embryoblast bordering the trophoblast, amniotic and chorionic mesoderm from extraembryonic mesenchyme and chorionic extravillous cytrophoblast cells from the trophoectoderm and decidua (endometrium
modified by pregnancy hormones). Shortly after implantation, decidua closes over the blastocyst. The decidua deep and lateral to the blastocyst (and eventually the placenta) is called the decidua basalis. The layer of decidua overlying the blastocyst is termed decidua capsularis. The rest of the decidua lining the uterus is termed decidua parietalis. The chori-

2.3.2 Gross Appearance
of the Extraplacental
Membranes

The membranes are usually slightly tan with a translucent amnion and thicker chorion. Velvety pink decidua may be present on the membranes of the delivered placenta. The amnion easily

Fig. 2.3 The umbilical cord stroma contains tangled bundles of spiralled collagen fibres set in a gelatinous matrix rich in hyaluronic acid. The umbilical vein has a thin subintimal layer of elastin and layers of longitudinal and circular fibres. The arteries have less elastin and overlapping spirals of vascular smooth muscle.
separates from the chorion and may seem almost gelatinous with its slippery consistency, especially with meconium exposure. Inflammation, infection, meconium and old haemorrhage may make the membranes opaque and discoloured.

### Section 2.3.3 Normal Histology of the Extraplacental Membranes

#### 2.3.3.1 Amnion

The amnion is usually a cuboidal to low columnar epithelium. Reactive changes cause a vacuolated appearance and may cause pseud Stratification. The amnion is closely related to the epidermis; rounded foci of squamous metaplasia are common in the mature placenta. The younger the gestational age, the more
notable the spindled to stellate mesenchymal cells of the mesoderm just below the epithelium basement membrane. The potential space between the amnionic mesoderm and chorionic mesoderm, once the site of the chorionic cavity, is usually visible as separation of the layers.

2.3.3.2 Chorion

The chorionic mesoderm also becomes less cellular appearing with increasing gestational age. One should not mistake the cellularity of first and second trimester chorionic mesoderm for inflammation. The extravillous cytotrophoblast cells of the cellular chorion are round to polygonal with round central nuclei. The cell borders are crisp and the cytoplasm typically eosinophilic on H&E stain, although clear-appearing cytoplasm is also common. Nuclear pleomorphism, evident as hyperchromatic, enlarged, irregularly shaped nuclei, is often seen. Cytotrophoblast cells are known to become aneuploid with numerous copies of the chromosomes from endoreduplication of the chromosomes without mitosis, likely manifest in these enlarged more bizarre-appearing nuclei [3].

The cellular chorion is typically 1–5 layers thick (Fig. 2.6). The cytotrophoblast cells may remain in layers or scatter amongst the decidual stromal cells. The term membranes may show few residual cytotrophoblast cells; other cases may show hyperplasia and microcyst formation [4]. Regressed villi of the chorion frondosum are nearly always seen, especially near the disc margin. Regressed villi may be surrounded by a variable amount of eosinophilic matrix-type fibrinoid.

![Image](image_url)

**Fig. 2.6** (a) Hyperplasia of membranous chorion cytotrophoblast cells, with clusters of cells deeper in the decidua. (b) Clear-cell change of membranous chorion cytotrophoblast cells. (c) Nuclear pleomorphism of membranous chorion cytotrophoblast cells
2.3.3.3 Membranous Decidua

The membranous decidua contains mostly modified endometrial stromal cells, (the plump, spindled “decidual cell”), scattered lymphocytes and histiocytes, occasional decidual glands, frequent spiral arteries that have undergone thinning of the vascular smooth muscle with pregnancy and larger thin-walled decidual veins (Fig. 2.7). Frequently at term, the uppermost decidua under the cellular chorion becomes necrotic appearing, with loss of nuclear basophilia of the decidual cells and vessels, termed laminar decidual necrosis [5, 6]. In some foci, the genesis of the process may show apoptotic change of vessels, with a gradual spreading of cell death into the surrounding decidual stroma. Long considered a degenerative change, the process appears surprisingly orderly, confined to a laminar layer of tissue. While this change may be a part of the mechanism of decidual shedding at parturition, not all placentas from spontaneous vaginal delivery show laminar decidual necrosis (Fig. 2.8). In the majority of cases, the process is bland, whereas in a subset, foci or bands of leukocytoclastic necrosis are seen. Near regions of decidual necrosis, one may see clotting, often in residua of veins, and a variable amount of fibrinoid accumulation.

Fig. 2.7 Spiral arteries of the membranous decidua do not undergo remodelling by invasive extravillous cytotrophoblast cells; they do show thinning of the vascular smooth muscle

Fig. 2.8 (a) Laminar decidual necrosis in the mature membranes. (b) Leukocytoclastic decidual necrosis in the mature membranes
2.4 Chorionic Plate

2.4.1 Development of the Chorionic Plate

At the implantation site, trophoblast in contact with maternal tissues fuses forming syncytiotrophoblast. Closer to the lumen of the blastocyst, they remain cuboidal (cytotrophoblast). This inner layer closest to the embryoblast gives rise to the primary chorionic plate. During the second week post-conception, extraembryonic mesenchyme forms an inner layer over the trophoblast shell. Vasculogenesis begins in the mesenchyme, eventually giving rise to the chorionic plate vasculature [7]. By the fifth week post-conception, when the heart begins to beat, these vessels are connected to the embryonic heart via the connecting stalk. By the fourth month after menstruation, the primary chorionic plate in the region of the cord has formed the definitive chorionic plate of the mature placenta; the remainder of the primary chorionic plate from the gestational sac becomes the membranous chorion (chorion laeve).

2.4.2 Gross Appearance of the Chorionic Plate

2.4.2.1 Shape and Colour of the Chorionic Plate

The chorionic plate is usually oval and consists of tan-white tissue overlying the villous parenchyma on the fetal side of the placenta [2]. The shape of the plate and placental disc can vary (Fig. 2.9). The first and second trimester chorionic plate appears thin, white and transparent with barely discernable chorionic plate vessels. Inflammation causes opacity at this early age. The consistency becomes

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**Fig. 2.9** Placental shape abnormalities. Multilobation (bilobed and succenturiate placenta) is relatively common, while the other abnormalities shown are rare. Placenta membranacea should be suspected when the disc is very broad and lacks significant associated extraplacental membranes. (Reproduced from Baergen, Manual of Benirschke and Kaufmann’s Pathology of the Human Placenta, 2005—Fig. 13.1 p. 209—with permission)
firmer throughout gestation as the plate thickens with collagenous connective tissue, especially around the larger vessels near the cord insertion. The plate becomes more tan and opaque near term in the third trimester.

The overlying amnion is continuous with the membranous amnion and covering of the umbilical cord. The amnion is translucent but may become opaque and discoloured with inflammation or meconium exposure. These processes also may cause the amnion to slide away from the chorionic plate, though it remains firmly attached to the umbilical cord. Accumulation of fibrin beneath the chorionic plate is a common finding at term, visible as tan-white plaques. Thrombohaematomas beneath the chorionic plate may be visible on the surface. Cysts lined by extravillous trophoblast often form beneath the chorionic plate. They may extend into the plate and appear as protuberant thin-walled cysts on the fetal surface of the disc.

### 2.4.2.2 Umbilical Cord Insertion

The umbilical cord may connect with chorionic plate vasculature in the centre of the disc or at any site moving further out the radius to the extraplacental membranes. The cord most commonly has an eccentric insertion [2]. Insertion within 1 cm of the disc edge is termed marginal umbilical cord insertion. Insertion within the extraplacental membranes is termed velamentous. The umbilical vessels typically diverge from the cord within the chorionic plate or occasionally within the membranes. Rarely, the vessels separate before connection to the chorionic plate (furcate insertion) (Fig. 2.10). The chori-
Chorionic plate vessels branch away from a central or peripheral umbilical cord insertion site in a radial distribution. Chorionic plate arteries typically cross over chorionic plate veins. The vessels progressively decrease in diameter with each branching, with substantial cover of the chorionic plate by pairs of an artery and vein diving down to supply at the villous vasculature. With more peripheral umbilical cord insertions, especially marginal and velamentous insertions, the chorionic plate vessels maintain a more constant diameter as the vessels course across the placenta, with fewer apparent ramifications in the chorionic plate. This sparser appearing distribution with large calibre vessels is termed a magistral distribution. The vessels are often “empty” appearing in the first and early second trimester specimen. Progressing to term they become more and more engorged with fetal blood.

2.4.2.3 Insertion of the Extraplacental Membranes

The membranes usually insert at the margin of the disc. Insertion of the membranes inside the circumference of the placenta is termed extrachorial placentation. In circummarginate insertion, the junction between chorionic plate and membranes is flat. In circumvallate insertion, the membranes are folded inwards towards the centre of the disc and a fibrin ridge is palpable. The amnion does not usually accompany the chorion in the fold of circumvallate membrane insertion. (Reproduced from Baergen, Manual of Benirschke and Kaufmann’s Pathology of the Human Placenta, 2005—Fig. 13.4 p. 212—with permission)

Extrachorial placentation. The membranes usually insert at the margin of the disc. Insertion of the membranes inside the circumference of the placenta is termed extrachorial placentation. In circummarginate insertion, the junction between chorionic plate and membranes is flat. In circumvallate insertion, the membranes are folded inwards towards the centre of the disc and a fibrin ridge is palpable. The amnion does not usually accompany the chorion in the fold of circumvallate membrane insertion. (Reproduced from Baergen, Manual of Benirschke and Kaufmann’s Pathology of the Human Placenta, 2005—Fig. 13.4 p. 212—with permission)

Fig. 2.11 Extrachorial placentation. The membranes usually insert at the margin of the disc. Insertion of the membranes inside the circumference of the placenta is termed extrachorial placentation. In circummarginate insertion, the junction between chorionic plate and membranes is flat. In circumvallate insertion, the membranes are folded inwards towards the centre of the disc and a fibrin ridge is palpable. The amnion does not usually accompany the chorion in the fold of circumvallate membrane insertion. (Reproduced from Baergen, Manual of Benirschke and Kaufmann’s Pathology of the Human Placenta, 2005—Fig. 13.4 p. 212—with permission)

2.4.3 Normal Histology of the Chorionic Plate

The amnion overlying the chorionic plate is like that described above for the free membranes. A potential space lies between the amnion and the chorionic plate. The chorionic plate consists mostly of fibrous connective tissue supporting