Practice Guideline for Examination of the Placenta

Developed by the Placental Pathology Practice Guideline Development Task Force of the College of American Pathologists

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• The Placental Pathology Practice Guideline Development Task Force, a multidisciplinary group, has prepared this guideline to assist those involved with placental examination. It provides recommendations related to indications and methods for placental examination as well as sample worksheets. An algorithm for the handling of placentas summarizes the recommendations of the guideline. A summary of specific findings of placental examination together with their pathogenesis and clinical associations is also provided. Recommendations related to reporting with sample reporting formats are included. The guideline is intended as an educational tool, and its use should be guided by the individual circumstances and care setting of specific cases.

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The American Medical Association is working cooperatively with national medical specialty societies, including the College of American Pathologists (CAP), to guide the development and implementation of practice guidelines. In an attempt to improve the quality and consistency of practice guidelines, attributes have been established to guide their development[1]:

1. Practice guidelines should be developed by or in conjunction with physician organizations.
2. Reliable methodologies that integrate relevant research findings and appropriate clinical expertise should be used to develop practice guidelines.
3. Practice guidelines should be as comprehensive and specific as possible.
4. Practice guidelines should be based on current information.
5. Practice guidelines should be widely disseminated.

Several definitions are important to the development, understanding, and use of practice guidelines.

Parameters.—Practice parameters are strategies for patient management developed to assist physicians in clinical decision making. Practice parameters include standards, guidelines, and other patient management strategies.

Standards.—Standards are accepted principles for patient management. Practice variation owing to patient- or physician-specific factors is not expected.

Guidelines.—Guidelines are recommendations for patient management that identify a particular management strategy or a range of management strategies. Practice variation is reasonable to the extent that definitions of management strategies or applicable clinical categories allow incorporation of patient- or physician-specific information.

Options.—Practice variation is expected because implementation of options requires incorporation of substantial patient- or physician-specific information.

Guideline development has become a high priority of organized medicine and the government. It attempts to provide a setting in which the effects of medical intervention on health outcomes can be evaluated, in terms of both human benefit and cost-effectiveness. It seeks to aid in the resolution of competing public, scientific, and personal interests, including the strong governmental and societal impetus to reduce health care costs, concerns for scientific and clinical validity, practical aspects of implementation, and public demand for high-quality health services.

INTRODUCTION

In response to a 1989 resolution of the House of Delegates of the CAP, the College of American Pathologists Conference XIX on The Examination of the Placenta: Patient Care and Risk Management,[2] organized by the CAP

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Surgical Pathology Committee, was held in September 1990. The resolution prompting this conference and the conference itself reflect the increasing awareness of the clinical importance of careful examination of the placenta. The conference brought together experts in placental pathology, perinatology, neonatology, epidemiology, and malpractice law. The conference attempted to arrive at a consensus with regard to the practical and clinically important aspects of examination of the second- and third-trimester placenta. The major conclusions of the conference along with a large body of supporting data were published in 1991.[2] Briefly summarized, these conclusions are the following.

1. All placentas should be examined grossly in the delivery room by the health care provider performing the delivery and a record of that examination should be made.
2. Some maternal and fetal conditions indicate the need for gross and microscopic examination of the placenta by a pathologist.
3. A standardized protocol for placental examination and the reporting of findings that has broad support among professional groups is possible.
4. There are consistently recognizable placental lesions that are reliably associated with maternal or fetal abnormalities.
5. The placenta can provide important evidence in obstetrical malpractice cases.

Following the conference, the Surgical Pathology Committee was charged with the development of a practice guideline for the examination of the placenta based on the consensus statements that were the product of that conference. The Placental Pathology Practice Guideline Development Task Force was formed to develop this guideline. Most, but not all, of the guideline committee members were participants in the consensus conference, and the committee is drawn from the community of health care providers concerned with placental examination, including pathologists, obstetricians, perinatologists and neonatologists, and physician's assistants in obstetrics. The committee also has consulted with the broad community of health care workers involved in obstetric care. The development of a guideline for examination of the placenta reflects a broad-based concern relating to poor pregnancy outcome.

This guideline is intended for use by individual practitioners in the provision of care to individual patients. It is also intended for use by institutions with delivery services to assist in the assessment of care as it relates to both maternal and infant outcome and in the development of appropriate outcome evaluations regarding such care. Moreover, it is designed for use by extrainstitutional reviews in the assessment of broader aspects of perinatal morbidity and mortality.

The CAP House of Delegates endorsed the Practice Guideline for the Examination of the Placenta on April 24, 1996, and the Board of Governors approved it on May 17, 1996. The guideline becomes effective as official CAP policy as of this publication.

Benefits and Outcomes

The well-being of the newborn infant depends on a large number of factors. Only some of these can be assessed by placental evaluation. However, the placenta is often the most accessible and readily evaluable component of the triad of mother, infant, and placenta. It shows the cumulative effects of pregnancy-related events, reflects the intrauterine environment, and can be examined to a degree that the infant usually cannot. There are many instances in which information gained from timely placental evaluation will assist in the timely management of individual patients and may prove exceedingly valuable to the infant and mother involved. However, such evaluation occurs in a very small percentage of deliveries, and the contribution of such cases to improved outcome for infants and their mothers may be too small in the aggregate to be effectively assessed statistically.

Placental examination includes the following benefits.

1. Clarification of the pathophysiology of adverse outcome with a categorization into its various etiologies and into acute and chronic processes.
2. Improved management of subsequent pregnancies for those shown to have conditions that have recurrence risks and may be either treatable or preventable.
3. Understanding of antenatal and intrapartum events that contribute to long-term neurodevelopmental sequelae, with early identification of such changes making possible early interventions and improvement in long-term outcome.
4. Assessment of factors contributing to poor outcome as a factual basis for resolving medicolegal issues.[3]

These benefits are quantifiable as improvements in the rates of prematurity, in the incidence of neurodevelopmental dysfunction from antepartum and intrapartum events, in the perinatal death rate, and in the costs of malpractice suits involving poor obstetric outcomes.

Understanding of the specific etiologies of adverse outcome can lead to specific treatments and preventive measures for those with risks for recurrence in subsequent pregnancies. It can optimize the management of those with nonrecurrent problems. Prematurity, a prominent cause of poor infant outcome, may be the result of many different processes. However, chorioamnionitis, which may be a recurrent condition, is the most important single contributing factor, accounting for about half of all prematurity. Examination of the placenta can identify these preventable or treatable conditions and those with implications for the immediate prognosis or long-term growth and development of the infant, improving outcome in subsequent pregnancies. These conditions include chorioamnionitis and complications of associated prematurity, as well as other less common conditions with risk for recurrence such as abruption, intrauterine growth retardation, and maternal floor fibrin deposition.

It has been known for some time that examination of the placenta is of considerable importance in understanding the factors involved in perinatal death. It has been shown that placental abnormalities can be identified in the overwhelming majority (92%) of perinatal autopsy examinations and that such examinations are diagnostic of the cause of death in a large proportion (32%) and are necessary for the diagnosis in 16%.[4] Although these figures are from an older study and the exact figures may have changed somewhat, the principle remains true.

Assessment of chronic prepartum prolonged and/or recurrent injury with the potential for neurologic impairment and poor neurodevelopmental outcome is an area in which examination of the placenta can contribute

greatly to an understanding of the events associated with such poor outcome and can provide early assessment of injuries associated with poor outcome leading to earlier investigation of infants and the possibility of earlier intervention. Early intervention is the best therapeutic practice currently available to optimize neurodevelopmental outcome. With experience, clinical correlation of placental lesions with neurodevelopmental outcome will be possible. More recently, placental examination has been shown to be valuable in the medicolegal arena, as it provides a set of facts that can be evaluated in assessing the contribution of various factors—maternal, fetal, placental, and obstetric management—to poor pregnancy outcome. This has been documented[5] and has been used as the basis for regional efforts to standardize placental examination (eg, by the Physicians Liability Insurance Company of the Oklahoma State Medical Association Perinatal Task Force and the Oklahoma Association of Pathologists [Oklahoma City, Okla] and the Arizona Medical Association [Phoenix, Ariz]). The decreased cost to physicians, insurance carriers, and society has been well documented in association with this approach[5] (*CAP Today.* April 1988:12–13).

Optimally, outcome measures related to placental examination should evaluate the maternal health status, the child health status, and the reproductive health status in terms of its influence on decisions related to child-bearing and on the outcome of subsequent pregnancies. Specific costs related to these issues should also be evaluated. Studies related to such issues are virtually nonexistent; however, this guideline is intended to provide a foundation for the accumulation of these important health measures.

Other Issues

As the fetus, placenta, and mother constitute the triad of contributors to pregnancy outcome, placental examination becomes an important, but not the only, assessment of pregnancy-related problems. The importance of this assessment is reflected in recent initiatives by state and regional agencies to develop "placenta registries." The use of these guidelines by registries would facilitate comparison of data and associated outcome issues. If registries have different criteria for submission and examination of placentas, their data will necessarily be skewed by the nature of their submissions.

The guideline reflects the best currently available knowledge related to those placental abnormalities that are *known* to be associated with neonatal mortality and morbidity. It seeks to place other abnormalities in perspective with respect to their *possible contribution* to poor pregnancy outcome. The guideline reflects the variable quality and availability of scientific data and experience regarding specific placental abnormalities and their association with neonatal outcome by characterizing such associations as "general acceptance/association," "varied acceptance/association," or "uncertain." The use of the term "general acceptance/association" implies that either there is a strong scientific foundation for the association or the experience of numerous individuals is similar with respect to such associations. The use of the term "varied acceptance" implies that, while there is scientific evidence to support these associations, these data are weakened by either conflicting data or varied experience; the term "uncertain" means that there is limited minority support for these associations. These issues are summarized in Table 2, which contains references to the relevant scientific literature.

The vast majority of placentas are from normal deliveries of healthy term infants, and the costs involved in placental examination, although modest individually, in the aggregate represent a significant health care expenditure. Therefore, this guideline provides an algorithm for the disposition of placentas (Fig 1); it suggests that initial examination, collection of basic information, and triage should be done for all placentas, either in the delivery room or in pathology. The guideline further identifies those conditions (maternal, fetal, and placental) that merit further placental examination and provides a protocol for the short-term storage of placentas that do not initially meet these conditions. Such placentas include those of infants who appear initially well yet have poor outcomes in the early neonatal period. In these situations, the examination of the stored placenta may yield significant information.

This guideline is not intended to preclude the examination of placentas from a wider variety of clinical circumstances than suggested, if those examinations serve specific scientific, clinical, or administrative functions. Indeed, such examinations in settings in which there is considerable scientific interest in the issues involved in placental function as a determinant of pregnancy outcome are necessary to provide data on which this guideline will be periodically refined and revised.

The impact of this guideline on current practice will vary from institution to institution depending on patient population and current policies or lack thereof. The impact of placental examination on the quality of health care provided is difficult to separate from the other components of prenatal and perinatal care; however, it has been shown in defined populations to have a considerable impact on another area of health care–related expenditure, that of malpractice actions for poor perinatal outcome. The Oklahoma experience with placental examination has shown a striking reduction in such actions with a clear overall savings in their associated costs.[5] While this is certainly a desirable outcome and a clearly defined measure of the impact of placental examination on the costs of medical care, it may ultimately lead a more significant outcome—attention focused on the multiple medical, social, and economic factors that contribute to poor outcome and that can be addressed by preventive care.

This guideline, like the conference that was its impetus, covers indications and methods for placental examination and indicates those lesions that are associated with functional abnormalities. It excludes from consideration the examination of the embryo or previable fetus and the examination of early placental tissue as "products of conception." The guideline is confined to examination of second- and third-trimester placental tissues.

It should be understood that adherence to guidelines does not guarantee a successful outcome. Rather, these guidelines are provided as an educational tool to assist pathologists and clinicians in providing quality care. If equally valid guidelines or views advanced by other respected groups are applicable, the pathologist and clinician are, of course, free to follow those authorities. Indeed, the ultimate judgment regarding the propriety of any specific procedure must be made in light of the individual

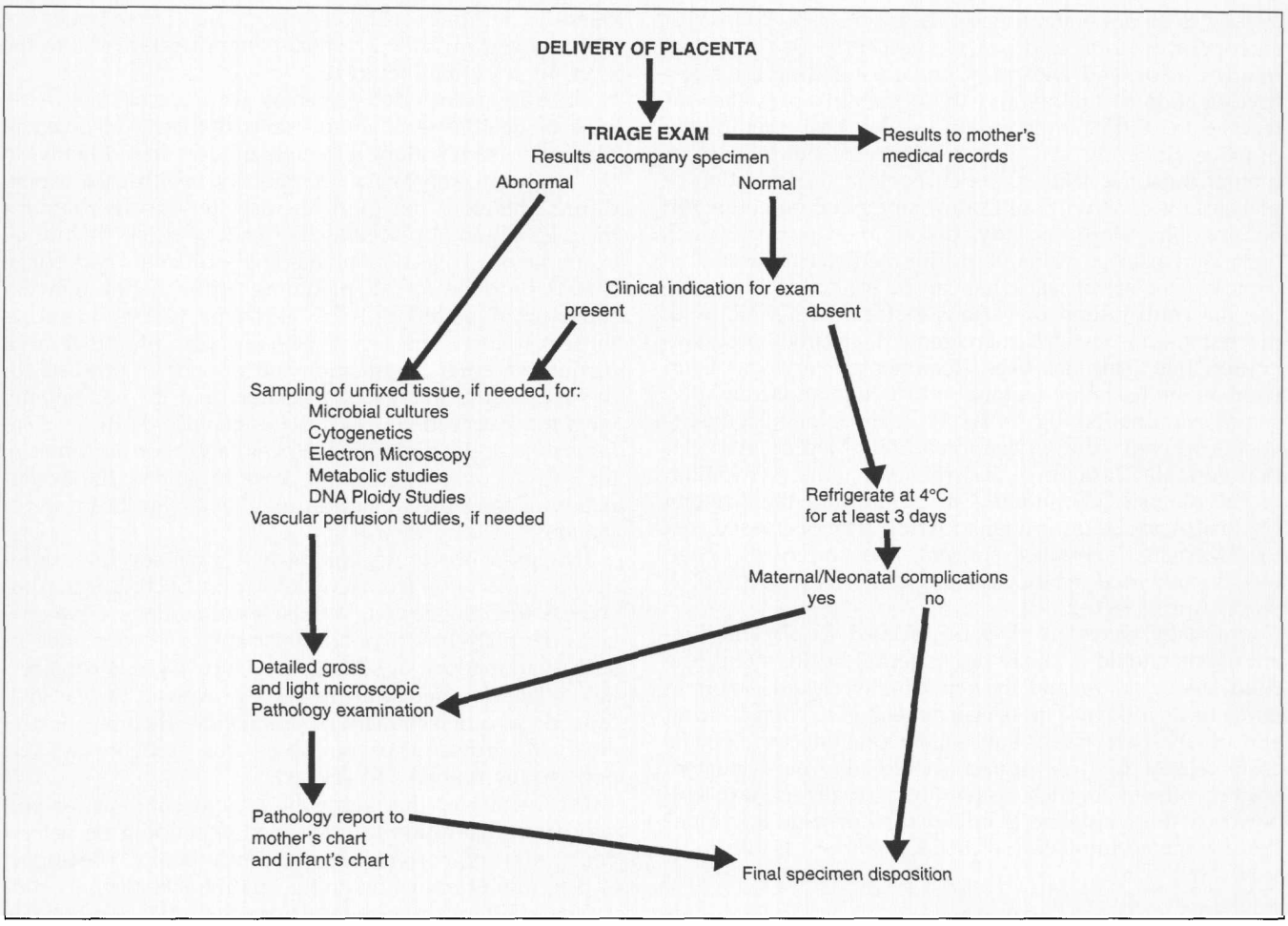

Fig 1.—*This algorithm for the handling of placental tissue summarizes the recommendations of the guideline in that area.*

circumstances presented by a specific patient or placenta in the specific care setting from which they arise.

As already noted, adherence to guidelines is voluntary; however, pathologists and clinicians should familiarize themselves with appropriate guidelines related to placental examination. When a choice is made to deviate from applicable guidelines based on the circumstance of a particular patient or specimen, the health care provider would be well advised to make a contemporaneous written notation of the reason for the procedure followed.

The College recognizes that this document may be used by hospitals and other institutions, managed care organizations, and insurance carriers and other payers; however, the document was not developed for reimbursement or credentialing uses. The College cautions that these uses involve considerations that are beyond the scope of this document.

INDICATIONS FOR PLACENTAL EXAMINATION

It was the unanimous conclusion of the Working Group on Indication for Placental Examination convened at the College of American Pathologists Conference XIX, The Examination of the Placenta: Patient Care and Risk Management,[2] that all placentas should be triaged. This may be done either in the delivery room or in the pathology laboratory (Fig 1). Those placentas that fulfill specific indications need further examination. The number of placentas further studied will vary with the patient population at different delivery sites. Indeed, each site must tailor the criteria outlined here to meet the needs of the patient population it serves. Clinical trials will then validate, refute, or more likely refine the criteria outlined, as they have with the testing of other guidelines.[6,7]

The clinician performing the delivery is privy to pertinent facts related to maternal history, delivery history, infant condition, and specific placental issues (eg, umbilical cord entanglement, prolapse, and length; location of the placenta within the uterus; abruption). That practitioner, therefore, is in the best position to appropriately direct placentas for further study by the pathologist. The clinician may prefer to designate the pathology laboratory as the site of triage. The minimum gross examination (see the Triage Worksheet, Appendix 1) by the delivering practitioner or designee should include total cord length, number of cord vessels, disc dimensions and estimation of disc weight, integrity of the maternal surface, and abnormalities of the free membranes. The most reliable measurement of total cord length is obtained when measured in the delivery room. This can be done using a sterile paper tape included in the delivery pack; alternatively, the delivering practitioner may specify the length of cord segments retained with the infant or submitted for other studies. When triage is done in the delivery room, findings should

be recorded in the maternal medical record and transferred to the infant's record along with other relevant information; in cases where further examination is indicated, the triage findings should accompany the specimen to the pathology laboratory.

More detailed gross and, often, microscopic examination of the placenta is indicated in the presence of certain maternal or neonatal complications of pregnancy, labor or delivery, or certain morphologic placental abnormalities. Issues addressed by placental examination include maternal and neonatal medical concerns for the current and subsequent pregnancies, quality assurance, and medicolegal considerations. The following lists of recommended and other maternal, fetal/neonatal, and placental indications for placental examination were formulated from the appropriate scientific literature.[6-11] and through a survey of placental pathology experts. The scientific literature and expert opinion agree on those conditions in which placental examination is recommended. There is less agreement on those indications listed as other. They should be evaluated in light of the clinical setting.

Recommended Maternal Indications for Placental Examination

Systemic disorders with clinical concerns for mother or infant (eg, severe diabetes, impaired glucose metabolism, hypertensive disorders, collagen disease, seizures, severe anemia [<9 g])
Premature delivery ≤34 weeks gestation
Peripartum fever and/or infection
Unexplained third-trimester bleeding or excessive bleeding >500 cm^3
Clinical concern for infection during this pregnancy (eg, human immunodeficiency virus, syphilis, cytomegalovirus, primary herpes, toxoplasma, rubella)
Severe oligohydramnios
Unexplained or recurrent pregnancy complication (eg, intrauterine growth retardation, stillbirth, spontaneous abortion, premature birth)
Invasive procedures with suspected placental injury
Abruption
Nonelective pregnancy termination
Thick and/or viscid meconium

Other Maternal Indications

Premature delivery from >34 to 37 weeks gestation
Severe unexplained polyhydramnios
History of substance abuse
Gestational age ≥42 weeks
Severe maternal trauma
Prolonged (>24 hours) rupture of membranes

Recommended Fetal/Neonatal Indications

Admission or transfer to other than a level 1 nursery
Stillbirth or perinatal death
Compromised clinical condition defined as any of the following: cord blood pH, <7.0; Apgar score, ≤6 at 5 minutes; ventilatory assistance, >10 minutes; or severe anemia, hematocrit < 35%
Hydrops fetalis
Birthweight <10th percentile
Seizures
Infection or sepsis
Major congenital anomalies, dysmorphic phenotype, or abnormal karyotype
Discordant twin growth >20% weight difference
Multiple gestation with same-sex infants and fused placentas

Other Fetal/Neonatal Indications

Birthweight >95th percentile
Asymmetric growth
Multiple gestation without other indication
Vanishing twin beyond the first trimester

Recommended Placental Indications

Physical abnormality (eg, infarct, mass, vascular thrombosis, retroplacental hematoma, amnion nodosum, abnormal coloration or opacification, malodor)
Small or large placental size or weight for gestational age
Umbilical cord lesions (eg, thrombosis, torsion, true knot, single artery, absence of Wharton's jelly)
Total umbilical cord length <32 cm at term

Other Placental Indications

Abnormalities of placental shape
Long cord (>100 cm)
Marginal or velamentous cord insertion

These general lists of indications for complete placental examination should not necessarily be considered all-inclusive but, rather, as a guide. Often more than one indication may apply. Every institution should establish its own specific list of indications by consultation among the clinical services involved. Delivery by cesarean section is not an indication for submission of the placenta for complete examination.

PLACENTA HANDLING AND TRANSPORT

When the placenta is submitted to the pathology laboratory for examination, the specimen requisition should contain information pertinent to interpretation of the pathologic findings. Information transmitted should include previous obstetric history, obstetric estimate of gestational age, route of delivery, birthweight, sex, Apgar scores at 1 and 5 minutes, maternal and fetal complications of pregnancy, labor, and delivery, and total length of umbilical cord. A copy of the birth record usually contains this information and can be sent with the placenta to the laboratory. Data from the triage examination (see the Triage Worksheet, Appendix 1) should also be provided if that was done in the delivery room. The indications for which the placenta is being submitted should be communicated.

Placentas not submitted for detailed examination should be stored in individual containers, labeled, and refrigerated in the fresh state at 4°C for at least 3 but optimally 7 days.[12] Freezing distorts histologic detail and should be avoided. If the clinical situation dictates that additional studies are needed, the placenta can be retrieved and will be suitable for histologic examination. If an infant is transferred to another facility for care, the placenta, if unexamined, should be examined. This can be done at the institution of either delivery or transfer; if the examination is performed at the institution of delivery, the results should be promptly communicated to the transfer

Table 1.—Special Studies Requiring Fresh Placental Tissue

Bacterial culture	
Indications	Suspected chorioamnionitis; premature rupture of membranes; maternal fever. Evidence suggesting infection in the neonate.
Source	Tissue or swabs from the chorionic plate or peripheral membranes.
Caveats	External amnion may be contaminated with vaginal flora. Such organisms may be significant, but cultures taken from the chorionic surface, beneath the amnion, may be more representative of the organism producing ascending infection.[48]
Type	Usually aerobic and anaerobic; special cultures as clinically indicated
Viral culture	
Indications	Clinical suspicion of viral infection.
Source	Villous tissue taken from the midportion of the placenta or from the subamniotic chorionic plate in a sterile manner, placed in appropriate media, and transported to the appropriate laboratory.
Cytogenetic studies	
Indications	Clinical suspicion of chromosomal/genetic abnormality; multiple congenital malformations.
Source	As for viral culture.
Metabolic studies	
Indications	Fetal hydrops; family history of metabolic disease; clinical suspicion or unusual clinical situations.
Source	At least 10 to 20 g of villous tissue rapidly frozen, retained frozen during transport to appropriate laboratory.
Electron microscopy	
Indications	As for metabolic studies.
Source	Minced villous tissue, 1-mm cubes, placed in glutaraldehyde.
DNA studies	
Indications	As for cytogenetic and metabolic studies.
Source	Fresh or frozen tissue; sterility is not an issue, but contamination is. Imprints of placental tissue, maintained in a clean manner.

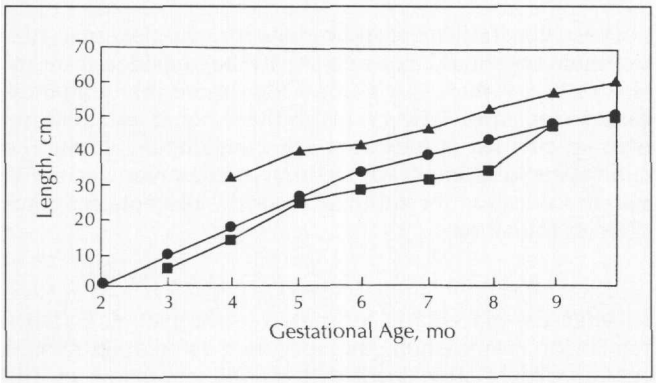

Fig 2.—*Comparison of published cord length studies (from Benirschke and Kaufmann[13]).*

site. If the placenta was examined prior to infant transfer, the report of this examination should accompany the infant.

Placentas should remain unfixed until after triage. Those that do not initially meet indications for further examination should be stored fresh and refrigerated, as already noted. Those that do meet indications for further examination should have any clinically indicated studies requiring fresh tissue done prior to fixation (Table 1), following which they should be handled as are other surgical specimens according to institutional policy (see Workplace Issues, later). If the placenta is placed in fixative prior to transport, an adequate volume of fixative must be used. The placenta should be transported in a large, flat container so that placental shape is not deformed. Fixative volume should be sufficient to completely surround and immerse the placenta.

METHOD OF EXAMINATION AND SAMPLING

The gross examination of the placenta entails careful review of the umbilical cord, placental membranes, fetal and maternal surfaces, and villous tissue.[13–15] Basic measurements are required[17] (see the Gross Examination Worksheet, Appendix 2). All significant changes and lesions should be described and measured, and their locations within the placenta should be noted. Estimates of the percentage of villous tissue or surface involved should be made when the processes are extensive. Photographic documentation of morphology is useful, particularly when there has been perinatal death, questionable or unusual findings, or an untoward outcome of a therapeutic intervention.

The gross examination may be performed in either the fresh or fixed state. Certain procedures, as noted earlier, can only be done on fresh tissue. In many instances, samples for such procedures are best obtained in the delivery room (Table 1). Injection studies in twin placentas can only be done on fresh tissue.[13–16] Surface changes are more apparent in the fresh state, and lesions are easier to palpate. Formalin exposure is minimized. The fixed placenta is more easily transported, less potentially infectious, and may better show infarcts.[14]

During the initial phase of examination of a fresh placenta, any unusual odors should be noted, as they may indicate possible infection. Abruption should be considered when large amounts of fresh clot (>200 cm^3) are present. The examination of the placenta proper should begin with a preliminary general overview of morphology. The fetal membranes should be placed in their usual anatomic position (maternal side outward), and a brief assessment of the membranes, cord, and fetal and maternal surfaces should be made to confirm that no particularly unusual morphology is present that would alter the usual examination procedure.

Examination of the Umbilical Cord

Examination of the umbilical cord should begin with measurement of its length and comparison of that length with standard tables (Fig 2).[13] The length of the umbilical

cord should be measured directly after delivery of the fetus, prior to excision of any segment. Alterations in the diameter of the cord may reflect edema, vascular abnormalities, or a lack of Wharton's jelly. The cord may be inserted in the placental disc centrally or eccentrically, at the margin, or in the membranes (velamentous). The length and intactness of any velamentous vessels in the membranes should be evaluated and noted. The first few centimeters of cord above its insertion into the placenta are frequently constrained by an enfolding web of amnion. The umbilical vessels may lose their covering of Wharton's jelly before insertion into the chorionic plate (furcate insertion). Helical twisting of the cord is usually present, most commonly in a counterclockwise direction as viewed from the fetal end. Aberrations such as excessive twisting or strictures should be identified. The presence and tightness of true cord knots should be noted as well as changes suggesting problems in umbilical cord blood flow such as thrombosis, congestion, thinning, or necrosis.

The number of umbilical vessels should be ascertained in the cord at least 3 to 5 cm above the chorionic plate, as the two arteries may fuse near the insertion. A fresh cut is helpful. At least two pieces of the umbilical cord should be taken for histologic section, one near the placental end and one closer to the fetus. These should be away from sites of trauma such as that of cord clamping. Any unusual lesions should also be sampled. Sites of in utero cord blood sampling identified should be examined.

Examination of the Extraplacental Membranes

The extraplacental membranes should be inspected for unusual coloration, hemorrhagic regions, extra lobes, membranous vessels, and other abnormal alterations (eg, flattened twin). With a relatively intact sac, the distance from the point of rupture of the membranes to the edge of the placenta should be noted. This notation provides some indication of placental location when the route of delivery is known. The mode of attachment of the membranes to the placenta should be noted. This can be at the margin, circummarginate, or circumvallate; the latter two processes can be complete or partial and can be approximately quantified as to extent and degree.

The preparation of the peripheral membranes for histologic sectioning is best done via a membrane roll to increase the surface area examined. A strip of membranes including the point of membrane rupture and the nearest placental edge should be obtained, as this is the region that most frequently shows inflammation with ascending infection. The rolling procedure can begin either at the point of rupture or at the placental edge with a small piece of villous tissue. The membranes should be wrapped around a long, thin object such as a forceps and may be secured with a pin. If the roll is made with the amnion inside, subsequent sectioning will be easier. Brief immersion in an alcohol-based ("hardening") fixative can be used either before or after roll preparation. The roll should be sectioned to provide a spiral cross-section.

Examination of the Placental Disc

The disc weight should be obtained once the membranes, cord, and extraneous clot have been removed. A major variable in placental weight is the amount of fetal blood present in the placenta; this is affected by cord-clamping practices and postdelivery drainage. Standards related both to gestational age and to birthweight are

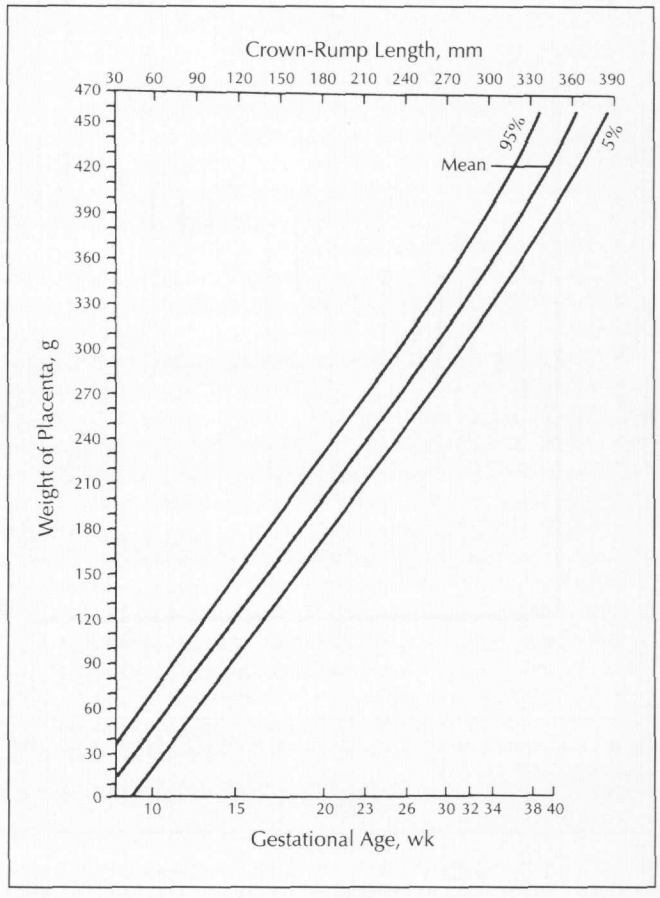

Fig 3.—*Placenta weight related to crown-rump length and gestational age (from Hall et al[17]).*

available (Figs 3 through 5).[17–19] Measurements of the generally ovoid disc should be taken along the greatest major and minor dimensions. Significant deviations of shape from the usual ovoid or round configuration should be noted, as should the presence of extra lobes. The thickness is best measured after cutting, noting unusual irregularity. The fetal surface should be inspected for color, opacity, subchorionic fibrin, and the presence of such lesions as cysts, large subchorionic hematomas, squamous metaplasia, and amnion nodosum. The vessels of the chorionic surface should be examined for thrombi and calcification.

The maternal surface also should be inspected for intactness and the presence of hematomas and/or depressions. Any such lesions should be carefully measured, and large amounts of loose or adherent clot should be weighed. Palpation may reveal other focal lesions that are not visible on the surface but that will appear on sectioning. The parenchyma should be sectioned in a "bread-loaf" fashion at approximately 1- to 2-cm intervals, cutting with either the fetal or maternal side down. The cut slices should be examined for the presence of focal lesions such as infarcts, thrombi, and excessive fibrin.

Small lesions can be individually measured and described. When such processes are extensive or multifocal, an estimate of the percentage of placental involvement should be made. Photographic documentation of the intact or sliced placenta may be useful in recording widespread multifocal processes and in other situations in which a permanent visual record of lesions is sought.

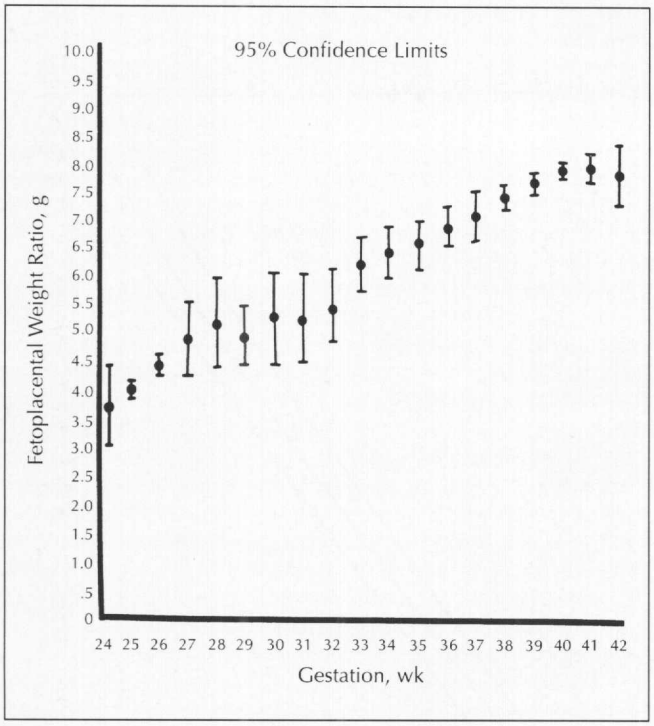

Fig 4.—*Mean fetoplacental weight ratios related to infant gestational age (from Molteni et al[18]).*

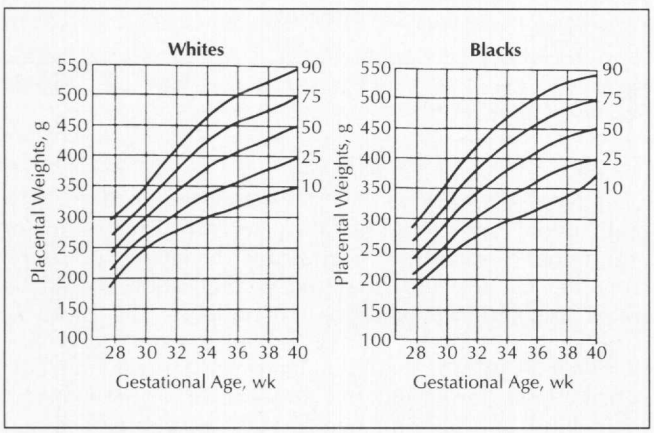

Fig 5.—*Placental growth curves for whites and blacks (from Naeye[19]).*

Examination of the Placentas of Multiple Gestations

Issues that should be addressed in the examination of placentas of multiple gestation include chorionicity, vascular anastomoses, discordance (variability of infant size and development and placental and cord findings), and the vanishing twin. In addition, all considerations for singleton placentas should be addressed. The features unique to the examination of placentas of multiple gestations include sections for chorionicity (dividing membranes) and studies for vascular anastomoses. It is important to maintain the relationship between the individual infant and its placenta and cord.[16]

The cords of multiple gestations should be identified in the delivery room. Institutions should establish a standard convention for identifying individual infants of multiple gestations and their separated umbilical cords. One such convention is for the cord of the first delivered infant to be designated A (with one clamp), the second B (with two clamps), and so forth. If no designation has been made, this should be noted; it is then necessary to make an arbitrary distinction and designation before further examination. In this circumstance, it may be less confusing to use designations other than A and B.

The next step is to identify the number of discs and the character of their dividing membranes to establish chorionicity (see the Twins Worksheet, Appendix 3). Monochorionic placentas always have a single disk. Their thin translucent membranes can be removed without disrupting the fetal surface. Dichorionic placentas may have separate or fused discs. Dividing membranes are relatively thick, forming a ridge where they meet the fetal surface. The line of division of the membranes may not correspond to the vascularization of each twin, as there can be irregular chorionic fusion. A histologic section of the dividing membrane should be taken. This can be in the form of a roll or a T section, the dividing membrane with the underlying chorionic plate; however, a T section may compromise studies for vascular anastomoses.

Vascular communications are present in virtually all monochorionic placentas and are responsible for manifestations of the "twin-to-twin transfusion syndrome" including fetal asymmetric growth, oligo/polyhydramnios, polycythemia/anemia, and disparate organ size. Documentation of vascular anastomoses is part of the evaluation of twin transfusion syndrome. Injection studies using air or liquid can only be performed on the fresh placenta. Even in the fixed placenta, many of the large direct vascular communications can be demonstrated by careful observation of the fetal surface, recognizing that arteries always cross over veins. The deeper connections through shared tissue, often those most significant in producing the twin transfusion syndrome, are frequently much harder to demonstrate, even with injection studies. If the placenta is torn or fragmented, injection studies are often not feasible. For all practical purposes, vascular anastomoses do not occur in dichorionic twins. Other placental abnormalities should be delineated, as there are many other causes of discordant twin growth.

If not already separate, dichorionic placentas can be manually separated, weighed, and examined individually (see the Fused Twin Placenta: Gross Examination Worksheet, Appendix 4). Monochorionic placentas should be weighed and measured intact. They may be separated along the approximate dividing vascular plane, which may not be the same plane as that formed by the dividing membranes. Separation of monochorionic placentas may not provide functional comparison, but separation of dichorionic placentas along the fusion ridge will represent functional comparison. In all cases, differences between the resulting portions should be carefully noted. The remainder of the placental examination is similar to that of the singleton placenta.

The placentas of multiple births greater than twins should be examined using a similar procedure, carefully documenting the type of dividing membrane for each infant pair and the placental and cord features, as already described.

Sampling the Placenta for Histologic Examination

The fresh placenta may be sectioned for histologic examination using a sharp blade or, alternatively, the placenta may be fixed after initial examination prior to sec-

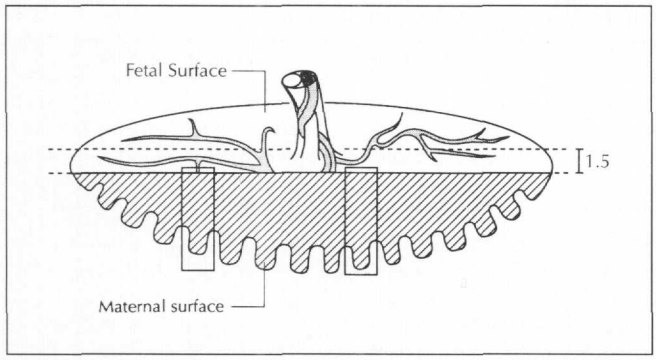

Fig 6.—*The placenta tissue that should be routinely fixed and saved is a transverse strip from the central region, which will often include the cord. This piece should be about 1 to 1.5 cm wide for adequate fixation and is identified by the dashed line in the diagram. Samples of villous tissue should be taken for histologic examination from two separate areas in the placental midzone; these should include the full thickness of the placenta, as indicated by the boxes in the diagram. Tissue near the margin shows substantial artifact due to poor perfusion in this area. Villous blocks ideally include small surface vessels and do not have substantial subchorionic fibrin. The appropriate submitted blocks from a normal-appearing, term, singleton placenta include two blocks of full-thickness placental tissue and one block with membrane roll and two sections of umbilical cord (from Kaplan[15]).*

tions being taken. If sections are obtained from the fresh placenta, rapid turnaround time is possible and formalin exposure is minimized. If fixation is done prior to sectioning, block preparation is usually easier and infectious hazards are minimized. Microwave methodologies to shorten fixation time are available. When sampling the placenta for histologic examination, the membrane roll and the two sections of umbilical cord can be placed together in one cassette, or the cord sections may be separated and designated as nearer the placental or fetal end. Sections of the villous parenchyma should be taken to include the full thickness of the placenta, extending from the fetal to the maternal surface, including both amnion and decidua. These sections should be from the central region, rather than from the margin, which is often nonrepresentative. Sometimes such sections will have to be divided to fit into the standard cassette. If no focal lesions are present, a minimum of two such sections should be taken from different locations of normal-appearing villous tissue (Fig 6). If focal lesions (eg, large infarcts, hematomas) are present, sections of these with a margin of adjoining uninvolved placenta should be taken in addition to at least two areas of uninvolved central villous tissue, as previously detailed. Isolated or very occasional small infarcts or thrombi, which have been clearly identified and described grossly, need not be sectioned; similar but more extensive processes, unusual appearances, and retroplacental hematomas should be documented with histologic examination. A transverse section of the maternal floor has been suggested as a means of obtaining samples of maternal decidual vessels and may be useful in appropriate cases.[20] Following the scheme outlined earlier, most normal-appearing placentas will have at least three blocks processed for histologic examination, whereas those with unusual findings will have more.

The component placentas of multiple gestations should be sampled in a manner similar to those of singletons, with the addition of sections of dividing membranes.

Histologic Processing and Examination of the Placenta

As for most surgical specimens, hematoxylin-eosin staining is routine for examination of the placenta. Special stains including those for organisms (eg, bacteria, fungi, spirochetes), iron, and immunohistochemical stains may occasionally be useful. Microscopic evaluation should include systematic assessment of the amnion, chorion, and attached decidua of the peripheral membranes, umbilical cord vessels and Wharton's jelly, fetal surface, villous tissue, including large and small fetal vessels and intervillous space, and basal decidua. Specific findings are provided in Table 2.

Specimen Retention

The fixed placenta and blocks and slides derived from it should be retained and discarded according to accepted institutional policy and regulatory requirements as for other surgical specimens, and the blocks and slides should be derived from them. Circumstances may occur, just as for other surgical specimens, in which such retention may be prolonged beyond the standard institutional requirements. Retention of placental blocks, slides, and reports should be consistent with state law relating to statutory limits for filing by minors.

Reporting and Distribution

The report concerning the preliminary examination of the placenta by the delivering practitioner or designee should be noted in an appropriate location in the medical record of the mother and transmitted along with other important clinical information to the infant's chart. The delivery room record is often the most convenient place for such notations, as it is generally included in the infant's chart and promptly transmitted to the infant's physician (see the Triage Worksheet, Appendix 1). When the initial examination is done outside the pathology laboratory and the placenta is submitted to pathology, this information should also be conveyed to the pathology laboratory.

For those placentas examined in the pathology laboratory, a pathology report should be issued. While this may have the usual format of other surgical pathology reports, inclusion of birth record information with the report facilitates current and subsequent review. The gross description should contain all observations made on gross examination, including such objective parameters as cord length, insertion, character of the membranes, placental size and weight, and any abnormalities noted. If further studies are done, they should be documented and pertinent findings should be reported. Definite statements regarding the placenta, membranes, and umbilical cord should be made (see the Singleton Report Form and the Twin Report Form, Appendices 5 and 6). When unusual or unfamiliar processes are encountered, a comment may be appropriate. Comments may also be employed to answer specific questions posed by clinicians and to provide appropriate correlation with clinical findings.

Except for transported infants, the placenta will usually be received in the laboratory labeled with the mother's name. In most institutions, this means that the ensuing report is transmitted only to the mother's chart and physicians. A mechanism for conveying this information to the infant's chart and physicians should be established in each institution. Certain placental findings may be unexpected or have immediate relevance to maternal and/or infant

Table 2.—Placental Findings: Description, Pathogenesis, and Clinical Associations†

Findings	Gross Description	Microscopic	Pathogenesis	Clinical Associations
Placenta as a whole				
Weight				
Abnormal fetoplacental ratio[8,13,14,18,19,21–23]	Ratio should increase from 4 at 27 weeks to 7 at term.		Abnormalities may be due to low or high placental weight or deviant infant weight.	Fetoplacental ratio can be abnormal when placental weight is normal in presence of deviant infant weight.
Low placental weight[8,19]	Small placenta for gestational age. Often thin (<1 cm).	May show normal villi. Often "hypermaturation" of villi. Rarely irregular maturation of villi.	Reduced uteroplacental blood flow. Impaired villous growth or development.	*Maternal vascular diseases: hypertension, diabetes mellitus with renal disease, etc. *Chronic infection. *Trisomy syndromes. Severe hemolytic anemia.
High placental weight[8,19]	Large placenta for gestational age. Often boggy, soft, pale, and thick (>3 cm). Retroplacental or intervillous hematoma may be noted.	Villous edema. May have large villi of "delayed" (retarded) maturation. Villous congestion. Pathology specific to diagnoses.	Increased placental "volume" of varied causes.	*Hydrops fetalis/fetal heart failure. *Maternal diabetes mellitus. Maternal anemia and/or malnutrition. Retention of excess fetal blood. Adherent blood clot, retroplacental hematoma. "Tumors" of placenta. *Syphilis and other chronic intrauterine conditions.
Placental coloration[13,24]			Dark color of placenta due predominantly to blood in fetal vessels and fetal hematocrit. Normally darkens with increasing gestational age.	
Diffuse pallor (pale)	Parenchyma pale.	Pallor of chorionic villi. Decreased blood in fetal vessels. Villous edema may be present.	Decreased blood in fetal vessels. Low fetal hematocrit. Excess drainage of fetal blood from placenta after delivery. Fetomaternal hemorrhage.	*Fetal anemia, eg, fetomaternal hemorrhage. (see Edema, under Fetal Placental Vasculature).
Diffuse deep red	Parenchyma congested.	Congestion of chorionic villi. Villous hemorrhage may be present. Differentiate from Chorangiosis, under Fetal Placental Vasculature.	Increased blood in fetal vessels.	*Placental congestion.
Abnormal shape or form[13,22,25,26]	Irregular nonovoid shapes. Includes all multilobed placentas.		Abnormalities of implantation. Failed involution of chorion laeve. Intracavity uterine abnormalities, eg, uterine septum, leiomyoma.	Maternal hemorrhage. Fetal hemorrhage with velamentous vessels.
Placenta membranacea[27,28]	Extensively thin placenta with entire or most of the gestational sac covered with functional chorionic villous tissue. There is no distinct membrane bag.	Very thin placenta.	Failure of involution of chorion laeve.	*Placenta previs is always present. *Maternal bleeding, both antepartum and postpartum.
Placenta extrachorialis (extrachorial placentation)[13,25,29]	Rim of placental tissue extends beyond vascular plate; fibrin at margin. Complete, partial, and mixed forms occur.	Not applicable.	Unknown. Possibly abnormal placental implantation and/or development.	See below for subtypes.

† Asterisk indicates general acceptance or association; question mark, uncertain; and unmarked, varied acceptance or association.

Table 2.—Placental Findings: Description, Pathogenesis, and Clinical Associations† (cont)

Findings	Gross Description	Microscopic	Pathogenesis	Clinical Associations
Circummarginate[13,25]	The marginal white/yellow ring of membrane attachment is flat and nonridged.	Not applicable.	As above.	?Any significant associations.
Circumvallate[13,25]	The marginal white/yellow rim of membranes is folded or rolled back on itself to form a distinct raised ridge. Blood clot often at margin.	Not applicable.	Unknown. Possibly abnormal placental implantation and/or development.	*Acute and chronic maternal hemorrhage. ?Premature rupture of membranes. ?Premature delivery. ?Reduced amniotic fluid. ?Intrauterine growth retardation.
Altered placental lobulation	Varying number of discrete lobes separate from the main disc. Velamentous cord insertion and intramembranous vessels common in all variants. Small lobes subject to infarction or perivillous fibrin deposition.	Not applicable.		*Velamentous/ intramembranous vessels at risk for rupture, hemorrhage, compression, thrombosis, and inflammation.
Multilobed placenta[13,25,26]	More than two lobes, usually of comparable size. Velamentous/ intramembranous vessels common.	Not applicable.		*Risk of fetal bleeding from velamentous/intramembranous vessels. *Maternal postpartum bleeding if lobe(s) retained in utero.
Bilobed (bipartite)[13,25,26,30]	Two lobes of about equal size. Cord insertion generally near junction (66%).	Not applicable.		As above.
Ring-shaped[13,25,26]	A distinct hole (defect) is present in the placental disc. Velamentous/intramembranous vessels may be present.	Not applicable.		As above.
Accessory lobe(s) succenturiate[13,25,26]	One or more lobes smaller than main disc and separate from each other and the main disc. Velamentous/intramembranous vessels often present.	Not applicable.		*Risk of fetal bleeding from velamentous/intramembranous vessels, especially if vasa previa present. *Maternal postpartum bleeding if lobe retained in utero.
Placenta accreta, increta, and percreta[21,15,26,31]	Rarely diagnosed in delivered placenta. Usually only diagnosed accurately when uterus removed. Villous tissue adhered to (accreta), invades (increta), or penetrates (percreta) the uterine wall.	Chorionic villi adhere to, invade, or penetrate the myometrium. There is absence of decidua basalis and a layer of fibrin is sometimes noted.	Deficient decidua basalis with penetration of villi into myometrium. Previous uterine "scar" from cesarean section or dilation and curettage. Low placental implantation.	*Retained placenta. *Postpartum hemorrhage that may require hysterectomy. *Placenta previa/low implantation. *Implants over previous uterine scar. *Recurrence risk.
Placenta previa				
Placenta previa "marginal"	Hemorrhage and blood clot at edge of placental disc. Membrane rupture at edge of placenta in noncesarean section delivery. Marginal cord insertion often present. Velamentous vessels may be present.	?Abnormal villous maturation. Hemosiderin-laden macrophages.	Implantation in lower uterine segment such that internal cervical os not completely covered by the placenta.	?Velamentous vessels. Antepartum and postpartum maternal hemorrhage.

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Table 2.—Placental Findings: Description, Pathogenesis, and Clinical Associations† (cont)

Findings	Gross Description	Microscopic	Pathogenesis	Clinical Associations
Placenta previa "complete"[21,31–33]	Hemorrhage and blood clot over maternal surface of placental disc. Delivery usually by cesarean section.	?Abnormal villous maturation. Hemosiderin-laden macrophages. Area of villous atrophy in region over os. Inflammation sometimes.	Implantation in lower uterine segment such that internal cervical os completely covered by the placenta.	*Antepartum and postpartum maternal hemorrhage. Retroplacental hematoma may also be present. ?Poor fetal growth. ?Central nervous system sequelae.
Umbilical cord				
Single umbilical artery[13,22,25,34–36]	A cord with only two instead of three vessels—one vein and one artery—due to total absence of one of the two arteries or remnant/hypoplasia of one artery.	Absent artery; remnant/hypoplasia confirmed microscopically.	Vessel absent due to primary agenesis or atrophy. In cases of caudal regression and sirenomelia, the single artery may be a persistent vitelline artery.	*Increased incidence of major congenital anomalies, 26%. *Increased incidence of perinatal mortality and morbidity.
Supernumerary vessels[37]	More than three vessels. Additional vessels may be arteries or veins. Differentiate from embryonic remnants (see below).	More than three vessels.	Developmental.	*Fetal anomalies with more than one vein. Be aware of artifact such as tangential section of a vessel loop or normal division of arteries near placental insertion.
Velamentous insertion (velamentous/intramembranous[13,38] vessels	Insertion of cord into membranes such that fetal vessels lie unprotected by Wharton's jelly on placental surface for varying distances in the membranes or between placental lobes. Laceration (hemorrhage) or thrombosis may be noted.	Hemorrhage or thrombosis of vessels.	Placental shape abnormalities and implantation events.	*Fetal blood loss from ruptured vessels. Fetal distress. *Vasa previa. *Common in twins. ?Increased incidence of fetal anomalies.
Furcate cord insertion	Cord insertion over placental disc, but cord vessels fan out over disc to form "tentlike" appearance with membranes. Forklike insertion onto placental disc.	No significant consequences.	No significant consequences.	No significant consequences. ?Compression, thrombosis.
Abnormal cord length	Length varies according to gestational age per percentile charts.			
Short cord[13,22,26,39]	<32 cm at term.	Hematoma or hemorrhage.	Decreased fetal movement. Abdominal wall defects. Primary anomaly.	*Increased in fetal anomalies, especially body wall. *Cord rupture/hematoma. *Fetal akinesia sequence/oligohydramnios. ?Long-term central nervous system sequelae.
Long cord[23,39,40]	>100 cm at term.	Thrombosis, congestion, cord edema.	?Hyperactive fetus.	*Knots, entanglements, and prolapse may cause fetal distress or demise. *Placental and cord vessel thrombus. ?Compression of cord may lead to oligohydramnios.
True knot[37]	Knot of the cord at various locations and degrees of tightness. Edema, grooving, narrowing, tightness, and difference of color or diameter on opposite sides of the knot indicate possibility of obstruction. Congestion on placental disc.	Intravascular thrombus and cord edema may be present. Congestion on placental disc. Thrombus may be present in the knot itself.	Long cord. Increased fetal movement in utero.	Fetal consequences depend on degree of cord obstruction. Intrauterine fetal demise if tight.

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Table 2.—Placental Findings: Description, Pathogenesis, and Clinical Associations† (cont)

Findings	Gross Description	Microscopic	Pathogenesis	Clinical Associations
False knot (varicosity)	A prominent, often tortuous aggregation of dilated ectatic vessels with reduced covering of Wharton's jelly.	Thrombus rarely present.	Lack of Wharton's jelly or tortuosity of vessels.	Insignificant.
Embryonic remnants[37]	Recognized microscopically. In rare instances a cord mass may be present. Microscopy defines lesion. Large ductal remnants must be distinguished from vessel (see Supernumerary Vessels, above)	Epithelial lined lumen in the cord lying either subamniotically (omphalomesenteric) or centrally between the arteries (allantoic). Rarely vascular remnants of the vitelline vessels may persist and be confused with a cord hemangioma. Lesions may calcify.	Persistence of embryonic structures that usually regress.	Usually of no clinical significance. Differentiate mass from teratoma, hemangioma, etc. Large mass (cyst) may cause cord obstruction.
Thrombosis[13,21,24,41,42]	True mural or luminal vascular thrombosis. Thrombosis may extend into placental surface and villous vessels. Recent thrombus soft and "red." Old thrombus firm and "yellow."	Mural or luminal thrombus. Inflammation or necrosis of vessel wall may be noted.	Abnormal fetal circulation. Infection/inflammation. Trauma.	Consequences depend on extent of thrombi and venous thromboembolization. No consequences may occur. In severe cases fetal distress/fetal demise. ?Growth retardation.
Hematoma of the[13,37] cord	Fusiform swelling of varying size and location. Cord may be short. Distinguish from hematomas from cord clamp at delivery (hemostat marks usually present) or traction in third stage of labor.	Hematoma and hemorrhage in Wharton's jelly. Rupture of vessel (usually vein) may be found. Distinguish from true hemangioma.	Short cord. Anomalous vessel. Trauma. "Artifact" due to cord clamp or cord traction after infant delivered.	Hemorrhage in confined space of cord leads to vascular compression, and blood loss leads to fetal anemia. *Both can cause fetal death when large.
Stricture[13,37]	Localized narrowing with loss of Wharton's jelly. Often near fetal body wall.	Narrowed cord. Look for thrombosis.	Primary anomaly vs result of torsion. ?A postintrauterine fetal death artifact. Look for amniotic bands.	?Fetal death.
Torsion[13,37]	Tight spiraling of entire cord length or at fetal abdominal surface. Spirals may be excessive. Spiral usually anticlockwise.	Thrombosis of vessels.	?Abnormal fetal movement. Long cord.	Thrombosis of surface and villous vessels may occur. Obstruction may cause fetal death. May occur after fetal death.
Reduced/absent spiraling	No spiraling of cord (straight).		Unknown. Short cord.	Two-vessel cord. Decreased or absent fetal movement.
Rare cord swellings	Cysts, teratoma, hemangioma.	Similar to these lesions occurring elsewhere. Microscopy defines lesion.	Cysts may be those of embryonic remnants. "Tumors."	In rare cases, large lesions may cause compression of cord.
Inflammation (acute vasculitis, arteritis, funisitis)[13,21,25,37]	Cord may be opaque and even yellow. "White," distinct microabscesses of candida can be recognized on the cord surface.	Neutrophils penetrate into (arteritis/phlebitis) or through (funisitis) vessel wall into Wharton's jelly. One, two, or all vessels may be involved. Vein involvement precedes arterial. Mural or luminal thrombosis may occur.	Fetal response to intraamniotic infection.	*Preterm labor and delivery. Fetal sepsis incidence low. *Vasospasm of cord and placental surface vessels. Fetal distress.
Inflammation (chronic necrotizing funisitis)[13,37]	"Barber-pole" or "cooked macaroni" appearance.	Perivascular rings of chronic and degenerating inflammatory cells and calcification.	Indicates long-standing infection (agent unknown); immune reaction. May be seen with acute chorioamnionitis.	Stillbirth. ?Growth retardation. Chronic infection (ie, syphilis).

† Asterisk indicates general acceptance or association; question mark, uncertain; and unmarked, varied acceptance or association.

Table 2.—Placental Findings: Description, Pathogenesis, and Clinical Associations† (cont)

Findings	Gross Description	Microscopic	Pathogenesis	Clinical Associations
Meconium effects[13,43]	Opaque to green, sometimes mucoid.	Pigment noted in macrophages in Wharton's jelly. Vascular medial necrosis may occur in umbilical and superficial placental vessels and indicates exposure to meconium of more than a few hours.	Passage of meconium by fetus in utero.	*Meconium aspiration. *Vasospasm of cord and placental surface vessels. Fetal distress.
Amniotic web (chorda)	Fold (web) of amnion extends from insertion of cord along one side of cord surface several centimeters. Differentiate from amniotic band (see Early Amnion Rupture Sequence, below, under Extraplacental Membranes).	Not applicable.	Developmental.	?Limits cord movement. No established consequences. May have subamniotic hemorrhage as consequence of traction during delivery of infant or placenta.
Cord edema[37]	Swelling of cord. Increased diameter, sometimes >2.5 cm. Cord translucency.	Edema fluid separates tissue to form numerous "spaces" in Wharton's jelly.		Fetal hydrops or generalized placental edema. ?Maternal diabetes mellitus. ?Abruption.
Extraplacental membranes				
Meconium staining (see also Meconium Effects, earlier)[13,43,44]	Variety of appearances from loss of membrane translucency (opaque membranes) through light green to diffuse dark green to brown often with edematous or mucoid features. Generally not seen in gestations <32 weeks. Pigments seen prior to 32 weeks are usually hemoglobin derivatives.	A variety of changes including vacuolation of amniotic epithelium, pseudostratification, epithelial disorganization, cell degeneration, and even epithelial necrosis with finely granular brown pigment in macrophages of amnion, chorion, and even decidua. Need to distinguish from other brown/yellow pigments—hemosiderin and lipofuscin. Pigment may disappear over time.	Passage of meconium in utero due to bowel peristalsis and relaxation of anal sphincter.	Often seen without obvious consequences to infant. May occur in presence of more reliable indicators of fetal distress. *Vasospasm of fetal and placental surface vessels. Fetal distress. *Neonate at risk for meconium aspiration. Precise timing cannot be determined with certainty because of concentration, repeated passage, and membrane response variables. *Chorioamnionitis and meconium may occur together, particularly in term pregnancy.
Acute inflammation (chorioamnionitis) of placental and extraplacental membranes[13,22,25,45–47]	In severe cases, extraplacental and placental membranes are yellow and even purulent. Milder cases show varying degrees of opacity of membranes with loss of normal shiny translucent appearance.	Extraplacental membranes: acute inflammation of membranes; microabscesses may be seen in severe cases. Placental membranes (over disc): subchorionic margination of inflammatory cells; infiltration in and through chorionic plate to amniotic epithelium; amniotic basement membrane thickening and "hyalinization"; fetal vasculitis/microabscesses. Some infections (ie, group B streptococci) typically show little inflammatory response.	Maternal and fetal response to intraamniotic infection. Pathogenic and commensal organisms may be causative agents.	*Strong association with preterm delivery and premature rupture of membranes. Maternal/fetal infection rate low. ?Vasospasm of cord and placental surface vessels may cause fetal distress. Risk for cord thrombosis. May have abnormal blood flow studies in utero. *Recurrence risk.

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Table 2.—Placental Findings: Description, Pathogenesis, and Clinical Associations† (cont)

Findings	Gross Description	Microscopic	Pathogenesis	Clinical Associations
Decidual lesions				
Inflammation (acute/chronic)	Opaque, yellow decidua.	Acute or chronic inflammation. Plasma cells in rare cases. Differentiate from common maternal lymphocyte response.	Decidual inflammation usually part of chorioamnionitis.	*Inflammation of uncertain significance in absence of chorioamnionitis.
Decidual necrosis	Red decidua.	Foci of necrosis. Microabscesses.	Necrosis with retroplacental hematoma.	Extensive necrosis with retroplacental hematoma. Maternal decidual vascular necrosis with lupus anticoagulant. Focal necrosis of uncertain significance.
Squamous metaplasia	Multiple pearly white, plaquelike hydrophobic lesions with central dimple. Generally small, <3 mm; on amnionic surface; cannot be removed easily; more common near cord insertion.	Stratified squamous epithelium, sometimes with keratinization. Distinguish from amnion nodosum.	Unknown. Normal in late gestation.	None.
Amnion nodosum[13,25]	Multiple warty white to yellow/gray nodules on fetal surface; easily removed. Usually small (<1 mm), but can aggregate into larger lesions. Present on both placental and extraplacental membranes. Uncommon <28 weeks gestation.	Flat amorphous aggregates of various components including degenerating squames, vernix, hair, and eosinophilic material resting on an intact amniotic basement membrane. Distinguish from squamous metaplasia.	Prolonged fetal contact with membranes due to long-standing oligohydramnios leading to aggregates of amniotic material on the amnion surface.	*Prolonged oligohydramnios from chronic leakage of amniotic fluid, oliguric renal anomalies, and prolonged retention of fetus in utero after intrauterine fetal death. *Pulmonary hypoplasia likely with long-standing oligohydramnios. *Fetal deformation from intrauterine compression.
Early amnion rupture sequence (amniotic band syndrome, amnion disruption sequence)[13]	Strands (bands) across fetal surface. Defects in amnion reveal shaggy opaque appearance of exposed chorion. Bands may attach to fetus or umbilical cord. Tight bands may constrict umbilical cord. Amniotic defect varies from focal to diffuse. Distinguish from body wall defect.	Amniotic epithelium is absent with adherent squames on sclerotic chorionic surface. Inflammation may be present.	Chronic rupture of amnion of undetermined cause.	*Fetal amputations, constrictions, and other disruptions often seen. *The more complex the fetal anomalies, the earlier the amnion rupture occurred. *Compression deformations from oligohydramnios. *Random event in classical case. Distinguish from body stalk defect, which may have recurrence risk.
Maternal uteroplacental vasculature				
Decidual vasculopathy[13,22,25,26]	No specific features. Placenta may be small for gestational age. Infarction may be present. Retroplacental hematoma may occur.	Alteration of decidual vessels with fibrinoid change and foamy intimal cells (atherosis); thrombosis. Intimal hyperplasia (occurs with hypertensive category.	Results from lack of or inadequate physiologic trophoblastic vascular implantation changes. Causes decreased blood supply to intervillous space.	*Hypertension. *Preeclampsia. *Anticardiolipin antibody. *Recurrence based on recurrence of maternal condition. *Intrauterine growth retardation. *Fetal demise.

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Table 2.—Placental Findings: Description, Pathogenesis, and Clinical Associations† (cont)

Findings	Gross Description	Microscopic	Pathogenesis	Clinical Associations
Infarction[13,21,24–26,48]	Features depend on age of lesion, ranging from dark red in the fresh infarct through brown-yellow-orange to white in old. Generally connected to the basal plate; have a lobular configuration with a distinct border and surrounding normal villous tissue. May vary in size, age, and location, and are often multiple. Central infarcts are more significant; small marginal ones occur incidentally and are of no clinical importance.	Cardinal features are obliteration of intervillous space with villous crowding. Early infarcts show congested, sometimes hemorrhagic villi. Necrotic changes include pyknosis and karyorrhexis of trophoblast with eventual loss of trophoblast structure. Trophoblast nuclear staining persists longer than stromal nuclear staining. Syncytial knots are often prominent. Final stage is coagulative necrosis with "ghost villi." Important negative features: absence of villous stromal fibrosis and of cytotrophoblast proliferation in early stages. Need to differentiate from perivillous fibrin deposition.	Caused by decrease/interruption of maternal blood supply to the intervillous space.	*Maternal hypertension, particularly preeclampsia and eclampsia. Maternal diabetes mellitus. *Systemic lupus erythematosus. Frequently found in absence of clinically apparent hypertensive or other disorder. *Clinical associations correlate with extent of lesion, particularly when >15% of placenta involved. *Intrauterine growth retardation. *Fetal distress. *Fetal demise. ?Cocaine abuse.
Intervillous thrombus[13,24,25,49]	Old to recent laminated thrombi usually central intervillous, often multiple, may be up to several centimeters in greatest dimension. Fresh lesions soft and bright red; older ones form a firm white plaque. Subchorionic ones lead to subchorionic fibrin deposition.	Laminated thrombus or a firm fibrin plaque. No villi present in the lension. Surrounding rim of compressed villous tissue, often with prominent syncytial knots.	May be caused by increased turbulence of blood in intervillous space.	?Associated with fetomaternal hemorrhage and large placentas of maternal diabetes mellitus or erythroblastosis. No fetal effects unless extensive. Blood may be of fetal, but more often of maternal origin.
Maternal hemorrhage			Bleeding originates from decidual vessels.	
Retroplacental hematoma[13,21,25,26,50,51]	A hematoma of varying size adherent to the basal plate that may bulge into the placental parenchyma to compress the adjacent villi (infarct). The bright red decidual surface under the hematoma is evidence of decidual necrosis. Multiple hematomas can occur. The degree of organization of the blood clot correlates with the age of the hematoma. Chronic variants may show extensive strongly adherent clot. Marginal and central locations occur.	Possible findings include organized blood clot, decidual necrosis, thrombosis of decidual vessels, and adjacent compression and infarction of villous tissue. Occasionally associated with acute inflammation (acute deciduitis), sometimes with microabscesses. Hemosiderin pigment present in older lesions.	Abnormal decidual vessels; may be related to preeclampsia, ?cocaine abuse, smoking, shearing from trauma, rapid decompression of uterus, or infection (chorioamnionitis).	*Fetal hypoxia; maternal blood loss; preterm delivery; maternal disseminated intravascular coagulopathy. Abruptio placenta is clinical term indicating hemorrhage and pain. Retroplacental hematoma and abruptio placenta can be present independent of each other. May occur in association with chorioamnionitis.
Marginal hematoma	Crescent-shaped hematoma at the margin of the placental disc with minimal extension over the placental disc surface. Little if any compression of adjacent villi. Varying degrees of clot organization may be present.	Blood clot with varying degrees of organization. Hemosiderin pigment often prominent in adjacent decidua.	Large marginal decidual venous thrombus. ?Other causes.	Maternal bleeding, but no other major effect.

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Table 2.—Placental Findings: Description, Pathogenesis, and Clinical Associations† (cont)

Findings	Gross Description	Microscopic	Pathogenesis	Clinical Associations
Retromembranous hemorrhage	Hemorrhage alongside placenta in membranes or on maternal (decidua parietalis) surface of free membranes. Often degenerating and yellow-tan.	Hemorrhage. Hemosiderin.	May be sign of abruption.	Minimal clinical significance. May be indication of abruption with movement of blood from retroplacental location.
Parenchymal fibrin deposition				
Minor perivillous (intervillous) parenchymal deposition[13,44]	Minor perivillous fibrin deposition is common and is generally not grossly visible. When more extensive it is white shiny, firm to hard. Lesions show a fine "infiltrate" surrounding villi in a linear (lacelike) manner rather than having a distinct border as with infarction. Lesions often surround a central portion of normal villi and are often near the basal plate. May be seen at periphery of infarct. Color may be yellow brown but is usually white due to age of the lesion. Increases toward term; often present at margin.	Villi are surrounded and separated by a pink layer of fibrin that to varying degrees obliterates the noncompressed intervillous space. Trapped villi show loss of nuclear staining of trophoblast and avascular villous stromal fibrosis.	?Caused by minor maternal circulatory disturbances with eddying. ?Coagulation disturbances. ?Maternal immune damage to trophoblast.	Minor forms of no clinical significance to fetal and neonatal outcome. Decreased incidence in preeclampsia/toxemia.
Massive perivillous (intervillous) parenchymal deposition[13,44]	In severe cases, almost entire placenta may be involved, including basal plate. Placental parenchyma shows aggregates of fibrin deposition forming linear and "nodular" lesions with indistinct borders. Color yellow-brown to white depending on age of lesion.	Villi "trapped" by surrounding perivillous fibrin that obliterates intervillous space. Villi show loss of nuclear staining of trophoblast and avascular villous stromal fibrosis. Differentiate from infarction.	Caused by extensive intervillous circulatory disturbances with eddying.	*When >30% of placenta, involvement may be associated with growth retardation and fetal death. *Recurrence risk. Association with chronic villitis.
Maternal floor fibrin deposition (previously maternal floor "infarction")[8,52,53]	Basal band of dense fibrin of varying thickness seen on maternal surface. Firm and yellow; hyalinized. Sometimes associated with massive perivillous fibrin deposition.	Basal band of acellular fibrin enmeshing ghost villi. May extend into parenchyma. Usually noninflammatory.	?Immunological cause, no maternal vascular disease. May be part of maternal blood flow abnormalities with excessive eddying. Lesion not a postmortem artifact. May have decidual vasculopathy.	*Recurrent poor pregnancy outcome, particularly midtrimester losses. *Intrauterine growth retardation. *Elevated maternal serum alpha-fetoprotein.
Subchorionic fibrin deposition (including massive subchorionic fibrin of Breus' mole)[54]	Laminated fibrin thrombus just below fetal surface. Firm, pearly white, laminated fibrin thrombi precede the older more typical triangular plaques recognized as subchorionic nodules from the fetal surface. Size varies from multiple small granules to larger nodules that bulge on the fetal surface. Older lesions may show progressive recent thrombus. When massive and recent may involve entire subchorionic area and bulge into amniotic cavity (Breus' mole). In rare instances, thrombus can extend to the basal plate.	Varies from the recent laminated thrombus through the dense fibrin aggregates.	Fibrin deposits generally increase with gestational age. Caused by eddying of maternal blood flow.	Breus' mole seen in missed abortion, but liveborn infants reported. May be seen on ultrasound.

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Table 2.—Placental Findings: Description, Pathogenesis, and Clinical Associations† (cont)

Findings	Gross Description	Microscopic	Pathogenesis	Clinical Associations
Calcification	White granular lesions, irregularly scattered over maternal surface.	Calcification.	Usually dystrophic variant.	*Higher socioeconomic status. Increased at term. *No adverse fetal outcome. Maternal cigarette smoking. ?Increased with maternal diabetes mellitus.
"Cystic lesions"				
Subchorionic cysts[13]	Cysts of varying size seen from fetal surface. Contain yellow serous or hemorrhagic fluid. Become gelatinous with fixation.	Lined by trophoblastic cells (X cells).	Unknown.	No clinical significance.
Septal cysts[13]	Seen in placental parenchyma sometimes in association with massive perivillous fibrin deposition. Gelatinous with fixation. Differentiate from "molar" changes of the placenta.	Present in stem villi and lined by trophoblastic cells (X cells). Surrounding fibrin deposition.	Unknown.	Clinical significance related to extent of associated lesions such as massive perivillous fibrin deposition.
Maternal blood in intervillous space	Various maternal blood dyscrasias can be recognized by examination of the maternal blood cells in the intervillous space.			
Sickle cell disease[13]	No specific gross features. Infarction may be noted. Placenta may be small for gestational age.	Sickled red blood cells in intervillous space.	Sickle cell disease/trait.	Clinical features as for sickle cell disease.
White blood cell dyscrasias (leukemia, lymphoma)[13]	Usually not recognized on gross examination.	Microscopic appearance of particular dyscrasia.	Blood cells from maternal circulation in intervillous space.	Per particular disease.
Malaria[13]	Usually no gross lesions.	Parasite in maternal red blood cells. Malarial pigment in mononuclear cells in intervillous space.	Maternal blood cells in intervillous circulation.	Fetal outcome depends on maternal condition. Maternal anemia affects fetus and newborn.
Fetal placental vasculature				
Chorangiosis[13,44]	No distinct gross features. May show congestion. May be associated with large placenta.	Diffuse increase in number of small caliber villous vessels (10 or more vessel lumens in each of 10 villi in 10 fields at ×10).	?Increased incidence in maternal diabetes mellitus.	?Increase in fetal vascular anomalies. Uncertain significance. ?Increase in neonatal mortality and morbidity.
Fetal vessel thrombosis (fetal vascular obliteration/obstructive fetal vasculopathy, including the entity of hemorrhagic endovasculitis)[21,25,55,56]	Distended fetal vessel(s) on placental surface. Older lesions white, firm, and easily palpated. On section vessel lumen distended with thrombus. Parenchyma supplied by vessel is pale but spongy and soft. Pale area well demarcated. Older lesions rubbery but not hard.	Mural or occlusive thrombus in fetal vessels (surface or deep). Obliterated, degenerated, or absent fetal vessels; extravasation with fragmented red blood cells in villous stroma; fibrotic and avascular villous stroma. Rim of prominent syncytial knots around avascular villi. Foci of intravascular calcification may be present.	Associated with poor/reduced fetal blood flow in villous circulation.	?Infectious (cytomegalovirus); ?disseminated intravascular coagulopathy; hypoxia, endothelial damage; long cord, especially if tortuous. May cause vascular disruptive defects, fetal growth retardation, and death. May be recurrent. Some variants considered to be postmortem event.
Subamniotic hemorrhage	Hemorrhage between amnion and chorion.	Hemorrhage.	Usually artifact after delivery due to cord traction. May be associated with tethered cord.	None unless fetal vessel ruptures before birth.

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Table 2.—Placental Findings: Description, Pathogenesis, and Clinical Associations† (cont)

Findings	Gross Description	Microscopic	Pathogenesis	Clinical Associations
Edema[13,21,24,57,58]	Pale, sometimes boggy placenta often large for gestational age.	Focal to diffuse vacuolization of stroma; increased numbers of vacuolated histiocytes. Increase in size of villi. Displacement of fetal vessels to center of villi. Differentiate from normal immature intermediate villi.	Particular variants of edema seen in partial and complete hydatidiform mole.	Depends on extent of edema. If excessive, can compromise fetal blood flow or maternal/fetal exchange. ?Increased compromise of fetus in chorioamnionitis.
Nucleated red blood cells in fetal vessels[13,59]	Not applicable	Easily identifiable nucleated red blood cells in umbilical or other fetal vessels. Normally limited to <5% after 12 weeks of gestation. Almost always increased in fetal circulation when seen in villous capillaries.	Increased release of fetal nucleated red cells into the circulation.	*Present in Rh and other blood group isoimmunization, fetal anemia, and infection (parvovirus and other viral infections). Fetal distress. Chronic hypoxia. Maternal diabetes mellitus.
Villous histologic lesions				
Development/maturation				
Abnormal maturation (accelerated for dates/"hypermaturation")[13,21,22,25]	Placenta may be small for gestational age.	Villi uniformly smaller than normal with increased syncytial knots, free "floating" syncytial snouts, abnormal proliferation of cytotrophoblast, and thickened trophoblast basement membrane. Focally seen at margins of placenta.	Cause ?immunological; ?infectious. Decreased uteroplacental blood flow.	May occur in absence of overt maternal disease. ?Hypertensive disorders, particularly preeclampsia. May be associated with fetal growth retardation and fetal death. Infant may be preterm. Fetus often normal.
Abnormal maturation (delayed/retarded for dates)[13,21,22]	Placenta may be very heavy, boggy, and large for gestational age.	Villi uniformly larger than expected for gestational age with "geographic" configuration. May have two-layer trophoblast; increased stroma and Hofbauer cells; nucleated fetal red blood cells; villous edema. Decrease in number of vasculosyncytial membranes and syncytial knots.	Unknown.	*Fetal anemia/congestive heart failure. *Maternal diabetes mellitus. *Hydrops fetalis. "Chromosomal" abnormality.
Irregular maturation ("dysmature placenta")[21,22,44]	No specific placental gross features. May be hydropic.	Irregular villous size, shape, stromal density, and vascularization. Chronic villitis may be present.	Intrinsic fetal abnormality. Variable uterine blood flow.	*Fetus may have congenital anomalies. *Fetus may be chromosomally abnormal (ie, trisomy 18). Chronic villitis may be present. Maternal diabetes mellitus.
Villitis				
Acute villitis and abscesses[13,25]	Usually no gross features of note. Occasional yellow, rounded, distinct lesions resembling infarcts.	Intervillous neutrophil aggregates and/or large areas of villous necrosis. Infarction may be present. Chorioamnionitis usually present.	Infection via maternal hematogenous route. Listeria or severe *Escherichia coli* infection.	*Usually indicates severe maternal infection. *Preterm labor. Fetal/neonatal infection.

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Table 2.—Placental Findings: Description, Pathogenesis, and Clinical Associations† (cont)

Findings	Gross Description	Microscopic	Pathogenesis	Clinical Associations
Chronic villitis—specific (<5% of cases of villitis)[13,25]	Placenta may be pale and in severe diffuse cases firm and even granular.	Varying degrees of villous inflammation and destruction. Infiltrate includes monocytes, lymphocytes, and plasma cells. Villous fibrosis, avascularity, and destruction are noted. Hemosiderin pigment frequent. Viral inclusions may be found. Specific features depend on underlying cause. Villous "dysmaturity" may be found.	Intrauterine hematogenous infections including cytomegalovirus, and toxoplasmosis, rubella, and syphilis.	*Fetal infection likely, but not invariable. Other diagnostic tests frequently required for specific diagnosis. *Intrauterine growth retardation. *Fetal distress and demise.
Chronic villitis—nonspecific (villitis of unknown etiology) (>75% of cases of villitis)[13,44]	Specific gross features minimal. In rare diffuse cases placenta may be pale, firm and even granular. Fibrinoid deposition in severe cases.	Infiltration of villi and/or intervillous space by mononuclear cells; vascular obliteration; villous fibrosis; variable involvement from rare scattered foci to widespread. Common focally at basal plate. Differentiate from microinfarction.	Cause ?immunological; ?infectious.	Fetal growth retardation; fetal death. May be recurrent.
Tumors of the placenta				
Primary tumors, nontrophoblastic				
Chorangioma[13,21,26]	Fleshy, firm, palpable, circumscribed, sometimes necrotic, calcified, and frequently subchorionic. May be infarcted, occasionally multiple or very large. May be confused grossly with infarct or intervillous thrombus.	Hamartoma (hemangioma) usually composed of small blood vessels. Infarction may be noted. Generally very congested. Covered by prominent trophoblast.	Is a placental hamartoma.	*Large lesions may trap fetal platelets or cause cardiac decompensation leading to polyhydramnios and hydrops fetalis. Occasionally other body site hemangiomas are present in the fetus.
Other	Hemangioma; teratoma; leiomyoma; hepatocellular adenoma.	Specific for tumor type.	Benign tumor of normally present placental elements.	All very rare.
Primary tumors, trophoblastic				
Complete hydatidiform mole[29]	Generalized hydatidiform swelling of villi resembling a bunch of grapes. No fetus present.	Generalized extensive trophoblastic hyperplasia of both cytotrophoblast and syncytiotrophoblast. Generalized cistern fomation of villi. Absence of embryo; therefore, no fetal blood in involuted fetal capillaries.	Conceptus is totally paternally derived (46XX or rarely 46XY).	Detected on ultrasound. *Uterus usually large for gestational age. *Increased human chorionic gonadotropin serum levels. *Maternal risk for preeclampsia. *Increased risk for choriocarinoma.
Partial hydatidiform mole[29]	Focal hydatidiform swelling of villi. Fetus present but seldom survives beyond 9 weeks. With prolonged intrauterine fetal demise, fetus might not be readily seen grossly.	Focal hydatidiform swelling with rare cistern formation. Focal moderate trophoblast hyperplasia confined to syncytiotrophoblast. Villous scalloping and trophoblastic inclusions. Dual population of villi: small fibrotic and larger hydropic.	Conceptus with triploid set of chromosomes (usually diandric). Conceptus may be 69XXY (most common), 69XXX, or 69XYY (most rare).	Human chorionic gonadotropin levels in maternal serum may be raised. *In rare cases in which fetus survives beyond the first trimester, an array of congenital anomalies occur. *Syndactyly of third and fourth digits of both hands and feet is highly characteristic. Intrauterine growth retardation. Nonmolar triploidy also occurs.

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Table 2.—Placental Findings: Description, Pathogenesis, and Clinical Associations† (cont)

Findings	Gross Description	Microscopic	Pathogenesis	Clinical Associations
"Secondary (metastatic) tumor"				
Maternal origin carcinoma of various organs including melanoma (multiple reports) and breast; leukemia (multiple reports); sarcoma[21,26]	Usually present as tumor masses in the intervillous space, sometimes attached to but generally not invading the villous stroma.	Histology specific for maternal tumor.	Hematogenous dissemination of maternal tumor to placental tissue.	
Fetal origin neuroblastoma (multiple reports), leukemia (multiple reports), hepatoblastoma[21,26]	Usually present in fetal vessels.	Histologically resembles fetal tumor.	Hematogenous spread of fetal tumor with implantation and growth of tumor cells in placenta.	
Congenital Nevus	Present in villous stoma.	Histologically resembles fetal lesion.	?Metastatic lesion or implant.	Benign.
Multiple gestation, classified according to the characteristics of the dividing membrane				
Monochorionic monoamniotic[13,15,25,26]	One placental disc. No dividing membrane on fetal surface (be aware of artifact due to amnion being stripped away). Vascular anastomoses present and often large. Surface anastomoses best demonstrated by injecting vessels with air, water, or dye.	No dividing membrane to sample.	Single ovulation and fertilization (monozygous) with "split" of fertilized egg on day 7 or later postfertilization.	Monozygous gestation. *Vascular anastomoses present in most if not all cases. Anastomoses may be between surface vessels or, more importantly, may occur within the villous tree. *Infants of same sex. Conjoined twins. Cord entanglement.
Monochorionic diamniotic[13,16,25,26]	Fused/single placental disc generally present. Dividing membrane on fetal surface translucent and thin. No ridge along fused border of placentas. Vascular anastomoses present. Surface anastomoses best demonstrated by injecting vessels with air, water, or dye.	Dividing membrane composed of two layers of amnion. No chorion present in dividing membrane.	Single ovulation and fertilization (monozygous) with "split" of fertilized egg on days 3 to 4 postfertilization.	Monozygous gestation. *Twin transfusion syndrome present in one out of three cases. *Vascular anastomoses present in most if not all cases. Anastomoses may be between surface vessels or, more importantly, may occur within the villous tree. Twins with chronic transfusion syndromes often lack large anastamoses. *Infants of same sex.
Dichorionic diamniotic[13,16,25,26]	Separate placentas that may grow together ("fuse"). Vascular anastomoses extremely rare. Dividing membrane opaque and thick. Distinct ridge can be palpated where discs fuse and placental discs can be "torn" apart along this ridge.	Dividing membrane shows two layers of amnion and one or two layers of chorion.	Multiple ovulation spontaneous or induced.	*Vascular anastomoses very rare. Monozygous or dizygous gestation. Dizygous: placenta is dichorionic in all cases. Monozygous: placenta is dichorionic in about one out of three cases. About one out of three cases of monozygotic twin gestation have a dichorionic placenta; the remaining two cases have a monochorionic placenta. Sex of infants: if sex is different, the gestation is dizygous; if sex is the same, gestation could be dizygous or monozygous. *Cannot determine zygosity from placental examination.

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Table 2.—Placental Findings: Description, Pathogenesis, and Clinical Associations† (cont)

Findings	Gross Description	Microscopic	Pathogenesis	Clinical Associations
"Missing twin" (vanishing twin)[60]	Early spontaneous loss of one twin represented by mummified nodule of tissue in fetal membranes. X-ray shows fetal skeleton. Underlying placenta degenerates. Either dichorionic diamniotic or monochorionic diamniotic.	Underlying placenta shows total replacement by perivillous fibrin deposition.	Death of one twin in early gestation. Disruptive lesions in cotwin if monochorionic.	*Source of raised maternal serum alpha-fetoprotein. Rarely has fibrinolytic problem "protective" feature of live fetus.
Iatrogenic lesions[13]	Trauma (amniocentesis, cordocentesis, chorionic villous sampling, etc) leading to hemorrhage, thrombosis, inflammation.	Microscopic features as for lesion in any nonplacental location. Old lesions related to percutaneous umbilical cord sampling may have hemosiderin laden macrophages in Wharton's jelly.	Depends on iatrogenic incident.	Fetal hemorrhage. Fetal injury. Infection. Placental trauma. ?Early amnion rupture sequence. ?Limb reduction defects.

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care. These should be communicated in a timely fashion, directly to the physicians caring for the mother and/or infant. Placental reporting should follow the same standards as other surgical pathology reports.

QUALITY CONTROL AND QUALITY ASSURANCE

Quality control and quality assurance activities for placental examination are a part of quality control and assurance activities as they pertain to surgical pathology in general. The following specific issues related to placenta examination might be considered in the development of such programs:

1. Written indications for examination of the placenta should be adopted by the medical staff.
2. The final pathology report should include critical descriptive and diagnostic features.
3. There should be a monitor of adherence to indications for submission of the placenta for examination.
4. Correlation of acute clinical outcome with placental findings should be made.
5. Intradepartmental quality assurance should be established for review of cases.
6. The adequacy of placental sampling and quality of sections (eg, full thickness) should be included in any monitor.
7. Adherence to Universal Precautions (see Workplace Issues, later) should be monitored for the gross examination of placentas, as for other tissues.

USE OF PLACENTAL TISSUE FOR RESEARCH

Placental tissue is subject to the same regulations that apply to the use of human tissue for research. Placental tissue is sought for a broad spectrum of research interests and even for therapeutic needs. This section is included to provide guidance for the collection and distribution of placental tissue for such purposes. A comprehensive list of potential uses for placental tissue is not provided here; each institution should follow appropriate procedures for the approval of research protocols in compliance with applicable regulations.

Diagnostic needs related to patient care always take priority over the provision of placental tissue for research. If the placenta is required for pathologic examination, close collaboration among the clinical services, pathology service, and the researcher will best serve the particular needs of each. Coordination by delivery room nursing personnel is critical for effective collaboration in this area.

The following issues need to be directly addressed.

General.—To ensure that tissues are appropriately handled and that patient care and diagnostic needs are met, all research protocols that use placental tissue should be approved in accordance with institutional policy.

Institutional Review Board Approval.—The use of placental tissue for research, whether obtained fresh, fixed, or as embedded tissue, may require Institutional Review Board approval. In many instances, such uses will qualify for expedited review, and specific patient-informed consent may not be necessary for most studies.

Commercialization.—When it appears that research on a particular placenta is yielding or is likely to yield a protein, enzyme, or other product of commercial value, appropriate informed consent should be sought from the patient. Because the scope of the consent will vary depending on the institutions involved and the nature of the research, the advice of legal counsel should be sought. In addition to obtaining informed consent, institutions should consider the ethical, business, and patient issues that may arise when a research protocol is likely to produce a commercial benefit.

WORKPLACE ISSUES

The placenta is subject to the same safety requirements as other tissues examined in pathology. Safe practices for labeling, transport, and storage of fresh and fixed tissue are an institutional requirement.

Fresh placentas after delivery and initial examination should be placed in clean containers, whose exterior surface remains clean or is disinfected prior to transport. Lids should fit securely, so that the container will not leak if it is accidently malpositioned. Containers should be labeled with all appropriate information and transported promptly to either temporary storage or the pathology laboratory. After transport, they should be refrigerated in a readily accessible site prior to examination. These issues remain

a consideration when the placenta accompanies an infant transfer. In these instances, the placenta, like other fresh tissues, should be transported in a clean and appropriately labeled container inside a cooler with an appropriate amount of ice to maintain a cool temperature throughout the transport process.

Placentas not examined in the pathology laboratory should be retained as described in Placenta Handling and Transport, earlier. Their ultimate disposal should be according to hospital guidelines for biohazardous material.[61]

The placenta, like all other tissues received in the pathology laboratory, should be considered as potentially infectious and should be treated according to the Universal Precautions employed in the institution in which they will be examined. "Universal Precautions" are based on recommendations issued by the Centers for Disease Control[62–64] and on recommendations of the National Committee for Clinical Laboratory Standards.[65] They are based on the premise that health care workers should consider blood and body fluids of all patients as potentially infectious. Appropriate protective clothing, as mandated by the Occupational Safety and Health Administration,[66] should be worn during examination of fresh and fixed tissues. This usually consists of a disposable overgarment impervious to fluids, surgical gloves, and eye protection, either goggles or face shields. Surgical masks, hats, and shoe covers, while recommended, are not mandatory. Protective coverings, containers, linens, and other items used in the transport and examination of fresh and fixed tissues should be disposed of according to institutional standards.

Sharps used during tissue examination should be disposed of in a puncture-resistant container provided for this purpose. Scalpel blades should be dropped into this container after use. In addition, all instruments used during examination should be disinfected prior to their reuse.

Retained tissues should be fixed in an appropriate volume of fixative. Appropriate consideration should be given to the storage conditions of fixed placental tissues, so that they will be appropriately maintained in a manner that does not allow formalin fumes to pose a chemical hazard to personnel involved in the storage and retrieval of such fixed archival material. External surfaces of containers used to store tissue specimens should be washed clean and wiped with disinfectant, such as diluted bleach, prior to transport and storage.

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See the April 1997 issue of the ARCHIVES for "Practice Guidelines for Autopsy Pathology: The Perinatal and Pediatric Autopsy" (pp 368–376)

Appendix 1.—Triage Worksheet

Triage Examination of Placenta
(circle correct response)

Accession no.: _____ **Name:** _____

	Normal		Abnormal		
Cord insertion:	Eccentric	Central	Marginal	Velamentous	Other: _____
Number of cord vessels:	3		2	>3	
Total cord length:	_____ cm		<32 cm	>100 cm	
Disc weight for gestational age:	Normal		Small	Large	
Dimensions:	___ × ___ × ___ cm				
Maternal surface:	Intact		Incomplete	Other: _____	
Fetal membranes:	Normal		Cloudy	Other: _____	

Other placental indications for exam:	None	Specify: _____	
Maternal indications for exam:	None	Specify: _____	
Fetal/neonatal indications for exam:	None	Specify: _____	

Appendix 2.—Gross Examination Worksheet

Accession no.: _____ **Name:** _____

History:	vaginal/cesarean	___ wk gestation	male/female	___ g (infant body weight)
	meconium/preeclampsia/diabetes			
Weight:	___ g (placental weight)	___ fixed	___ fresh	
Cord:	length ___ cm	central/eccentric/marginal/velamentous		
	web ___ cm	true knot/congestion		
	twist right/left/absent	2 vessels	torn cord/vessels	furcate ___ cm
Membranes:	___ cm	disrupted	absent	
% Circummargination:	___ rim ___ cm			
% Circumvallation:	___ rim ___ cm			
Subchorionic Fibrin:	0/1/2/3			
Subchorionic Hemorrhage:	___ % old/recent			
Opacity/Green Staining/Hemolysis/Betadine/Subamniotic Hemorrhage:	slight/fresh/thick/edema			
Dimensions:	___ × ___ × ___ cm	bilobate ___ × ___ cm	irregular	
Succenturiate Lobe:	1. ___ × ___ × ___ cm	2. ___ × ___ × ___ cm	atrophic/partial	
Maternal Surface:	disruptive focal/extensive			
Calcification:	0/1/2/3			
Thrombus:	1. ___ cm	2. ___ cm	3. ___ cm	fresh/recent/old
Infarcts:	marginal	1. ___ cm	2. ___ cm	3. ___ cm
	central	1. ___ cm	2. ___ cm	3. ___ cm
Hemorrhage:				
Old:	1. ___ × ___ cm	RM/RP mar/cm	2. ___ × ___ cm	RM/RP mar/cm
Fresh/Recent:	1. ___ × ___ cm	RM/RP mar/cm	2. ___ × ___ cm	RM/RP mar/cm
Fibrin Deposition:	diffuse	1. ___ × ___ cm	2. ___ × ___ cm	
Atrophy:	diffuse	1. ___ × ___ cm	2. ___ × ___ cm	
Parenchyma:	pale/deep red	soft/firm		
Accompanying clot:	soft clot ___ g			

Appendix 3.—Twins Worksheet

Accession no.: _____ **Name:** _____

History:
A ___ g male/female
B ___ g male/female

Separated/Fused: Diamniotic Dichorionic/Diamniotic Monochorionic/Monoamniotic Monochorionic

Cord Designation: none/clamps

Total Weight (Fused): ___ g

Overall Dimensions: ___ × ___ × ___ cm

Vascular Anastomoses: artery-artery vein-vein artery-vein (___ to ___) artery-vein (___ to ___)

(provide diagram if complicated)

Maternal Surface Coloration: similar/___ paler

Singleton Report forms can be attached for detailed evaluations of each portion of the placenta.

Appendix 4.—Fused Twin Placenta: Gross Examination Worksheet

Accession no.: _____ **Name:** _____

History: gestation ___ wk vaginal/cesarean
A ___ g male/female
B ___ g male/female

Hematocrit: A B meconium/preeclampsia/diabetes

Diamniotic Dichorionic/Diamniotic Monochorionic/Monoamniotic Monochorionic

Cord Designation: none/clamps

Total Weight: ___ g

Overall Dimensions: ___ × ___ × ___ cm

Dimensions: Sac A
Sac B

Vascular Anatomoses: artery-artery vein-vein artery-vein (___ to ___) artery-vein (___ to ___)

(provide diagram if complicated)

Maternal Surface Coloration: similar/___ paler

Singleton Report forms can be attached for detailed evaluations of each portion of the placenta.

Appendix 5.—Singleton Report Form

Accession no.: _____ **Name:** _____

History: vaginal/cesarean ____ wk ____ g male/female

Received fresh is a ____-g placenta with a ____-cm 3 vessel left-twisted (central/eccentric/marginal/velamentous/furcate) cord with a ____-cm amniotic web. Membranes are (incomplete/essentially complete) and ruptured ____ cm from the placental margin. Membrane insertion is (at the margin/____ % circummarginate/circumvallate with a rim to ____ cm). Subchorionic fibrin is (minimal/slight/moderate/abundant). There is (opacity/green coloration/no unusual coloration) of the fetal surface. The placental disk is ____ × ____ × ____ cm (approximate greatest dimensions). The maternal surface is (apparently complete/disrupted focally). There is (no/slight/moderate/abundant) calcification. On cut section, color and consistency are unremarkable. (No) other gross lesions:

retromembranous/retroplacental hemorrhage
intervillous thrombus
succenturiate lobe
marginal infarct
central infarct

Soft clot present.

Microscopic performed. Gross only. Tissue blocks available.

Diagnosis:

Placenta, delivery: No pathologic diagnosis
Immaturity
Ischemic change
Subchorionic intervillositis/chorionitis/chorioamnionitis
Retroplacental/marginal hemorrhage
Intervillous thrombus
(Marginal) infarct
Succenturiate lobe

Fetal membranes, delivery: No pathologic diagnosis
Acute inflammation/chorioamnionitis
Meconium pigmentation
Retromembranous hemorrhage
Circumvallate/circummarginate

Umbilical cord, delivery: No pathologic diagnosis
Marginal/velamentous insertion
Acute phlebitis/vasculitis/funisitis

Appendix 6.—Twin Report Form

Accession no.: _____ **Name:** _____

History: vaginal/cesarean ___ wk A (1 clamp) male/female ___ g B (2 clamps) male/female ___ g

Received fresh is a diamniotic di/monochorionic twin placenta with separate/fused disks. Cords are unlabeled and arbitrarily designated/labeled with 1 and 2 clamps. Overall, the placenta is ___ g and ___ × ___ × ___ cm. Injection studies are performed revealing ___ artery to artery and ___ artery to vein anastomosis from ___ to ___. The placentas are divided along the approximate vascular plane.

I. (___ clamp) is ___ g and ___ × ___ × ___ cm with a ___-cm 3 vessel left-twisted central/eccentric/marginal/velamentous cord. Membranes are incomplete/ruptured ___ cm from the margin. There is opacity/green staining/no unusual color of the fetal surface. Maternal side is apparently complete/disrupted focally. No/slight/moderate calcification. No other gross lesions.

II. (___ clamps) is ___ g and ___ × ___ × ___ cm with a ___-cm 3 vessel left-twisted central/eccentric/marginal/velamentous cord. Membranes are incomplete/ruptured ___ cm from the margin. There is opacity/green staining/no unusual color of the fetal surface. Maternal side is apparently complete/disrupted focally. No/slight/moderate calcification. No other gross lesions.

Representative sections including dividing membranes. Microscopic performed.

Diagnosis:

Placenta, delivery: Twins, dichorionic/monochorionic (identical)
Immaturity
Subchorionic intervillositis/chorionitis/chorioamnionitis

Fetal membrane, delivery: No pathologic diagnosis
Acute inflammation/chorioamnionitis

Umbilical cord, delivery: No pathologic diagnosis
Marginal/velamentous insertion
Single artery

Note: Although like-sexed dichorionic twins may be monozygotic or dizygotic, about 80% are dizygotic.