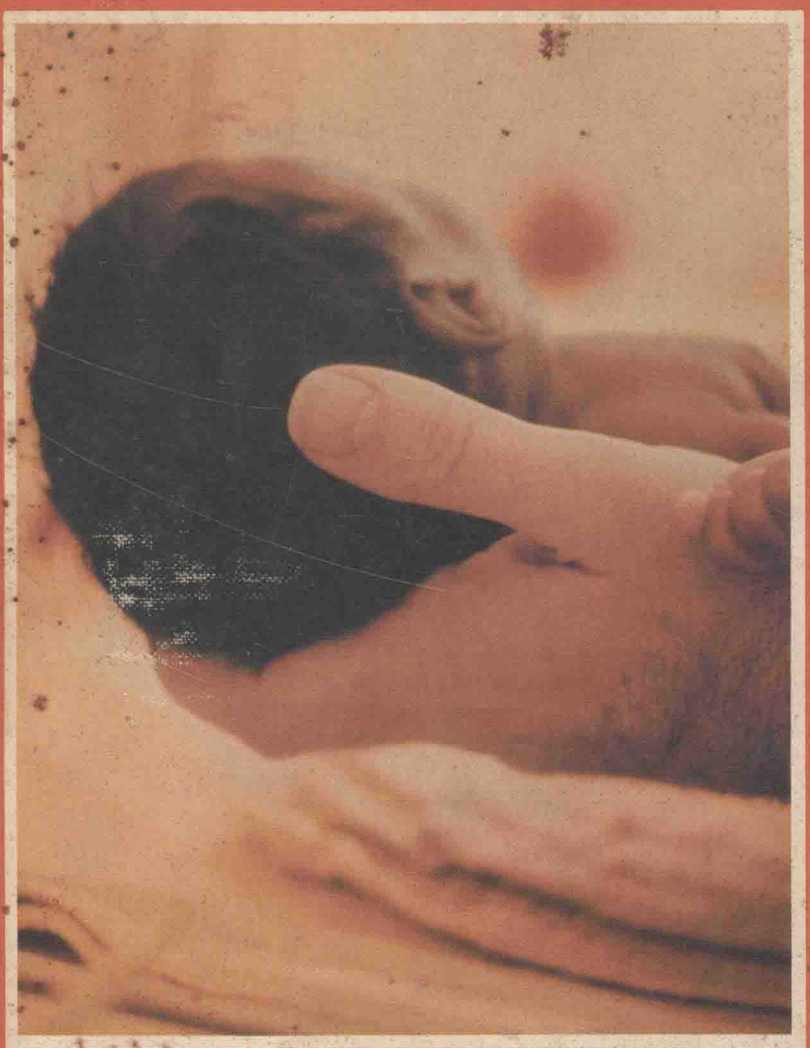
High-risk newborn infants

The basis for intensive nursing care



SHELDON B. KORONES

SECOND EDITION

High-risk newborn infants

The basis for intensive nursing care

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High-risk newborn infants

The basis for intensive nursing care

To

The infants who suffered from our past inadequacies
and to those who will benefit from our newly acquired skills

AND

Judy and David and Susan, who motivate me;
and to my parents, who started everything

Preface

This second edition was written because advances in perinatal medicine have unfolded at a dizzy pace since publication of the first edition. It was also written to meet the increasingly sophisticated requirements of nurses in educational programs and those already in the field. The book is thus presented to keep its readers apace of recent developments and to keep apace of the development of its readers.

Like the first edition, the second is not intended as a "how-to-do-it" manual; neither is it offered as an encyclopedic reference. I have discussed the major topics of neonatal medicine with the unchanged conviction that although nurses are eager to know *what* must be done, they are more eager to know *why*. As in the first edition, I have attempted to maintain a narrative that is as conversational as the subject permits. Tables of data are presented when reasonably full exposition of a topic is impractical or when a tabulated summary of the accompanying narrative serves to implement the learning process.

This new edition is longer than its predecessor because material has been added throughout. Discussions are more extensive in the chapters devoted to the fetus, the relationship of birth weight and gestational age, and physical examination of the neonate. The chapter on respiration was virtually rewritten and considerably expanded. The discussion of thermoregulation, which appeared as a section in the first edition, has now been expanded into a separate chapter. I particularly enjoyed adding a substantial number of illustrations to enhance the effectiveness of corresponding discussions. The great majority of them were taken in our own unit. If there are any inadequacies in the photographs, I accept full responsibility for them; a long-standing compulsion to take all kinds of pictures was simply uncontrollable.

The preface is an appropriate place to record some personal observations on the role of the nurses for whom this book is written. Although the drama and genuine importance of equipment are inescapable, the reader will note throughout the book a relentless emphasis on the role of personnel. The role of the nurse cannot be an ancillary one. Intensive care is primarily a nursing activity. Without superb nursing, effective intensive care is impossible, the caliber of medical supervision notwithstanding. One need not work long in a neonatal intensive care unit to be fully convinced. I have been repeatedly gratified by the success of my own efforts when working with nurses whose performance was admirable by any standard. On the other hand, I have been frustrated by the futility of the same effort while working with nurses who could not perform at such a level.

The nurse is in a better position than anyone else to detect the subtle signs that often herald catastrophe. No one else can continuously provide and monitor the multiple facets of therapy that support the infant through critical illness. This is a big job. It cannot be done without understanding the bases of intensive care. Ventilatory support, oxygen therapy, and manipulation of the thermal environment, for example, cannot be performed reliably unless nurses have adequate knowledge of fundamentals. Without such knowledge, today's correct maneuvers may be performed only by chance and for the wrong reasons, whereas tomorrow the

baby may not be so lucky; the maneuver may not be correct, for whatever reasons. The nurse's need to be well informed is no different from the physician's; in neonatal intensive care they are indeed colleagues.

The job is being done daily. It can be seen in hundreds of units in this country and in others, and more babies are now surviving because of it. The uninitiated nurse generally approaches the task with fear and trepidation; yet if scrupulous guidance is provided in a well-structured training program, nursing responsibilities are ultimately well met. The apprehensive and insecure become calm and confident. The uninformed become knowledgeable. The metamorphosis is something to behold, and this book is intended to implement it.

Books cannot be written or revised without the kindness and help of others. The task was eased and the deadline met because a number of individuals provided much-needed support.

Only a rare physician (and I am not one of them) could write a book like this without the guidance of an exemplary nurse such as Jean Lancaster, R.N., M.N. Her expert advice is an indispensable contribution for which I am deeply grateful. It is derived from her effective activities as Clinical Supervisor of Nurses in our unit. In this position she functions as teacher of nurses, caretaker of babies, counselor of parents, and mollifier of the unit's Director—for all of which I am even more grateful.

Drs. Loraine Evans, Fabien Eyal, and Charles Sarasohn relieved me of clinical responsibilities during the protracted period of time that was required to complete this revision. The numerous extra sleepless nights for which they spontaneously volunteered are reason enough for profound appreciation, but the friendship that motivated them was a particularly moving experience.

Through her capacity for gentle leadership, Ann Lamb, R.N., has steadfastly brought cohesion and stability to all the nursing functions in our unit. As Head Nurse, even before the inception of intensive care at this institution, she was the one primarily responsible for bringing to sick babies the best care that was then available. An unsurpassed intensive care nurse, the intelligent and compassionate example she sets has long been an object of my admiration.

I am indebted to Kathleen Ambrose and Marion Haynes for their expert handling of all the details that comprised the actual preparation of this manuscript. I am thankful for their forbearance in the face of repeated impositions and for the impressive, well-ordered manuscript they produced. Their uncanny ability to decipher hundreds of pages of my scribblings is a noteworthy feat in itself.

I mention Patti Lechman's art work because it is perfect and was produced just a few days after my urgent request.

Without exception, the comments of reviewers of the first edition were constructive. I have attempted to respond to most of their suggestions whenever possible.

My thanks to Doll. And thanks, too, to Susan Poo.

Sheldon B. Korones

Contents

Chapter 1

The fetus

NEONATAL DISORDERS, especially those which occur soon after birth, are usually the result of prenatal difficulties. Effective management of these illnesses requires familiarity with the intrauterine events that give rise to them. This chapter is concerned with the important aspects of fetal growth and function and the maternofetal relationships on which they depend.

THE PLACENTA: JUXTAPOSITION OF FETAL AND MATERNAL CIRCULATION

Fundamentally, the placenta provides bidirectional passage of substances between the mother and the fetus, whose circulations are contiguous but separate. The placenta is comprised of a maternal contribution called the decidua basalis and an embryonic one called the chorion. The maternal tissue contains uterine blood vessels, endometrial stroma, and glands; the embryonic tissue is composed of chorionic villi anchored to the chorionic plate.

The placenta (from Latin for "flat cake") is shaped like a round biscuit. At term it is about 15% of the fetal weight, approximately 6 to 8 inches in diameter, and 1 inch thick. Throughout pregnancy it occupies approximately one third of the inner surface of the uterus. One side, the fetal surface, is covered by glistening amniotic membrane, which receives the umbilical cord near its center. The

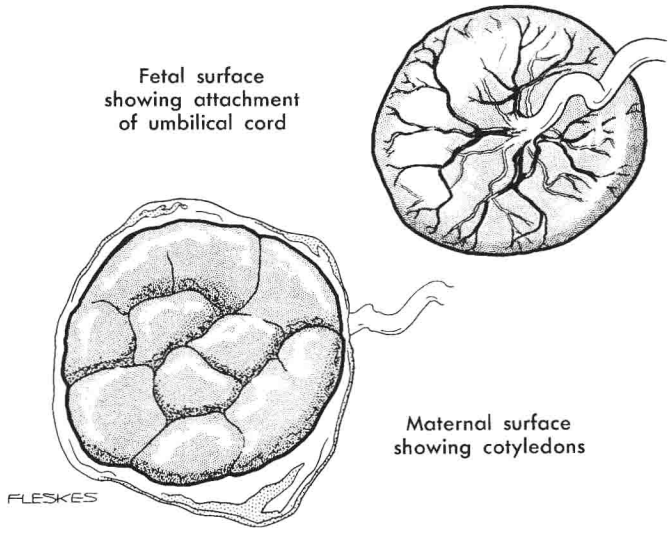

Fig. 1-1. Maternal and fetal surfaces of the placenta. The cotyledons are visible on the maternal surface, which is implanted into the uterine wall. The fetal surface glistens, being covered by amnion. Branches of the umbilical vessels are on the fetal surface. (Modified from Netter: In Oppenheim, E., editor: Ciba collection of medical illustrations, vol. 2, Reproductive system, 1965.)

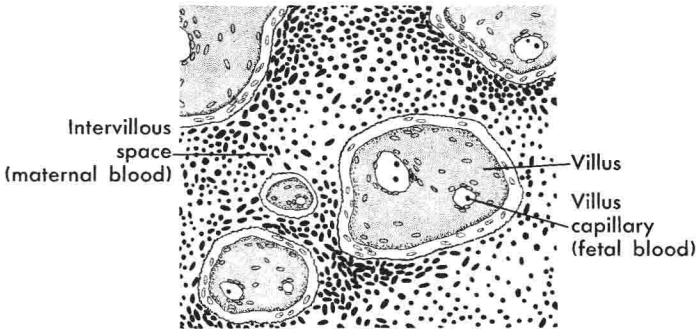

Fig. 1-2. Microscopic appearance of the villi in an intervillous space. Fetal capillaries permeate the villi, which are immersed in maternal blood within the intervillous spaces.

opposite side, the maternal surface, remains embedded in the uterine wall until it begins to separate during labor and is extruded during the third stage (Chapter 2). The placenta is composed of fifteen to twenty-eight grossly visible segments called cotyledons (Fig. 1-1), which are of identical structure and are separated from each other by connective tissue septa. Each cotyledon contains fetal vessels, chorionic villi, and an intervillous space. The dark red color of the placenta is due to fetal hemoglobin rather than to maternal blood, which is usually drained by the time the placenta is delivered. Thus, if the placenta appears pale, the fetus was significantly anemic.

Chorionic villi, intervillous space, and placental circulation

Within a week after implantation the embryo elaborates fingerlike projections of tissue known as the chorionic villi, which invade uterine endometrium at the site of implantation. They enlarge and branch considerably, becoming more deeply embedded into uterine tissue as they grow. Each of these projections is ultimately comprised of an outer layer of epithelial cells and a connective tissue core that contains fetal capillaries. During villous invasion of the endometrium, erosion of uterine vessels and supporting tissue occurs, and as a result irregular spaces are formed in the uterine wall. They are filled with maternal blood and surround the chorionic villi. These comprise the intervillous space, and the exchange of substances between maternal and fetal blood takes place at these sites. Transfer of any given substance from the maternal to the fetal circulation thus requires sequential passage from the mother's blood through the outer epithelial layer of the chorionic villus, through the connective tissue, and then through the endothelial wall of the capillary into fetal blood (Fig. 1-2). As pregnancy progresses, the villi decrease in size but increase considerably in number. This change in both size and number of villi provides a progressive increase in fetal surface area within the placenta for the greater needs of the growing fetus. The villous surface at term is estimated at 13 to 14 sq M, an area that is ten times greater than the total skin surface of the adult. This enhanced capacity for maternofetal exchange is further implemented by simultaneous thinning of the tissue layer in the villus, thus reducing the distance between fetal capillaries and the intervillous space.

Maternal blood in the uterine arteries enters the base of the intervillous spaces through spiral arterioles. At term there are approximately 100 arteries supplying the placenta. Spurting jets of oxygenated blood diffuse upward and laterally to surround the villi, passing out of the intervillous spaces in a deoxygenated state by way of venous orifices that are situated at the base of the

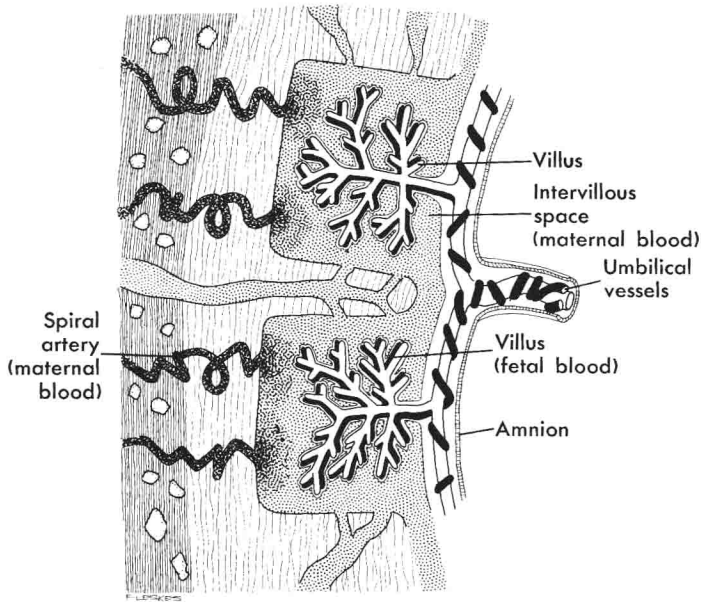

Fig. 1-3. Section through the placenta, showing the spiral arteries that supply maternal blood to the intervillous space, the branching villi immersed in the intervillous space, and the umbilical vessels that branch repeatedly to terminate as villous capillaries. (Modified from Netter: In Oppenheim, E., editor: Ciba collection of medical illustrations, vol. 2, Reproductive system, 1965.)

intervillous space adjacent to the spiral arteries. The direction of blood flow is normally maintained by an arteriovenous pressure gradient, that is, from higher arterial pressure to lower venous pressure (Fig. 1-3). Pressure in the spiral arteries is about 70 to 80 mm Hg. In the draining veins it is 8 mm Hg. Lateral flow between cotyledons is negligible.

Fetal blood leaves the body through two umbilical arteries that course through the umbilical cord to the placenta. Once they contact the fetal surface of the placenta, the arteries branch to supply each of the cotyledons. After continued branching and diminution in size, the vessels finally terminate in capillary loops within each chorionic villus. Villous blood receives oxygen from the maternal circulation, returns from capillary loops to venules, and goes thence to larger veins that leave each of the placental cotyledons. Coalescence of veins results in the formation of a single umbilical vein, which

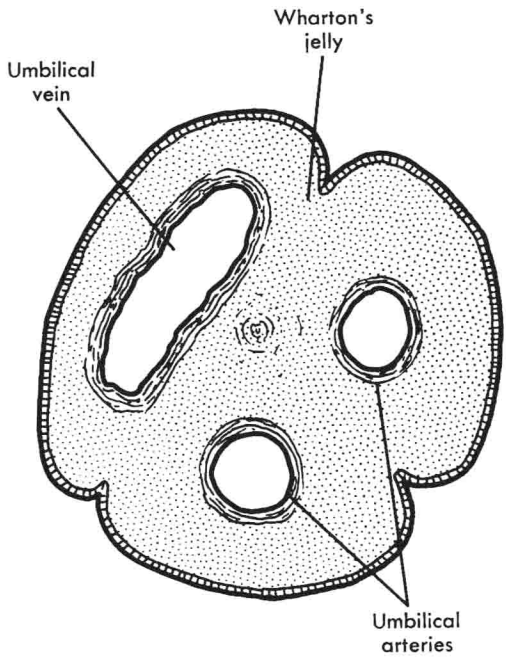

Fig. 1-4. Cross section of umbilical cord. The arteries have thick walls; the lumen of the vein is larger than those of the arteries, and its wall is thin.

passes from the placenta through the length of the umbilical cord to the body of the fetus. A cross section of the umbilical cord reveals two thick-walled muscular arteries and one vein. The vein is noticeably larger than the arteries, although it has a considerably thinner wall (Fig. 1-4). The umbilical vessels are surrounded by a white gelatinous material called Wharton's jelly.

FETAL CIRCULATION

Two major circuits, pulmonary and systemic, comprise the circulation in the fetus as well as in the adult. A third component, the placental circuit, is peculiar to the fetus. Pulmonary circulation begins at the pulmonic valve at the origin of the main pulmonary artery, which arises from the right ventricle. It continues through the lungs and terminates at the pulmonary vein orifices in the wall of the left atrium. The systemic circulation includes all other arterial and venous channels elsewhere in the body. The placental circuit is comprised of the umbilical and placental vessels as described earlier.

In any discussion of circulatory patterns a conceptual subdivision into "right" and "left" sides is often invoked. In the mature individual the right-sided circulation contains venous blood and the left side, arterial blood. Anatomically the right side begins at the venous end of capillary beds in all organs except the lungs. It continues through progressively enlarging veins to the inferior vena cava into the right atrium, to the right ventricle, into the pulmonary arteries, and finally to the pulmonary alveolar capillaries, where oxygenation occurs. Blood now continues on to the left side, which begins at the alveolar capillaries and progresses through the pulmonary veins into the left side of the heart, through the aorta to smaller arteries, and to the arterial side of capillary beds in all organs except the lungs. At the arterial side of the capillaries, blood gives up oxygen and continues to the venous side, where it reenters the right side of the circulation.

The normal mature circulation is characterized by flow of unoxygenated (venous) blood into the right side of the heart, which continues into the pulmonary circuit, where oxygenation occurs, and then returns to the left side of the heart for distribution to the rest of the body by way of the aorta. Thus the only connections between the right and left circulations are at the capillary beds in the lungs and in the capillaries of other organs of the body.

When, as in certain cardiovascular malformations, a connection between the right and left sides exists at sites other than the capillaries, an anatomic shunt is said to be present. Examples of such anomalies are persistent patency of the ductus arteriosus, arteriovenous aneurysm, and numerous malformations of the heart itself. Blood flow through an abnormal vascular channel from the venous to the arterial circulation constitutes a right-to-left shunt. Conversely, flow of blood through an anomalous channel from artery to vein is a left-to-right shunt. In either case blood enters one side of the circulation from the other before reaching the capillary bed for which it is normally destined, and the result is an admixture of oxygenated and unoxygenated blood.

Fetal circulation differs from the neonatal or adult pattern in three major respects: (1) presence of anatomic shunts, one within the heart at the foramen ovale, one immediately outside the heart in the ductus arteriosus, and another at the juncture of the ductus venosus and the inferior vena cava; (2) presence of a placental circulation; and (3) minimal blood flow through the lungs (3% to 7% of cardiac output).

The course of fetal circulation is as follows (Fig. 1-5): Blood that has been oxygenated in the chorionic villi leaves the placenta through the umbilical vein, which enters the fetal abdomen at the umbilicus and then courses between the right and left lobes of the liver. Some of this blood perfuses the liver through branches of the umbilical vein, whereas the rest continues past it to the ductus venosus and empties into the inferior vena cava at a point just below the diaphragm. Blood now enters the heart through

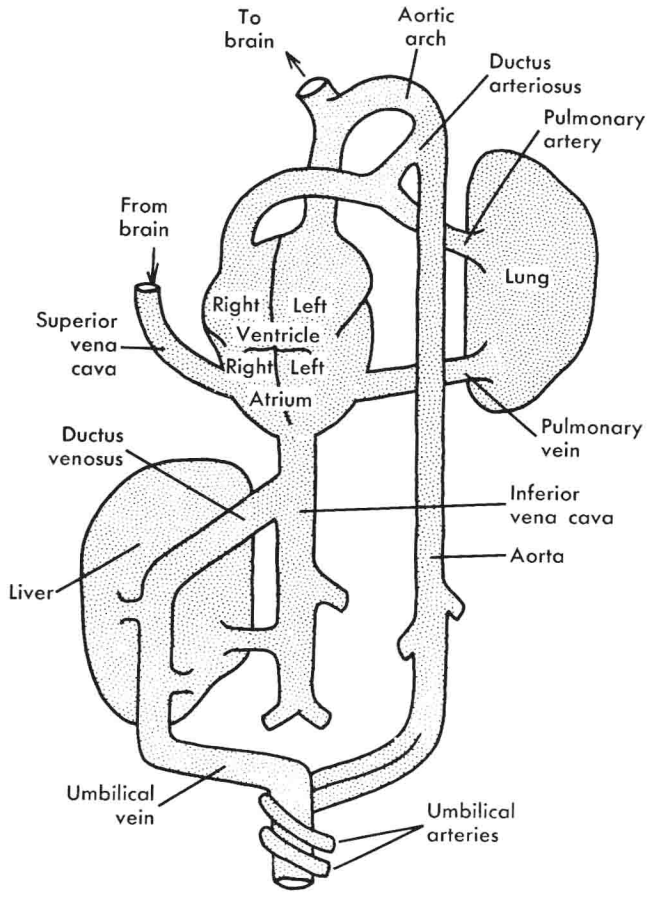

Fig. 1-5. Fetal circulation. (Modified from Lind, J., Stern, L., and Wegelius, C.: Human foetal and neonatal circulation, Springfield, Ill., 1964, Charles C Thomas, Publisher.)

the inferior vena caval orifice, where a direct communication exists between the right and left atria (foramen ovale). Here the caval bloodstream is divided, the major portion flowing directly into the left atrium, whereas the remainder enters the right atrium to join blood from the superior vena cava, which drains the head, neck, and upper extremities. The left atrium also receives blood that has perfused the lungs from pulmonary veins, and this mixture enters successively the left ventricle, the ascending aorta, and the aortic arch, from whence most of it is distributed to the coronary arteries and the vessels of the head and upper extremities.

The smaller quantity of blood directed to the right atrium from the inferior vena cava is added to the superior vena caval flow, enters the right ventricle and then the main pulmonary artery. Most of it is conveyed to the descending aorta through the ductus arteriosus. Some continues through the pulmonary circuit to perfuse the lungs and return to the left atrium by way of the pulmonary veins. The descending aorta bifurcates at the lower end of the body into the right and left iliac arteries. The two hypogastric arteries are branches off the latter vessels. They each course upward around the bladder and leave the abdomen through the umbilical cord, where they are called the umbilical arteries.

Admixture of blood occurs at three sites

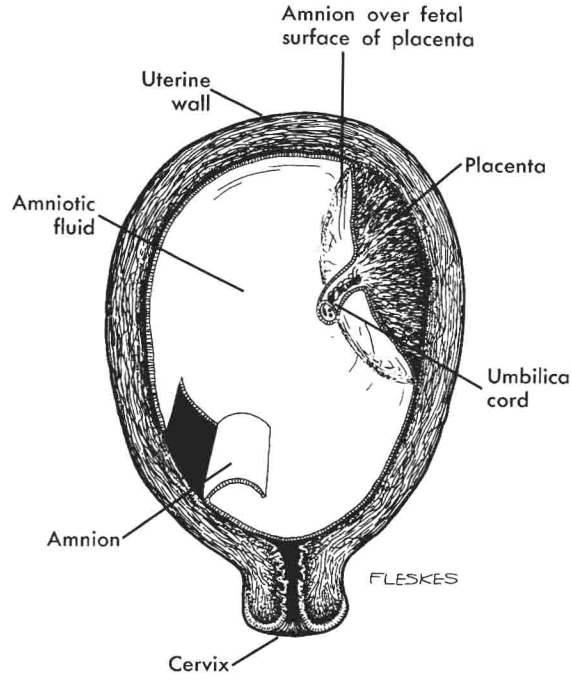

Fig. 1-6. Uterine cavity at term. The amnion lines the uterine wall. It continues over the placenta and reflects from it to envelop the umbilical cord. Amniotic fluid surrounds the fetus to fill the cavity. (Modified from Hillman, L. M., and Pritchard, J. A.: Williams obstetrics, ed. 14, New York, 1971, Appleton-Century-Crofts.)

in the fetal circulation: (1) the foramen ovale, (2) from the pulmonary artery to the descending aorta through the ductus arteriosus, and (3) at the junction of the ductus venosus and inferior vena cava.

THE AMNION AND AMNIOTIC FLUID

The amnion and the amniotic cavity develop in the embryo during the first week, appearing as a small elongated sac adjacent to the dorsal embryonic surface. As the fluid-filled cavity enlarges, it spreads around the embryo in all directions and ultimately comes to surround the fetus. This cavity is traversed only by the umbilical cord, which courses from the umbilicus to its insertion into the placenta. The amniotic membrane is 0.5 mm or less in thickness and is comprised of five microscopic layers. Fully developed, it lines the entire inner surface of the uterine wall, except at the site of placental attachment, where it covers the fetal surface of that organ, reflects onto the umbilical cord, and completely envelops it (Fig. 1-6). The fluid that surrounds the fetus (amniotic fluid) is contained by this membrane, and when the latter ruptures at the onset of labor, the fluid is released.

Amniotic fluid accumulates progressively throughout pregnancy, attaining a volume of approximately 1000 ml at term (normal range: 400 to 1500 ml). It continuously enters and leaves the amniotic cavity, creating a constant turnover of approximately 600 ml/hr involving complex water and solute exchanges that have yet to be precisely delineated. Amniotic fluid is derived primarily from maternal serum and fetal urine. The latter constitutes the major source of amniotic fluid during the last half of pregnancy. Some fluid originates in the fetal lung. Egress of fluid occurs largely by absorption into the fetal bloodstream from the gastrointestinal tract, into which it gains entry as a

result of repeated fetal swallowing. That the fetus swallows effectively has been shown often by amniography, a procedure that entails injection of radiopaque dye into the amniotic fluid for the primary purpose of demonstrating the site of placental implantation for the possible presence of placenta previa. At term, the fetus swallows 500 ml of amniotic fluid per hour. Normal volumes of amniotic fluid are thus largely regulated by contributions of the fetal urinary tract and evacuation through the gastrointestinal tract. Thus, if the elimination of fluid is obstructed, as in esophageal atresia, the quantity of fluid may be abnormally increased to more than 2000 ml. This increase is called *hydramnios* or *polyhydramnios*. Conversely, if little or no urine is excreted, as in renal agenesis, amniotic fluid may be virtually absent or greatly reduced in quantity. This abnormality is known as *oligohydramnios*.

Amniotic fluid affords a milieu that allows relatively free fetal movement. It protects the fetus from externally inflicted trauma, and it contributes to the stability of fetal body temperature. During early labor, hydrostatic forces created by uterine contractions are mediated by the fluid to aid in normal dilatation of the cervix and descent of the fetus.

MATERNOFETAL EXCHANGE OF GAS ACROSS THE PLACENTA

Fetal respiration occurs by transfer of oxygen from maternal blood in the intervillous space to fetal blood in the villous capillaries and by release of carbon dioxide in the opposite direction from fetus to mother. The normal flow of these gases is governed by their respective partial pressures in maternal and fetal blood and by unimpeded fetal and maternal blood flow in the placenta.

Gases have a tendency to expand, and they exert measurable pressure in the process. This pressure is expressed in millimeters of mercury (mm Hg). Thus pressure exerted by gases in the atmosphere at sea level is 760 mm Hg. In any mixture of two or more gases the pressure exerted by each of them is termed their partial pressure. The sum of the partial pressures of each constituent gas in a mixture is the total pressure exerted by that mixture. The expression symbolizing partial pressure of a gas is written P (or p) preceding its chemical formula. The partial pressures of oxygen and of carbon dioxide are written as P_{O_2} and P_{CO_2}. This terminology is utilized routinely in reports from clinical and research laboratories.

Table 1-1. Mean quantitative changes in fetal blood gases resulting from fetomaternal exchange*

	Umbilical artery (blood from fetus)	*Intervillous space (maternal blood)*	*Umbilical vein (blood to fetus)*
P_{O_2} (mm Hg)	16	40	29
P_{CO_2} (mm Hg)	46	38	42

*Based on data from Seeds, A. E.: Pediatr. Clin. North Am. **17**:811, 1970.

Assuming normal placental blood flow, the differences in the partial pressures of gases (P_{O_2}, P_{CO_2}) that exist between maternal and fetal blood determine the direction in which these gases move. Gases are transferred in response to pressure gradients, that is, from a higher pressure to a lower one. Tissues intervening between the maternal and fetal circulations (normally 3.5 to 5.5 microns in total thickness) seem to offer more resistance to the movement of oxygen than to carbon dioxide; the latter is thus considerably more diffusable. Table 1-1 gives P_{O_2} and P_{CO_2} values of fetal blood in the umbilical artery, maternal blood in the intervillous space, and fetal blood in the umbilical vein as reported by a number of investigators. The acquisition of oxygen and the surrender of carbon dioxide by fetal blood are indicated in the alterations of P_{O_2} and P_{CO_2} that result from the exchange of gases with maternal blood in the intervillous space. The influence of partial pressure gradients can be appreciated by noting, in Table 1-1, that a higher maternal P_{O_2} in the intervillous space moves oxygen into the fetal circulation to raise P_{O_2} from 16

mm Hg in the umbilical artery to 29 mm Hg in blood returning to the fetus via the umbilical vein. The flow of oxygen is therefore from mother to fetus. The direction of carbon dioxide transfer is from fetus to mother because P_{CO_2} in the intervillous space (38 mm Hg in maternal blood) is lower than in the umbilical artery (46 mm Hg in fetal blood); thus carbon dioxide moves from fetus to mother with a resultant diminution of P_{CO_2} in the umbilical vein (42 mm Hg) compared to that in the umbilical artery (46 mm Hg).

Maintenance of these pressure gradients depends on normal maternal blood gas levels and normal blood flow in the mother and fetus. A number of disorders may alter maternal blood gas levels sufficiently to hamper gas exchange at the intervillous space. Thus severe pneumonia, asthma, congestive heart failure, and apnea during convulsions (epilepsy, eclampsia) may result in maternal hypoxemia that endangers the fetus. Impairment of blood flow to the intervillous space may occur as a consequence of maternal shock or congestive heart failure. Local impediment to placental perfusion may result from abnormally intense and frequent uterine contractions during labor or from compression of the inferior vena cava by the overlying heavy uterus when the mother is supine (supine hypotension syndrome).

FETAL GROWTH

Growth of the fetus involves an increase in the number of cells (hyperplasia) and an increase in their size (hypertrophy). Growth progresses through three stages: (1) hyperplasia, (2) a declining rate of hyperplasia with increasing hypertrophy, and (3) predominant hypertrophy. Embryonic growth is largely hyperplastic; hypertrophy becomes increasingly prominent later in pregnancy. Growth may be impeded during any or all of these progressive stages, resulting in different types of growth failure. Thus the number of cells may be diminished although their size remains relatively normal, or cell size rather than quantity may be reduced, or both size and number may be diminished. The net result is subnormal size and weight of affected organs. These effects are dependent on the stage of growth during which an insult is inflicted. For example, intrauterine rubella infection, which occurs early in pregnancy, produces a severe reduction in the quantity of cells in many organs. On the other hand, the effects of toxemia, which often appear late in pregnancy, are characterized by a significant reduction in cell size, although cell number is relatively normal. All these changes have been produced in pregnant animals that were starved at various stages of pregnancy. Interference with cellular proliferation (hyperplastic growth) may cause a permanent diminution in the quantity of cells, although impaired hypertrophy is apparently reversible to some extent if proper feeding is instituted after birth. There is evidence that these phenomena may also be operative in humans. The human brain continues to grow by cellular proliferation for at least 8 postnatal months after 40 gestational weeks. Postnatal malnutrition impairs this process. Whether maternal malnutrition permanently limits organ growth in humans has yet to be documented. Data from animal studies and clinical observations of human infants suggest that permanent limitation of growth does indeed occur.

The characteristics of normal cellular growth are of importance in understanding the abnormalities of birth weight and size that are peculiar to prematurity and intrauterine growth retardation. (These details are discussed in Chapter 5.)

ASSESSMENT OF FETAL STATUS (FETAL DIAGNOSIS)

The growing ability to assess fetal conditions in utero continues to be one of the most exciting areas of advance in perinatology. The diagnosis of certain disorders of the fetus is feasible; approximation of fetal maturation is aided significantly by examination of chemical and cellular constituents of amniotic fluid and by accurate estimation of fetal head size. Identification of hypoxic

stress in utero is possible by monitoring fetal heart rate and by sampling specimens for blood gas and pH determinations. The overall health of the fetus is assessable by estriol determination. The diagnosis of placenta previa can be made by amniography (injection of radiopaque dye into the amniotic cavity) or by ultrasound to demonstrate placental position. Postnatal microscopic examination of the amniotic membrane and the umbilical cord for the presence of neutrophilic infiltration has been used to determine the presence of fetal exposure to intrauterine bacterial infection (Chapter 12). Insofar as intrauterine therapy is concerned, transfusion is the only procedure currently in common use. A description of the most widely utilized methods for fetal diagnosis and assessment follows.

Examination of amniotic fluid

Amniocentesis. Amniotic fluid may be withdrawn simply and safely by amniocentesis, that is, insertion of a needle through the abdominal and uterine walls into the amniotic cavity. Amniocentesis has been performed as early as the twelfth gestational week. Amniotic fluid components can be analyzed for the severity of erythroblastosis (Rh incompatibility); for predictions of postnatal appearance of hyaline membrane disease, certain genetic disorders, and inborn errors of metabolism; and for fetal age. Although the complications of amniocentesis are uncommon, a variety of them have been reported. These risks include abortion, maternal hemorrhage, infection, fetal puncture wounds, pneumothorax, laceration of the fetal spleen, damage to placental and umbilical vessels, and sudden death from fetal exsanguination.

Bilirubin. Determination of bilirubin in amniotic fluid is valuable for assessment of the severity of Rh disease. Bilirubin is derived from the breakdown of red blood cells (Chapter 10), and it is thus present at certain levels in normal amniotic fluid. Peak levels are attained between 16 and 30 weeks of gestation, and a steady decline thereafter usually culminates in its disappearance by 36 weeks. In most instances there is no bilirubin in amniotic fluid beyond 36 weeks of gestation. This finding is variable, however, and bilirubin determination is not valuable for estimation of fetal maturity. If, as in fetal erythroblastosis, an excessive rate of hemolysis occurs, bilirubin levels in amniotic fluid rise abnormally. These values are plotted on a graph that delineates three zones of fetal involvement (mild, moderate, and severe), depending on the bilirubin concentration. If the results indicate severe disease, an intrauterine transfusion or an immediate termination of pregnancy is urgently indicated. If moderate involvement is present, repeated frequent determinations are required to monitor the course of fetal disease. Values falling into the zone of mild involvement contraindicate intrauterine transfusion or interruption of pregnancy, since postnatal therapy is effective in these circumstances. The amniotic fluid of hyperbilirubinemic mothers often contains abnormally increased amounts of bilirubin, even in the absence of Rh disease.

Creatinine. Levels of creatinine in the amniotic fluid increase with gestational age and are thus useful in the assessment of fetal maturity. Creatinine in amniotic fluid is derived from fetal urine. It is excreted through the kidneys across the glomeruli, which increase substantially in number during the third trimester. Thus it is reasonable to expect that creatinine levels will rise steadily as pregnancy progresses. In normal pregnancies, 1.6 to 1.8 mg/100 ml of creatinine is indicative of 36 or 37 gestational weeks in approximately 94% of patients. However, the usefulness of this determination for estimation of fetal age is unfortunately limited in complicated pregnancies. Thus, in toxemic mothers, amniotic fluid creatinine concentrations are often misleadingly higher than in normal pregnancies of like duration. The toxemic gravida's impaired kidney function causes some degree of creatinine retention, and therefore her serum level is elevated. This increase is reflected in amniotic fluid as an