CHAPTER

19

Anatomy and Physiology of Pregnancy

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Introduction

Pregnancy is a time of profound anatomic and physiologic change. In addition to the reproductive organs, all maternal physiologic systems make adaptions needed to support the developing fetus and, at the same time, maintain maternal homeostasis. A thorough understanding of these changes is an essential foundation for all healthcare providers—including midwives—who care for women during pregnancy. This chapter provides an overview of changes in the reproductive organs, the effect of the major hormones of pregnancy, fetal development, maternal adaptations that take place during pregnancy.

The Reproductive Organs During Pregnancy

Pregnancy lasts approximately 266 days or 38 weeks from ovulation (postconceptional weeks). This translates into 10 lunar months or 9 calendar months (because some months have 5 weeks). If dating occurs from the last normal menstrual period, some sources note the duration of human pregnancy is 280 days or 40 completed weeks' gestation. When counting gestation in weeks, it is important to remember that the number changes after the week is completed. Therefore 23 5/7 weeks' gestation is 23 completed weeks since the last menstrual period and an additional 5 days. This translates to 21 completed weeks and 5 days since conception.

Over the course of pregnancy, a woman's breasts grow and prepare for lactation. The uterus increases to approximately 5 times its normal size; at 38 gestational weeks, the uterus measures approximately 32 centimeters long, 22 centimeters across, and 24 centimeters wide. The cervix must first act as a barrier maintaining the uterine contents. However, at the end of pregnancy, the cervix becomes soft and short, opening to allow passage of the fetus during birth. These remarkable changes are the result of a complex interplay of hormonal stimulation that has important clinical implications for all women, but especially those who have congenital uterine anomalies, as well as those who experience miscarriage, preterm labor, preeclampsia, and other pregnancy complications.

The Breast

Under the direction of several different pregnancy hormones, the breast undergoes two distinct developmental changes to prepare for lactation during pregnancy. Both stages—mammogenesis and lactogenesis I—include hyperplasia and hypertrophy.[1] Hyperplasia refers to an increase in the number of cells, or cellular proliferation. Hypertrophy refers to enlargement of the cells; that is, cells grow in size.

Mammogenesis begins early in pregnancy. Breasts enlarge via cellular hyperplasia, and the breast lobules increase in size. The nipples become erectile, the areola becomes proportionately larger and darker, and superficial veins can become visible. During this

process, an individual may feel her breasts are tender or even painful. Alveoli expand and proliferate at the end of breast lobules; the lobules proliferate as well.

Toward the middle of pregnancy, the alveoli epithelial cells change into secretory epithelium, which is the first stage of lactogenesis. Toward the end of pregnancy, the alveoli secrete colostrum but are primarily quiescent secondary to inhibition by progesterone, one of the primary pregnancy hormones. After birth, the influence of progesterone abruptly ceases and lactogenesis II—that is, the onset of milk production—begins. A more detailed description of lactogenesis II is presented in the *Breastfeeding and the Mother–Newborn Dyad* chapter.

The Uterus

The three layers of the uterus (endometrium, myometrium, and perimetrium) become clearly defined over the course of pregnancy. The uterus grows at a steady, predictable rate during pregnancy, with its expansion first becoming detectable at approximately 5 weeks' gestation. The initial uterine growth occurs in the anteroposterior diameter, while the isthmus or lower segment of the uterus can become very soft. This softening results in marked compressibility in the lower uterine segment that is present for a short period of time at approximately 4 to 6 weeks after the first day of the woman's last menstrual period started, and approximately 2 weeks after conception. This unusual compressibility, called *Hegar's sign*, is a probable sign of pregnancy. As seen in **Figure 19-1**, during a bimanual examination, the lower segment of the uterus is so soft that it can be easily compressed between the fingers of the examiner's two hands (one on the abdomen and the other gloved hand in the posterior fornix of the vagina).

The uterine shape changes from the nonpregnant pear shape to a ball or sphere in the first trimester, and then expands to an elongated cylinder. The anatomic location of the uterus in relation to maternal anatomy is illustrated in **Figure 19-2**.

The uterine round ligaments attach on either side of the uterus just below and in front of the insertion of the fallopian tubes; they then cross the broad ligament in a fold of peritoneum, pass through the inguinal canal, and insert in the anterior (upper) portion of the labia majora on either side of the perineum. The ligaments are composed largely of smooth muscle that is continuous with the smooth muscle of the uterus. The round ligaments hypertrophy during pregnancy and stretch as the uterus enlarges. During periods of rapid uterine growth, women may feel stretching or sharp pain in the inguinal area when moving or turning, which is secondary to additional torsion or stretching of the round ligament.

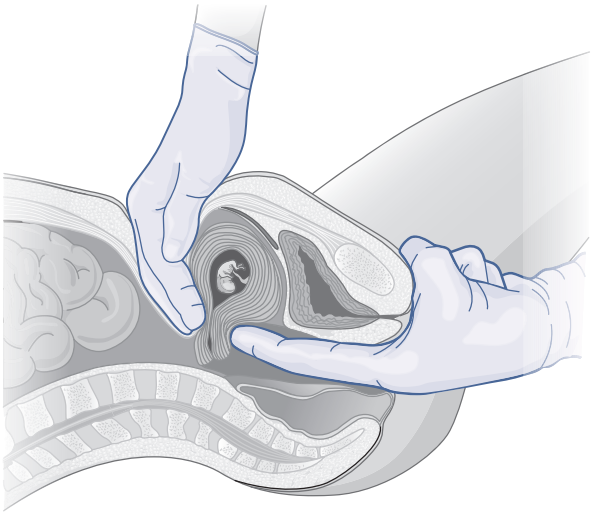

Figure 19-1 Hegar's sign.

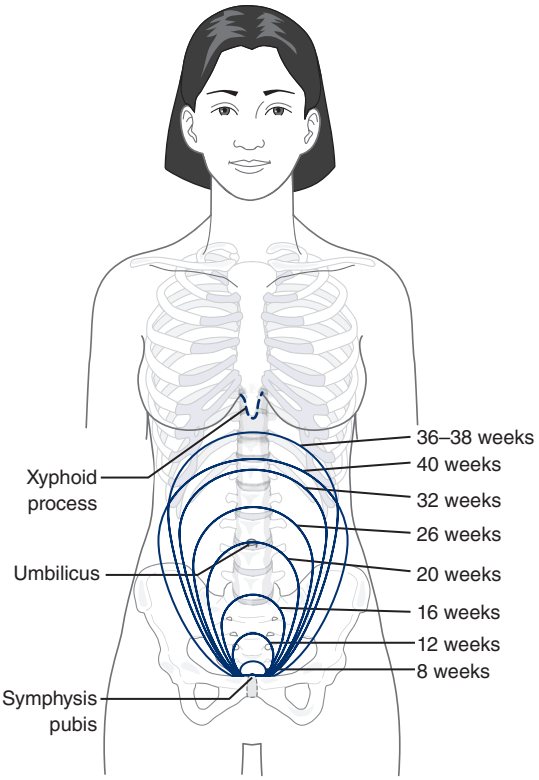

Figure 19-2 Approximate normal fundal heights during pregnancy.

Growth of the uterus is due to two processes: (1) estrogen- and progesterone-induced hyperplasia

of uterine smooth muscle cells within the myometrium during early pregnancy and (2) hypertrophy of the uterine muscles later in pregnancy. The muscles increase their content of actin, myosin, sarcoplasmic reticulum, and mitochondria, which collectively serve as the machinery used to contract the muscles during labor and birth, as described in the *Anatomy and Physiology During Labor and Birth* chapter. The myometrium thus has both properties of contractility and elasticity. Contractility allows for lengthening and shortening, whereas elasticity refers to the ability to stretch.

Transformation of the Endometrium into the Decidua

The secretory endometrium contains columnar epithelium, epithelial cells, and nonresident or migratory immune cells and spiral arteries. The radial arteries that supply the myometrium branch into basal arteries, which become the spiral arteries that supply part of the myometrium and the endometrium.[2]

The endometrial changes that allow and facilitate implantation are collectively termed the *decidual reaction*. The name "decidua" was chosen for this process because, like the leaves on deciduous trees, the transformed tissue is shed after birth.[3] The decidual reaction occurs in response to estrogen, progesterone, and a complex dance or "cross-talk" of locally produced chemicals generated by the blastocyst and maternal endometrium.

Approximately 8 days after ovulation, the secretory endometrium provides a 4- to 5-day "window of opportunity" for blastocyst implantation.[4] During this short span of time, immune cells congregate, the endometrial glands become more secretory, and the epithelial surface develops small protrusions called pinopods and cell-adhesion molecules.[5] The pinopods absorb fluid from the uterine cavity and likely play key roles in attracting the blastocyst. Once the blastocyst is in contact with the endometrium, the pinopods and trophoblastic protrusions interact to facilitate implantation (**Figure 19-3**).[6] It is thought that physiologic variations in the development of the endometrial "window of opportunity" could result in failure of implantation; this hypothesis is currently the subject of infertility research.

The decidual reaction starts once the blastocyst is present at the endometrial site of implantation. The endometrial stromal cells become larger, round, decidual cells that have a membrane to which the trophoblast can anchor itself, and another surge of chemical "cross-talk" occurs.[7]

The decidua fulfills multiple roles, including a key role in directing trophoblastic invasion and

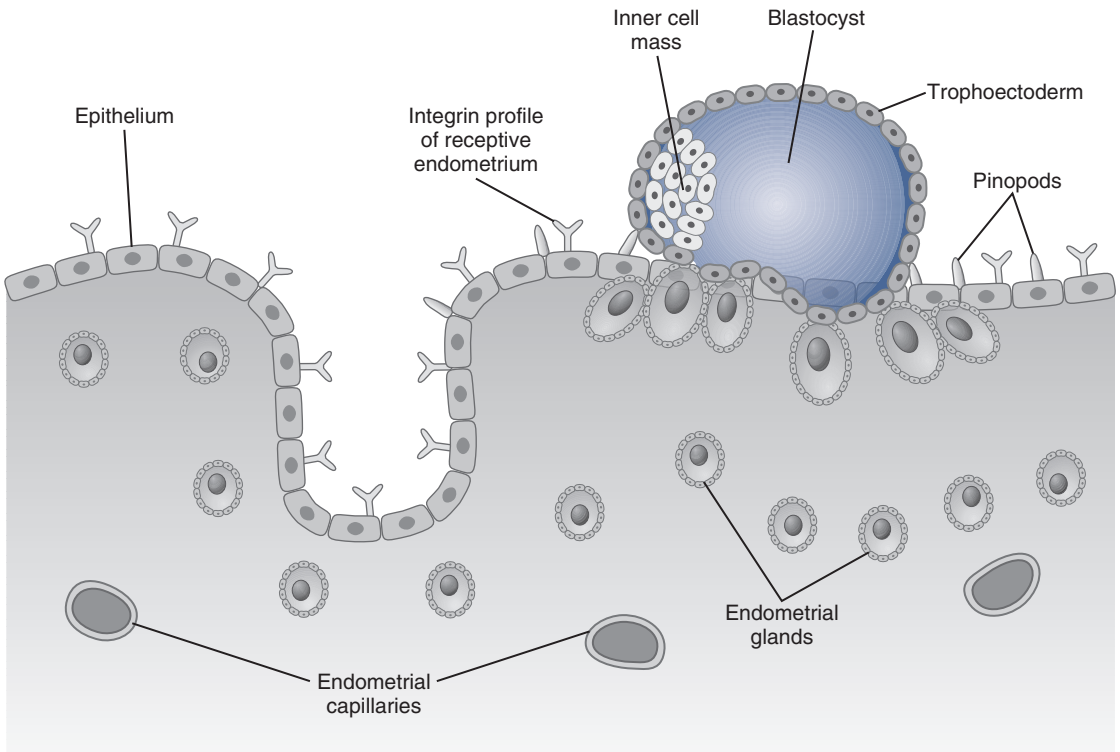

Figure 19-3 Implantation of the blastocyst.

provision of nutrition to the embryo in the early stages of embryology. The entire maternal decidua is divided into three regions: the *decidua basalis*, the *decidua capsularis*, and the *decidua parietalis*. These three regions are named for their positional relationship to the conceptus. The decidua basalis is the site where implantation occurs and is the future site of the maternal potion of the placenta. It is subdivided into a *zona compacta* and a *zona spongiosa*, the latter of which is the site of detachment of the placenta in the third stage of labor. The decidua capsularis lies like a capsule around the chorion, whereas the decidua parietalis is found on the opposite uterus wall. At approximately the fourth month of gestation, the decidua capsularis comes into contact with the decidua parietalis. The merging of these two causes the uterine cavity to become obliterated.

Uterine Innervation

Changes in uterine innervation are among the most interesting physiologic effects of pregnancy. The uterus is primarily innervated by the sympathetic nervous system, with some fibers coming from the parasympathetic nervous system and cerebrospinal tract. Innervation is not consistent throughout the uterus; for example, more nerve fibers are found near the cervix as compared to the fundus. Sympathetic nerve fibers in the uterine fundus virtually disappear during pregnancy, whereas those in the cervix remain.[8,9] Although the reasons for these changes are not clear, it is postulated that the lack of sympathetic innervation protects the uterus from sympathetic stimulation via catecholamines and resultant contraction activity. Uterine contractions during pregnancy and labor occur secondary to endocrine stimulation rather than via muscle fiber nerve stimulation.

Cervical Remodeling During Pregnancy

In nonpregnant women, the cervix is an average of 3 centimeters long.[10-12] In essence, it is a tubular structure, made up of two major structures: the *ectocervix* and the *endocervix*. The ectocervix, which is visible from the vagina, comprises an external layer of columnar epithelial cells and a layer of squamous epithelial cells. The endocervix is the internal, canal-like portion of the cervix, which opens into the uterus. It is covered with a single layer of columnar epithelial cells. The cervical openings are referred to as the *internal os* and *external os*, as described in the *Anatomy and Physiology of the Female Reproductive System* chapter. In nulliparous women, the opening of the endocervix at the external os is pinpoint and circular in size; in contrast, in multiparous women, the external os has a slit-like appearance.

Glandular tissue in the cervix produces thick, tenacious mucus, which forms the mucus plug sealing the endocervical canal. This mucus plug helps prevent ascending bacteria or pathogens from entering the uterine cavity during pregnancy.

During pregnancy, the cervix is composed primarily of connective tissue covered by a thin layer of smooth muscle, which itself is covered by the columnar epithelial cells. The extracellular connective tissue within the cervix contains protein (in the form of collagen and elastin) and proteoglycans (primarily hyaluronic acid and decorin); it is referred to as the *extracellular matrix*. Approximately 80% of the cervix comprises extracellular matrix and 15% is smooth muscle, but the distribution is quite heterogeneous.[10-14] The area closest to the internal os has more smooth muscle, whereas the area closer to the external os has more extracellular matrix. The strength of the collagen tissue depends on the type and number of crosslinks between the collagen microfibrils.

Over the course of pregnancy, labor, birth, and the postpartum period, the cervix normally undergoes four distinct phases: (1) softening, which is also called *remodeling*; (2) accelerated softening at the end of pregnancy, referred to as *ripening*; (3) dilation; and (4) repair.[13] Under the influence of estrogen in pregnancy, the cervix first begins to soften approximately 4 weeks after the first day of a woman's last menstrual period. This cervical softening, called *Goodell's sign*, is one sign of pregnancy.[11] As vascularization increases, a cyanosis or bluish-purple discoloration called *Chadwick's sign* develops. Chadwick's sign is usually first evident at 6 to 8 weeks' gestation.

Following the initial relatively rapid softening, the cervix continues to soften throughout the pregnancy, albeit at a slower rate. The collagen becomes more soluble and compliant, but does not lose structural integrity. Several factors can adversely influence cervical architecture during pregnancy, including infection, genetic factors, and previous surgical procedures that involved the cervix. Usually the cervix remains between 30 and 40 millimeters in length throughout gestation. Women whose cervix shortens to less than 20 millimeters are at increased risk for preterm labor.

Cervical Ripening Prior to Labor

"Cervical ripening" is the term used to describe the process of accelerated remodeling and softening that begins weeks prior to the onset of labor. Cervical remodeling and activation of uterine contractions are the two primary physiologic events that

are associated with the initiation of parturition or labor. Cervical ripening results from a series of interactions between hormonal and mechanical factors that have not been fully elucidated. As progesterone levels fall and estrogen levels rise, the water content and vascularization of the cervix increase and the collagen cells become disorganized, which results in a marked reduction in the mechanical strength of the collagen bundles.

The local paracrine activity of prostaglandins PGE2 and PGF2a also influence the cervical ripening and the onset of labor. Fetal production of corticotropin-releasing hormone and cortisol causes an upregulation of prostaglandin receptors in the cervix and uterus. PGE2 facilitates cervical vasodilation. Finally, production of pro-inflammatory cytokines leads to infiltration of the cervix by leukocytes and macrophages. These cells release enzymes that facilitate alterations in extracellular matrix proteins, loosening of collagen fibers, and a reduction in collagen content.

Cervical Effacement and Dilatation

Toward the end of gestation and extending into early labor, the cervix shortens or thins so that there is no discernable length between the external os and the internal os. This process, which occurs in response to uterine contractions, is referred to as *effacement*. The cervix also begins to *dilate* as the process continues. Effacement and dilatation generally occur during early labor in nulliparous women but may occur prior to progressive labor in multiparous women. However, the onset of labor is not often discrete. There is significant variability between individuals with regard to the degree of effacement and dilatation that has occurred when labor begins.

Many women notice an increase in vaginal secretions during pregnancy called *leukorrhea*. This phenomenon likely occurs because, in addition to normal vaginal secretions produced by the epithelial cells, the cervical glands secrete an increased amount of mucus at this time in order to form the cervical mucus plug. These cervical and vaginal secretions likely form the basis of the physiological discharge of leukorrhea.

Hormones of Pregnancy

The placenta is the primary physiologic interface between the maternal and fetal compartments and is a central mediator for chemical messages between the fetus and the pregnant woman. The primary hormones of pregnancy produced by the placenta are estrogen, progesterone, human chorionic gonadotropin (hCG), and human placental lactogen (hPL). Each of these hormones has a major role in supporting pregnancy (**Table 19-1**). In addition, the placenta and the fetus synthesize other chemical mediators that act both locally and systemically to support growth and development of the fetus and placenta itself; many of these mediators are still being discovered.

Human Chorionic Gonadotropin

Human chorionic gonadotropin is a glycoprotein with both alpha and beta subunits. The alpha subunit is structurally the same as luteinizing hormone (LH), follicle-stimulating hormone (FSH), and thyroid-stimulating hormone (TSH). The beta unit can have various forms depending on the tissue that produces it and is the basis of modern qualitative pregnancy tests.[15-17] Serial plasma levels of beta-hCG can be used to provide quantitative levels used to diagnose and monitor development of the early conceptus.

Human chorionic gonadotropin is secreted by the syncytiotrophoblast tissue within the blastocyst before implantation occurs, and then later by the placenta. The first role of hCG is to sustain estrogen and progesterone production in early pregnancy by preventing degeneration of the corpus luteum. The level of the free beta unit of hCG (beta-hCG) rises in early pregnancy and is first detectable approximately 8 to 10 days after ovulation, or shortly before the first missed menses. This timing coincides with implantation of the fertilized ovum.[18] This level doubles approximately every 48 to 72 hours in 85% of women with normal pregnancies, maintaining this rate of increase until it reaches a peak of approximately 100,000 mIU/mL at 8 to 11 gestational weeks. At this point, the plasma level of beta-hCG slowly decreases to a stable level of approximately 20,000 mIU/mL (**Figure 19-4**).[18]

The characteristic doubling time of hCG has been used in serial measurements of blood values to assess the viability of pregnancy. Because there is wide individual variability in hCG levels, however, some patterns of serial hCG levels (e.g., slower than expected increases) can be difficult to interpret. Moreover, although an hCG level may be obtained every 2 days if clinically indicated, assessing these values every 48 hours can sometimes be too soon to see a full doubling of the hCG plasma level.[19] Serial quantitative measures of the beta subunit are used to determine the viability of a pregnancy, as described in the *Prenatal Care* and *Medical Complications in Pregnancy* chapters. Plasma levels of the beta subunit are also used as a marker for tumors that have an embryologic origin, such as choriocarcinoma and hydatidiform mole.

Table 19-1 Major Hormones and Functions During Pregnancy

Hormone	Source	Selected Functions During Pregnancy
Human chorionic gonadotropin (hCG)	Syncytiotrophoblast Placenta	Stimulates production of progesterone from the corpus luteum Prevents degeneration of the corpus luteum, thereby ensuring ongoing estrogen and progesterone production Promotes formation of syncytiotrophoblast during trophoblastic invasion Aids spiral artery remodeling during the process of trophoblastic invasion Promotes angiogenesis in uterine vasculature Stimulates thyroid production of thyroxine in the first trimester Suppresses myometrial contractions
Human placental lactogen (hPL)	Placenta	Increases insulin resistance Stimulates production of growth hormones
Progesterone	Corpus luteum Placenta	Promotes systemic vasodilation Has anti-inflammatory actions to protect trophoblast from being rejected Supports decidualization within the endometrium Prevents myometrial contractility Inhibits uterine production of prostaglandins Supports mammary growth for lactation Withdrawal at term leads to uterine contractions
Estrogen	Ovaries Corpus luteum Placenta Fetus	Softens collagen fibers in cervix and ligaments Increases uterine blood flow Promotes growth of the uterus and breast glandular tissue Increases production of insulin-like growth factors Enhances myometrial contractility Increases myometrial sensitivity to oxytocin, may upregulate oxytocin receptors

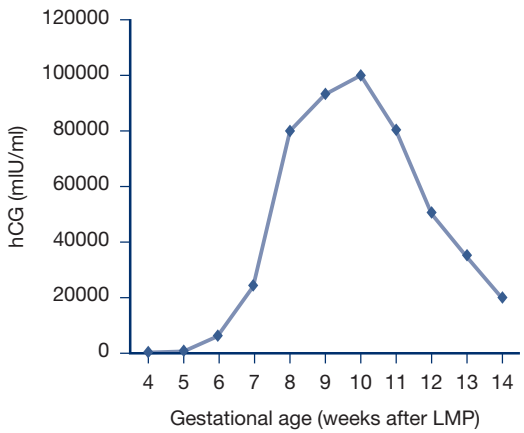

Figure 19-4 Human chorionic gonadotropin values in the first trimester of pregnancy.
Abbreviations: hCG, human chorionic gonadotropin; LMP, last menstrual period.

The natural rise and fall of hCG levels also corresponds to the natural history of nausea and vomiting in the first trimester. Although the direct etiology of nausea and vomiting in pregnancy is not known, it has been postulated that hCG may play a role. Nevertheless, studies that have linked hCG levels to nausea symptoms have not shown a direct correlation between the two. Women who have severe nausea and vomiting or hyperemesis may also have subclinical hyperthyroidism; in such women, the alpha subunit of hCG is able to stimulate the thyroid gland as though it were TSH.[20]

Human Placental Lactogen

Human placental lactogen, which is structurally similar to growth hormone, ensures adequate fetal nutrition by altering maternal glucose metabolism

so that glucose is available for fetal uptake. This transformation is achieved primarily by mobilizing fatty acids from lipids to provide an alternative fuel for the pregnant woman while sparing glucose for the fetus. hPL also increases maternal insulin resistance, thereby assuring consistent blood levels of glucose for fetal use. Glucose is transported across the placenta via facilitated diffusion.

The fetus relies primarily on glucose for nutritional needs; fetal production of glucose either does not occur or is undetectable.[21] It appears that the fetal liver does not develop the mechanism necessary for gluconeogenesis until just before birth.

The hPL-induced insulin resistance results in an increase in maternal insulin levels. This, in turn, stimulates amino acid production, thereby ensuring that amino acids are also available for fetal growth and development.

hPL is secreted by the placental syncytiotrophoblast. This agent is detectable in maternal circulation at 6 to 8 gestational weeks, with levels increasing in direct proportion to placental growth.

Progesterone

Progesterone and estrogen—both steroid hormones—act as intracellular chemical messengers by binding to intracellular receptors. These hormones influence many aspects of DNA transcription and cellular activities.

Progesterone is essential for maintenance of pregnancy in all mammals, and its name is derived from this function: "pro-gestational steroidal ketone." This hormone maintains the uterus in a quiescent state and helps suppress the maternal immune response to fetal antigens so that the fetal tissue is not rejected. Progesterone is produced by the corpus luteum in early pregnancy. The placenta then assumes the progesterone production at some point during gestational weeks 7 to 9, an event called the "luteal-placental shift." Additional functions of progesterone during pregnancy are listed in Table 19-1.

Low levels of progesterone in pregnancy are associated with an increased risk of spontaneous abortion. Women with luteal-phase defects can have progesterone levels that drop in early pregnancy, leading to spontaneous abortions. Progesterone supplementation has been shown to be effective for treating threatened miscarriage.[22,23] Progesterone is also used to prevent preterm birth in women who had a previous preterm birth.[24] Alternatively, administration of mifepristone, a progesterone receptor antagonist, will cause miscarriage.

Estrogen

Three types of naturally occurring estrogen have been identified: estrone, estradiol, and estriol. Estriol (E_3) is the primary estrogen of pregnancy. Estrogen production during pregnancy entails a three-part interplay between the woman, fetus, and placenta; each of these three separate but interrelated factors completes part of estrogen synthesis but not all of it. Estrogen is initially synthesized in the corpus luteum until the eighth or ninth week of gestation. At that point, the fetal adrenal glands are mature enough to produce the necessary estrogen precursors, and the placenta is able to produce and excrete the active forms of estrogen. Placental estrogen production depends on input from both the fetal and maternal adrenal cortex because the placenta cannot produce the androgenic C19 steroid dehydroepiandrosterone (DHEA), and its sulfoconjuate, DHEA-S, which are essential substrates of estriol.

Selected effects of estrogen during pregnancy are listed in Table 19-1. Estrogen encourages growth of breast tissue, stimulates uterine contractility, and increases uterine receptiveness to oxytocin. For most of pregnancy, however, the uterus remains refractory to the effects of estrogen—a consequence of the influence of progesterone, which ensures that the uterine myometrium has very few estrogen receptors.

Fertilization and Implantation

The processes that make it possible for the single cell that results from the fusion of the ovum and the sperm to develop and mature within the course of months are the subject of many fields of study, including genetics, embryology, and fetology. It is beyond the scope of this chapter and this text to cover these topics in detail. Instead, this chapter presents an overview of the knowledge needed by a practicing midwife with regard to the milestones of embryologic and fetal development and the intricate and vulnerable steps that create organs and organ systems. This understanding will help the midwife discuss embryonic and fetal development with the woman and her family and also provides general knowledge regarding the causes of congenital malformations.

Fertilization

Fertilization is the process of fusion of two haploid (containing 23 chromosomes) cells—a sperm and an

ovum—to form a diploid (containing 46 chromosomes) cell or zygote. Once a sperm cell binds to receptors on the oocyte membrane, its nucleus is pulled into the cytoplasm of the oocyte and the oocyte membrane depolarizes, causing destruction of other sperm cells. This prevents the binding of other sperm cells and, therefore, polyspermy. Once the sperm enters the ovum, the oocyte, which was arrested in the metaphase of the second meiotic division, completes metaphase. As the nuclei of the ovum and the sperm swell, fertilization occurs, the nuclear membranes disappear, and pairing of the chromosomes occurs to create the diploid zygote (**Figure 19-5**).

Fertilization usually occurs in the fallopian tube and takes approximately 18 to 24 hours. The fertilized oocyte becomes known as a *zygote*. The zygote begins as a single cell that contains 46 chromosomes: 23 from the ovum and 23 from the sperm. At this stage, the genetic code for that individual is formed. The 23rd pair of chromosomes determines the fetus's sex. The ovum contains only an X chromosome, so the X or Y chromosome that pairs with the maternal X chromosome is donated by the sperm.

Blastocyst

As the zygote moves through the fallopian tube into the uterine cavity, the cells divide (rapid mitotic activity) and create additional cells. These cells, known as blastomeres, are held together by the zona pellucida (an extracellular glycoprotein matrix) and form a solid ball of 12 to 16 cells known as the *morula*. As the morula enters the uterus, which occurs approximately 4 days after fertilization, intracellular fluid increases and a central cavity forms. The zygote is now called a *blastocyst*. The blastocyst is composed of four components: (1) the zona pellucida, (2) an inner cell mass, (3) an outer layer of cells called the trophoblast, and (4) a fluid-filled cavity. The trophoblast eventually forms the placenta and chorion, while the inner cell mass develops into the embryo and amnion.

The blastocyst is said to "hatch" when it sheds the zona pellucida. At approximately 10 to 12 days post conception or 1 to 2 gestational weeks, the blastocyst implants in the decidua.[25] The blastocyst is guided to a receptive area, usually in the fundus, for implantation. It orients itself so that the embryonic pole is closest to the endometrium, and the process of implantation begins.

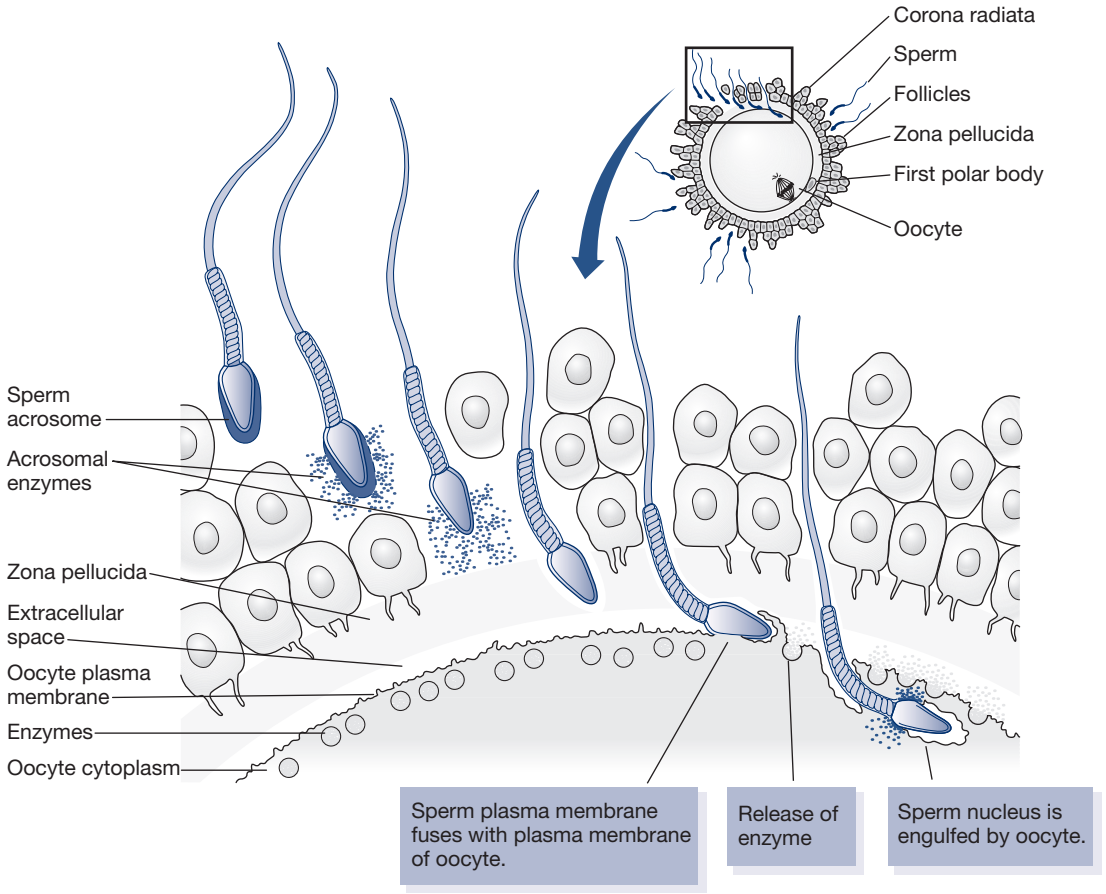

Figure 19-5 The process of fertilization.

The Trophoblast and Implantation

The trophoblast is unique tissue playing an essential role in pregnancy. The trophoblast, in contact with the endometrium, first differentiates into two distinct tissues: the syncytiotrophoblast and the cytotrophoblast. The *syncytiotrophoblast* is a syncytium or multinucleate protoplasmic mass formed by the fusion of cells, some of which were endometrial decidual cells. The syncytiotrophoblast burrows into the endometrium and remains in contact with maternal blood throughout gestation.[25] This tissue becomes the primary source of hCG.[26] The implantation process can result in light vaginal bleeding or spotting, referred to as "implantation bleeding," and can be mistaken for a light menses.

As the syncytiotrophoblast invades the endometrium, it disrupts maternal endometrial capillaries, portions of which are engulfed and become the lacunae. This process initiates the lacunar stage, which is marked by growth of small vacuoles in the syncytiotrophoblast that multiply and eventually fuse to form a system of *lacunae*. Initially, these lacunae fill with a substance derived from glandular secretions of the endometrium and a filtrate of maternal blood that diffuses through the trophoblastic tissue and serves to nourish the embryo. The lacunar networks enlarge and communicate with each other as they evolve into the intervillous space (IVS). *The intervillous space* will fill with maternal blood and bathe placental villi as they form into this space (**Figure 19-6**).[27]

The *cytotrophoblast* tissue is the point of contact between fetal and maternal tissues. The cytoblast differentiates into extravillous trophoblasts, and these trophoblasts then form columns of cells that become the anchoring villi of the placenta (**Figure 19-7**).[17,25] Proliferative extravillous cytotrophoblast cells also invade the spiral arteries, that developed in the luteal phase to provide blood to the endometrium. When the cytotrophoblastic cells reach the spiral arteries, the cytotrophoblastic cells replace the endothelial layer of the spiral artery and begin the process of remodeling those arteries into the low-pressure, high-volume deinnervated open vessels needed to sustain the developing fetus.[28-30]

This cytotrophoblastic invasion of the spiral arteries occurs in two waves. The first wave takes place in the first weeks following implantation, as cytotrophoblast cells move down the endothelial lining of the spiral arteries in the decidua. The second wave occurs between 12 and 20 weeks of gestation.[25] During this wave, the cytotrophoblast extends into the myometrial portion of the spiral arteries (**Figure 19-8**).[28-31] The changed spiral artery architecture accommodates a remarkable change in uterine blood flow. Prior to pregnancy, uterine blood flow is approximately 50 mL per minute; by comparison, at the end of pregnancy, uterine blood flow is approximately 750 mL per minute.

Complications of pregnancy can occur if cytotrophoblast cells fail to fully invade or deinnervate the spiral arteries.[29] The genesis of preeclampsia is cytotrophoblast invasion, wherein the second wave does not occur and the maternal spiral arteries remain small, high-resistance vessels. These vessels culminate in a small placenta secondary to limited maternal blood flow. The effects of preeclampsia become evident when the nutritional needs of the fetus are no longer met by the small placenta.[31] When the trophoblastic tissue invades indiscriminately, without the normal complex immunologic checks and balances that occur between the maternal and trophoblastic tissues, placenta accreta can develop. Abnormal placentation can also cause fetal growth restriction and has more recently been associated with preterm premature rupture of membranes and preterm labor.[31]

The Placenta

The placenta has been recognized as an important organ for centuries and has been imbued with many different meanings. In some cultures, the placenta is considered the alter ego or "secondary self."[32] In early Egypt, the placenta was described as the "external soul." In other cultures, it is referred to as "the tree of life," because the vessels that branch out from the insertion point on the maternal surface of the placenta look like branches of a tree.

At term, the placenta weighs approximately 500 grams, is 18 to 22 centimeters in diameter, and is approximately 2 to 2.5 centimeters thick.[33,34] This organ has a villous surface area of approximately 1.8 m^2 at term.[33] Clinical implications of common variations in placental gross anatomy and systematic evaluation of the placenta following birth are reviewed in the *Third Stage of Labor* chapter.

The placenta is a vital endocrine, hemochorial, villous organ with four well-known functions:

- Produces hormones critical for maintenance of pregnancy, and other bioactive substances.[25]
- Transports substances between the maternal and fetal circulations, including acting as the respiratory organ for gas exchange.
- Metabolizes and synthesizes agents necessary for sustaining pregnancy.
- Provides an immunologic barrier between the maternal and fetal systems.

Many of these functions overlap (**Figure 19-9**).

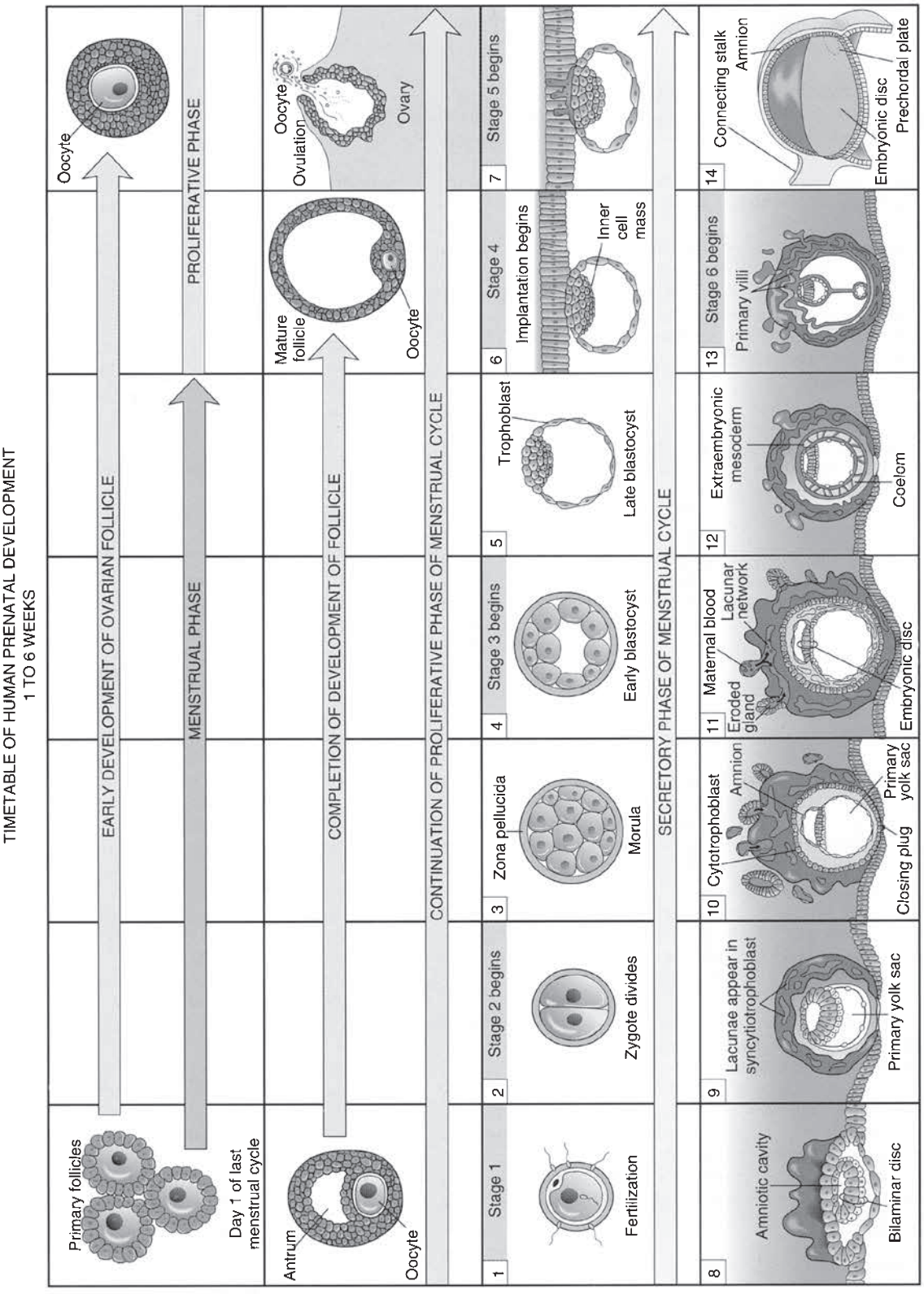

Figure 19-6 Development of an ovarian follicle containing the oocyte, ovulation, and phases of the menstrual cycle are illustrated. Human development begins at fertilization, approximately 14 days after the onset of the last menstruation. Cleavage of the zygote in the uterine tube, implantation of the blastocyst, and early development of the embryo are shown.
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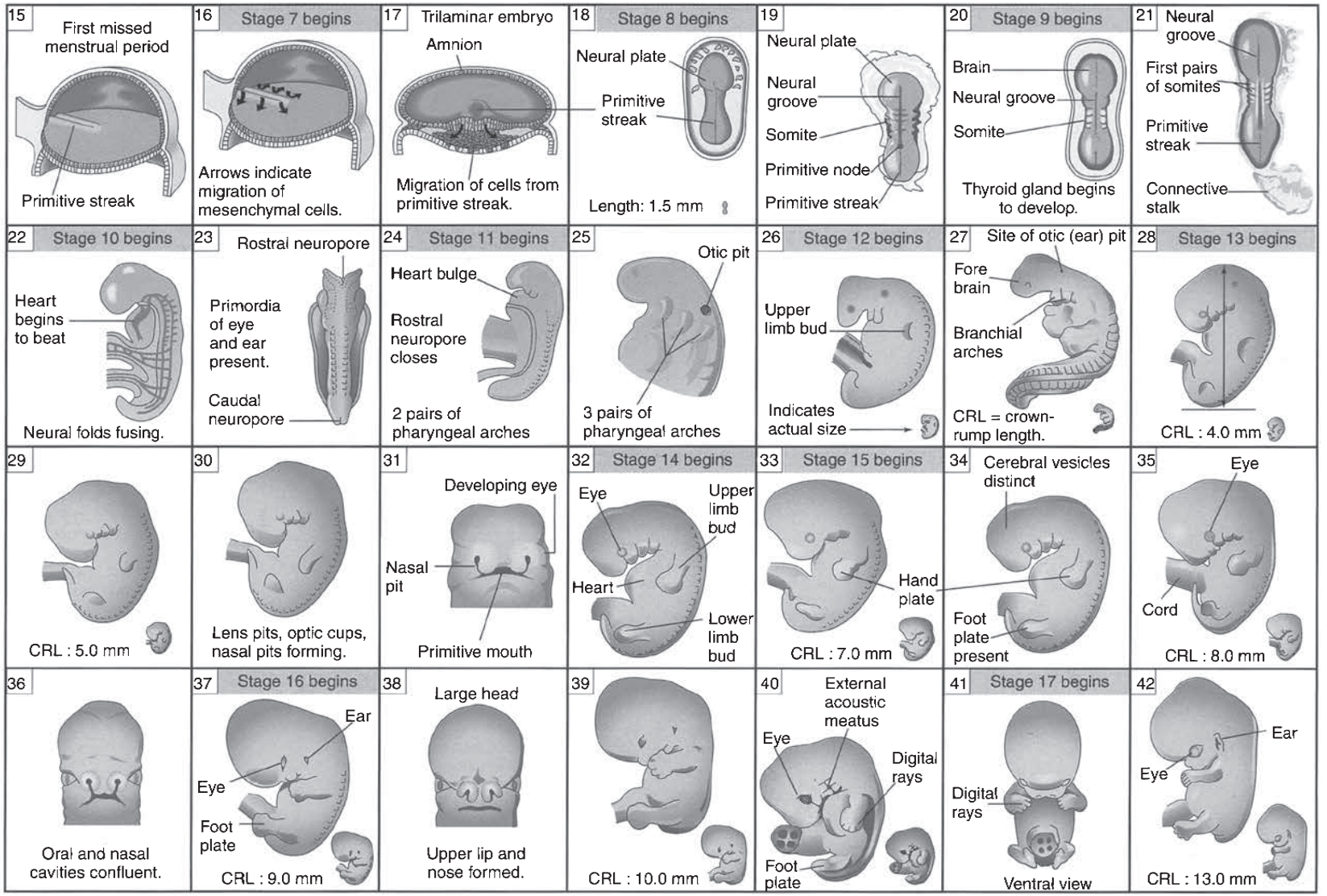

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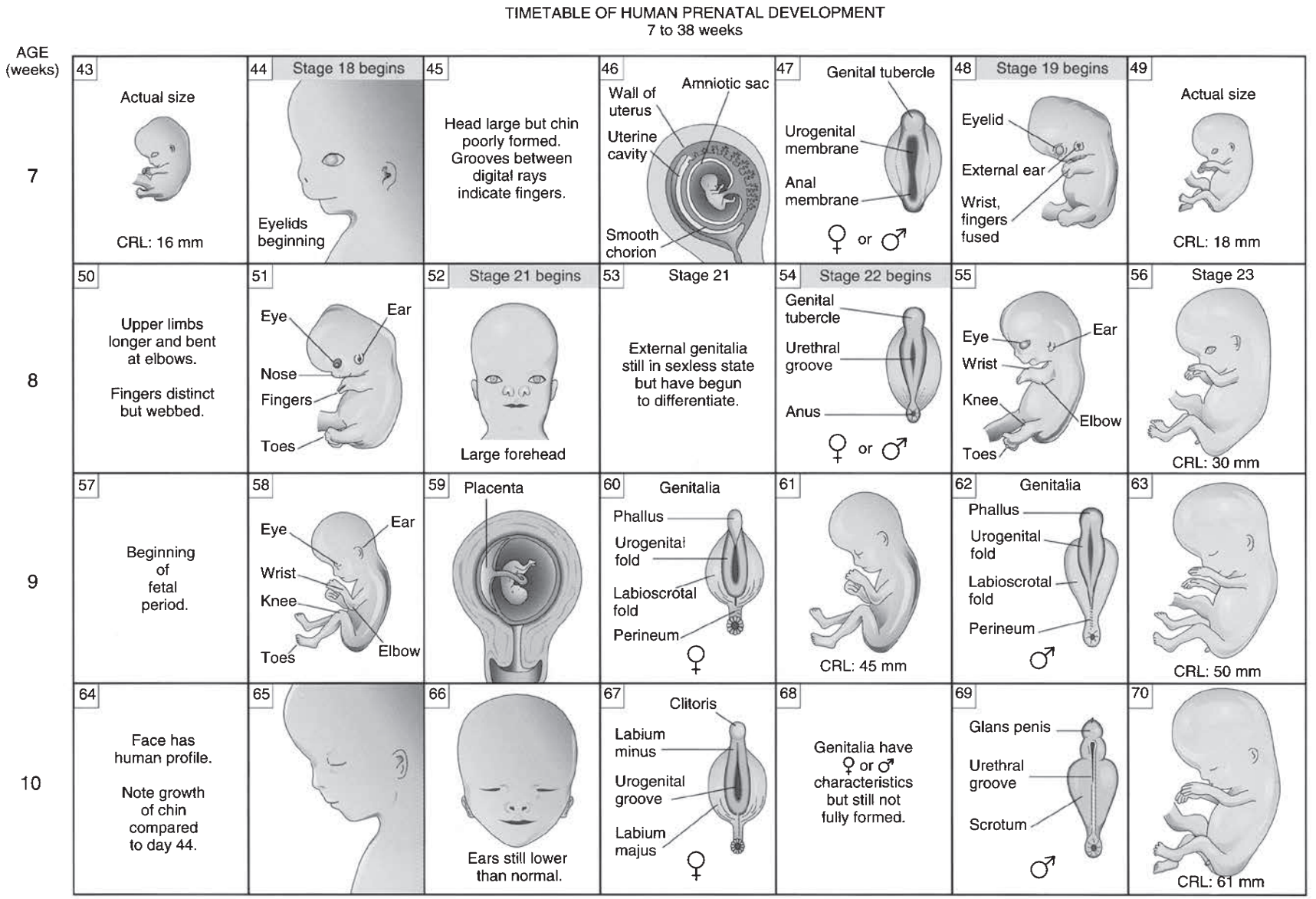

Figure 19-6 (*Continued*)

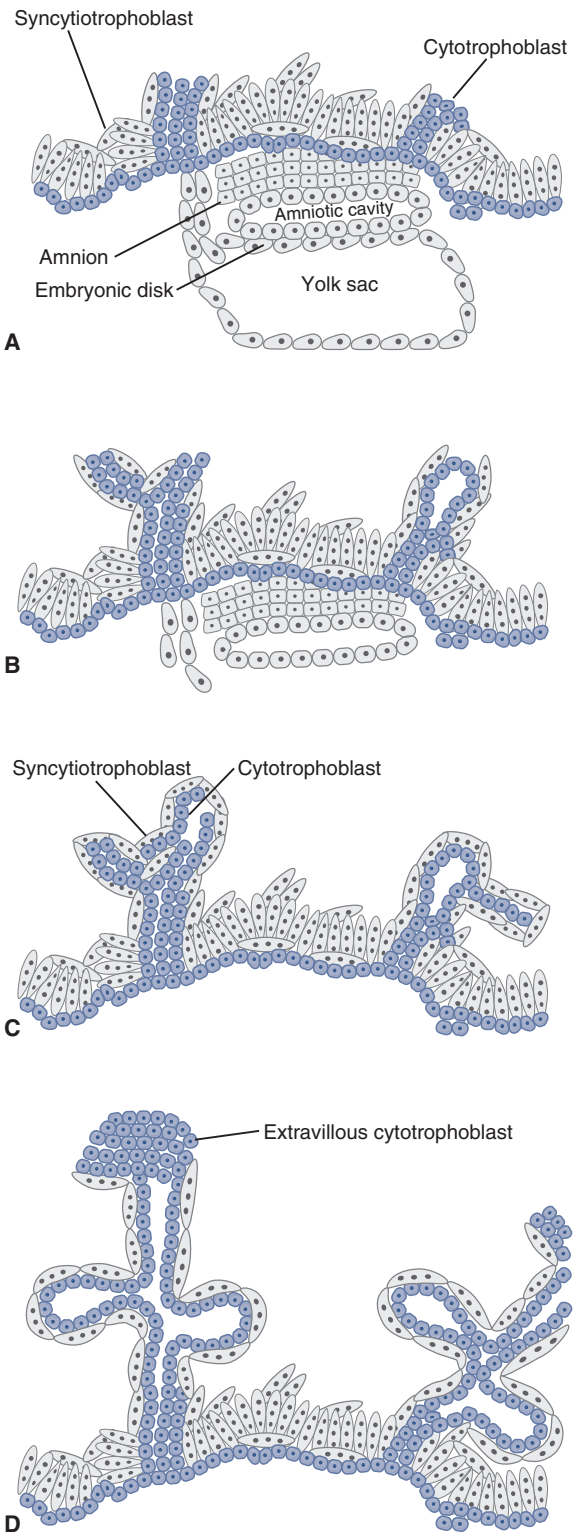

Figure 19-7 **A.** Cytotrophoblast columns in early implanted embryo. **B.** Extension of columns and differentiation of peripheral cells. **C.** Folding of extensions caused by shape of syncytiotrophoblast cells. **D.** Formation of trophoblastic villi.

Reproduced with permission from Cole LA. hCG, the wonder of today's science. *Reprod Biol Endocrinol.* 2012;10:24-28.[17] Published by BioMed Central.

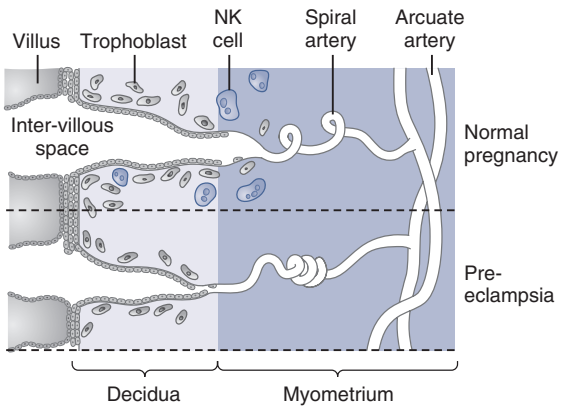

Figure 19-8 Spiral artery invasion in normal and preeclamptic pregnancies.

Based on Jain A. Endothelin-1: a key pathological factor in pre-eclampsia. *Reprod BioMed Online.* 2012;25:443-449.[31]

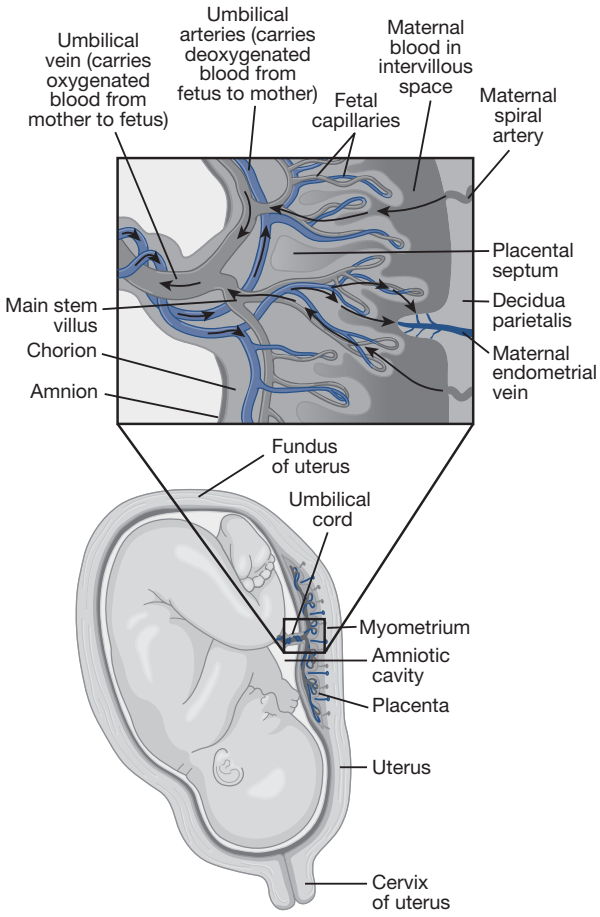

Figure 19-9 Transverse view of a placenta at a full-term pregnancy. Maternal blood flows into the intervillous spaces in spurts from the spiral arteries, and exchange of nutrients and gases occurs with the fetal blood as the maternal blood flows around the branch villi. The two umbilical arteries carry poorly oxygenated fetal blood to the placenta; the one umbilical vein carries oxygenated blood to the fetus. The placental cotyledons are separated from each other by septa projections of the decidua basalis. Each cotyledon consists of two or more main stem villi and branches.

Recent research regarding placental function suggests that the placenta may also play a more complex role during pregnancy. Instead of being just a passive filter or barrier, which has historically been the perceived role of the placenta, this amazing organ integrates a wide range of chemical messages produced by the fetal and maternal systems and actively adapts to different conditions.[25,35] In short, the placenta plays an active and independent role in fetal growth and development.

Development of the Placenta

The cytotrophoblast lines the decidua and firmly anchors the placenta. The extraembryonic mesoderm—another tissue from the original blastocyst—grows into the cytotrophoblastic columns to form villi that float freely in the intervillous space. The fetal vessels that form within the villi are separated from maternal blood by three layers of cells: (1) fetal endothelial cells, (2) connective tissue, and (3) cytotrophoblastic cells that are eventually called the chorionic villi epithelium.

Blood flow begins in the placenta when the intervillous spaces become filled with maternal blood. Initially, the preponderance of fluid in the intravillous space consists of maternal serum and secretions from the endometrial glands, until fetal vessels begin to function at 8 to 9 weeks' gestation. Common variations in placental morphology that have clinical implications are reviewed in the *Third Stage of Labor* chapter.

Placental Transport Mechanisms

Placental function involves multiple transport mechanisms, including facilitated diffusion, passive diffusion, active transport, pinocytosis, endocytosis, bulk flow, solvent drag, accidental capillary breaks, and independent movement.[25,35] These mechanisms are described in Table 19-2.

Uteroplacental Circulation and Fetal Respiration

The uteroplacental circulation is the lifeline for the fetus. As such, physiology affecting fetal respiration deserves detailed attention. Fetal respiration depends on four factors: (1) adequate maternal blood flow into the intervillous space; (2) sufficient functional placental villi for gas exchange; (3) adequate diffusion, facilitated diffusion, and active transport of gases, nutrients, and fetal waste products; and (4) unimpaired fetal circulation through the placenta and umbilical cord.

Table 19-2 Placental Transport Mechanisms

Transport Mechanism	Description of Mechanism	Examples of Substances Transported via This Mechanism
Simple diffusion	Transfer of substances across a membrane down a concentration gradient (from an area of higher concentration of the substance to an area of lower concentration of the substance)	Oxygen, carbon dioxide, electrolytes, water, certain medications including analgesic and anesthetic agents
Facilitated diffusion	Transfer of substances across a membrane down a concentration gradient (such as diffusion), but in a manner that allows for more rapid or more specific transfer	Substances such as glucose that are essential for rapid fetal growth but are present in low concentrations in the maternal blood
Active transport	Transport against a concentration gradient, which requires energy	Transfer to the fetus of substances that are present in higher concentrations in the fetus than in the pregnant woman, such as iron and ascorbic acid
Pinocytosis	A form of endocytosis that allows small particles to be brought into a cell. The cell membrane folds around the particle and becomes an intracellular membrane.	Immunoglobulin G (IgG) maternal antibodies, phospholipids used to make cell membranes, lipoproteins used to transport cholesterol
Breaks between cells	Active capillary breaks allow fetal and maternal cells to mix	Cell trafficking may play a role in fetal immunity and in maintaining maternal tolerance to the fetal allograft
Bulk flow	Movement of water and some solutes in water via aqueous pores	Free movement of water and some solutes maintains equal osmolality in the fetal and maternal compartments

The fetus can use either oxygen or glucose to produce the energy needed for growth and metabolic processes. Carbon dioxide (CO_2) and water (H_2O) are the end products of using oxygen for aerobic metabolism. When the fetus does not have sufficient oxygen, glucose is used to create adenosine triphosphate (ATP) for energy. This process is called anaerobic metabolism, and one of its end products is lactic acid ($C_3H_6O_3$), which has important clinical implications that are reviewed in more detail in the *Fetal Assessment During Labor* chapter.

Immunology of the Placenta

The fetus is considered a semi-allogenic graft, meaning that it consists of foreign tissue from the same species but has different antigens. The processes that ensure the woman's body does not reject the fetus have not been yet fully identified. Most importantly, the trophoblast does not express the cell surface antigens that are the usual targets for maternal antibodies. Additionally, the hormones of pregnancy alter the maternal immune response in ways that enable tolerance to contact with the cytotrophoblast and syncytiotrophoblast.

There appears to be more cellular traffic between the fetus and the pregnant woman than was originally hypothesized.[36] Fetal cells can be found in many maternal tissues and can persist in a mother for decades. Similarly, maternal cells enter the fetal circulation via mechanisms that have not been fully elucidated and may support the development of fetal immunity. The implications of this microchimerism are unknown.[36]

Role of the Placenta in Fetal Development and Programming

The placenta synthesizes many substances, including glycogen, cholesterol, and fatty acids, which contribute nutrients and energy for the embryo and growing fetus. Placental metabolism uses approximately 40% to 60% of the oxygen and glucose that is delivered into the intervillous spaces.[35] The placenta can also alter its metabolism in the presence of stressors such as hypoxia.[35] More importantly, the placenta is instrumental in making heritable changes in gene expression (e.g., epigenetic changes) of the fetal genome that affect postnatal life.[37,38] This developmental plasticity in placental function is an area of current research interest.

Fetal Origins of Adult Disease/ Fetal Programming

In the 1990s, physician and researcher David Barker published a theory that was later known by various names. Initially, it was best known as "Barker's hypothesis." Today, alternative nomenclature—such as "developmental origins of health and disease" (DOHaD), "fetal programming," and "the thrifty genotype"—is more commonly used because research has established that Barker's work is no longer a hypothesis. Barker's major finding was that fetuses who were undernourished in utero were at increased risk for chronic diseases such as obesity, diabetes, coronary heart disease, and hypertension when they grew to adulthood in a country with abundant food.[38] His work linked birth records with later health and death records in the United Kingdom. Approximately a decade after his initial publication, Barker described his basic methodology and acknowledged his debt to the meticulous and complete records of midwives, without whom he would not have had adequate data to discover the associations.[39]

Subsequent to Barker's initial work, many links have been discovered between the fetal environment and several major chronic diseases that arise during adulthood.[40-47] When a pregnant woman experiences poor nutrition, such as during war or related to severe poverty, her placenta will lower production of enzymes that protect the fetus from cortisol. More exposure to cortisol in utero enables the fetus to survive low protein intake during gestation, but increases the risk of cardiovascular disorders when that fetus becomes an adult.[43] Specific physiologic changes related to these processes are under investigation and much remains to be learned.

Current research suggests many types of prenatal stresses—not just nutrition—can permanently affect fetal physiology and infant development, especially in the areas of vascular, metabolic, and endocrine changes. Prenatal stressors may include stress hormones, environmental toxins, and even paternal genes. Currently, DOHaD is the subject of a great deal of research, including that which focuses on the evaluation of epigenetic mechanisms and the investigation of maternal, paternal, and fetal genes.

Barker's work is of importance to midwifery because it underscores the importance and complexity of preconceptional and prenatal care. In an era when prenatal visits are becoming fewer in number and shorter in duration, it is the midwife's responsibility to promote health care both for the current generation of women and for the future of society's health.

Role of the Placenta in Pregnancy-Related Complications

In the short-term, the placenta plays a central role in many pregnancy-related complications. As described, the origins of preeclampsia occur early in pregnancy although signs and symptoms develop much later.

Poor maternal nutrition can result in small, poorly functioning placentas that provide inadequate nutrients to the fetus, resulting in fetal growth restriction. Women with diabetes and hyperglycemia may experience microvascular and macrovascular placental changes, such as thickening of the basement membrane of cells, and these placentas also may not sustain normal fetal growth.

A more complex example is illustrated in the relationship between obesity and poor maternal and newborn outcomes. The placentas of women who are obese express placental genes differently than the placentas of women who are lean, and they tend to show metabolic and oxidative stress leading to cell injury and inflammation of the placenta.[48] The low-level inflammation damages the endothelial cells that line blood vessels, which in turn, adversely affects delivery of oxygen and nutrients to the fetus.

Amniotic Fluid

Amniotic fluid serves multiple functions during pregnancy, including cushioning and protecting the fetus, providing space for fetal movement and growth, and maintaining consistent temperature and pressure.[49] Substances found in amniotic fluid include electrolytes, urea, creatinine, bile pigments, renin, glucose, hormones, fetal cells, lanugo, and vernix caseosa. The fluid's osmolality and composition evolve throughout the course of the pregnancy and are similar to the characteristics of dilute fetal urine in a term gestation.

In the latter half of pregnancy, amniotic fluid is primarily produced by the fetus in the form of urine (700–800 mL) and lung fluid. The average amount of amniotic fluid at term is 700–800 mL.[50] Amniotic fluid is removed via fetal swallowing and diffusion across the placenta. The fluid secretions from the fetus's lungs contain phospholipids, including lecithin and sphingomyelin, which are components of surfactant—a substance that is essential for the function of the neonatal lungs. As pregnancy advances, the absolute and relative amounts of lecithin in amniotic fluid increase. The ratio of lecithin to sphingomyelin is used as means of assessing fetal lung maturity to help guide decisions about delivery prior to term. While the amount of fluid varies in the third trimester and begins to decrease after 40 gestational weeks, the amount of fluid that is turned over (produced and removed) remains relatively constant, at approximately 1000 mL per day.

Amniotic fluid is essential for normal fetal growth. *Oligohydramnios* at term is defined as an amniotic fluid index of 5 centimeters or less (via ultrasound assessment). Oligohydramnios in the second trimester can inhibit normal fetal lung development. Etiologies of this disorder include fetal renal abnormalities, heart disease, and fetal growth restriction. Maternal conditions that can cause oligohydramnios include severe hypertension, dehydration, and renal disorders.

Polyhydramnios is defined as an amniotic fluid index of more than 24 centimeters and is caused by too much production or too little removal of amniotic fluid. Modest polyhydramnios can be idiopathic, whereas oligohydramnios is most likely to be associated with an abnormal condition in the fetus or the pregnant woman. Conditions that can cause polyhydramnios include maternal hyperglycemia, obstructed fetal swallowing, fetal cardiac failure, and severe fetal anemia.

The Umbilical Cord

The umbilical cord has two arteries and one vein surrounded by a gelatinous collagen material called *Wharton jelly*. The vessels within the cord are longer than the cord itself, so they coil in a spiral fashion as the cord lengthens. This coiling may protect the blood flow within the cord if it is subjected to tension or compression. The maximum length of the cord averages 55 to 60 centimeters. The cord epithelium, which is formed by the amnion, typically inserts into the placenta centrally but may insert at many portions of the placenta. The only clinically relevant aspect of cord insertion occurs when the cord inserts marginally (at the placental edge) or when the insertion is velamentous, wherein the cord inserts into the membranes. With velamentous cord insertion, fetal vessels run through the membrane unprotected by Wharton's jelly. When umbilical cord vessels run through the membranes across the cervix, the presentation is termed vasa previa. Although vasa previa is very rare, fetal mortality occurs approximately 50% of the time when the vessels rupture.[51]

The Embryo

Organogenesis

Returning to the blastocyst, the *embryonic period*, which is the period of organogenesis, occurs between 2 and 8 weeks following fertilization. The *fetal period*, marked by growth and tissue differentiation, starts at 8 weeks after fertilization and extends to birth. The age of the embryo in "gestational weeks" refers to

the number of weeks after fertilization in the embryology and basic science literature. Clinical obstetric texts use the term "gestational weeks" to define the number of weeks after the woman's last menstrual period. This 2-week discrepancy can be confusing unless the reader understands which terminology the author is using. This chapter follows the convention used by embryologists for this section of this chapter.

The embryonic disc within the blastocyst gives rise to three germ layers: (1) the endoderm, (2) the mesoderm, and (3) the ectoderm. The third week of embryo development is marked by a period of rapid growth, during which the mesoderm, ectoderm, and endoderm begin to undergo the dramatic transformations that form specific embryonic structures (**Table 19-3**). Most functional organs and organ systems are formed from all three embryonic germ layers. Each germ layer contributes a specific feature, but the germ layers do not produce specific structures separately from each other.

Morphogenesis

The genetically controlled process during which cells and cell groups take on a specific form, shape, and function is known as *morphogenesis*. Initially, the cells in the embryoblast are all the same; that is, they are stem cells, capable of becoming any type of body cell. These unspecialized cells must proceed through two distinct phases: (1) determination, which restricts the cell to a specific type, and (2) differentiation, in which the morphologic and functional characteristics specific to that cell type develop.

Cell differentiation often involves a process called induction wherein cells in a local environment signal one another to develop in a specific manner. The signaling cell is called the inductor, and the cells that respond to induction are called the inducers. If disruption occurs during any of the differentiation sequences, the next step in the process will not proceed in the usual fashion and an abnormality will develop. Complete organ agenesis may occur when the process is disturbed at an early stage. For example, anencephaly is a form of organ agenesis wherein the brain does not form. Some disruptions in differentiation sequences will result in termination of the pregnancy, whereas others may produce a fetal defect that can be undetectable, minor, or clinically significant.

In addition to cell differentiation and induction, several other cellular mechanics are involved in morphogenesis, including proliferation, migration, adhesion, and folding. Some of these processes are summarized in **Table 19-4**.

The Fetus

Fetal Growth

All major organs have formed by the beginning of the fetal period. Fetal growth involves both hyperplasia (cellular division that yields a significant increase in cell numbers) and hypertrophy (an increase in cell size). At approximately 32 weeks' gestation, hypertrophy dominates.[52] The rate and amount of fetal growth are determined by many factors, including genetics, placental metabolism, maternal conditions, maternal behavior, and environmental factors. For example, there is a well-known correlation between maternal smoking and impaired fetal growth.[53]

Adequate fetal growth is also directly associated with optimal functioning of the placenta and the uterine vascular system. Alterations in the trophoblastic invasion of the maternal spiral arterioles as well as inadequate development of the chorionic villus can result in impaired fetal growth.

Table 19-3 Differentiation of Embryonic Germ Layers

Ectoderm	Mesoderm	Endoderm
Central and peripheral nervous system	Connective tissues	Epithelium of the digestive system (except the mouth and anus, which are involutions of the ectoderm)
Epidermis including hair, nails, and sebaceous glands	Muscle tissue	Liver and pancreas
Epithelium of sensory organs	Skeleton (bone)	Respiratory system, including alveolar cells of the lung
Nasal and oral cavities	Cardiovascular	Thymus, thyroid, parathyroid, and pancreas
Salivary glands	Lymphatics	
Adrenal medulla	Urogenital structures (gonads and kidney)	
Parts of the pituitary gland	Serous lining of body cavities (peritoneum, pleural, and pericardium)	

Table 19-4 Selected Cellular Mechanisms Involved in Morphogenesis

Cell Mechanism	Description	Congenital Defects That May Result from Faulty Mechanism
Cell proliferation (hypertrophy)	A rapid increase in the number of cells by cell division and growth	Inhibition of cell proliferation can occur when there is a lack of space. A diaphragmatic hernia will allow abdominal contents to be in the thoracic cavity; pulmonary hypoplasia occurs when the lungs are not allowed to continue cell proliferation.
Cell differentiation	Process by which pluripotent cells become more specialized cells	Prenatal exposure to high levels of methylmercury is thought to result in failure of cell differentiation in the central nervous system, causing neurologic and developmental defects.
Apoptosis	A genetically determined process of cell self-destruction. Cells produce enzymes that lead to their dissolution.	Excessive enzymatic release can lead to excessive cellular destruction and result in defects such as limb shortening; deficient enzymatic release can lead to defects such as bowel atresia, syndactyly, or imperforate anus.
Migration	A dynamic and cyclical process in which layers migrate to a strategic location along the developing embryo. The cell extends protrusions at its front and attaches to the substratum on which the cell is migrating.	Central nervous system abnormalities, such as lissencephaly (lack of grooves on the brain)
Adhesion	The interaction of specific mechanisms on one cell and complementary adhesion molecules on the membrane of another cell	Cleft palate and neural tube defects occur as a result of alteration in cell recognition and the adhesion process.
Folding	As new cells form, the embryo is forced to conform to the available space. The embryo folds in both the transverse and longitudinal planes. Structures within the embryo also must fold to conform to the space available to them.	Congenital heart defects Diverticula

Fetal Movement and Behavioral States

Limb movement develops at 9 weeks, with reflective leg movements occurring at 14 weeks. Hand-to-face movements become apparent by 12 to 13 weeks, while limb, head, and torso movements develop by 12 to 16 weeks. Fetuses begin to suck on fingers by 15 weeks and continue to develop more complex movement patterns after 24 weeks, when respiratory movements occur.[54]

Discrete fetal behavior states involving sleep–wake patterns and behavioral patterns begin to occur by 32 weeks. Distinct fetal heart rate patterns, eye movement, gross body movement, and quiet states have been catalogued.[55,56] Although women often report less fetal movement close to term, the underlying fetal physiology of this maternal perception is likely the development of fetal cycling, in which the fetus exhibits distinct quiet states and distinct active states. The average time the fetus spends in the quiet state ranges from 20 to 40 minutes.[54,55]

Maternal Adaptations to Pregnancy

Pregnant women experience a wide variety of anatomic and physiologic alterations over the course of pregnancy and postpartum. While many of these changes are normal symptoms of pregnancy, others herald the onset of an abnormal processes. A thorough knowledge of the maternal anatomic and physiologic adaptations that occur during pregnancy is, therefore, an essential foundation for midwifery practice. This section presents a broad overview of some of the clinically important anatomic and physiologic pregnancy adaptations. Management of pregnancy symptoms are described in the *Prenatal*

Care, *Pregnancy-Related Conditions,* and *Medical Complications in Pregnancy* chapters.

Musculoskeletal Changes

The musculoskeletal changes that occur during pregnancy are primarily related to weight gain, the growing uterus, the softening effects of progesterone on cartilage in joints, and the laxity of ligaments induced by estrogen and relaxin.[56] These changes result in lordosis, kyphosis, and altered gait that gradually increase as pregnancy progresses.

Changes within the pelvic girdle are the most profound. The sacroiliac joint widens and has more mobility. The symphysis also widens, and the pelvis develops an anterior tilt. Many of the common complaints of pregnancy can be attributed to these anatomic changes, including pelvic pain, sciatica back pain, and carpal tunnel syndrome. A careful history and physical examination is needed to rule out more serious problems for which pregnant women are at increased risk, such as herniated discs and peripheral nerve injury.

Integumentary Changes

Pregnancy is associated with many changes in the integument. Hyperpigmentation occurs as estrogen, progesterone, and melanocyte-stimulating hormone induce melanocytes to make and deposit pigment. This phenomenon results in darkening of the areola, the change of the linea alba to the *linea nigra*, and *melasma* or *chloasma* (irregular areas of pigmentation on the cheeks), which is also called the "mask of pregnancy."[57-59] Intertriginous areas such as the axillae, genitalia, perianal region, and inner thighs may also become darker during pregnancy.[59]

Thinning of the elastin fibers in connective tissue under the skin predisposes pregnant women to striae gravidarum (stretch marks). As the size of the abdomen and breasts increase, elastin fibers at the dermal–epidermal junction stretch and shift from perpendicular to parallel, which can create striae.

Vascular nevi called spider angiomas are common as blood vessels dilate and proliferate. These small red lesions have a central puncta and branches that extend from the center and disappear after birth.

Cardiovascular Changes

Cardiovascular changes in the pregnant woman begin early in the first trimester.[57] Blood volume increases by 40% to 50% over the course of pregnancy, reaching a maximum by 32 weeks' gestation. Plasma accounts for 75% of this increase. Cardiac output increases 30% to 50% to approximately 4 to 6 L/min, primarily as a result of increased stroke volume.[60] The increase in cardiac output begins in the first trimester and peaks at approximately 25 to 30 weeks' gestation, when the total blood volume is approximately 5000 to 6000 mL. Heart rate increases by approximately 10 beats/min, and blood pressure decreases gradually from prepregnancy values as early as 7 weeks' gestation. This decrease reaches a nadir in the second trimester. The decrease in blood pressure is presumed to occur in concert with expansion of the low-pressure placental compartment, and lower systemic vascular resistance induced by progesterone.[60]

Anatomically, the heart is displaced upward (cephalad) and rotated to the left as the uterus enlarges. Mild pulmonic or tricuspid regurgitation often occurs. Several common signs and symptoms are related to these changes. Systolic ejection murmur that is loudest along the left sternal boarder is a common finding in pregnant women and is attributed to the dramatic increase in cardiac output. These murmurs are clinically benign. A third heart sound may be auscultated in many pregnant women. The decrease in systemic vascular resistance in combination with pressure on the vena cava from the growing uterus is responsible for the dependent edema that most pregnant women experience in the third trimester and contributes to the development of varicosities, hemorrhoids, labial varicosities, and increased risk for venous thrombosis.

Careful monitoring of cardiovascular changes can facilitate early detection of abnormalities. For example, if a woman's blood pressure fails to decrease during the second trimester, she may have chronic hypertension or be at increased risk for developing preeclampsia. Cardiovascular changes can also increase the risk for adverse outcomes in women who have preexisting cardiomyopathies; these women should be referred promptly to a physician.[58]

Hematologic Changes

Two aspects of hematologic changes in pregnancy have important clinical implications. First, pregnancy is a hypercoagulable state, as evidenced by the increased clotting factors, decreased fibrinolysis, and decreased anticoagulant activity noted in the pregnant woman. Clotting factors I, II, VII, VIII, IX, and XII are more abundant in such women, whereas protein S levels and activated protein C levels fall. These alterations in the coagulation cascade are likely protective, geared toward preventing hemorrhage at birth. Nevertheless, they increase a woman's risk for venous thromboembolism in the prenatal and postnatal periods. In addition, significant placental damage and obstetric hemorrhage are associated with early onset of disseminated intravascular coagulation (DIC), which is

not a common component of surgical or traumatic hemorrhage. DIC occurs because the tissue factor in the placenta acts as a potent activator of the coagulation cascade, which is already in a hypercoagulable state.[58]

The second clinically important hematologic change in pregnancy relates to iron metabolism and iron-deficiency anemia. During pregnancy, the plasma volume increases and this expansion exceeds the increase in red cell mass, which results in a physiologic hemodilution. The physiologic hemodilution has a positive effect on placental perfusion, with blood becoming less viscous.[61] In addition, the maternal hemoglobin (Hgb) concentration decreases by approximately 2% to 10%. The drop in hemoglobin occurs because the fetal uptake of iron is usually more than maternal absorption of iron can replace. The net result is a decrease in the woman's hematocrit of approximately 3% to 5%, which reaches a nadir late in the second trimester or early in the third trimester.

Because iron is not easily absorbed, fetal uptake can deplete a woman's iron reserves despite the fact that iron absorption in the second and third trimesters increases more than fivefold.[62] Thus iron stores can be easily depleted in pregnancy and, in turn, iron-deficiency anemia is common in pregnancy.

Additional hematologic changes include a decrease in the concentration of plasma proteins, especially albumin. Lower albumin result in lower colloid oncotic pressure, which in tandem with decreased venous resistance, facilitates the development of dependent edema.

Respiratory Changes

Pregnant women commonly experience dyspnea even when at rest. The actual etiology is unknown, but most sources assume this condition arises secondary to the added respiratory effort and work of breathing.[63] Pregnancy is associated with an increase in minute ventilation (i.e., the volume of air inhaled and exhaled in a minute) and in pulmonary blood volume, both of which can contribute to the sensation of dyspnea. The etiology of these changes is largely unknown, but it is thought to relate to an enhanced sensitivity to carbon dioxide levels as well as to hypoxia. As the uterus expands into the abdominal cavity in the second and third trimesters, the respiratory effort required by the woman further contributes to dyspnea. Physiologic dyspnea can be distinguished from pathologic dyspnea by the respiratory rate. Tachypnea is a sign of possible respiratory compromise.

An increase in thoracic diameter and cephalad rise in the diaphragm (as much as 4 centimeters) changes the pregnant woman's lung capacity. Tidal volume increases by 30% to 40%, and vital capacity increases slightly (**Figure 19-10**).[63,64] This hyperventilation probably occurs secondary to the effects of progesterone and places the woman in a state of respiratory alkalosis, which has the effect of improving carbon dioxide transfer from the fetus to the maternal circulation.

Under the influence of estrogen and increased blood volume, nasal passages become edematous and hyperemic in pregnancy. Women often report more congestion and/or rhinitis, sometimes confusing the symptoms with an upper respiratory infection or allergies.

Changes in the Oral Cavity

During pregnancy, bleeding gums, especially after brushing teeth, is related to estrogen- and progesterone-mediated inflammation and hyperemia. Elevated levels of estrogen create a favorable environment for the growth of bacteria that can cause gingivitis and gingival inflammation. Women should receive regular dental care during pregnancy.

Gastrointestinal Changes

During pregnancy, decreased peristalsis caused by relaxation of the smooth muscle of the large bowel in the presence of increased amounts of progesterone, as well as changes in fluid reabsorption increases the risk of constipation. The displacement and compression of the bowel by the enlarging uterus or presenting part may also contribute to decreased motility in the gastrointestinal tract and, therefore, to constipation. Specifically, the stomach is moved superiorly and the intestines are displaced laterally. Another factor that may contribute

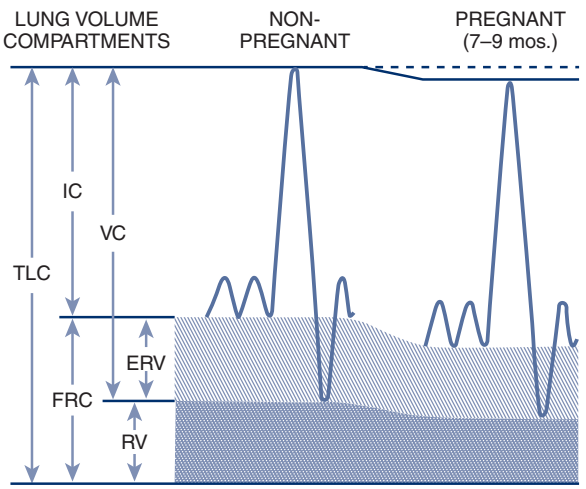

Figure 19-10 Changes in lung volumes in women who are 7–9 months pregnant compared with volumes in nonpregnant women. Abbreviations: ERV, expiratory reserve volume; FRC, functional residual capacity; IC, inspiratory capacity; RV, residual volume; TLC, total lung capacity; VC, vital capacity.

to constipation is the decrease in gastric emptying time and gastric motility secondary to the smooth muscle relaxation effects of progesterone. Thus, constipation is common, especially in the first trimester when the growing uterus also places pressure on the descending colon.

Several physiologic changes of pregnancy predispose the pregnant woman to heartburn (pyrosis): The lower esophageal sphincter is relaxed, gastrointestinal motility is slower, esophageal function and peristalsis change, and the angle of the gastroesophageal junction is altered as the stomach is displaced by the enlarging uterus. Women with preexisting gastroesophageal reflux disorder may find the condition aggravated throughout pregnancy. Flatulence and gas pain is more common during pregnancy due to decreased motility from the effect of progesterone relaxing smooth muscle and from the displacement of and pressure on the intestines by the enlarging uterus.

Renal Changes

Two different aspects of renal changes in pregnancy are responsible for symptoms that must be carefully evaluated to determine normal versus abnormal alterations. First, a marked increase in renal plasma flow is the natural consequence of arterial vasodilation and increased cardiac output. Renal blood flow increases 60% to 80% above prepregnant levels in the first and second trimesters, and 50% above prepregnant levels in the third trimester. The glomerular filtration rate (GFR) increases by 50% over prepregnant levels, peaking at 12 gestational weeks.[65] The obvious physiologic consequences of these changes are the commonly reported symptoms of urinary frequency and nocturia that frequently occur at two different times during the prenatal period. Frequency during the first trimester is due to hormonal changes affecting levels of renal function as well as developing hypervolemia and bladder compression within the pelvis due to uterine growth. Bladder compression resolves as the uterus becomes an abdominal organ in the second trimester. Urinary frequency during the third trimester occurs most often among primiparous women, after engagement has occurred when the presenting part descends into the pelvis and causes direct pressure against the bladder.

Nocturia also can be caused by increased urine production at night. Venous return from the extremities is facilitated when the woman lies in a recumbent lateral position while sleeping at night, when the uterus is not pressing against the pelvic vessels and inferior vena cava, which results in an increase in urinary output. Additionally, pregnant women have an increase in sodium excretion at night, with an associated increase in fluid excretion, which may also explain nocturia.

The increased flow may be great enough that the descending tubule is unable to reabsorb all glucose. This resultant physiologic glycosuria is usually intermittent, but it affects as many as 20% of pregnant women. Similarly, protein reabsorption is not as efficient as it is in the nonpregnant state. A small amount of urinary protein in a sample of concentrated urine can cause a dipstick test to be positive, which may be falsely interpreted as a urinary tract infection or even proteinuria associated with preeclampsia. Serum creatinine likewise falls; thus, plasma values for creatinine that would be considered normal in a nonpregnant woman may actually reflect renal dysfunction in a pregnant woman.

Pregnant women are at increased risk for urinary tract infections because the ureters, urethra, and bladder dilate under the influence of progesterone and ascending infections can occur, including pyelonephritis. The bladder becomes hyperemic and urinary stasis can occur, which occasionally results in stress incontinence. Thus, physiologic changes in pregnancy cause symptoms that may be normal, a sign of urinary tract infection, or a sign of renal dysfunction. Urinary tract infections are also associated with preterm labor. Therefore, careful attention to the history, physical examination, and adjunct measures of urinary function are necessary to adequately care for women with urinary symptoms.

Metabolic Changes

Among the many important endocrine and metabolic changes in pregnancy are changes that occur in the hypothalamus, pituitary, and adrenal glands, often in an interrelated manner. Calcium metabolism and the renin–angiotensin system both exhibit significant alterations in ways that facilitate fetal growth and development.

Thyroid Metabolism

Although all endocrine organs undergo changes in pregnancy, thyroid changes are particularly of note. Because both hypothyroid and hyperthyroid states can adversely affect the fetus, it is critical that a euthyroid state be maintained throughout pregnancy.

The thyroid is the first endocrine gland to appear in the fetus, but the fetus does not start secreting thyroid hormone until approximately 18 to 20 weeks' gestation.[66,67] Consequently, the fetus is dependent on maternal thyroid hormone for the critical metabolic functions fulfilled by this hormone. A woman's thyroid slightly increases in size early in pregnancy and may be palpable on an initial prenatal visit as a smooth and regular-shaped mass. The basal metabolic rate also increases by 20% to 25% during pregnancy.

Several changes occur in the production and transport of thyroxine during pregnancy. The alpha unit of hCG stimulates the thyroid in the same manner as TSH, which causes an increase in total thyroxine (T_4) levels (i.e., subclinical hyperthyroidism). Sensing the increase in total thyroxine, the pituitary reduces production of TSH. At the same time, higher levels of plasma albumin and thyroxine-binding globulin (TBG) bind more thyroxine in serum. Overall, the level of free thyroxine remains normal despite lower levels of TSH and higher levels of total thyroxine (**Figure 19-11**).[67,68]

The TSH level reaches a nadir at approximately 10 gestational weeks.[66-68] Subsequently, as hCG levels decline, TSH levels rise to reach the nonpregnant level by the third trimester and total thyroxine levels decline to a normal value. Plasma values for thyroid function are trimester specific and cannot be determined via one measurement. In general, an assessment of TSH and free T_4 or total T_4 is needed to interpret thyroid function during pregnancy.[66-68]

Glucose and Lipid Metabolism

Glucose metabolism is significantly altered in pregnancy. Glucose is the primary energy source for the fetus and placenta, and is transferred across the placenta via facilitated diffusion. Thus, glucose delivery to the fetus depends on a concentration gradient between the maternal and fetal circulations. The hormone hPL induces maternal insulin resistance and hepatic glucose formation, both of which raise glucose levels in maternal circulation. hPL acts by initiating hyperplasia within the beta cells of the maternal pancreas, which in turn enables greater production of insulin. Hepatic glucose production increases by as much as 30% in the third trimester. Because of the ongoing fetal use of glucose provided by the maternal circulation, a pregnant woman will experience fasting glucose levels that are lower than usual, yet because of the insulin resistance, postprandial glucose levels can be higher.[69]

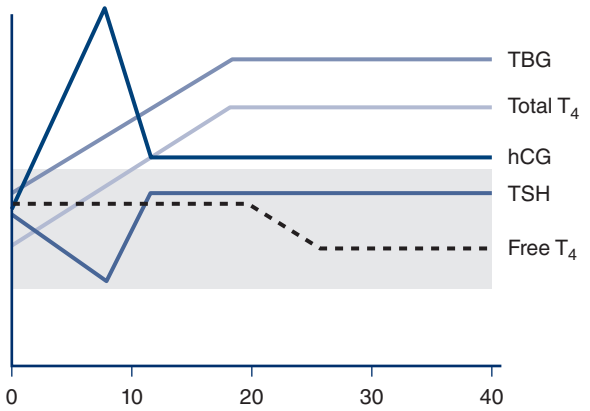

Figure 19-11 The pattern of changes in serum concentrations of thyroid function studies and hCG according to gestational age. The shaded area represents the normal range of thyroid-binding globulin, total thyroxine, thyroid-stimulating hormone, or free T_4 in the nonpregnant woman.
Abbreviations: hCG, human chorionic gonadotropin; T_4, thyroxine; TBG, thyroid-binding globulin; TSH, thyroid-stimulating hormone.
Reproduced with permission from Casey MB, Leveno K. Thyroid disease in pregnancy. *Hil Obstet Gynecol.* 2006;108:1283-1292.[67]

Lower fasting blood glucose levels can worsen nausea and vomiting in early pregnancy but may also contribute to the pregnant woman's enhanced appetite and her need to eat more often. As the placenta increases in size and function, maternal hyperinsulinemia must likewise increase to keep pace. Women who are unable to sufficiently raise their insulin production will develop gestational diabetes in the latter half of pregnancy.

Lipid metabolism is also altered significantly during pregnancy.[70] Body fat accumulates during the first two-thirds of pregnancy, but this growth then stops or declines during the last third of pregnancy. Thus, the pregnant woman is first in an *anabolic* state and then later in a *catabolic* state.[71] During the period of rapid lipolysis, free fatty acids and glycerol are generated. Free fatty acids are converted to ketones, and glycerol is converted to glucose. The free fatty acids are used to synthesize triglycerides, which causes triglyceride levels to increase.[71]

Immunologic Changes

Pregnancy is essentially an immunologic paradox: First, how does the pregnant woman avoid rejecting the fetus given that fetal cells and maternal cells are in direct contact in the maternal spiral arteries and intervillous space? Second, how do the immunologic changes that take place to accommodate the fetus, a semi-allograft, affect the maternal immune response? Immunologic changes in pregnancy are implicated in several important clinical disorders, including recurrent miscarriage, Rh sensitization, and preeclampsia.

The immune response is usually subclassified as either innate immunity or adaptive immunity; the latter is subdivided into two components called cell-mediated immunity and antibody-mediated immunity. The innate immune response is the first line of defense against "non-self" invaders, which includes inflammation and phagocytosis. *Cell-mediated immunity* is responsible for elimination of intracellular microbes and involves several immune lymphocytes, including natural killer (NK) cells and T cells. *Antibody-mediated immunity* involves the production of antibodies by B cells; these antibodies then target extracellular microbes or antigens.

In general, innate immunity is enhanced during pregnancy, whereas adaptive immunity is less

Table 19-5 Changes in the Immune System During Pregnancy

Primary Host Defense Mechanisms
Increased number of white blood cells (primarily polymorphonuclear leukocytes), which enhances the pregnant woman's nonspecific immune response.
Delayed chemotaxis (the movement of phagocytes to the site of foreign invasion), which may delay the maternal response to infection.
Decreased number of natural killer cells, which may delay the maternal response to infection.
Reduced levels of plasma IgG. The hemodilution of pregnancy and passive transfer of Ig antibody to the fetus reduces maternal blood levels of IgG.
Cell-Mediated Immunity
Although the overall number of lymphocytes remains unchanged, there is a decreased number of T-helper cells (CD4 cells) relative to the number of T-suppressor cells (CD8 cells).
With fewer CD4 cells, the B-cell function may be slightly impaired.
Antibody-Mediated Immunity
Overall, the antibody-mediated response is unchanged.
Clinical Implications
Small increase in risk for developing gram-negative organism infections and mycotic or fungal infections
Increased morbidity from gram-negative infections, H1N1 flu virus, and varicella if infection occurs
Increased infectivity with certain pathogens, including herpes simplex virus, poliovirus, cytomegalovirus, malaria, and hepatitis
Changes in autoimmune disease characteristics (rheumatoid arthritis often improves during pregnancy, while systemic lupus erythematosus is more likely to flare)

Abbreviation: IgG, immunoglobulin G.

functional. In particular, cell-mediated (T helper 1) responses are somewhat suppressed compared to antibody-mediated (T helper 2) responses, which are more responsive. This change in the cell-mediated and antibody-mediated responses is referred to as a *Th1 to Th2 shift*.[71-73] These changes increase the risk of maternal infection, especially with regard to viral infections such as influenza or varicella. At the same time, the Th1 to Th2 shift results in improvement of some autoimmune disorders such as rheumatoid arthritis.

Chemotaxis is delayed in the pregnant woman, which can delay the maternal response to some infections. The total white blood cell count is elevated largely due to increased numbers of polymorphonuclear neutrophils, monocytes, and granulocytes. Many of the bioactive agents produced by the fetus and placenta effect subtle shifts in maternal immunity. Although little overall change occurs in the maternal immune response, subtle changes in each of the three types of immunity have important clinical implications (Table 19-5).

The Fetus as an Allograft

The first part of the pregnancy immunologic story occurs during implantation and is not yet fully understood. In simple terms, trophoblastic tissue does not express the cell membrane proteins that would stimulate an innate or cellular immune response. The mechanisms by which the fetus eludes detection as a foreign object are quite complex.[72] In addition, changes in the cell-mediated immune response result in a concentration of NK cells in the decidua; these cells, however, are a variant of NK cells that have minimal cytotoxic abilities but a refined ability to control trophoblast invasion and remodel uterine vasculature. T cells and B cells become scarce in the uterine environment during this period.

Fetopelvic Relationships

The fetus can lie in numerous positions in relationship to the maternal abdomen pelvis. A few of these positions preclude a vaginal birth; others are associated with a longer labor. For this reason, it is important to know all possible fetopelvic relationships and their clinical significance. The terminology used to describe fetopelvic relationships is listed in Table 19-6.

Table 19-6 Fetopelvic Relationships

Term	Definition
Asynclitism	Oblique presentation of the fetal head. When the fetal head is tilted laterally toward the fetal shoulder, the biparietal diameter is not parallel to the planes of the pelvis. The sagittal suture will not be palpable as midway between the front and back of the pelvis. Asynclitism is called anterior when the anterior parietal bone is the point of presentation; it is called posterior when the posterior parietal bone is the presenting part of the fetal head.
Attitude	Relation of fetal parts to each other. The basic attitudes are flexion and extension. The fetal head is flexed when the chin is close to the chest; it is extended when the occiput is closer to the cervical spine.
Cephalic Prominence	Fetal part of head most easily felt (prominent) during Leopold's maneuvers and used to determine attitude. When the cephalic prominence is felt on the same side as fetal small parts, the head is flexed; when on the same side as fetal back, the head is extended. When the cephalic prominence is not palpable on one side or the other, it often is called a military attitude.
Denominator	An arbitrarily chosen point on the presenting part of the fetus that is used to describe fetal position. The denominator for a vertex presentation is the occiput; for a breech presentation, it is the sacrum. The denominator of a face presentation is the mentum or chin.
Engagement	The point at which the widest diameter of the presenting part is at or below the pelvic inlet.
Lie	Relationship of the long axis of the fetus to the long axis of the pregnant woman. The three possible lies are longitudinal, transverse, or oblique.
Position	Relationship of the denominator to the front, back, or sides of the maternal pelvis.
Presentation	The part of the fetus that presents first to the maternal pelvis. The three possible presentations are cephalic, shoulder, and breech. Breech presentations are further subdivided based on presentation of the buttocks or feet.
Presenting part	The most dependent part of the fetus that is closest to the maternal cervix.
Station	The number of centimeters above or below the plane between the ischial spines of the presenting part. The ischial spines are designated as 0 station; the centimeters above the spines are –1, –2, –3, –4, and –5; and the centimeters below the ischial spines are +1, +2, +3, +4, and +5, which is when the fetal presenting part is visible at the vaginal introitus.

The Fetal Skull

The fetal skull is composed of five bones—two frontal bones, two parietal bones, and one occipital bone—that may be palpated during labor to identify the position of the fetus and assess labor progress. In addition, the two temporal bones are located inferior to the parietal bones on each side but are not involved in the anatomic markers important during labor assessment (Figure 19-12). The bones meet at the frontal suture, located between the two frontal bones; at the sagittal suture, located between the two parietal bones; at the two coronal sutures, where the parietal and frontal bones meet on either side of the head; and at the two lambdoid sutures, where the parietal bones and the upper margin of the occipital bone meet on either side of the head.

Two fontanels—areas formed by the meeting of sutures—are found on either end of the sagittal suture. The anterior fontanel is the largest, formed

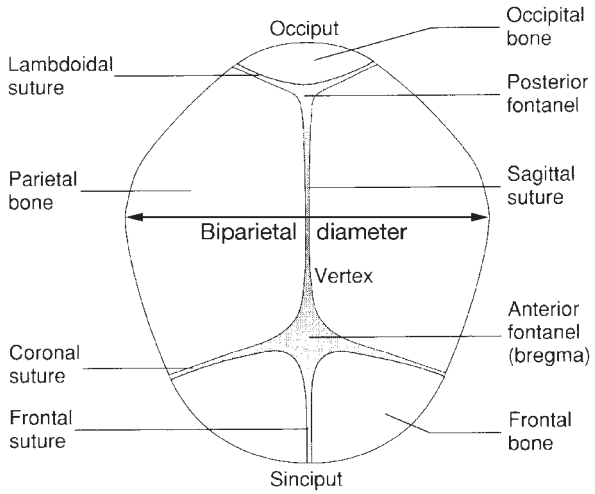

Figure 19-12 Fetal skull: landmarks, bones, fontanelles, sutures, and biparietal diameter.

as the frontal, sagittal, and two coronal sutures come together in a diamond-like shape.

The four sutures can be palpated coming from the four corners of diamond shape, with markers of the frontal suture being more subtle and sometimes difficult to palpate. The sagittal and two lambdoid sutures meet to form the posterior fontanel in a triangle shape; the sagittal and lambdoid sutures can be palpated from the three corners of the triangle.

The fetal head also has several diameters of importance in providing maternity care, which are shown in Figure 19-12 and **Figure 19-13**.

Lie, Presentation, Denominator, and Position

Determination of the lie, presentation, and position of the fetus require an understanding of terms and the anatomic landmarks of the fetal skull in relation to the maternal pelvis.

Lie is the relationship of the long axis of the fetus to the long axis of the pregnant woman. Three possible lies are longitudinal, transverse, and oblique (**Figure 19-14**). Transverse and oblique lies in labor are abnormal conditions requiring collaboration with or referral to a physician because they will likely necessitate cesarean section.

Presentation is determined by the presenting part—that is, the part of the fetus that first enters the pelvic inlet. The three possible presentations are cephalic, breech, and shoulder. Cephalic and breech presentations are each further subdivided: A cephalic presentation can be vertex, sinciput, brow, or face (**Figure 19-15**), and a breech presentation can be frank (legs extended), full/complete (legs flexed), or footling (single or double). Approximately 3.0% to 3.5% of women enter labor with a breech presentation and 0.5% with a face presentation. Approximately 0.5% of women enter labor with a shoulder presentation. The midwife collaborates with a physician in the management of women with a noncephalic presentations.

The *attitude* of the fetus is its characteristic posture, determined by the relationship of the fetal parts to one another and the effect this has on the fetal vertebral column. The attitude of the fetus varies according to its presentation. For example, a fetus in a vertex presentation has a well-flexed head, flexion of the extremities over the thorax and abdomen, and a convex curved back. By comparison, the straight upright attitude of a fetus with a sinciput presentation has resulted in the classically defined military attitude. Finally, a fetus with a face presentation has an acutely extended head, flexion of the extremities on the thorax and abdomen, and a vertebral column that is arched to some degree.

Fetal *position* is named using three letters in the following order: the first reference is to the side of the maternal pelvis (**L**eft or **R**ight); the second reference is the denominator (**O**cciput, **S**acrum, or **M**entum); and the third reference is where in the maternal pelvis the denominator lies (**A**nterior, **T**ransverse, **P**osterior). These designations serve as a shorthand description for describing the lie, presentation, and position of the denominator within the circle of the pelvis (**Figure 19-16**). For example, the designation LOA indicates that the lie is longitudinal, the

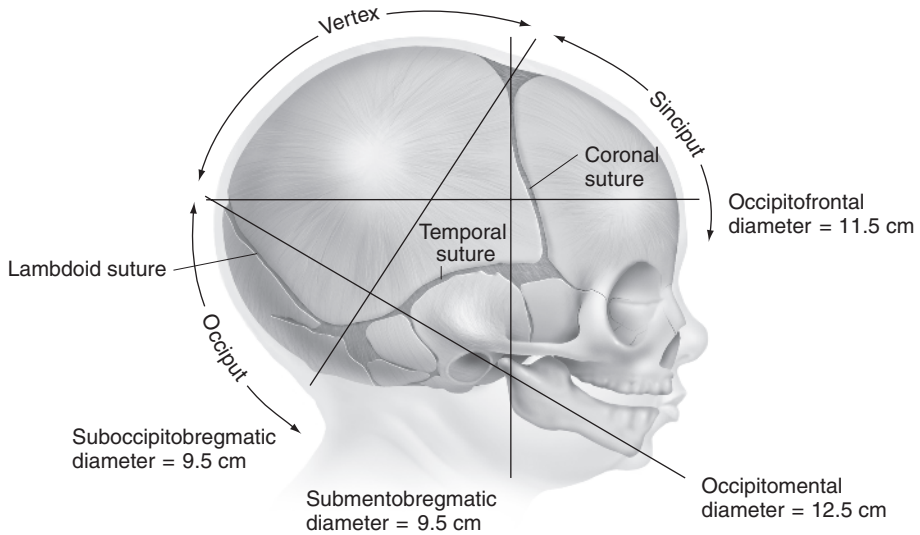

Figure 19-13 Average diameters of the full-term fetal head.

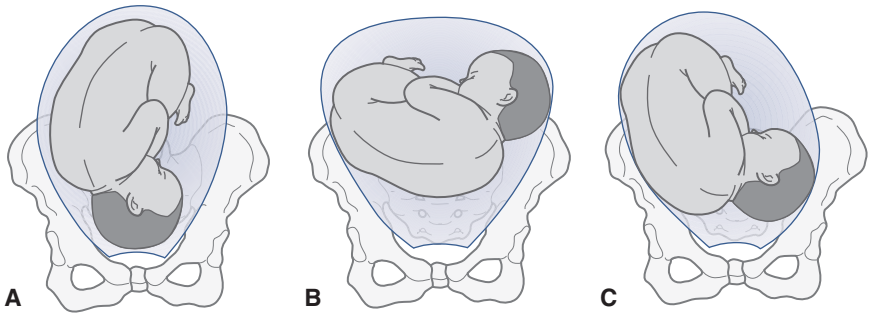

Figure 19-14 Lies. **A.** Longitudinal. **B.** Transverse. **C.** Oblique.

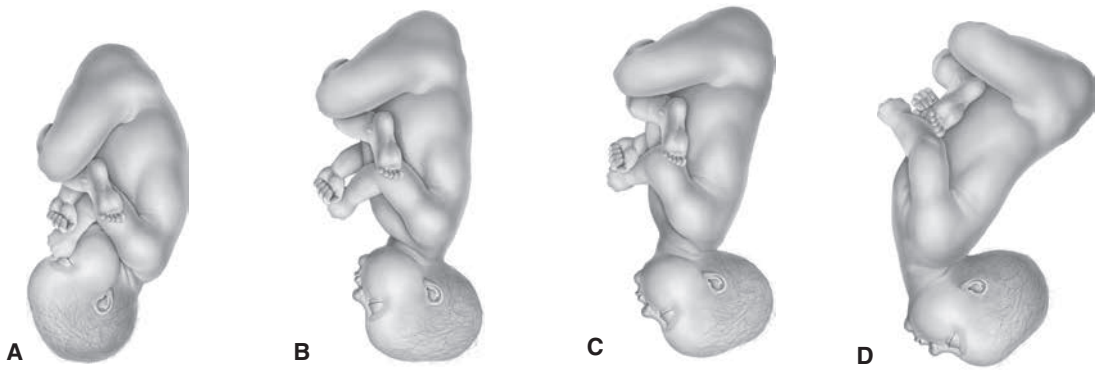

Figure 19-15 Attitude of the fetus in various presentations. **A.** Vertex. **B.** Sinciput (military). **C.** Brow. **D.** Face.

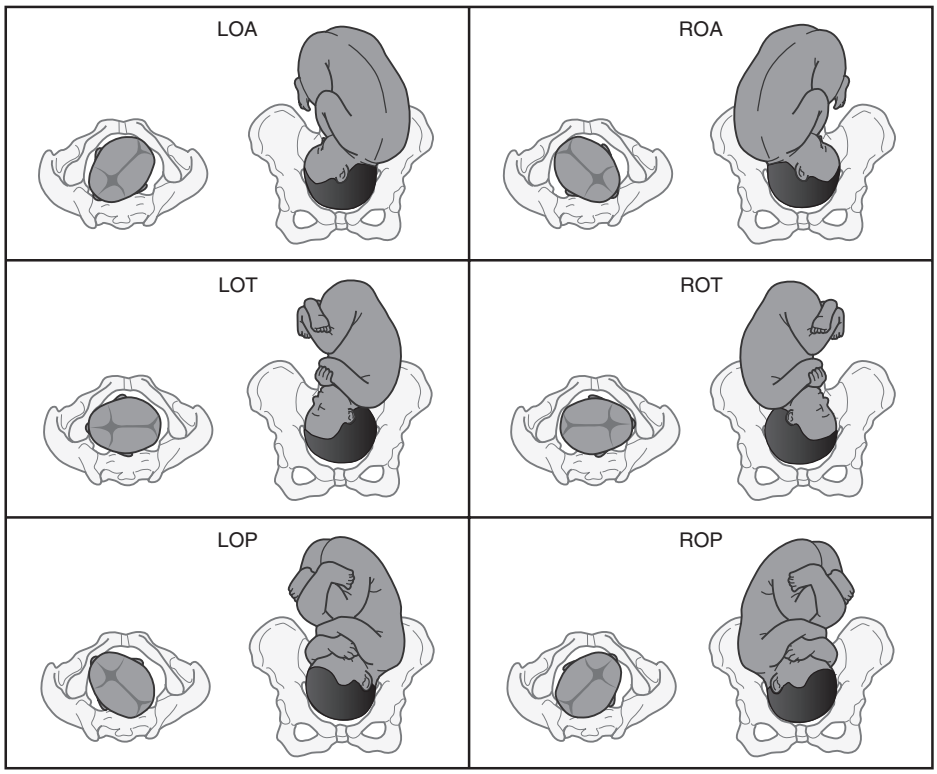

Figure 19-16 Fetal position for occiput presentations.
Abbreviations: LOA, left occiput anterior; LOP, left occiput posterior; LOT left occiput transverse; ROA right occiput anterior; ROP, right occiput posterior; ROT, right occiput transverse.

presentation is cephalic, and the denominator, which is the occiput, is in the anterior portion of the left side of the pelvis. The possible fetal relationships to the maternal pelvis for each lie and presentation are summarized in Table 19-7.

Vertex is the presentation most often associated with a longitudinal lie; it has an incidence of approximately 95%. Approximately two-thirds of all fetuses will be positioned with the occiput in the left side of the maternal pelvis (LOA, LOT, LOP) by the last month of pregnancy; one-third will be positioned with the occiput in the right side of the woman's pelvis (ROA, ROT, ROP). Because the head usually enters the inlet with the occiput directed to the transverse portion of the maternal pelvis, the most common position of the fetus at the onset of labor is left occiput transverse (LOT).

In a vertex presentation, the fetal head usually enters the pelvis with the biparietal diameter parallel to the plane of the pelvis. If the fetal head is tilted laterally toward the fetal shoulder, the head will enter the pelvis at an oblique angle which is referred to as *asynclitism*.

Estimated Fetal Weight

When assessing fetopelvic relationships, a midwife is also able to estimate a fetal weight (EFW). The average EFW at 30 gestational weeks is approximately 3 pounds or 1360 grams. A fetus usually reaches 4 pounds at approximately 33 weeks (1900 grams); and at 37 weeks it is 5 pounds 8 ounces or 2500 grams. The latter is important since it is the international definition for low birth weight. Fetal weight is important for fetopelvic relationships, since a small fetus (either constitutionally or due to disease) is more likely to present abnormally such as in a breech presentation. Conversely, a large fetus can have difficulty navigating even an average-sized pelvis.

Table 19-7 Possible Fetal Relationships to the Maternal Pelvis for Each Lie and Presentation

Lie	Presentation	Denominator	Designation for Position
Longitudinal	Cephalic		
	Vertex	Occiput	ROA LOA
			ROT LOT
			ROP LOP
	Sinciput	Sinciput (bregma, anterior fontanel)	Sinciput and brow presentations usually convert to either a vertex or a face presentation.
	Brow	Brow	
	Face	Mentum (chin)	RMA LMA
			RMT LMT
			RMP LMP
	Breech		
	Frank	Sacrum	RSA LSA
			RST LST
			RSP LSP
	Full/complete	Sacrum	Same as frank presentation
	Footling	Sacrum	Same as frank presentation
Transverse	Shoulder	Acromion	RAA LAA
			RAP LAP
			A transverse variety is not possible.
Oblique	With an oblique lie, the midwife will feel nothing at the inlet. There is no presentation, position, or variety associated with an oblique lie, which is usually a transitory condition.		

Abbreviations: LMA, left mentum anterior; LMP, left mentum posterior; LMT, left mentum transverse; LOA, left occiput anterior; LOP, left occiput posterior; LOT left occiput transverse; LSA, left sacrum anterior; LST, left sacrum transverse; ROA, right occiput anterior; ROP, right occiput posterior; ROT, right occiput transverse; RSA, right sacrum anterior; RSP, right sacrum posterior; RST, right sacrum transverse; RMA, right mentum anterior; RMP, right mentum posterior; RMT, right mentum transverse.

Conclusion

Remarkable physiologic changes occur during pregnancy. Knowledge regarding physiologic pregnancy changes must inform every aspect of pregnancy care. A deep understanding of these changes will allow the midwife to interpret signs and symptoms accurately, enabling reassurance and guidance regarding managing common pregnancy discomforts or initiation of assessment for pathology. These are essential steps in the provision of quality maternity health care.

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