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IN YOUR formal training, you were intensively exposed to the challenging area of obstetrics and gynecology (OB/GYN). The knowledge you gained from learning new concepts and procedures was augmented by heavy immersion in OB/GYN realtime imaging. This course will not attempt to repeat or add to the visual knowledge, which cannot be separated from the hand and eye coordination of the sonographer. Instead, we will clarify and slightly expand your mental knowledge of the various areas in OB/GYN sonography.

This volume is divided into three units. Unit 1 covers general gynecological information, specifically focusing on the most commonly encountered pelvic abnormalities. Most of these abnormalities are grouped by structural differences. Unit 2 provides a brief overview of the events in the female reproductive system after fertilization up to the end of the first trimester. Abnormalities that may affect the normal early pregnancy are briefly discussed. The final unit touches on obstetrics. Concepts and principles concerning the sonographer's approach to imaging second and third trimester pregnancies are clarified and, in some areas, expanded.

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This volume is valued at 12 hours and 4 points.

NOTE:

In this volume, the subject matter is divided into self-contained units. A unit menu begins each unit, identifying the lesson headings and numbers. After reading the unit menu page and unit introduction, study the section, answer the self-test questions, and compare your answers with those given at the end of the unit. Then complete the unit review exercises.

Please read the menu for Unit 1 and begin. ➔

Unit 1. Gynecological Sonography

GYNECOLOGY IS THE study of normal function and diseases of the female reproductive system. Because most of that system (along with other systems) is located in the pelvis, sonography of the female reproductive system is frequently referred to as pelvic sonography. In this unit we will examine the anatomy and function of the female pelvis, focusing on the reproductive structures. We will then take a close look at pelvic sonography of the ovaries and uterus. Some of the most common abnormalities of these structures will also be outlined.

1–1. Pelvic Anatomy and Physiology

The pelvis and its organs constitute a significant portion of the female body. Not only does the female pelvis contain much of the large bowel but it also contains nearly all of the reproductive organs and half of the urinary system. In this section we will discuss the structure and physiology of the female reproductive system *within the female pelvis*. Because of the complexity of the female reproductive system, we will briefly cover topics common to most sonographers. This broad coverage should provide you with the necessary background for performing accurate sonographic images of the female pelvis.

401. Pelvic anatomy

The female pelvis is orientated at an obtuse angle to the long axis of the spine; that is, its anterior portion (the symphysis pubis) is lower than its posterior section (the sacral promontory, or the point where the 5th lumbar vertebra meets the superior edge of the sacrum). An imaginary axial plane from the symphysis pubis to the sacral promontory forms the superior border of the true pelvis. Apart from the individual bones that make up its boundaries, the pelvis is bordered with muscle. On the lateral margin, the *obturator internus* muscles form the pelvic wall. Posteriorly and extending to either side from the sacrum lies the *piriformis* muscles.

The final border is inferiorly located, the floor of the pelvis. The muscle that forms the floor is the *levator ani* muscle. The levator ani is a compound muscle formed by the *pubococcygeus* (anteriorly) muscle and the *iliococcygeus* (posteriorly). Another floor muscle stretching from the ischial spine posteriorly and attached to the sides of the coccyx is the *coccygeus*. The floor muscles support pelvic organs, provide resistance to the downward force of intraabdominal pressure from actions such as coughing or defecation, and provide for contraction and relaxation of the rectum, urethra, and vagina.

Ovaries

The most important organs in the female pelvis are the ovaries. Like male testes, ovaries are gonads, or organs that produce reproductive (sex) cells. Ovaries are located lateral to either side of the uterus in the space sonographers call the adnexa. They are suspended in this area by two ligaments. The *suspensory ligament* (or infundibulopelvic ligament) attaches to the lateral wall of the pelvis and the superior pole of the ovary. It contains ovarian blood vessels and nerves. The other ligament, called the *ovarian ligament*, anchors the ovary to the superolateral corner of the uterus. You should realize that

these two ligaments are flexible enough to allow the ovaries to be in any location of the adnexa. Commonly, in fertile women who have not had children (nulliparous), the ovary is lodged within a triangular depression called the *ovarian* fossa. This fossa is located in the broad ligament close to the pelvic wall and anterior to the internal iliac artery but inferior to the external iliac vein. The section of broad ligament between the ovary and the uterine tubes is called the mesovarium, which is continuous with the hilum of the ovary. The hilum is the opening of the ovary through which passes blood vessels, lymphatics, and nerves.

The ovaries are oval-shaped organs which vary in size as a female ages and, secondarily, as hormonal status changes. Thus, as we discuss the anatomical size, we will briefly mention some of the physiological activities influencing the ovary. Ovarian physiology will be covered more in depth in the next lesson. Ovarian size, based on age, is classified under three periods in a female's life:

- Prepubertal, or before puberty (which can occur anywhere from 8 to 16 years of age).
- Premenopausal, or the period when menses occurs cyclically at regular 4-week intervals. This period is often referred to as the reproductive period.
- Postmenopausal, or the period after menses has stopped, usually around 50 years of age (menopause).

In children, physicians frequently assess the size of the ovaries in terms of volume. This is largely because the size and shape of the ovaries vary considerably from about 5 years of age to the start of menses. Thus, measuring volume rather than linear dimensions (length, width, and height) alone allow better reproducibility; that is, accurate measurements of the ovaries can be constantly obtained from sonographer to sonographer and despite compression of the ovaries from a full bladder or mass. Sonographers obtain ovarian volumes using the formula for an elliptical (oval) shape:

$$V = d_1 \times d_2 \times d_3 \times 0.523$$

where *V* is volume and *d* represents the maximum lengths of the three ovarian dimensions—length, width, and height. Ovaries in children can grow from less than 1 cubic centimeter (cc or cm^3) up to approximately 4 cc in 13 year-olds. All three linear dimensions of most pediatric ovaries rarely exceed 2 centimeters (cm).

For women of reproductive age (roughly 15–55) the ovarian size, obtained through three linear dimensions, is dependent on hormonal and pregnancy status. For women who have never been pregnant, the ovarian size is approximately 3 cm × 2 cm × 2 cm (L × W × H, respectively). In women who have delivered a child or children, the ovaries are slightly larger at approximately 5 cm × 3 cm × 2 cm (L × W × H, respectively). In post-menopausal women, from about 50 years of age, ovarian size usually decreases with each decade to a volume similar to that of premenstruating girls. This decrease is due to atrophy of the ovarian tissue.

A single layer of cells called the germinal epithelium covers the surface of the ovary. At one time it was thought that sex cells, or oocytes, arose from this layer (hence the name, "germinal"); but now we know cells that develop into ova arise from a layer of the yolk sac (endoderm) and migrate (travel) to the ovaries within the embryo. At birth, the female has 1 to 2 million oocytes in the ovaries, most of which die off (atresia) steadily until puberty. Thus, before a female is born, all of the primary oocytes destined to mature are in place within follicles. From birth to puberty, the primary oocytes are in a suspended state of development. At puberty, 200,000 to 300,000 primordial (primitive) follicles, containing primary oocytes, exist within the ovaries. Of these, only approximately 400 to 500 of these oocytes will ever be released (ovulation) during a woman's reproductive years. When puberty occurs, development continues in up to a dozen oocytes each month until one clearly outgrows the others. This mature follicle will be the one to undergo ovulation.

Just beneath the germinal epithelium covering the ovary is a dense layer of connective tissue called tunica albuginea. The tissues directly beneath this tunica are functional cells, or parenchyma, of the

ovary, composed of two layers: the cortex and medulla. Most of the ovarian parenchyma is cortex, where oogenesis takes place. The cortex is made of follicles surrounded by dense connective tissue (stroma). Near the hilum of the ovary, the medulla is made of looser connective tissue that contains most of the vascular structures as well as nerves and lymph channels.

Uterus, cervix, and vagina

Apart from the ovaries, other accessory structures play a part in reproduction. They are the uterus, cervix, and vagina. These three structures form a continuous pathway from outside the body into a point within the pelvis just above the ovary.

Uterus

The uterus is located centrally within the pelvis, between the bladder anteriorly and the rectum posteriorly. The uterus is literally suspended within the center of the pelvis by a double fold of peritoneal membrane called the broad ligament. Extending from the lateral sides of the uterus to the lateral wall of the pelvis, the broad ligament houses the adnexal structures within its two layers. Two round ligaments also suspend the uterus, superiorly and laterally. From both corners of the uterus, anterior but below where the uterine tubes begin, the round ligaments extend anteriorly and up through the inguinal canal to attach to an area in the anterior wall of pelvis.

The uterus is a hollow, tube-shaped, muscular organ divided into three major parts: the fundus, body, and cervix. The fundus is the rounded projection at the superior end of the uterus. The uterine tubes extend from either side of and below the fundus. The body of the uterus is the main portion of the uterus. Hollow and containing the functional tissue, the body extends below the level of the uterus' upper corners (*cornua* or *uterine horns*) and tapering down to a narrow constrictive area known as the isthmus. Immediately inferior to and continuous with the isthmus is the cervix.

The uterus is oriented within the pelvis according to either of two basic positions: version or flexion. Version is a tilting of the entire uterus and cervix forward (anteversion), backward (retroversion), or to the side of midline (lateroversion). With an empty bladder, the uterus normally conforms into an anteverted position, causing the cervix to form a 90-degree angle with the long axis of the vagina. The other basic position is flexion, where the uterus is bent either forward (anteflexion) or backward (retroflexion) at the isthmus with the cervix.

Anterior and posterior to the uterus are empty spaces called cul-de-sacs. The anterior space between the bladder and the uterus, lined with peritoneum, is called the *vesicouterine pouch*. The space is normally empty but occasionally loops of bowel may be found within it. The space between the posterior surface of the uterus and the anterior rectum is called the *rectouterine pouch*, often known as the pouch of Douglas.

Apart from the ovaries, the other adnexal structure within the broad ligament is the *uterine tube* extending from the cornua areas. Uterine tubes (also called fallopian tubes, oviducts, or salpinges) are two 7–12 cm hollow tubes that course in a tortuous or coiled manner out toward the ovaries. They have four major divisions:

1. Interstitial. The narrowest portion of the tube located entirely within the muscle of the uterus and opening directly into the uterine cavity. The interstitial region is a few centimeters in length.
2. Isthmus. Just over a few centimeters in length and extending straight beyond the lateral uterine wall.
3. Ampulla. More winding and tortuous than the isthmus segment, this portion of the uterine tube is 5–8 cm in length.
4. Infundibulum. Shaped like a funnel with an opening (ostium) of about 3 mm (millimeter) in diameter, the infundibulum has fringes (fimbria) that extend over the ovary.

The uterine tubes contain the same three layers of tissue as the uterus, which we will discuss below. The important thing to remember about the inner lining, which is mucosal, is that it is continuous with the peritoneal membrane lining the pelvis wall and organs. Thus, the inside of the uterine tubes open directly into the abdominal cavity.

The three main layers of the uterus are, from outer to inner, the serosa, the myometrium, and the endometrium. The serosa is the outermost coat or layer of the uterus, made of peritoneum and fibrous connective tissue. The layer beneath the serosa is the myometrium, itself made of three layers of smooth muscle and connective tissue: a thin outer layer, a thick intermediate layer, and a thin inner layer. Overall, the myometrium is thickest at the level of the fundus and thinnest at the level of the cervix. The innermost layer is the endometrium, a mucous membrane lining the inside of the uterus. The endometrium has two layers: the *basal layer*, or *decidua basalis*, a thin layer that connects the deep endometrium to the myometrium; and the *functional* layer, or *decidua functionalis*, is a relatively thicker layer that contains glands and capillaries supported by loose connective tissue (stroma). The functional layer is shed every month and regenerated from the basal layer after menses. The endometrium lines the entire cavity of the uterus, which is flat and triangular in shape (fig. 1–1).

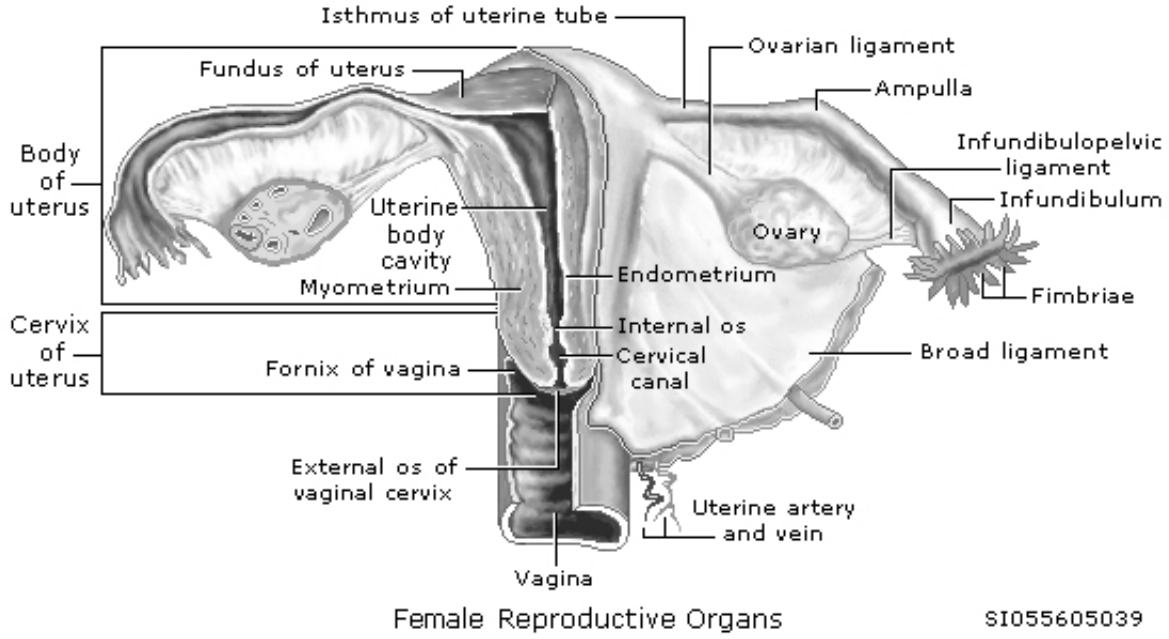

Figure 1–1. Reproductive organs.

Uterus size typically changes with age and pregnancy status. In children, the uterus is tubular shaped with a length from 2 cm in neonates up to approximately 4 cm just before puberty. From 2 years old to 10 the uterus is generally 3.5 cm in length. After puberty the size of the uterus grows above 5 cm in length and forms a shape similar to that of a pear. The nonpregnant adult uterus is typically 7–8 cm in length, 3–5 cm in width, and 3–4 cm in height (anteroposterior [AP] diameter). After the first pregnancy, the typical uterine size increases a few centimeters in all dimensions. With the ceasing of menses (menopause), the uterus begins to atrophy down to a size comparable with that of a child.

Typical Uterine Sizes in Centimeters		
Age	***Nulliparous (never pregnant)***	***Parous (one or more pregnancies)***
Premenstrual (before puberty)	(L = 2 – 4) x (W < 2.5) x (AP < 2)	
Puberty and adolescence	(L = 5 – 7) x (W = 2.5 – 3) x (AP = 2 – 3)	
Adult	(L = 7 – 8) x (W = 3 – 5) x (AP = 3 – 4)	(L = 8 – 11) x (W = 4 – 6) x (AP = 5 – 6)
Postmenopausal	(L = 3 – 7) x (W = 2– 4) x (AP 1.7 – 3)	

Cervix and vagina

The lowest portion of the uterus is called the cervix, which extends into a tubular canal called the vagina. The superior boundary of the cervix is called the internal os, which is identical to the isthmus of the uterus. The inferior boundary is the external os, which opens into the vagina. The walls of the cervix contain the same muscle layers as the body of the uterus, but thinner. However, unlike the body of the uterus, the cervix has no serosa. As with the cavity of the uterus containing endometrium, the canal within the cervix (cervical canal) also contains a mucous membrane called the endocervix.

The vagina is a tubular structure attached to the uterus above the level of cervix. Thus, the vaginal walls envelope the cervix, forming spaces on all sides called fornices. The anterior and lateral fornices are shorter than the posterior fornix. This fact makes the typical adult, anterior vagina wall 5–7 cm in length compared to its posterior wall length of 7–10 cm. The walls are made of elastic fibromuscular tissue, which contain an inner lining made of a mucous membrane. When relaxed, muscles of the walls form folds called rugae. In females who have not had sexual activity through the vagina, the external opening of the vagina may be partially covered by connective tissue called the hymen. A totally closed vaginal opening is called an imperforate hymen and occurs usually before puberty. In order for normal menses flow to occur the hymen must be perforated.

Vasculature

The reproductive organs are supplied directly by two main artery branches, the uterine artery and the ovarian artery. Coursing roughly parallel and next to these arteries are corresponding veins. Vascular anatomy of the pelvis varies from person to person, but a general pattern exists in most people.

Ovarian vessels

The ovaries are supplied primarily via the ovarian arteries, which branch directly off the anterolateral surface of the abdominal aorta slightly inferior to the level of the renal arteries. The ovarian arteries cross the common iliac vessels as they enter the pelvis. Coursing medially along the infundibulopelvic ligaments (suspensory ligaments), the arteries enter their respective ovaries through the hilar slits along with nerves and lymph channels. The ovarian artery also has branches that feed the uterine tubes and portions of the broad ligament.

Also, veins run through each ovarian hilum. The right ovarian vein courses from the ovary parallel with the suspensory ligament and the ovarian artery. It drains directly into the anterior inferior vena cava (IVC). Conversely, the left ovarian vein continues superiorly and drains into the left renal vein.

Uterine vessels

The uterine artery courses off the anterior branch of the internal iliac artery. Running to either side of the cervix, the uterine arteries turn in a cephalic direction and course along the lateral edges of the uterus to the cornua. At the cornua, the uterine arteries branch, feeding portions of the broad ligament, and the uterine tubes, as well as connecting (anastomosing) with ovarian arteries entering ovarian hila. Both uterine arteries lateral to the uterus also send branches into and across the body of the uterus. These arteries, which run parallel with the surface of the uterus and within the myometrium, are called arcuate arteries. Branching off the arcuates to penetrate deeper into the uterine myometrium are the radial arteries. The radial arteries become straight arteries that feed the basal layer of endometrial tissue. At this point, the straight arteries branch off coiled vessels called spiral arteries, which feed the functional layer and glands of the endometrium. The veins of the uterus parallel the arteries and are paired closely.

402. Pelvic physiology

The functioning of the female reproductive system and its effect on anatomy is of considerable complexity. Thus, for the sonographer, a basic understanding of pelvic physiology can only help you to obtain quality sonographic images in this challenging area. In this lesson, we will briefly touch on a broad range of pelvic physiology and discuss some of the techniques applied by physicians for

abnormal functions. Being familiar with general pelvic physiology will prepare you for the rigorous imaging of abnormal pathology you are certain to encounter.

Ovarian and uterine functions

As previously stated, the ovaries are the principal reproductive organs in the female. Their primary function is to produce the sex cells, called gametes or ova. Secondarily, the ovaries produce the hormones estrogen and progesterone. The hormones help produce and maintain female characteristics, prepare the endometrium for implantation of a fertilized ovum, and develop the mammary glands.

The uterus serves as the pathway for sperm to reach secondary oocytes that travel along the uterine tubes. The fertilized ovum is swept through the tubes into the cavity of the uterus. The endometrium serves as the site of implantation for the fertilized egg. If fertilization does not take place the endometrium becomes the source for menstrual flow, which is the monthly sloughing off and bleeding of the endometrial lining. The final function of the uterus, perhaps its main function, is to serve as the environment for the developing embryo and fetus until delivery.

Reproductive cycle

During the monthly cycle, events occur simultaneously within the ovary (further development of oocytes and subsequent ovulation) and within the uterus (menstruation). For our purposes it will be easier to consider these functions separately.

Hormonal control

The reproductive (sometimes called the menstrual) cycle typically spans every 28 days. Low levels in the blood of a particular type of estrogen, estradiol, drives the cycle. Estradiol matures and maintains the reproductive organs and sex characteristics as well as prepares the uterus for implantation. Low blood levels of progesterone also affect the cycle. Progesterone plays a major role in preparing the uterus for implantation. The hypothalamus gland, located within the brain, detects a drop in the levels of estrogen and progesterone in the blood, which prompts it to release a hormone called gonadotropin-releasing hormone (GnRH). This hormone causes the anterior portion of the attached pituitary gland to secrete two sex hormones: large amounts of follicle-stimulating hormone (FSH) and a relatively small amount of luteinizing hormone (LH). FSH causes the follicles of the ovaries to grow, especially the dominant follicle, and to increase the secretion of estrogen. LH causes the final stage of maturation of a dominant follicle, stimulates the follicle to begin secreting progesterone, causes the release of an ovum (ovulation), and helps convert the ruptured follicle cells into the corpus luteum. However, production of these hormones occurs at different levels and at different times throughout the cycle, affecting the ovary and uterus differently. A certain level of estrogen and progesterone also has the affect of suppressing FSH and LH production.

Ovarian cycle

The ovarian cycle is divided into two separate periods: the follicular phase and the luteal phase. The follicular phase involves the first 14 days of the cycle preovulation, and the luteal phase involves the post-ovulation 14 days.

Follicular phase of the ovarian cycle

Between day 1 (the start of endometrial menstruation or blood flow) and day 13 of the reproductive cycle, FSH causes the growth of approximately 8 to 12 follicles, which secrete estrogen. One of the follicles outgrows the others and becomes mature. The excessive amount of estrogen production acts as a feedback on the pituitary and causes the reduction of FSH. This, in turn, causes the other follicles to degenerate (atrophy). The follicle that continues to grow larger and secrete estrogen is referred to by various names such as vesicular ovarian follicle, dominant or mature follicle, and occasionally, the graafian follicle. This dominant follicle reaches a size of 1.5 cm and migrates close to the surface of the ovary before rupturing on day 14. The rupture event (ovulation) is caused by a surge in LH, which

occurs at the highest point of estrogen production. Ovulation causes the stored secondary oocyte to be released into the peritoneal cavity and immediately swept up into the opening of the uterine tubes. Not only is the oocyte released from the dominant follicle but also the fluid, which then settles into posterior cul-de-sac between the uterus and rectum (fig. 1–2).

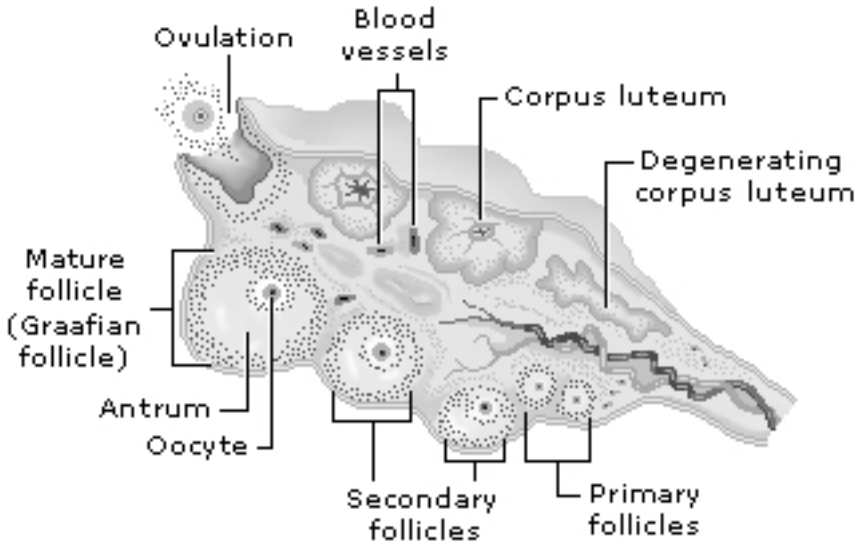

Figure 1–2. Ovarian follicle development.

Luteal phase of the ovarian cycle

After ovulation, the cells in the ruptured follicle wall collapse and begin, with the help of LH, to transform into lutein cells, which expand and become yellowish (thus 'luteal') while secreting progesterone and some levels of estrogen. This process is controlled largely by the increase in LH in the blood, which lasts about seven days. The collapsed mass, or body, of yellow cells is called the corpus luteum. The corpus luteum continues to secrete hormones, which prepare the endometrial lining for implantation of a fertilized egg. The corpus luteum secretes a particular hormone (inhibin) that, along with progesterone and estrogen, inhibits the anterior pituitary gland from producing FSH and LH, levels of which begin to drop in the blood. This low blood level of FSH and LH causes the corpus luteum to degenerate (involute), a process that is completed at approximately day 26 of the cycle. As the corpus luteum degenerates, secretion of progesterone and estrogen also decrease until they cease altogether at complete involution. The absence of the hormones causes the uterus to menstruate, as explained below. Further, the sudden reduction of progesterone and estrogen in the blood provides feedback to the anterior pituitary gland, prompting the production of FSH and LH and beginning the cycle again.

Endometrial cycle

While the ovary goes through its physical and hormonal changes during the cycle, the endometrium also undergoes changes in response to the hormones produced. The two phases of the ovarian cycle correspond with the phases of the endometrium. The ovarian follicular phase (days 1–14) occur simultaneously with the proliferative phase of the endometrium. The ovarian luteal phase after ovulation (days 15–28), occur with the secretory phase of the endometrium.

Proliferative phase of the endometrial cycle

The proliferative phase (sometimes considered as starting on day 6 of the cycle) begins with the menses on day 1. That is, if pregnancy does not occur, the endometrial tissue is shed over a 5-day period. During this time, the absence of progesterone and estrogen causes the anterior pituitary to begin producing FSH and LH. As stated above, the resulting growth and development of the ovarian

follicles causes them to secrete increasing amounts of estrogen. It is the rising levels of estrogen that, after the 5-day menses, causes new glands within the endometrium to develop and grow, or proliferate. As the levels of estrogen rise, so too does the thickness of the endometrium in response to glandular growth. A certain high level of estrogen brings on an LH surge, which causes ovulation to occur about day 14 of the cycle.

Secretory phase of the endometrial cycle

After ovulation and the formation of the corpus luteum, progesterone and estrogen further increase the development of the endometrium. The endometrial glands and blood vessels become tortuous and swollen (edematous), and the glandular cells secrete and store fluids of lipids and glycogen (fats and sugars). These secretory fluids are nutrients prepared for implantation of a fertilized ovum. If implantation does not occur, the secretory phase then lasts from about the 15th day to the 27th day of the cycle, when the corpus luteum suddenly ceases to produce hormones and involutes. After the 27th day, the endometrium begins to degenerate in the absence of hormone influence and the subsequent constriction (ischemia) of the spiral arteries feeding it. Thus, the degenerative changes in the tissue and blood vessels of the endometrium cause both hemorrhaging into the cavity and shedding. Menstruation continues for about 5 days into the next cycle (fig. 1–3).

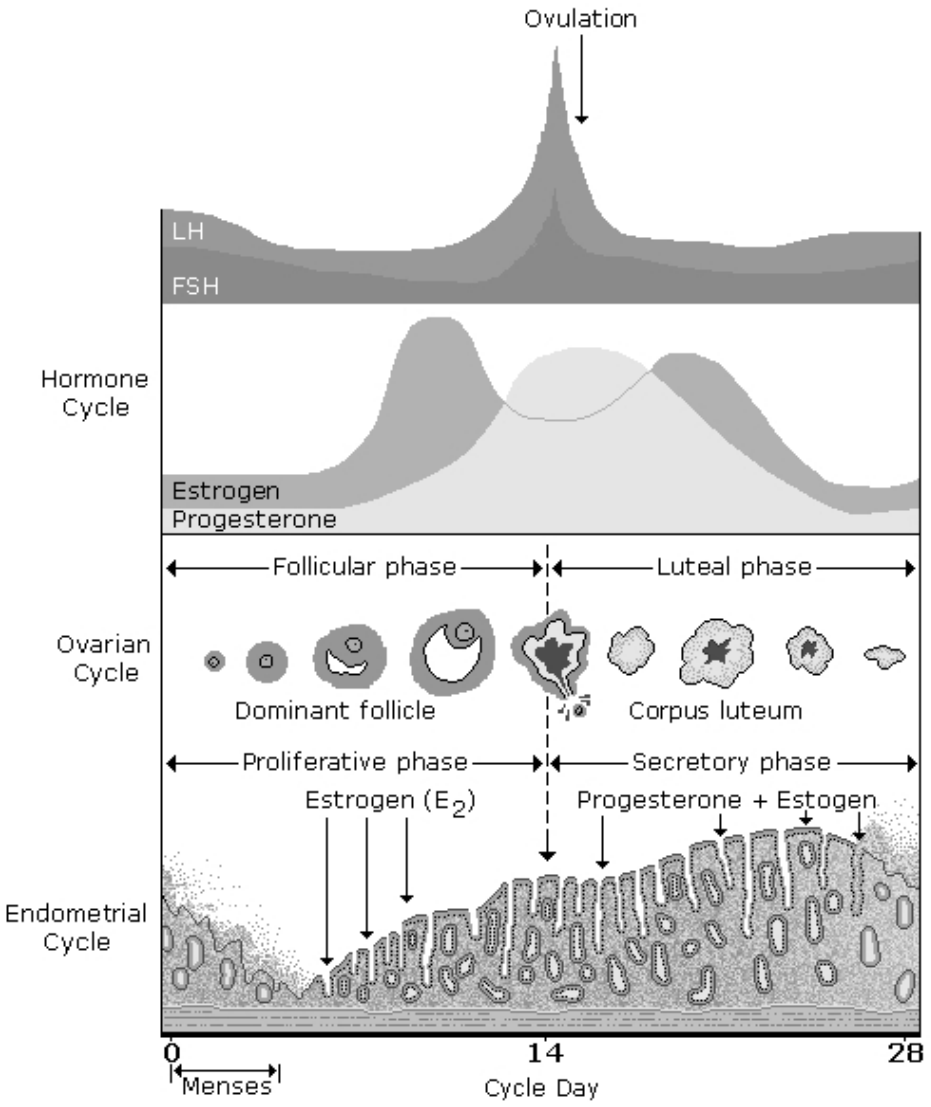

Figure 1–3. Menstrual cycle.

Reproductive Cycle	
Ovarian	***Uterine***
Follicular phase (Day 1–14) • Follicle growth from FSH • Mature follicle development • Increased secretion of estrogen by follicles • LH surge from high level of estrogen preovulation • Ovulation on day 14	Proliferative phase (Day 1–14) • Day 1–5 menses • Day 6–14 proliferation of endometrial glands
Luteal phase (Day 15–28) • Corpus luteum forms and produces progesterone and estrogen • FSH and LH levels drop • Corpus luteum involutes and stops secreting hormones	Secretory phase (Day 15–28) • Progesterone and estrogen cause thickening of endometrium and storage/secretion of glandular fluids (lipids and glycogen) within the glands • Day 27, endometrium begins degeneration due to absence of hormones • Day 28/1, the endometrial tissue hemorrhages and begins to shed

Postmenopausal considerations

At approximately 40 years of age, the ovaries begin to slow their production of estrogen and progesterone. This is a normal part of aging and occurs at different rates and times from woman to woman. Eventually, the ovaries cease hormonal function altogether, which causes monthly menstruation to stop, too. The cessation of menstruation *for one year* is called menopause and typically occurs after a woman's 50th birthday. During the time around menopause (perimenopausal), ovulation is also reduced, thus the drop in progesterone.

In the postmenopausal woman, the supply of ovarian follicles is depleted, creating a lack of response to FSH. Thus, the sizes of the ovaries decrease significantly. Also, the endometrium becomes a thin, inactive layer. Irregular bleeding can happen up to a year *after menopause*, but any bleeding after a year without hormone influence is likely abnormal.

Frequently, some women will suffer symptoms of estrogen deficiency and may require administration of manufactured estrogen to prevent chronic diseases such as osteoporosis and cardiovascular abnormalities. The estrogen given alone tends to not only thicken the endometrium; it also causes abnormal hyperplasia, an abnormality that can lead to carcinoma. Thus, unopposed estrogen is now often supplemented with progesterone, which is the hormone that eventually causes endometrial atrophy. This therapy, called hormone replacement therapy (HRT), is given to the patient either continuously or cyclically, depending on the tolerance of irregular or regular bleeding.

Self-Test Questions

After you complete these questions, you may check your answers at the end of the unit.

401. Pelvic anatomy

1. What purpose does the floor muscles of the pelvis serve in relation to the pelvic organs?

2. What formula is used to measure volume rather than linear dimensions of the ovary?

3. List the layers of the ovary from outer to inner.

4. What distinguishes the basal layer of the endometrium from the functional layer?

5. What are the dimensions of the nonpregnant adult uterus?

6. What feature of the body of the uterus is absent in the cervix?

7. What is the difference between the right ovarian vein and the left ovarian vein?

402. Pelvic physiology

1. What are the functions of estradiol and progesterone?

2. What causes the reduction of FSH during the follicular phase of the ovarian cycle?

3. What are the phases of the endometrial cycle and their corresponding ovarian cycle phases?

4. What occurs to the ovary and endometrium in a postmenopausal woman?

1–2. Pelvic Imaging

Sonographic imaging of the female pelvis can be one of the most challenging to perform. The complexity of the female reproductive system is such that the sonographic appearance of structures can vary significantly from person to person, within the same person over time, and even cyclically. In the last ten years, sonographic technology has vastly improved our ability to track normal changes and variations, as well as to better detect the host of abnormalities that can occur within the female pelvis.

403. General pelvic imaging

As with other areas of the body, male or female, the approach with sonography remains relatively the same. We still require a transducer, an ultrasound unit, patient preparation, a valid patient history, a review of any previous studies, and standard images to perform a complete sonographic examination of the female pelvis. However, we need to keep in mind some points in each of these areas.

Equipment

Sonography of the pelvis is generally performed using both a transabdominal approach and an endovaginal (also called *transvaginal* by sonographers) approach. Because the transabdominal technique requires a full bladder and endovaginal procedures are best performed with an empty bladder, the transabdominal portion is usually accomplished first. This allows the patient to void the bladder and allow the exam to be completed in a reasonable amount of time using an endovaginal approach. The reverse would require over a half hour of waiting for the bladder to fill.

The transabdominal approach is normally performed using a sector or curvilinear array transducer of up to 5 MHz (megahertz). With coupling gel placed on the skin of the anterior abdomen just above the symphysis pubis centrally, the transducer is used to transmit sound through the bladder to the reproductive organs beneath. The primary advantage for using the transabdominal technique is the large field-of-view that allows rapid location of nearly all of the pelvis's major structures. Also better located in this view are large masses, which can then be measured and compared in relation to the reproductive organs. Another advantage to this approach is the bladder displacing sound-scattering bowel loops superiorly and out of the way of the beam. Ovaries situated high in the pelvis are seen better transabdominally than with the more limited field-of-view of endovaginal approaches.

Disadvantages to transabdominal sonography of the pelvis also exist. Having a bladder too full may compress the uterus and cervix, distorting the true size and shape. Another big disadvantage is a full bladder can be quite uncomfortable for some patients. Also, transabdominal approaches cannot yield anatomic detail sufficient for accurate diagnosis of the endometrium and ovaries. Finally, an obese patient may be extremely difficult to exam transabdominally because of the tendency of fat to scatter sound and to increase the distance between the transducer and the pelvic organs.

The endovaginal approach requires a probe with at least 7 MHz but normally no greater than 10 MHz in frequency for excellent resolution. Before examining a patient with an endovaginal probe, the instrument must be disinfected with an antibacterial solution recommended by the manufacturers. Gel is normally applied to the tip of the probe and a latex or a nonlatex probe cover (condom) is placed over it. Either sterile lubricant or water (in infertility cases, of which the properties of ultrasound gel or lubricant tend to interfere with the action of sperm) is applied to the tip. If the patient's bladder is completely empty, the probe is then inserted using real-time sonography as a guide. Your institution may have a specific protocol for this part of the procedure. Male sonographers may find it more appropriate to have the patient insert the probe herself (while she is covered below the waste with a sheet). Let the patient know that you will be taking the handle after insertion.

Advantages for the use of endovaginal probes are better resolution of details in pelvic organs or masses, close viewing of the cervix without bladder compression distorting its true structure, better viewing of a retroverted or retroflexed uterus, and increased ability to detect fetal heart motion and details of first trimester anatomy sooner than with the transabdominal approach.

Disadvantages to endovaginal techniques include a limited field of view; patient refusal (for psychological, ethical, or physical reasons); the patient may be too young and or sexually inactive with an intact hymen; a male sonographer should require a female chaperone present for medicolegal reasons; and endovaginal is not as maneuverable as a transabdominal approach due to the constricting environment of the vaginal canal.

Endovaginal vs. Transabdominal

Method	***Advantage***	***Disadvantage***
Transabdominal	• Large field-of-view allows rapid location of the pelvis major structures and large masses • Bladder displaces sound-scattering bowel loops • Ovaries situated high in the pelvis better seen	• Bladder too full may compress uterus and cervix, distorting the true size and shape • Full bladder can be quite uncomfortable • Cannot yield anatomic detail of endometrium and ovaries • An obese patients difficult to examine

Endovaginal vs. Transabdominal		
Method	***Advantage***	***Disadvantage***
Endovaginal	• Better resolution • Close viewing of cervix without bladder compression distorting its true structure • Better viewing of a retroverted or retroflexed uterus • Increased ability to detect possible fetal heart motion and details of first trimester anatomy	• Limited field of view • Patient refusal (for psychological, ethical, or physical reasons) • Patient may be too young and or sexually inactive with an intact hymen • A male sonographer should require a female chaperone present for medicolegal reasons

Despite the advantages and the disadvantages, most radiologists prefer both methods in nearly all pelvic sonogram patients, providing a comprehensive evaluation. However, you may encounter some radiologists who will demand only one or the other be used in routine cases. Also fairly common is the use of transabdominal technique only for investigation of premenstrual and some postmenstrual patients. For these cases, this is largely due to the difficulty or impossibility of introducing the endovaginal probe into the vaginal canal.

Patient considerations

Before examination of the female pelvis begins, you must prepare. Most sonographic departments have administrative protocols for patient and staff preparations. Radiologists have different requirements, and you should be familiar with the ultimate goal of any protocol: to obtain diagnostic images for the radiologist to interpret.

Patient instructions

In general, and if not an emergency, patients should be given instructions for drinking prior to arriving for examination. Commonly, patients are asked to drink 32 ounces of water 1 hour prior to the start of the exam. Drinking instructions should be flexible enough for people who may have difficulty with the amount or rate of drinking. Frequently, 32 ounces of water 1 hour prior causes pain in women. A good way to modify this plan for maximum patient comfort is to ask the patient to empty the bladder 1 hour prior to exam start. Then ask them to begin *sipping* or *slowly* drinking 20 to 32 ounces of water until about 15 minutes before the exam. The bladder should fill gradually and comfortably in most patients, as well as enough to accomplish the exam.

No fasting preparation is necessary. The patient should wear two-piece clothing, as the bottom piece will be removed for the endovaginal portion of the exam. Medications can be taken and should not interfere with the examination. Put a patient at ease about menstrual cycles and that bleeding does not interfere with the exam. However, after menses is a better time to examine the pelvis.

Patient history

Physicians and other providers send patients for pelvic sonograms for a host of reasons. The validity of these reasons is critical and has a lot to do with the patient's history. The principal reasons for most referrals of pelvic sonograms are pain in the pelvis, abnormal bleeding, and suspected or confirmed masses in the pelvis noted clinically or from computed tomography (CT) and magnetic resonance imaging (MRI). Other reasons include follow-up of previously diagnosed problems, localization of contraceptive intrauterine device (IUD), and infertility patient monitoring.

Questionnaires are extremely helpful to radiologists diagnosing the pelvic patient. They also help focus the sonographer into performing a more accurate exam. Whether using a questionnaire, or some other method such as interviewing the person, the important thing is to obtain key information before the start of the exam.

- Age (related to menstrual status: premenstrual, premenopausal, and postmenopausal).
- First day of the last menstrual period (LMP) or last *normal* menstrual period (LNMP).

- Gravidity (the number of pregnancies) and parity (the condition of having given birth).
- Current complaint.
- Previous problems.
- Surgery.

Other helpful items would be personal and family history of cancer, laboratory values (if appropriate), clinical findings, previous imaging studies, and hormone status (oral contraceptives for premenopausal patients or regimen therapy for postmenopausal patients).

Standard pelvic images

In one of the images of the study annotate the patient's LMP. Also, appropriate labeling of each image is necessary along with a systematic presentation. With the transabdominal approach, the exam should be completed as rapidly but as accurately as possible. Once completed, have the patient empty the bladder and prepare the patient for the endovaginal portion of the exam.

Obtaining standard images transabdominally

Standard transabdominal images of the uterus and ovaries contain longitudinal and transverse views. The minimum that should be documented of the uterus is the size, location, and orientation. The length of the uterus is the most important, measured from the edge of the fundus to the edge of the cervix (technically, the outer wall of the vaginal fornix as it wraps posteriorly around the external os of the cervix). The endometrium (endometrial stripe) can be measured; however, a more accurate assessment of the stripe is accomplished endovaginally. On sonography, the uterus has medium level echoes with a homogeneous echotexture (fig. 1–4). However, prominent blood vessels within the outer third of the myometrium (often arcuate arteries or veins) may give the uterus a heterogeneous quality. The stripe is normally echogenic, the thickness of which depends upon the stage of the menstrual cycle.

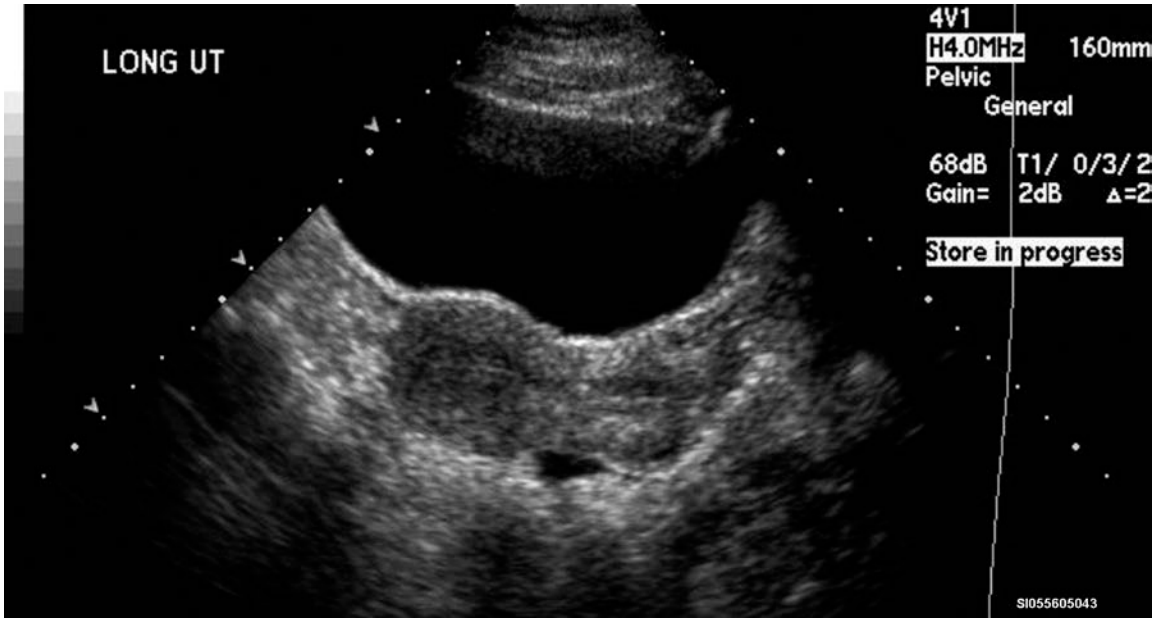

Figure 1–4. Longitudinal uterus, transabdominal.

Longitudinal and transverse images of the ovaries should be obtained. In the absence of ovaries, the adnexa should be imaged. The ovaries are sonographically more hypoechoic than the uterus. Transabdominal is particularly valuable for locating ovaries situated high in the pelvis and beyond an endovaginal probe's field-of-view. During the reproductive years, the ovaries contain follicles, which appear anechoic. Some departments require a few images of color Doppler to determine the vascular flow in and just around the ovary.

Typical Transabdominal Pelvic Protocol		
Uterus	Longitudinal views • Midline (with/without measurements of the length from fundus to cervix, and AP of enodmetrium); the cul-de-sac should be seen on the same view • Right and left lateral portions of the uterus • A view of the cul-de-sac	Transverse views • Inferior views of the vagina and cervix • View of inferior portion or isthmus of uterus • Midline width (with/without measurements) • Superior view of the fundus
Ovaries	Longitudinal view • With and without measurement of length and AP in the longest dimension	Transverse view • With and without measurement of the width in the widest dimension 90 degrees to the length measurement

Obtaining standard images endovaginally

For obtaining sonographic endovaginal images of the ovaries you should recognize that the ultrasound beam orientation is different. The direction is from within the vaginal canal up into the pelvis. The organs are closer and thus the display on the screen should be of mostly the anatomy in question. Also, the views are now sagittal instead of longitudinal, and coronal (or transaxial) instead of transverse.

For the uterus and cervix, the first image seen should be a sagittal midline view (fig. 1–5). This will demonstrate the orientation of the uterus in the body without a full bladder. Measurements of length from the fundus to the cervix and the AP of the widest portion of the body of the uterus can be obtained in this view. The endometrial stripe is also measured in this view (as discussed below). The left and right portions of the sagittal uterus should be documented. Coronal images of the cervix, isthmus, inferior, midline, and superior are acquired in successive order (fig. 1–6). A midline image of the uterus of width measurements should also be obtained at the widest portion. Keep in mind that the uterus may not be oriented in an anteverted (tilted forward) fashion as with transabdominal, but may be situated with fundus above, below, or straight ahead to the probe tip. For the uterus without a tilt or bend, you may have to use labels such as anterior and posterior.

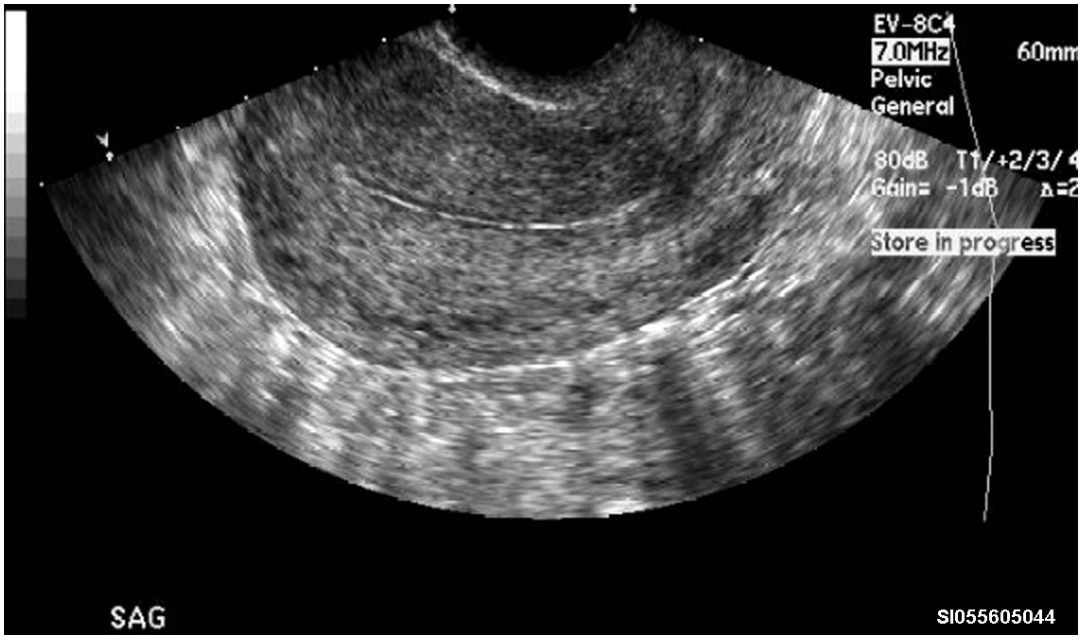

Figure 1–5. Sagittal uterus, midline.

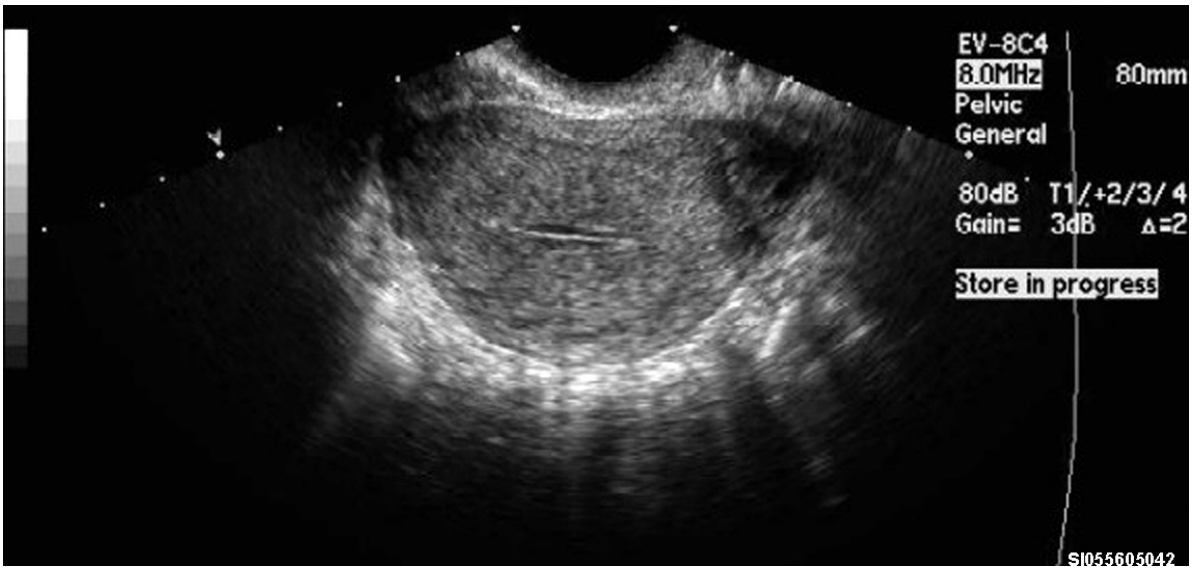

Figure 1–6. Coronal uterus, midline.

Sweeping the sonographic beam through either adnexa will bring the ovaries into view. Manipulate the probe in such a way to obtain the longest dimension of the ovary, which will represent the length. The AP should be measured perpendicular to this. These length and AP measurements are considered sagittal images of the *ovary* (not the pelvis). Turn 90 degrees to the sagittal measurement and obtain a coronal (transaxial) view of the ovaries for a width measurement at its widest point. Some radiologists or department protocols will require you to measure the dominant follicle in all three dimensions. Also, do not be surprised if color Doppler of the ovaries is requested, as blood flow to the ovaries is a critical factor in distinguishing from normal and certain abnormalities.

Sonographic appearance of the endometrium throughout reproductive cycle

Helpful to an interpreting radiologist is the appearance of the endometrium, commonly referred to as the endometrial stripe, on endovaginal sonography. Because the menstrual cycle is hormonally driven, corresponding changes in endometrial tissue prompt changes in its sonographic appearance. Generally, the endometrium thickens throughout the proliferative phase and up to ovulation. The thickening continues beyond ovulation to about halfway through the secretory phase. With the involution of the corpus luteum from the ovary, and a subsequent cessation of progesterone and estrogen, the thick endometrium immediately becomes ischemic and starts to break down. Thus, hemorrhaging begins and the shedding of the functional endometrium leaves only an extremely thin basal layer. This process occurs cyclically and is a normal occurrence. Variations in the cycle provide clues to the radiologist about potential abnormalities.

Although variations exist in women, typical sonographic appearances of the normal endometrial stripe throughout the cycle are known. Thickness and characteristics of the stripe outside known normal parameters may prompt radiologists into closer follow-ups. Measurements of the endometrial stripe are taken endovaginally. A single AP measurement of the thickest portion of endometrium is taken, usually within the body of the uterus. Calipers are placed only at the outer edges of the echogenic stripe.

Early in the menstrual portion of the proliferative phase (frequently days 1–5), endovaginal sonography reveals the stripe stretching from the cervix to the fundus and appearing as a hyperechoic yet broken line with anechoic spaces representing blood in the cavity. AP measurements during menstruation should be no more than 3 or 4 mm, usually depending on the amount of fluid present within the cavity. After menstruation, the early proliferation of endometrial tissue (often days 6–14) displays sonographic thicknesses in the range of 4 to 8 mm. During this time of expanding thickness, the echogenicity of the endometrium is hypoechoic throughout, yet noticeably distinct from the

central hyperechoic line of two opposing endometrial walls as ovulation approaches. Late in the proliferative phase, another hyperechoic line appears around the stripe at the basal layer, where endometrium meets the myometrial tissue of the uterus. The AP measurements are usually taken at this hyperechoic basal line. Thus, three hyperechoic lines appear in the endometrial stripe, most apparently just before ovulation. After ovulation, the endometrium enters the secretory phase (days 15 to 28) and continues thickening up to at least day 25 of the cycle. Sonographic measurements of the stripe range between 8 and 15 mm. The glandular secretions as well as fluid and proliferation of tortuous spiral arteries within the endometrium cause the secretory stripe to change from a hypoechoic appearance to an extremely hyperechoic appearance.

For a year after menopause, the endometrium is generally considered to appear as a hyperechoic line no more than 5 mm thick in normal, asymptomatic women. After this point, anything greater than 8 mm causes concern for abnormality. Women on hormone regimen therapy (HRT) of synthetic estrogen (opposed with synthetic progesterone) will often have thickened endometria up to 15 mm, which is normally the only exception. Otherwise, consider any thickness greater than 8 mm to be an ominous sign, which we will discuss later.

Typical Sonographic Endometrial Appearances

Cycle Phase	***Day***	***Stripe Appearance***	***Thickness***
Proliferative (menstrual)	1–5	Thin, hyperechoic, and broken with anechoic spaces	3–4 mm
Proliferative	6–14	Early: thin hyperechoic line representing the interface between opposing endometrium Late: thick hypoechoic endometrium with central hyperechoic line and surrounding hyperechoic line on the periphery representing basal layer	4–8 mm
Secretory	15–28	Increasing thickness with completely hyperechoic appearance	8–15 mm
Postmenopausal **Note:** not part of cycle	N/A	Thin hyperechoic line	< 5 mm and no greater than 8 mm

Sonographic appearances of congenital malformations and normal variants

Malformations are usually the results of error in development before birth. The structural defects that appear from birth (congenitally) are anomalies that occasionally can cause problems later in adult life. In the female, congenital malformations of the reproductive organs are rare, but can be occasionally detected sonographically. More common, slight variations (called normal variants by sonographers) occur in the orientation of the uterus in the pelvis. You should at least be familiar with these variants and aware of the potential for encountering a congenital malformation.

There are several variants of sonographic uterine appearances. Most women have a normal orientation of the cervix with vagina of 90 degrees (called *version*) and a tilting forward (anteversion) of the uterus and cervix (in line with each other) against the vaginal tube, typically seen on transabdominal with the bladder full. The anteverted uterus is seen with superior detail with endovaginal sonography. Other variants of the anteverted position in the pelvis are:

- Anteflexion—The body of uterus is bent forward, with an acute angle at the isthmus, *against the cervix*. This variant can be difficult to demonstrate on sonography. Following the endometrial stripe as it bends sharply at the isthmus is the clue, and the angle should almost fold back to 45 degrees.
- Lateral versions—The body of uterus and cervix deviate to the right (dextroversion) or to the left (levoversion) in relation to the midline orientation of the vaginal tube.
- Retroversion—The body of the uterus and cervix together are tilted backward away from the vaginal tube and toward the posterior pelvis and rectum. This variant is particularly difficult

to appreciate transabdominally and is best seen endovaginally, where the fundus of the uterus will be situated posteriorly.

- Retroflexion—The body of the uterus is bent backward, with an acute angle at the isthmus, *against the cervix*.

Congenital malformations that you may occasionally see mostly occur within the uterus and cervix. Because these anomalies originate before birth during the embryologic stage, a period of rapid development, extreme variations occur in the type of malformation in the uterus. Congenital malformations of the uterus are due to failure of the paramesonephric ducts in the embryo to fuse or to develop. This failure can be partial or complete. Paramesonephric ducts (müllerian ducts) are embryologic structures that will later develop into reproductive organs; that is, in the female, into the uterine tubes, uterus, and vagina. Because the paramesonephric ducts run parallel with early urinary system structures (mesonephros), failures can sometimes cause or occur with congenital renal abnormalities. Thus, pelvic malformations are usually associated with renal defects, especially absence of a kidney (unilateral renal agenesis). On sonography, we can detect the signs through both the appearance of the endometrium and the outer contour of the uterus. When seen, we should also scan the abdomen to document the appearance and location of the kidneys.

- Uterus didelphys—Rare, complete duplication of the uterus and cervix, and sometimes also the vagina. Each uterus has all three layers separating the endometria. Readily apparent on transverse transabdominal images.
- Uterus bicornis—Most common; typically called *bicornuate uterus*, although some radiologists and sonographers use the term even with types didelphys and septus. The fundus and most of the body of the uterus are duplicated, but the inferior body and cervix are intact. Rarely a bicornuate uterus will also have a duplicate cervix (uterus bicornis bicollis). Bicornuate malformations also have all three layers of the uterine walls surrounding duplicate endometria, with a deep notch in the outer contour of the uterine fundus. On sonography, the bicornuate uterus is best seen in coronal endovaginal plane, where the endometria will be seen in cross section as you sweep down from the fundus to cervix (fig. 1–7). The best time to see this is when the patient is in the secretory phase of the cycle, which causes the endometria to be seen at their most echogenic.

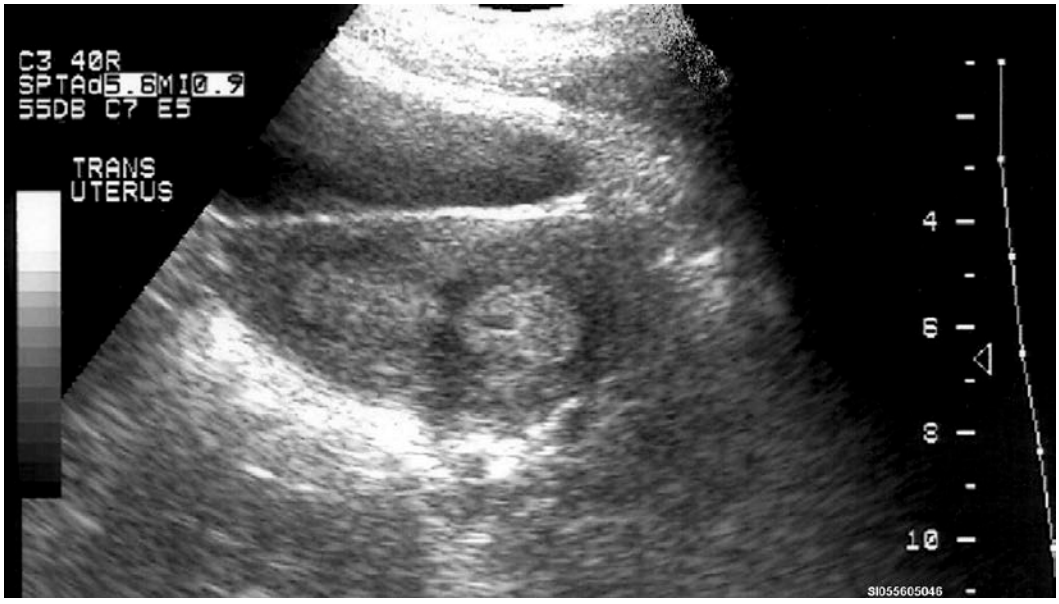

Figure 1–7. Bicornuate uterus, coronal.

- Uterus septus/subseptus—Typically called septate or subseptate uterus. The uterus and cervix appear completely fused but have a thin or thick septum running down the length of the

endometrial cavity, or running down most of the length in the subseptate type. The outer contour of the uterine fundus is smooth and unaffected. This malformation causes most of the problems in women associated with congenital anomalies, such as infertility and spontaneous abortions. It is usually the result of the failure of the fusing tubes to resorb the septum.

- Uterus unicornis—Unicorn uterus results from the failure of a uterine tube to develop or form. This abnormality is difficult to see sonographically due to the undetectable nature of the uterine tubes. A clue may be a small uterus that deviates to one side; different from version in that the uterus is, again, smaller than normal.

404. Pelvic pathology

A cascade of abnormalities can occur within the area of the female pelvis, affecting the reproductive organs as well as the other structures. Sonographers who have an understanding of normal pelvic physiology and the normal sonographic appearance of pelvic anatomy should also be familiar with interruptions to function caused by disease and other abnormal occurrences. Such familiarization with pelvic pathology will prepare the sonographer to recognize corresponding alterations of pelvic anatomy.

Pelvic inflammatory disease and torsion

Inflammation and physical twisting of the ovarian pedicle can cause pain. For the twisted ovary, not only are ligaments twisted but also nerves and blood vessels, which can cause loss of blood to the ovary (*ischemia*) and congestion of return flow. Pelvic inflammation can also cause the formation of abscesses that can make the peritoneal cavity toxic. In this lesson, we will look at these two common adnexal abnormalities.

Pelvic inflammatory disease

Pelvic inflammatory disease (PID) is the name clinically given by gynecologists to a spectrum of abnormalities affecting the female pelvic organs caused mostly from sexually transmitted bacterial infection. Thus, PID is the combination of consecutively or concurrently inflamed endometrium (*endometritis*), uterine tube (*salpingitis*), uterine tube with ovary (*tubo-ovaritis*), and pelvic peritoneum (*peritonitis*). Inflammation of each one of these structures is usually—but not always—the cause of pain, particularly in the uterine tube and peritoneum.

The organisms most responsible (there are others) for this infectious process are *Neisseria gonorrhoeae* and *Chlamydia trachomatis*, which are introduced into the cervix through sexual activity. According to the National Institutes of Health, one million women in the United States suffer from PID annually. One hundred thousand of these women (10 percent) develop infertility problems resulting from damage the disease causes to the uterine tubes. Risk factors for PID are: multiple sexual partners, IUDs used for contraception, post-partum endometritis, or infection developing from instrumentation used to terminate pregnancy (curettage).

Endometritis causes the endometrium to thicken and become irregular. Typically, fluid, debris and occasionally gas will collect within the cavity. Because early PID often produces only mild symptoms, most women will initially feel little or no pain at all. As the infection worsens and spreads into the upper reproductive tract, pain increases; yet this symptom will vary significantly from woman to woman in intensity. Abnormal bleeding, vaginal discharge, fever, and occasional nausea and vomiting will occur.

The organisms travel up into the uterine tubes and infect the lining, causing swelling (edema) and damage to the tissue. Again, infection in the tubes is called *salpingitis* and usually occurs bilaterally. Untreated, an infected tube may produce inflammatory pus and debris, a condition called *pyosalpinx*. Adhesions can form, obstructing the tube and causing pus to build up and painfully increase tube diameter. The pain from the tubes causes most women to seek treatment, which is normally with antibiotics. Recurrent infections can cause chronic PID, which may produce pyosalpinx or clear

serous fluid in the uterine tube, known as *hydrosalpinx*. The diameter of the uterine tube with hydrosalpinx can grow from a few millimeters up to 6 cm, often becoming tortuous.

When the infection involves both the tube and the ovary, it is called *tubo-ovaritis*. Specifically, the peritoneal lining covering the ovary becomes inflamed, which may produce adhesions that cover the entire ovary or adhesions that attach the ovary to the end of the uterine tube. An inflamed ovary attached to an inflamed uterine tube is also called a *tubo-ovarian complex*. If untreated, a complication called *tubo-ovarian abscess* (TOA) may form. A TOA results from adhesions and inflammatory pus extending from a tube and attached ovary, which breaks down and disorganizes surrounding pelvic tissue, creating a massive abscess (a collection of pus) around both tube and ovary. A TOA frequently generates more adhesions that attach to surrounding peritoneal structures, such as the outer surface of bowel loops, ureters, and surrounding pelvic walls. These adhesions fix the TOA in place as well as freeze bowel walls, preventing normal peristalsis. Thus gastrointestinal (GI) problems may result. By this point, pelvic pain is nearly intolerable to most women. Further, a danger of the TOA rupturing exists. If rupture occurs, inflammatory fluid can spread throughout the peritoneal cavity, causing life-threatening peritonitis.

TOA can also be caused by non-PID infection. For example, an infection from elsewhere in the body may spread from the peritoneum to the ovary and into the tube. These peritoneal infections can sometimes form abscesses, which may or may not rupture, such as pelvic abscesses (located usually in the cul-de-sac) or ruptured appendicitis.

Spectrum of Events in Pelvic Inflammatory Disease	
Cervix	Entry point of *N. gonorrhoeae* and *C. trachomatis* sometimes with mild infection of endocervical canal.
Uterine cavity (endometrium)	Endometritis causing thickened and irregular endometrium with possible fluid, devris, or gas.
Uterine tube	Salpingitis causing edema of the tubes which may progress to production of pus (pyosalpinx) or serous secretions (hydrosalpinx), painfully distending and kinking the tube.
Uterine tube and ovary	Tubo-ovaritis which may or may not cause inflamed peritoneal covering of the ovary to form adhesions, attaching the ovary to the tube (tubo-ovarian complex). Complication: TOA may form with complex producing inflammatory pus. Adhesions may fix TOA to surrounding peritoneal structures causing intense pain.
Pelvis	Pelvic abscess formation in the cul-de-sac or other location as a result of peritoneal spread from the tube and ovary

PID abnormalities are generally treated with antibiotics. Treatment for TOA is either with antibiotics or drainage, depending on the size and severity of the collection. Most PID treated with antibiotics will recur in women with PID resulting from gonorrhea.

Torsion

Torsion in the adnexa of the pelvis is the twisting of the ovarian pedicle. The pedicle involves the long axis of the broad ligament, the ovarian branches of the uterine artery and vein, lymph channels, and, occasionally, the uterine tube. The affected pedicle also can involve the terminal end of the ovarian artery as it courses from the suspensory ligament. The twisting can be either complete or partial, with some spontaneously detorsing and torsing recurrently. Torsion causes the obstruction of outflowing venous blood and lymphatic drainage. This causes the ovarian parenchyma to become backed up (congested) with fluid, which in turn increases the size from swelling (edema). Frequently, an accompanying cyst will begin to hemorrhage and areas of necrosis may appear. Arterial supply of blood is completely cut off as well, resulting in an infarcted ovary. However, the presence of persistent arterial flow does not rule out torsion. The presence of dual arterial supply from an untwisted vessel, either the ovarian branch of the uterine artery or the ovarian artery, has been known to provide flow in confirmed cases of torsion.

Torsion is most common in women younger than 30 but can occur at anytime from childhood to postmenopausal. The most common causes of torsion are large ovarian masses, usually dermoid and paraovarian cysts. Freely mobile ovaries within the adnexal spaces can worsen the situation. Less commonly, the adnexa involving a normal ovary will twist, but this is seen more in prepubescent children and adolescents. There is also an increased risk of torsion occurring in pregnant women.

Typically torsion is accompanied with severe abdominal pain, mostly on the right side. This is thought to be due to the right ovary and pedicle having sufficient room to become twisted. Conversely, the left ovary is not as mobile due to the compactness of the left lower quadrant filled with sigmoid colon. The complaint of pain is similar to that of acute appendicitis as well as other acute pelvic diseases. Frequently, nausea and vomiting will accompany the intense pain. Pain can be chronic with intermittent episodes occurring over weeks before an acute event. The referring physician will sometimes describe the presence of a palpable mass, which subsequent scanning should correspond with an edematous ovary. The treatment of choice for torsion is immediate surgical intervention.

Ovarian cysts

Ovarian cysts are usually classified as either functional or nonfunctional, with the majority being the former. Being familiar with the functional cysts and some of the more common nonfunctional cysts will give you more information with which to help form a diagnosis.

Functional cysts

Functional cyst implies a cyst that responds or is influenced by the normal physiologic (functional) process of the reproductive organs, specifically the ovaries. Because they tend to disappear or regress after one or two menstrual periods, they are considered responsive to the hormonal fluctuations that accompany the cycle. Functional cysts are the most common of the ovarian cysts. Three types of functional cysts occur: follicular, corpus luteum, and theca-lutein.

Follicular cysts

Follicular cysts are mature or dominant follicles that fail to ovulate. They are considered the most common of *anechoic* ovarian cysts. Follicular cysts are responsive to the hormonal cycle and will thus grow to a certain size before spontaneously regressing in one or two cycles. These cysts can occur singularly or multiple, unilateral or bilateral. Unless they grow too large or hemorrhage, the follicular cysts are usually asymptomatic.

Corpus luteum cysts

Less common than follicular cysts are the corpora lutea cysts. A corpus luteum cyst results from a failure of the corpus luteum to collapse or involute. This abnormality occurs after ovulation and is usually, *but not always*, a hemorrhaging of the corpus luteum. The corpus luteum cyst can also be filled with serous fluid. Recall that during the luteal phase of the ovarian cycle the corpus luteum secretes progesterone, preparing the endometrium for possible implantation of a fertilized ovum. Increased vascularization of the walls of the corpus luteum may cause hemorrhage, which can cause a cyst to form. Normally, the corpus luteum begins to regress late in the luteal phase, but with hemorrhaging, it can persist and continue to produce progesterone, which will delay or cause abnormal menstruation.

Corpora lutea cysts are usually large and generally cause patients pain, mostly as a result of rupture or leakage. In fact, the hemorrhagic corpus luteum cyst is more prone to rupture than hemorrhagic follicular cysts. They occur unilaterally and ultimately regress after one or two cycles.

You should be aware that if pregnancy occurs, a *corpus luteum* will persist and continue to produce progesterone; thus, it is called the corpus luteum *of pregnancy*. If the corpus luteum of pregnancy hemorrhages, it becomes a corpus luteum *cyst* and may grow to a considerably large size. Again, the cyst will usually, but not always, resolve by 16 to 18 weeks.

Theca-lutein cysts

The largest of the functional cysts are theca-lutein cysts; however, they are not as common as follicular and corpora lutea cysts. When the internal layer of an ovarian follicle (theca interna) becomes hyperstimulated by high blood levels of human chorionic gonadotropins (hCG), the follicles grow at an exaggerated rate. Just as the gonadotropin LH causes luteinization of a mature follicle, hCG also causes the theca interna to transform into luteal tissue, as if ovulation has occurred and the body is trying to produce a corpus luteum. The high concentrations of hCG is normally due to several conditions such as gestational trophoblastic disease (hydatidiform moles) and drug-induced ovarian stimulation for the treatment of infertility. Multiple gestations can also cause highly elevated levels of hCG in the blood.

The excessive stimulation of the follicles produces multiple cysts that replace most of the ovarian tissue. Theca-lutein cysts are bilateral in half of patients. They usually contain multiple septations and can grow beyond 10 cm in size, particularly in infertility patients with ovarian hyperstimulation syndrome. With the removal of the source of stimulation, hCG, a theca-lutein cyst can persist for up to 4 months before regressing. HCG removal usually consists of the cessation of drug therapy for infertility patients and the removal of molar pregnancies for trophoblastic disease.

Other adnexal cysts

You will probably encounter other common ovarian cysts, which tend not to regress or resolve with the menstrual cycle. Some are congenital, such as the paraovarian cysts, while others may be part of a spectrum of problems, such as the polycystic ovaries seen with *polycystic ovarian syndrome* (PCOS).

Paraovarian cysts

Cysts outside the ovary and adjacent or near (para-) to it are called paraovarian. Most are simple cyst filled with serous fluid and are remnants from embryolic development, usually of the mesonephric duct. Thus, they arise from the surface of the broad ligament that stretches from either side of the uterus. Paraovarian cysts can appear at any age, but are usually present in early adulthood. They are asymptomatic unless they hemorrhage or rupture, causing pain. Internal hemorrhage may cause the paraovarian cyst to become larger, which may cause painful ovarian torsion.

Polycystic ovaries

Polycystic ovaries are simply ovaries that contain many cysts; that is, they contain multiple follicles large enough to resemble small cysts. They are normally associated with PCOS. Sometimes called Stein-Leventhal syndrome, PCOS is a spectrum of endocrine related disorders with the most obvious being hyperandrogenism (excessive secretions of androgens, hormones that promote male characteristics). Other disorders of PCOS are: oligomenorrhea (infrequent episodes of menstrual bleeding), amenorrhea (absence of menstrual bleeding), hirsutism (abnormal hairiness), and anovulation (inability to ovulate). Some women are obese in PCOS cases. Not every case of PCOS will have polycystic ovaries. In fact, some women with PCOS may only show absent or abnormal bleeding. Usually, PCOS-related polycystic ovaries are treated hormonally.

Endometrial cysts (endometriomas)

Like paraovarian cysts, endometrial cysts do not arise from the ovaries; however, unlike paraovarian cysts, they usually attach to or completely replace the ovary. Endometrial cysts develop from endometrial foci or lesions that occur with *endometriosis*, the presence of endometrial glands outside of the uterus. Patients with endometriosis may have foci of endometrial tissue scattered throughout the pelvis, with the most common location being the ovaries. Because the endometrial tissue is active during the menstrual cycle, the lesion will shed during the menses portion of the cycle, resulting in blood contacting the pelvic peritoneum, which in turn can cause painful inflammatory reaction in severe cases. With ovarian involvement, a cyst may develop from cyclic hemorrhaging of glandular focal tissue. However, the endrometrial cyst does not regress with the menstrual cycle, and the blood within it clots with age. Thus, physicians call these cysts “chocolate cysts” because of the brownish-red color of old blood discovered after removal. Sonographers sometime call the cyst an *endometrioma*, which has inaccurate connotations of a tumor or neoplasm.

Women with endometriosis are sometimes without symptoms, but most typically have a range of clinical symptoms from dysmenorrhea (painful menstruation) to low back pain. Women may have other conditions such as infertility. The abnormality can affect women at any period in their lives, but mostly those of reproductive age who are nulliparous. Most are treated through surgical removal of the lesions.

Ovarian neoplasms

A neoplasm is an abnormal growth or mass (tumor) that may be either benign or malignant (cancer). Most neoplasms, particularly the malignant variety, are asymptomatic.

Most ovarian *cancers* are found in postmenopausal women and, thus, many of the neoplasms seen in women of reproductive age are either benign or potentially malignant. In the United States, ovarian cancer (carcinoma) appears in at least 20,000 women annually, with at least 6 percent resulting in death each year. Of all gynecological cancers, ovarian carcinoma is the main cause of death, primarily because it is detected too late at advanced stages III (spread into the abdomen from the pelvis) and IV (spread beyond the abdomen). As a result, the survival rates for the advanced stages are poor. Ovarian carcinoma, stage I and stage II, have the best chances for survival as the cancer remains within the ovaries and pelvis, respectively.

Although appearing in women sporadically at all ages, but particularly in women over 45 to 50 years of age, ovarian carcinoma is not as common as breast cancer. Nulliparous women, women with a history of breast, colon, or endometrial cancer or with a family history of carcinoma are at risk.

Primary (arising from the organ) ovarian neoplasms are divided up into three tumor types, usually along the cell-structure of the gonadal tissue from which they arise: epithelial, germ cell, sex cord-stromal tumors. Metastatic ovarian neoplasms, those that originate elsewhere in the body, are relatively rare. Most metastatic tumors found in the ovary are from the GI tract, specifically the bowel, and usually referred to as *Krukenberg* tumors. Other sources for metastases to the ovaries are breast carcinoma, skin (melanoma), and lymphoma.

Epithelial ovarian tumors

Epithelial ovarian tumors arise out of the layer of cells that cover the ovary, the epithelium. Most neoplasms of the ovary are epithelial tumors, which also gives rise to most of the ovarian cancers. If malignant, epithelial tumors can spread or metastasize through three pathways: peritoneal cavity, lymph channels, or the vascular system. However, in general, most malignant epithelial tumors rarely appear in women younger than 40 years of age. Thus, benign and low-malignant-potential epithelial tumors are found in premenopausal women. Epithelial tumors do not usually generate symptoms in women unless they grow to enormous sizes. Early stage malignant tumors can grow to sufficient size that causes compression of the bladder and rectum, which in turn can generate constipation and urinary problems. With more advanced stages, ascites can cause abdominal bloating, anorexia, nausea, and vomiting.

Most epithelial tumors can be grouped into several categories: serous, mucinous, endometroid, clear cell, and transition cell (Brenner). Of these major categories, most are serous and mucinous. Benign forms of either are called *adenomas* and malignant forms are called *adenocarcinoma*. Because most serous and mucinous tumors are predominately cystic structures, they are usually referred to with the prefix, *cyst-*, as in *cyst*adenoma and *cyst*adenocarcinoma.

Benign and borderline (low malignant potential) serous tumors are called serous cystadenomas and malignant tumors are called serous cystadenocarcinomas. Serous tumors are the most common of all epithelial ovarian neoplasms.

The second most common epithelial ovarian tumor is the mucinous tumor, most of which are benign. As with the serous tumors, mucinous tumors are found in two types: the mucinous cystadenoma and the mucinous cystadenocarcinoma.

Serous cystadenoma and cystadenocarcinoma

The second most common *benign* ovarian tumor is the serous cystadenoma (the first being the cystic teratoma, discussed below). They usually occur in women before the age of 50. The tumors are mostly unilateral but a third of patients will have bilateral appearances.

The most common ovarian malignancy is the serous cystadenocarcinoma, appearing mostly in women older than 45. Because this malignancy typically develops without symptoms, it is usually quite large by the time the patient is referred to you. Typical symptoms of large serous cystadenocarcinomas are of abdominal distension and pressure, weight loss, or nausea. These masses may be unilateral or bilateral and are typically as large as its benign counterpart, the serous cystadenoma.

Mucinous cystadenoma and cystadenocarcinoma

Occurring in women younger than 50 and mostly in their thirties, mucinous cystadenomas are typically unilateral. Like its benign counterpart, the malignant form of mucinous tumor also is unilateral or multilocular. Mucinous cystadenocarcinomas are found in women between 30 and 60 years of age.

Germ cell ovarian tumors

Tumors not only arise from the surface layer of ovaries, but also from deep within. A class of ovarian tumors arises from ovarian germ cells, the primitive cells surrounded by primordial follicles that develop into ovum. Germ cell tumors are the second most common ovarian tumors, appearing predominately in children and young adults. They are the most common ovarian neoplasms in females before the age of 20. Women in the twenties and thirties typically will have germ cell tumors that are mature and benign. Although they do occur, rarely will you encounter a germ cell tumor in a female beyond 40 years of age. In children, about a third of germ cell tumors are malignant, making them the source of most childhood ovarian malignancies. Most germ cell tumors cause elevated laboratory values of alpha fetoprotein (AFP) and hCG.

Germ cell tumors are divided up into several types depending upon the type of germinal cell composition. Of the following types, we will discuss only the first two, as you are more likely to encounter these than the last two extremely rare and aggressively malignant types.

- Teratomas: (mature, immature, monodermal).
- Dysgerminoma.
- Endodermal sinus tumor (yolk sac carcinoma).
- Embryonal carcinomas, nongestational choriocarcinomas of the ovaries, or mixed germ cell tumors.

Teratomas

Teratomas are congenital tumors composed of any type of embryonic (and therefore primitive) body tissue and can occur anywhere in the body. Most are composed of two out of three primary germ layers: *ectoderm* (nervous tissue and skin), *mesoderm* (connective tissue), and *endoderm* (epithelial lining of the GI system, glands, and lungs). In infants of both sexes, they frequently appear in the sacrococcygeal area. In males, the teratomas usually appear in the testes. In adolescent and adult females of reproductive age, they occur mostly in the pelvis.

The germ layer composition of the teratoma determines its type. Most ovarian teratomas are composed of *mature* ectodermal tissues and are quite benign. Some are composed of *immature* germ layers, which are highly malignant. A few will be composed strictly of one type of tissue (monodermal). For instance a particular teratoma, called a *struma ovarii*, is composed mostly of thyroid gland tissue (endoderm).

Again, two types of ovarian teratomas will predominately occur, mature and immature. The mature teratoma is further divided up into a solid or a cystic mass, with the cystic mass being the most

common germ cell ovarian tumor. The mature solid teratoma is a rare, slow growing solid tumor that has sonographic features similar to immature teratomas, discussed below.

The mature cystic teratomas are called, interchangeably: cystic teratomas, dermoids, dermoid tumors, or dermoid cysts. They are the most common benign germ cell tumors and the most common *benign* ovarian neoplasms in women of reproductive age. Cystic teratomas are discovered incidentally on routine pelvic sonograms and do not cause symptoms unless complications develop, such as torsion or rupture. Either complication can produce pain. Torsion from a dermoid, the most common complication, is more prevalent with pregnancy. The less common rupture will generally cause chemical peritonitis.

Dermoid cysts are composed primarily of skin, fat, and hair. Half of these tumors will contain bone, teeth, muscle, cartilage, and other tissue. Typically, a dermoid, particularly a large one, will move an ovary anterior to the uterus and level with the fundus. Most dermoids are unilateral, with less than a fourth appearing bilaterally.

The immature teratoma is a malignant form of teratoma that occurs primarily in children and adolescents. The malignancy tends to proliferate aggressively and spreads through the lymphatic system. Generally uncommon among teratomas, most are unilateral tumors.

Dysgerminoma

The most common germ cell malignancy in females between the ages of 10 and 30 years, dysgerminomas are identical at the cellular level to seminomas found in male testes. Despite it being the most common of *germ cell* malignancies, the dysgerminoma is relatively rare when compared to the more prevalent *epithelial* carcinomas. However, keep in mind that epithelial carcinomas occur mostly in women older than 45, while germ cell malignancies are largely seen within the first two decades of a female's life.

The dysgerminoma is composed of abnormally proliferating germ cells. Within the dysgerminoma are lobules separated by septations laced with blood vessels. Also, most of the tumors are unilateral.

Sex cord-stromal tumors

You may see a few of the third type of ovarian tumors, the sex cord-stromal tumors. These masses arise out of the ovarian sex cords (columns of germinal and follicular cells) and ovarian stroma (fibrous connective tissue). Sex cord-stromal tumors divide into six major types: fibroma, thecoma, granulosa cell, Sertoli-Leydig cell, steroid cell, and hilus cell. Together, these tumors constitute a tenth of all ovarian tumors. They also are responsible for most of the functional (estrogen-producing) ovarian tumors. Of the sex cord-stromal tumors, only fibromas and thecomas are likely to be encountered by sonographers, followed by the adult form of granulosa cell tumors.

Ovarian fibromas appear in women of all ages, but principally occur in middle-aged women around the time of menopause (perimenopausal). They are the most common of sex cord-stromal tumors. Fibromas are benign stromal masses that, unlike the other stromal tumors, are not typically associated with estrogen production. The tumors are mostly fibrous and solid, appearing unilaterally. Their sizes range from very small to extremely large. Up to half of fibroma cases with tumors larger than 5 to 10 cm are generally associated with ascites in the pelvis and abdomen. An extremely small percentage of the large fibromas will appear with both ascites and pleural effusion, a combination called *Meigs syndrome*.

The major differences between thecomas and fibromas are thecomas appear mostly in postmenopausal women, are functional, and are composed mostly of lipid (fat) and thecal cells instead of fibrous tissue. The thecoma is unilateral and benign.

Granulosa cell tumor

Despite its rarity, the granulosa cell tumor is the most common tumor associated with estrogen production. Mostly seen in its adult form in the postmenopausal period, the granulosa cell tumor has

the potential to transform into malignancy. The estrogen production can produce abnormal uterine bleeding as well as abnormally thickening endometrium (hyperplasia) in the postmenopausal patient. A tenth of these cases may develop into endometrial carcinoma.

An extremely rare juvenile form of granulosa cell appears mostly in young adults and in prepubescent children. Estrogen production in these cases tends to cause abnormal female development and menstrual bleeding (precocious puberty).

Uterine, cervical, and vaginal abnormalities

Along with the ovaries, the uterus can also develop abnormalities that tend to interfere with the reproductive cycle. Masses in the muscle of the uterine wall can compress against the inner cavity, upsetting the function of the endometrium. Abnormalities of the endometrium also can have a profound effect on the reproductive functioning. Cervical and vaginal abnormalities can prevent reproduction from ever taking place and interrupt menstrual flow.

Uterine abnormalities

Most uterine abnormalities originate within the muscular wall of the uterus. Normally, this is rarely a problem for the female if it is not too large and does not affect blood flow or the endometrium. The presence of masses in the uterine wall is usually of concern only as it relates to the proximity of the endometrium.

Leiomyoma

Leiomyomas are the most common uterine tumors and are seen in up to half of women of reproductive age. They are frequently referred to as *fibroids*. The tumors arise from the smooth muscle of the myometrium. The proliferating muscle cells of the tumor are arranged in a circular pattern interlaced with fibrous connective tissue. The compression of the surrounding myometrium forms a pseudocapsule around the leiomyoma. Other less common sites for leiomyomas are the cervix and pelvic ligaments. The lesions are considered benign.

Leiomyomas respond to estrogen hormones with growth, which explains why some (but not all) will rapidly increase in size during the hormone elevations of pregnancy. Throughout the reproductive years, a leiomyoma may gradually grow larger and then begin to shrink in the postmenopausal period due to the cessation of hormones. Occasionally, the tumors can grow so large that they may outgrow their own blood supply, causing ischemic changes and degeneration of tissue. The resulting necrosis tends to liquefy and produce cystic areas within the tumor.

Leiomyomas are frequently asymptomatic; however, some can cause general symptoms due to enormous size and specific symptoms due to location. Types of leiomyomas are classified by their location in the uterus.

- *Intercavitary*—Located within the endometrial cavity, causing heavy (menorrhagia) or irregular (metrorrhagia) menstrual bleeding and may interfere with pregnancy implantation.
- *Submucosa*—Located beneath the endometrium against the basal layer, causing menorrhagia or metrorrhagia and deforming the endometrial cavity, which can cause spontaneous abortion.
- *Intramural* (also called *interstitial* or *myometrial*)—Located within the middle of the uterine wall; the most common location for leiomyomas and the one presenting the least symptoms unless hemorrhaging or necrotic.
- *Subserosal*—Located on the surface of the uterus but beneath the serous membrane that covers it; may grow large enough to adversely press against other organs such as the bladder or bowel, which can cause dysuria and constipation; some subserosal tumors extend out into the pelvis and are attached to the uterus by a slender peduncle, and thus they are called *pedunculated fibroids*; the pedunculated variety may twist on the pedicle (torsion), cutting off blood flow to the leiomyoma, which can generate enormous pain.

Adenomyosis

Another benign condition of the myometrium actually originates in the endometrium. Adenomyosis is the presence of clusters of endometrial glands and stroma scattered throughout the myometrium of the uterus. It occurs mostly in multiparous women in their forties and fifties. This is a highly suggestive way to distinguish this condition from endometriosis, which typically occurs in nulliparous women before the age of 30 and is usually a distinct contributor to infertility. Similar to endometriosis, adenomyosis is a scattering of endometrial glands outside of the endometrium. However, with endometriosis, the ectopic glands are distributed throughout the entire pelvis. The symptoms of adenomyosis are painful menses (dysmenorrhea) and, often, heavy menses (menorrhagia).

Endometrial hyperplasia

The abnormal increase in the number of endometrial glands is called endometrial hyperplasia. Continuous (unopposed) stimulation of estrogen causes the hyperplasia. Several conditions give rise to high levels of estrogen that may cause the endometrium to proliferate. For instance, in late premenopausal women, estrogen elevations are caused by anovulation, polycystic ovarian disease, obesity, and some estrogen-secreting ovarian tumors. In postmenopausal women, the estrogen administration of hormone therapy increases the risk of hyperplasia, particularly if unopposed estrogen is given in women who have *not* had a hysterectomy (though this is rarely done today).

Endometrial hyperplasia commonly causes abnormal endometrial bleeding in pre- and postmenopausal women. The condition is usually divided up into three general categories: simple (cystic), complex (adenomatous or glands crowding out stroma), and atypical complex (nearly complete crowding of endometrial glands with little stroma). The atypical complex is the type that has the higher risk of transforming into carcinoma. Endometrial biopsy is usually performed to distinguish between these tissue types. Sonography, however can detect the simple cystic hyperplasia, but other conditions such as polyps and even carcinoma can also contain cysts. Because most endometrial abnormalities have similar sonographic appearances, the gold-standard for confirmed diagnosis remains the biopsy.

Endometrial polyps

Seen in women of all ages, endometrial polyps are common, asymptomatic lesions of the endometrium that can cause menorrhagia or metrorrhagia. They are normally benign growths arising from the endometrium composed of glands, stroma, and blood vessels. Polyps can have stalks attaching them to the endometrium or they can be flattened. Most appear singularly but can also be multiple.

Endometrial carcinoma

Found mostly in postmenopausal women, endometrial carcinoma is the most common gynecologic malignancy. Yet it is the least likely to result in death; as the late detection of ovarian carcinoma is the leading cause of gynecological cancer deaths. Common risk factors for endometrial carcinoma (specifically adenocarcinoma) are diabetes, nulliparity, obesity, hypertension, late menopause, history of breast disease (and also tamoxifen administration for breast cancer treatment), and prolonged estrogen stimulation. The primary symptom for endometrial carcinoma is uterine bleeding.

Cervical and vaginal abnormalities

The most common benign abnormality of the cervix is the presence of *nabothian cysts*. You will frequently see these cysts referred to as *Nabothian* cysts or *Naboth's* cysts, with the latter being more accurate if the proper name is used. These cysts are formed when one or more cervical glands are blocked off or occluded, causing the retention of glandular fluid. Thus, they are sometimes called *retention* cysts. They may be singular or multiple. Unless inflamed from infection or hemorrhaging, the nabothian cyst is usually asymptomatic.

The most common *solid* masses in the cervix are leiomyomas. As with uterine leiomyomas, these can form within the muscular layer of the cervix or on the surface. They are also of concern if they

impinge or block the cervical canal. Within the cervical canal itself, polyps can form, which may cause abnormal vaginal hemorrhage.

Vaginal abnormalities are rarely encountered in sonography. Occasionally, a benign cyst will form congenitally within the anterolateral wall of the vagina, called a *Gartner cyst*. Usually, you will see and hear it referred to as *Gartner's duct cyst*. These cysts, like the nabothian cysts of the cervix are usually asymptomatic.

405. Imaging pelvic inflammation and torsion

Infection is the usual cause of inflammation in the body, which normally is accompanied with pain. Aside from laboratory and clinical diagnosis, most physicians will refer the pelvic inflammation or pelvic *pain* case to the sonography department for either confirmation or diagnosis. In this lesson, we will briefly look at what it takes to recognize and image the inflamed pelvis.

PID

Normally, sonography is requested not to diagnose PID, but to determine the presence of a TOA, which may dangerously rupture if untreated. Sonography plays an adjunctive role with PID cases. The combined sonographic appearances of inflamed endometrium, uterine tubes, and ovaries *assist* with making a diagnosis of PID. Sonography results coupled with patient history, lab cultures, and clinical examination are necessary before PID is confidently diagnosed.

Recall that PID involves a spectrum of related abnormalities affecting the reproductive pathway. Infection and inflammation is spread through the endometrial canal, the uterine tubes, and finally the ovaries. Thus, PID can affect the anatomic structure of the reproductive organs and leave clues that appear sonographically. As you scan, you should see endometritis as a thickened and irregular endometrial stripe, often accompanied with anechoic fluid. Gas and debris within the cavity may cast echo-scattering shadows. Endometritis is usually seen endovaginally. As with normal endometrial imaging, measure the AP diameter of the thickened endometrium in the sagittal endovaginal view.

You will likely *not* see salpingitis until it becomes a *pyosalpinx* or *hydrosalpinx*. On sonography, the presence of a tortuous, anechoic or hypoechoic tubular structure in the adnexa provides the clue of an inflamed tube. Unlike the anechoic lumen seen with hydrosalpinx, pyosalpinx usually contains internal echoes, making the lumen hypoechoic. Septations span halfway across the tube, which represent folds of tube wall. You should attempt to locate the ovary and document its relationship to the pyo- or hydrosalpinx. Some women will find this extremely painful. The compression from the probe tip may cause the ovary to move in a direction different from that of the distended tube. This separation of tube from ovary with pressure will rule-out the presence of a tubo-ovarian complex. However, if a recognizable ovary and tube move together as a unit when pressed with the vaginal probe, then tubo-ovarian complex should be considered. Document this situation by using side-by-side panels that show before and after images of the complex moving as a unit. Your radiologist may find it helpful if you measure the diameter of the distended tube. Also, measure the diameter of the wall, which in nearly all cases of acute PID with pyosalpinx, is greater than 4 mm. The walls of a hydrosalpinx are usually a few millimeters. Place color Doppler over any tubular structure located within the pelvis, as it may turn out to be an abnormally enlarged blood vessel instead of a distended uterine tube. As much as possible, provide standard images with ovarian measurements.

When a heterogeneous mass with no recognizable ovary is present in the adnexa or the cul-de-sac, a TOA is the likely reason, specifically if the mass moves as a unit with vaginal probe pressure. A TOA can also be present with the ovary identifiable, yet the tissue around it is disorganized, moving with the ovary as a unit. Normally, a woman suffering from TOA will find vaginal probe pressure of any kind extremely painful. Attempt to measure the TOA length, width, and AP depth. Abscesses may form elsewhere in the pelvis and up into the flanks of the abdomen, so make sure to sweep the entire pelvis for abnormal masses. Transabdominal is particularly helpful for performing a general survey of the pelvis. To measure and document details of a TOA, use endovaginal sonography.

Torsion

In cases of torsion, sonography can be helpful but is generally not as specific as once thought. The classic sonographic criterion for ovarian torsion is the absence of a Doppler signal (color and spectral) within the ovary tissue, signifying absence of blood flow. This criterion has been modified over the years, largely as a result of the improvement in Doppler sensitivity on ultrasound equipment. Blood flow (no matter how slight) is now known to occur in the presence of some cases of torsion, reducing Doppler's usefulness as a criterion for the condition. Most sonographers and radiologists find the combination of a patient's right-sided pain, an enlarged and hypoechoic ovary with or without small follicles on the periphery, to be highly suggestive of torsion. Keep in mind, however, that Doppler is useful information, and should be performed on not only the affected ovary but also the unaffected ovary.

Sonographic images of ovarian torsion are usually of an enlarged ovary of 4 cm or more in size, reaching 10 cm in some cases. Your measurement of the ovaries may be difficult due to the presence of cysts. The affected ovary may be located superior to the fundus of the uterus and is commonly seen in the midline. The presence of a cyst or solid tumor within the ovary can have a range of appearances depending on if it hemorrhages. The cyst can appear from anechoic to complex to completely echo-filled, resembling an endometrioma or tubo-ovarian abscess. Also, the ovarian pedicle itself may twist to a point where the ovary itself is wrapped up in broad ligament, vessels, and even a dilated uterine tube. This entire mass may present a range of echotextures that can be quite confusing when combined with hemorrhage and the presence of a cyst. In suspected torsion, Doppler sonography can help make some sense of the adnexal mass if a small amount of blood flow is detected in a whirlpool or spiral configuration in the adnexa. The swirl of reduced flow represents the twisted vascular part of the pedicle. Thus, torsion may be present even within the twisted pedicle. Finally, you will usually see free fluid in the cul-de-sac, particularly if there is hemorrhaging.

Realize that sonography is helpful and suggestive for detecting torsion. However, certain factors must be in place before a high probability of torsion is found. The presence of any one of the above sonographic findings may prompt some radiologists to suggest torsion, whereupon the next step is surgery. Some radiologists will determine that a normal appearing ovary, in size and echotexture, is suggestive enough to rule out torsion. Others will insist on the presence of blood flow to rule out torsion. Your job will be to make sure that your sonographic equipment, particularly Doppler sensitivity, is set to optimum levels. By obtaining clear images of ovaries, masses, or unusual adenexal structures, you can do your part in assisting radiologists with the diagnosis by showing them what is there.

406. Imaging ovarian abnormalities

Most pelvic masses seen outside the uterus with sonography are cysts, with the most common being those that involve the ovary. Many ovarian cysts usually encountered change according to the hormonal status of the female, such as the functional cysts, and most are benign. The appearances of ovarian cysts range from simple anechoic cysts to nearly solid-appearing complex masses. Most of the complex cysts are due to hemorrhage and are usually the cause of pain. Otherwise, ovarian cysts are generally asymptomatic.

Functional cysts

The sonographic appearances of functional cysts are variable, depending upon the type of cyst and whether it is hemorrhagic. Understand that any type of functional cyst can hemorrhage, either in the form of leaking or rupture, with blood flowing to the outside or inside of the cyst. Once convincing sonographic evidence is seen of hemorrhage, most radiologists would label the cyst a *hemorrhagic cyst* (fig. 1–8). For you to properly recognize the sonographic appearance of a hemorrhagic cyst depends on how long bleeding has occurred. Fresh or acute bleeding of a functional cyst nearly always displays a hyperechoic mass, with a solid-looking homogeneous appearance. A telltale sign is

the presence of distal acoustic enhancement posterior to the cyst. Also, the acute hemorrhagic cyst is almost always accompanied with pain.

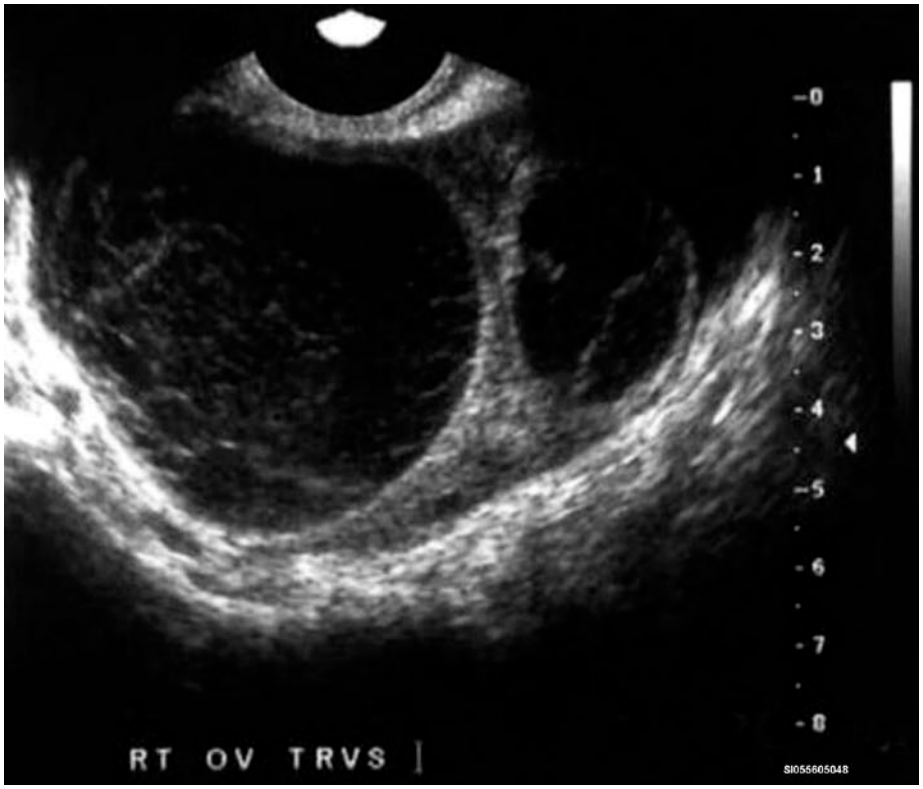

Figure 1–8. Hemorrhagic cyst.

As a hemorrhagic cyst ages, the blood begins to congeal and clot, which changes the sonographic appearance. You should see old blood displayed on sonography as a heterogeneous mass with extremely thin hyperechoic strands in a reticular or branching pattern throughout the cyst. Older blood will progressively become more anechoic as time goes on. Occasionally, fluid-fluid levels will be seen, with the low-level echoes of old heavier blood cells settling according to gravity and the more anechoic fluid component resting above. Generally, hemorrhagic cysts regress and are reabsorbed, which you should notice on follow-up sonograms after approximately a month.

Follicular cyst

Because follicular cysts are responsive to the hormonal cycle, they will usually change in sonographic appearance. You should recognize the appearance usually seen with follicular cyst, which is that of the classic sonographic criteria for a simple cyst: round or ovoid, smooth thin walls, unilocular (one chamber or cavity), anechoic, and posterior acoustic enhancement. Hemorrhage into the cyst causes low-level internal echoes, which you may be able to note by examining the change in echogenicity according to age and subsequent clotting. If the cyst is hemorrhagic and is leaking or has ruptured, you may find a slightly abnormal amount of anechoic or hypoechoic fluid in the cul-de-sac.

Using endovaginal sonography, obtain measurements of the length, AP, and width of the folliclar cyst. Follicles have varying sizes at maturity, from a few millimeters up to approximately 2.5 cm; therefore, your scan measurements of follicular cysts may yield sizes from 2.5 cm up to as large as 20 cm, with most falling into the 2.5 to 8 cm range. Be sure to place a color Doppler box over the cyst to determine if blood flow is present within it, particularly when the appearance is complex. Presence of blood flow within it rules out the possibility of a follicular cyst and may instead suggest a malignant neoplasm.

Corpus luteum cyst

Sonographically, your scans should reveal low-level internal echoes with the corpus luteum cyst. Occasionally, you should recognize a thick and notched cyst wall, and echogenic septations (fig. 1–9). Otherwise, its appearance may be similar to a follicular cyst and can vary considerably. The corpus luteum cyst usually has an extremely hypervascular rim, notable by placing color Doppler over the cyst, revealing bright colors relative to the surrounding tissue. The size of the cysts can grow beyond 10 cm, particularly during the first trimester where it may or may not regress. Usually, the size is no larger than 5 cm and the corpus luteum cyst will spontaneously collapse into a solid echogenic mass that *may* retain the hypervascular rim seen with color Doppler. Measure the corpus luteum cyst in the length, AP, and width dimensions.

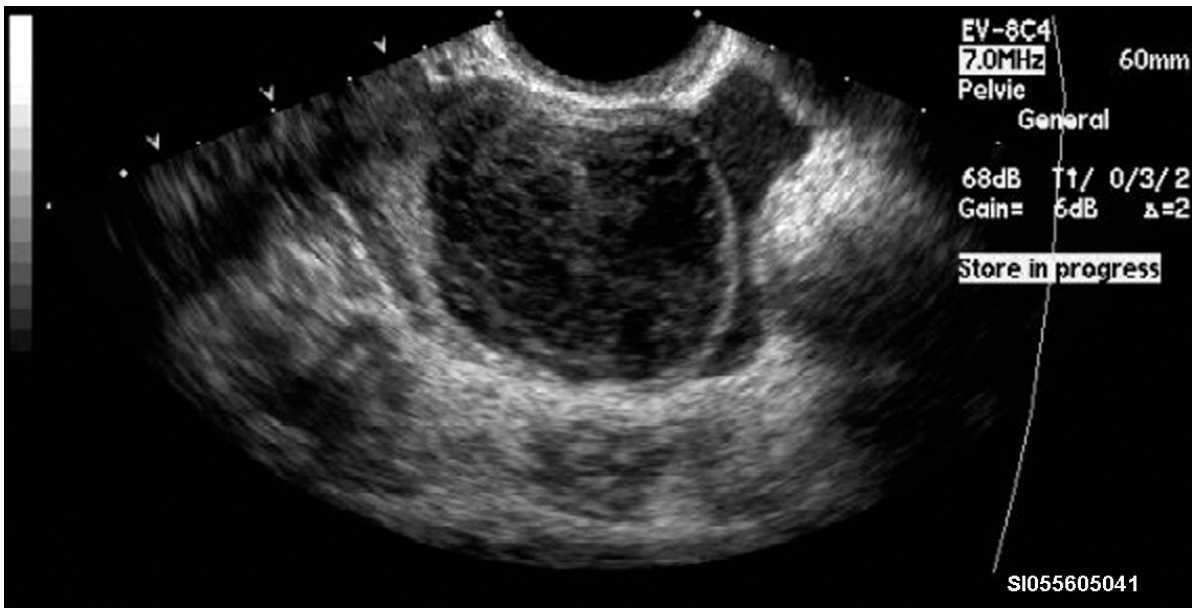

Figure 1–9. Cyst with low-level internal echoes.

Do not be confused by the various ways used by radiologists and sonographers to name this abnormality. You will generally hear corpus luteum and corpus luteum cyst being used interchangeably. Either manifestation produces progesterone. Remember that the *corpus luteum* is the remnant yellow body or mass of a mature follicle that has ovulated. Once ovulation occurs, the follicle's fluid escapes into the pelvis and eventually settles into the cul-de-sac (fig. 1–10). The mass left behind is the transformed (luteinized) wall of the follicle, which is called the corpus luteum. If the central cavity within the corpus luteum begins to build up fluid (*serous or blood*), the mass should be considered a corpus luteum *cyst*. If the cyst ruptures, it is likely due to continuous internal bleeding; that is, a *hemorrhagic* corpus luteum cyst has ruptured. If it doesn't rupture, the corpus luteum cyst will eventually regress or resolve after one or two cycles.

The single most important *sonographic* factor to distinguish the corpus luteum cyst, or sometimes even the corpus luteum, from any other ovarian cyst or mass is the hypervascular peripheral rim within the ovary. Be aware that the hypervascular rim is also similar to that seen with ectopic pregnancies, which we'll discuss in a later unit. The best way for you to distinguish between the two is to know that ectopics are mostly seen outside of the ovary. Finally, the low-level internal echoes, if present, of corpora lutea cysts may resemble that of endometrial cysts (described below). However, the echotexture of the endometrial cyst is more homogeneous throughout than the coarser internal echoes of the corpus luteum cyst. Also, endometrial cysts do not have hypervascular rims.

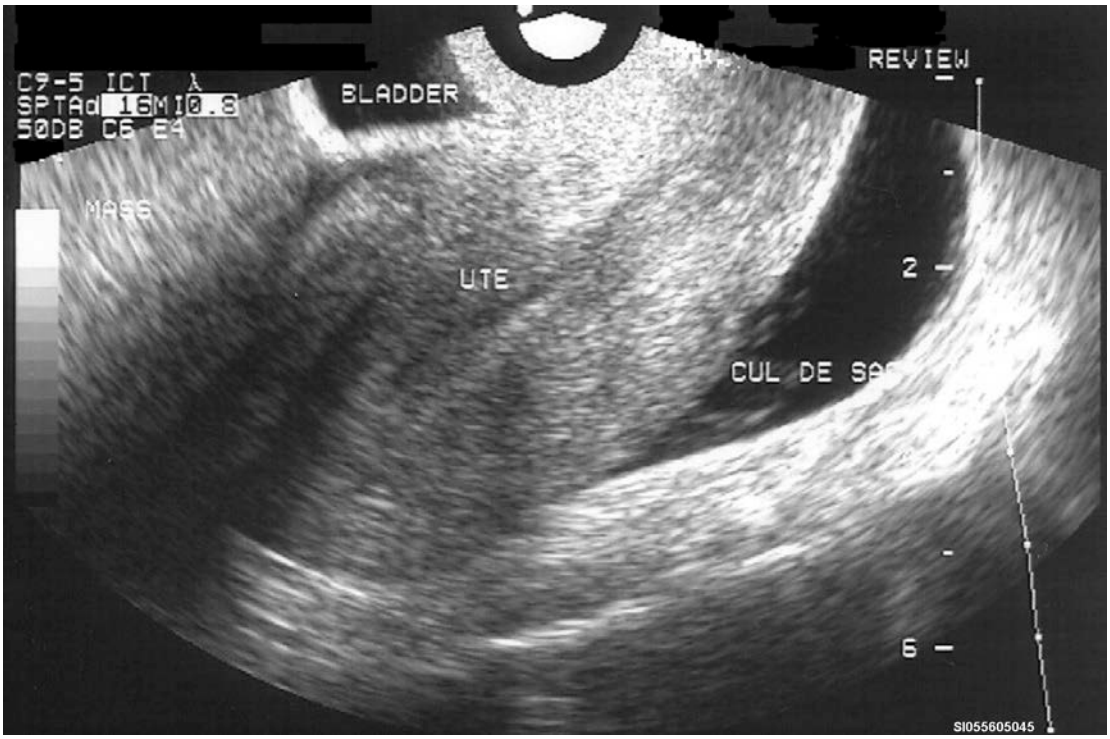

Figure 1–10. Fluid in the cul-de-sac.

Theca-lutein cyst

On sonography, the usual clue that suggests you may be seeing theca-lutein cysts is the characteristic appearance of bilateral, large, multiloculated cysts resembling a cluster of grapes obscuring the outline of the ovaries. If seen, measure the entire mass of cysts in three dimensions. The radiologist may also require you to measure the largest cyst. Use color Doppler to document the presence of any blood flow within the mass.

Other adnexal cysts

Accurate identification of ovarian cysts depends on a combination of patient history and the images you document that display cyst characteristics. However, another important factor that you must determine is whether the cyst is located within the ovary or outside it. Because of the compactness of the area surrounding the ovary (adnexa), the sonographic distinction is not always readily apparent. Cysts which arise from the surface of the ovary and protrude outward into the adnexa may appear to separate from the ovary. The cysts discussed briefly below represent some of the adnexal cysts that can easily be confused with typical ovarian cysts on sonography. Polycystic ovaries are the exception in that they are not true cysts, but rather represent an increase in the number of follicles with cystic appearances. We include them because the condition is also confused with ovarian cysts.

Paraovarian cysts

Usually, you will discover a paraovarian cyst incidentally during a pelvic sonogram. They have an identical sonographic appearance to classic, simple functional cysts of the ovaries. With hemorrhage or rupture, they may also become complex with internal echoes, as with functional cysts. As you scan, you should be able to locate most paraovarian cysts at a level above the fundus of the uterus. They are usually a few centimeters in diameter, but can also appear as large as 10 centimeters. Because they resemble a functional cyst so much in appearance, the best way for you to distinguish the paraovarian cyst is to sonographically prove that it is unconnected to the ovary. The most convincing way to do this is to demonstrate, on the same endovaginal image, the ovary and paraovarian cyst separate from each other. Also, place color Doppler over the cyst to ensure that

blood flow is not a part of its composition, as the paraovarian is avascular. Measure the cyst in all three dimensions.

Polycystic ovaries

Sonographically, polycystic ovaries present as bilaterally enlarged and rounded ovaries, usually twice the normal size, with a number of small follicles seen at the periphery or scattered throughout the ovary. In most cases you should see a central, echogenic ovarian stroma or parenchyma, which you should document. Significantly, the number of follicles, which varies from 5 to 15, is not really a sensitive indicator of PCOS, but rather the *absence* of a dominant follicle. However, since dominant follicle sizes vary considerably from woman to woman, this type of criterion is not specifically useful during a sonographic evaluation.

Other factors, such as ovarian size, peripheral location of the follicles, follicles no more than 4 or 5 mm in diameter, and particularly echogenic stroma, serve to help you sonographically confirm the disorder. Do not be surprised if your radiologist or department sets differing criteria for diagnosing PCOS. For instance, with endovaginal sonography, more than 5–11 follicles is an indicator of PCOS in some departments. Other radiologists may consider PCOS to be present *only* when the follicles are arranged peripherally. PCOS is usually diagnosed biochemically through laboratory values and sonography is normally only helpful for confirmation.

Naturally, measuring the ovary is routinely required. However, consider also measuring one of the follicles in an AP manner. This will helpfully give the radiologist the general sizes of the other follicles.

Endometrial cysts (endometriomas)

On sonography, you will not see evidence of endometriosis without cyst formation, as the lesions are too small for ultrasound resolution. With endometrial cysts, you may see one or multiple cysts, which you should measure. This should yield sizes generally 5 to 8 cm in diameter, but can reach sizes up to 15 cm. Recognize and document certain features of the endometrial cyst. For instance, the walls of the cysts are usually thick and hyperechoic, but can be thin-walled. Bowel loops may be attached to the cysts as a result of adhesions that form from inflammatory reactions to menstrual shedding. The internal characteristic of the endometrial cyst is variable, but usually is of homogeneous low-level echoes throughout.

You should be able to distinguish the endometrioma from the hemorrhagic cyst by noting the absence of septations and fluid-fluid levels usually seen within hemorrhagic cysts. More importantly, the endometrioma does not regress with the menstrual cycle. Typically, hyperechoic foci are seen in the walls of the cyst. Up to half of endometrial cyst cases will be bilateral. As with any mass seen within the pelvis, place color Doppler over it to determine possible blood flow. Measure the endometrial cyst in all three dimensions. Many times, the ovarian measurement will be greatly expanded as a result of the endometrial cyst nearly replacing the ovary.

Ovarian neoplasms

For sonographers, ovarian neoplasms may be encountered incidentally during an examination performed for other reasons. Most, however, are referred due to pain or discomfort from increasing size of the mass, known or unknown. When the mass is solid or solid with cystic areas, physicians become concerned with determining if the mass is benign or malignant. Although sonography cannot distinguish malignant tumors from benign, it can display suggestively useful clues. As you scan, you may be able to recognize sonographic masses in the adnexa by noting the following appearances that generally suggest malignancy:

- Complex mass (mostly cystic or multiloculated cysts with solid elements such as mural nodules, thick septations, and irregular wall thickening).
- Solid masses (may be with poorly defined margins, signifying spread to nearby structures).

- Doppler flow demonstrates new blood vessel formation (neovascularization) in the center of the mass (color Doppler may reveal tortuous vessels and spectral Doppler may reveal low resistance to incoming flow to the tumor, with a resistive index of 0.40 or less and a high diastolic signal).
- Ascites in the pelvis and abdomen (sometimes called malignant ascites in stages III and IV, especially if solid masses are seen attached to bowel loops or other surfaces, which is called peritoneal metastases).

Normally, the sonographic appearance for metastatic disease to the ovaries is of solid, hypoechoic, and bilateral tumors. If necrotic, they can have a cystic appearance that mimics primary ovarian cystic carcinomas. Ascites, if present, frequently is seen in the pelvis with fluid in the cul-de-sac. Lymphomatous tumors to the ovary usually have a solid but mottled appearance on sonography. Again, metastasis to the ovary is relatively rare, and thus, we will concentrate on the epithelial tumors.

Epithelial ovarian tumors

If you wind up detecting serous or mucinous tumors in your scan of the ovaries, you will routinely document the dimensions of the masses. However, the radiologist will also be concerned about features which may provide *clues* that masses are either serous or mucinous, and features which may further *suggest* the masses are benign or malignant.

Serous cystadenoma and cystadenocarcinoma

The sonographic appearance of the serous cystadenoma is typically that of a unilocular, anechoic cyst with thin walls and, in some, a few thin septations. Be sure to note the occasional tiny projections (papillae) seen protruding from the wall or septa; however, more fully developed papillae indicate a borderline or low malignant potential mass. Serous cystadenomas generally grow to approximately 10 cm in diameter, with some reaching 30 cm. Measure the serous cystadenoma in all three dimensions. Use color Doppler to determine vascularity of the mass, as flow detected within the mass having low resistance suggests malignancy.

With serous cystadenocarcinoma, you will usually see pelvic and abdominal ascites. The walls and septations of the mass may be thicker, but are usually just as thin as serous cystadenomas. However, *multiple* septations distinguish serous cystadenocarcinomas from serous cystadenomas, making the mass usually multilocular. Another distinguishing feature is the large number of fully developed papillary projections seen extending from both wall and septations of the serous cystadenomacarcinoma, along with the occasional calcification. Color and spectral Doppler reveals low resistance flow in both walls (Resistive Index [RI] less than 0.40) and in thickened septations. As with other masses, measure the serous cystadenocarcinoma in three dimensions. Also survey the tissue around the lower abdominal aorta for lymph node metastasis as well as other areas such as the liver and kidneys.

Mucinous cystadenoma and cystadenocarcinoma

On sonography, the benign mucinous cystadenoma appears as a well-defined, cystic mass with somewhat thicker and more numerous septations than those found in serous tumors. It can resemble a serous cystadenocarcinoma because of its multilocular nature. Thus paying particular attention to the thickness and quantity of septations will help distinguish between the two. Also, debris may be seen within the separate cyst-like chambers of the mucinous cystadenoma. Perhaps a bigger discriminator would be the sizes of the mucinous cystadenomas, which are typically larger than the smaller serous tumors. Sizes can reach up to 50 cm. Thus, sonographic measurement may be extremely difficult with the small field-of-view of endovaginal probes. In such a situation, use the transabdominal probe to acquire the entire mass for measurement.

More solid areas and septations than that found in mucinous cystadenomas tend to suggest malignancy. On sonography, the walls of the mucinous cystadenocarcinoma may be ill-defined with

papillary projections. The septations will usually be thick and packed with solid areas, which will be hyperechoic or heterogeneous from hemorrhage and necrosis. The tumor, being mucinous, is of an enormous size; frequently, it can reach beyond 30 cm. Doppler will reveal low-resistance blood flow with an RI of 0.40 or less. Along with measurement and Doppler assessment of the tumor, a survey of the abdomen for metastasis is appropriate.

You may detect ascites, which can produce symptoms resembling that found with serous cystadenocarcinomas. However, if the apparent ascites seen in the pelvis or abdomen contains multiple septations, it is likely the gelatinous material (mucin) of a ruptured mucinous cystadenocarcinoma. This condition is called *pseudomyxoma peritonei*. Document thoroughly.

Epithelial Ovarian Neoplasms		
Type	***Characteristics***	***Sonographic Appearance***
Serous cystadenoma (benign)	Second most common *benign* ovarian neoplasm	• Unilateral/unilocular • Thin wall, very thin septations with occasional papillary projections • 10 cm but can reach 30 cm (smaller than mucinous)
Serous cystadenocarcinoma (malignant)	Most common ovarian malignancy	• Unilocular or multilocular/unilateral or bilateral • Thicker irregular walls with thicker septations • Increased papillary projections with solid nodules as well as calcifications • Ascites
Mucinous cystadenoma (benign)	Second most common epithelial ovarian neoplasm	• Unilateral/ multilocular • Well defined margin, more septations than serous tumors, debris may be seen within separate chambers. • Enormous mass can reach up to 50 cm
Mucinous cystadenocarcinoma (malignant)	Uncommon	• Unilateral/multilocular • Ill-defined border, with increased number of septations and solid areas than mucinous cystadenoma • Papillary projections, complex areas representing hemorrhage and necrosis • Massive tumor beyond 30 cm • Ascites or pseudomyxoma peritonei after mass rupture

Germ cell ovarian tumors

Teratomas are primarily the only germ cell tumors of the ovary you are likely to encounter. Again, they come in two varieties, mature and immature. Distinction between these tumors and various complex cysts can be difficult. The following approaches to imaging germ cell tumors can be helpful to suggest the type and help the radiologist rule out other masses.

Mature cystic teratoma (dermoid cyst)

As you scan the suspected cystic teratoma, keep in mind that the sonographic appearance ranges from completely anechoic to complex to completely hyperechoic. Typical appearances of a dermoid mainly are of a fluid-fat level seen with one half of the tumor echogenic and homogeneous (fat) and the other side anechoic or hypoechoic (fluid). Occasionally the echogenic area will be anterior to the anechoic area, despite gravity; suggesting the presence of a dermoid rather than a hemorrhagic cyst, where the echogenicity corresponds to heavy blood clot that settles with gravity. Most dermoids are also accompanied by an echogenic mural nodule protruding from the wall of the tumor toward the center. This solid material is called either a *dermoid plug* or a *Rokitansky nodule*. Some will have bright hyperechoic structures which shadow, which are typically mixtures of hair, fat, and sometimes bone. Frequently, a bright echogenic line will lie along the fluid-fat interface level. This represents hair. Any of the above appearances must be carefully rendered in grayscale and clearly labeled for the radiologist.

Apart from these classic appearances, you should be able to recognize several other characteristics of the dermoid cyst, which may help distinguish it from endometrioma and hemorrhagic cysts.

- Anechoic cystic tumor with a mural nodule containing shadowing calcification.
- Tip of the iceberg sign (highly echogenic crescent seen of the anterior portion of the tumor, with significant attenuation of the ultrasound beam posteriorly; the result is shadowing that obscures most of the body and the posterior portion of the tumor).
- Hyperchoic lines and dots (hair) distributed throughout an anechoic cystic area.
- Most dermoids attenuate rather than enhance sound transmission.
- Completely hyperechoic, solid appearing tumor.

When you measure the dermoid, remember that the size ranges from 0.5 cm to an enormous 40 cm. However, most of the dermoids you will encounter will fall within 5 to 10 cm, and will hardly ever get above 15 cm. When you place electronic calipers to measure the cystic teratomas, displaying the tip of the iceberg sign can be challenging. A useful method will be to measure the length and width of the tumor, and thus estimating the AP dimension. Place color Doppler over the tumor to ensure that blood vessels are not present (which will indicate either a struma ovarii or a malignancy).

Immature teratoma

On sonography, the clue suggesting an immature teratoma will usually be limited to the age of the patient and the presence of a solid tumor. Again, they occur in children and adolescents and are malignant. Thus, if a young female has a solid mass in the ovary, the potential for it to be an immature teratoma is increased. As you scan, be aware that some immature teratomas will contain multiple tiny cysts and a complex appearance, making it difficult to determine its nature. A few will contain calcifications. Most immature teratomas are small in size but can grow up to 25 cm in diameter. Measure the tumor as you would any other mass of the body. Ensure that you use color Doppler to document the presence of feeder blood vessels.

Dysgerminoma

You may see the occasional dysgerminoma on sonography to be rounded, solid, and echogenic. Your use of color Doppler should reveal the highly vascular characteristic of the dysgerminoma, a feature which tends to cause hemorrhaging and necrosis. They typically measure no more than 15 cm in diameter. Some, however, have been known to reach over three times that size.

Germ Cell Ovarian Neoplasms		
Type	***Characteristics***	***Sonographic Appearance***
Mature cystic teratoma, (*dermoid*) (benign)	• Most common benign germ cell tumor • Most common benign ovarian neo-plasm in women of reproductive age • Composed mostly of skin, fat, hair, teeth, and bone	• Classic fluid-fat level, or anechoic cyst with dermoid plug • Tip-of-the iceberg appearance • Hyperechoic foci with shadowing • 5–10 cm
Immature teratoma (malignant)	• Appears mostly in women in the first two decades of life	• Primarily solid tumor (some with multiple cysts or calcifications) • Up to 25 cm
Dysgerminoma (malignant)	• Most common ovarian malignancy of childhood	• Round, echogenic, solid, and lobulated with septations • Highly vascular • Typically no more than 15 cm

Sex cord-stromal tumors

Sonographic appearances of the typical *fibroma* are of a hypoechoic mass with an echotexture *similar* to that of a uterine fibroid; that is, you will see multiple edge shadows distally and note heterogeneous sound attenuation throughout the mass. Recognize the potential for this and document accordingly. Some fibromas are hypoechoic with significant degrees of attenuation, so you may have to adjust

your gain or switch to a lower frequency for beam penetration. Fibromas that nearly replace entire ovaries can be difficult to detect sonographically or missed altogether, particularly if they are highly attenuating. Therefore, if you are having trouble locating an ovary *and* you notice an area of profound shadowing, suspect a fibroma. Occasionally, you may see cysts and calcifications. Measure the mass in all three dimensions.

Sonographically, the *thecoma* is also similar to fibromas. The sizes of both these solid tumors are wide ranging from very small to enormously large, but most will fall in between 5 and 10 cm. Because most of the thecoma tends to attenuate sound, sonography cannot reliably distinguish between fibromas and thecomas.

Granulosa cell tumor

On sonography, the granulosa cell tumor is unilateral. They have varying sizes and can grow quite large, typically round 12 to 20 cm in diameter. Small granulosa cell tumors are generally hypoechoic and solid with an echotexture similar to that of a fibroid. Larger tumors usually will be multiloculated and cystic, making it difficult for you to distinguish it from an epithelial cystadenoma. These masses, if large enough, can hemorrhage, rupture, or cause torsion.

407. Imaging uterine, cervical, and vaginal abnormalities

Apart from the ovaries, the uterus is also a primary concern during sonography. The cervix and vagina, when abnormal, are usually quite obvious on sonography. Otherwise, most of your attention will be on abnormalities of the uterus.

Myometrium

Several abnormalities of the uterine wall, or myometrium, can be seen through sonography. Leiomyomas and adenomyosis are usually the only myometrial abnormalities to typically appear while myometrial malignancy will rarely be seen.

Leiomyoma

Most leiomyomas appear either focally or diffusely. Multiple, focal leiomyomas is the usual pattern which can be seen in most cases with endovaginal sonography. They can grow from tiny yet discrete nodules to massive sizes of at least 20 cm. Other tiny nodules are diffusely spread throughout the uterus, causing a general enlargement of the uterus.

On sonography, the classic leiomyoma is a round, hypoechoic, heterogeneous, smooth or irregular bordered, attenuating mass. Significant variations can occur from this basic structure. For instance, the internal echoes can be echogenic with increased amounts of fibrous connective tissue. Diffusely spread nodules may not readily show discrete borders, giving the entire uterus a heterogeneous appearance. Calcifications that cast shadows may appear within a leiomyoma, and some tumors will have a calcified rim that obscures much of the tumor through attenuation. Hemorrhaging leiomyomas may show anechoic cystic areas where blood has pooled or where necrotic tissue is present. Most of the above characteristics are readily seen by endovaginal sonography. Assessment of the leiomyoma becomes increasingly difficult the more the tumor attenuates the sound beam and with the increase in size.

With transabdominal sonography, the bladder affords a view of the anterior surface of the uterus, where the contour may be lumpy with subserosal or large intramural leiomyomas. Truly large leiomyomas are sometimes only assessed transabdominally. Use transabdominal technique to ease locating a pedunculated leiomyoma, but be aware that it may resemble an extrauterine pelvic mass. Using an endovaginal approach coupled with color Doppler, you should be able to assess where the stalk or peduncle branches out from the wall of the uterus toward the leiomyoma. Normally, you should be able to detect blood flowing through the stalk.

Sonographic measurements of the uterus should be obtained even in the presence of massive fibroids that distort the uterine size. Also, a submucosal or intercavitary leiomyoma should not prevent you

from attempting to measure the endometrial stripe, however distorted by the mass. Give the radiologist measurements of the widest portion of the stripe, which will probably contain most of the tumor, and measurements of nearby normal stripe thickness. Be careful of the intercavitary leiomyomas, as they can mimic an endometrial polyp. Look for continuation of echogenicity and echotexture in the mass with the wall of the uterus and the abrupt interruption of the endometrial line.

Accurate measurements of the leiomyoma can be taken as with any other tumor. Be aware that measurements can seem near impossible to obtain with truly massive tumors that attenuate much of the sound or cast multiple linear bands of shadows from within the tumor. Both conditions tend to obscure the posterior border of a leiomyoma. The situation can be even more complicated with the presence of a retroflexed or retroverted uterus. Useful methods for dealing with this situation are to use a transabdominal transducer with low frequency settings and to significantly increase the overall gain. The rest of the uterus will be brightly obscured, yet you may be able to obtain what would be obscured by normal settings.

Adenomyosis

Usually, distinct endometrial glands cannot be seen through sonography and instead the entire uterus is enlarged. The echotexture of the uterus will likely appear normal, but may be heterogeneous because of pooling blood from endometrial glands bleeding throughout the myometrium. This occurs because the glands actively correspond with the menstrual cycle, causing extreme pain. When a cluster of glands is large enough to be detected by sonography, it is called an *adenomyoma*, which can be extremely difficult to distinguish from a leiomyoma. However, its echogenicity and location in the intramural portion of the uterine wall may suggest adenomyosis if the patient is also complaining of intense pain. Further, cysts may appear within the adenomyoma or even in the uterine wall. Adenomyosis is the primary cause of such cysts, which are usually collections of blood from glandular bleeding. Although this can be challenging, you should attempt to measure the adenomyoma as well as any evidence of cysts. Most adenomyomas have ill-defined borders and can sometimes be nearly isoechoic with the surrounding uterus. Be careful of suggesting this condition if most of the above factors are not present and fully documented on sonography.

Endometrium

Recall that normal endometrial thickness in a woman of reproductive age depends on the menstrual cycle. The endometrium is thus at its widest diameter during the secretory phase of the cycle, which is normally no more than 15 mm. This will be fairly obvious to you. As you scan, you should note the bright echogenic endometrium which appears to fill most of the central portion of the uterus. Ensure the first day of the last *normal* menstrual period is known. Abnormal cycles are not very helpful in these cases.

During the postmenopausal years, the stripe should be no more than 8 mm in diameter and is usually less than 5 mm in asymptomatic women. Be sure to magnify your image slightly for accurate AP placement of your calipers. This is particularly important in borderline cases where the measurement hovers near 8 mm. The stripe may or may not be hyperechoic.

You should keep in mind that a lot of the postmenopausal thickness depends on which *hormone therapy regimen* is applied. Three types exist: *unopposed estrogen* administration, *continuous combined estrogen and progesterone* administration, and *sequential estrogen and progesterone* administration. Unopposed estrogen (that is, estrogen only is administered) is usually provided for women who have had a hysterectomy, or the removal of the uterus. Of the three, most postmenopausal women are given sequential hormones for the estrogen benefit. Be sure to either check the medical record or ask your patient what her hormone status is according to the regimen just described. This is important because, in postmenopausal women who use sequential hormone therapy, the thickness can increase up to 15 mm during the estrogen phase of the therapy regimen (first half of the monthly cycle). Without sequential administration, a measurement above 8 mm will be abnormal. With progesterone administration during the latter half of the month, the endometrium thickness

should gradually diminish. You should clearly note the thickness as being less than 5 mm during the bleeding phase. Thus, it is important to find out the type of regimen and the dates of administration and subsequent bleeding in the postmenopausal woman undergoing HRT.

Any endometrial thickness beyond the above normal limits is suspicious for hyperplasia, carcinoma, endometritis, leiomyomas, or even abnormalities associated with early pregnancy in reproductive age women. Normally, you should note the contour of the stripe as being smooth with a sharp interface between the endometrium and the myometrium. Any focal lumps or irregularities seen along the endometrial border are probably abnormal.

When you scan the inner cavity of the uterus, be vigilant for varying amounts of fluid, which can be present for both benign and ominous reasons. Anything that affects the endometrial lining can generate fluid within the cavity. As you scan, be aware that the most normal reason for small amounts of fluid seen within the endometrium in premenopausal women is the menstrual cycle, where the fluid is outflowing blood. If you see increasing amounts of fluid, the likelihood of an abnormality (benign or malignant) as the source also increases. Be sure that you document the fluid collection in three dimensions. The following abnormalities we will look at have the capability to generate abnormal amounts of fluid within the endometrium (usually abnormal blood).

Endometrial hyperplasia

On sonography in the *premenopausal* patient, the criterion suggestive for endometrial hyperplasia is an endometrial thickness greater than 15 mm. Keep in mind that the secretory phase may have thicknesses greater in some women, which may be due to other factors in the patient's medical history. The best thing to do in such situations is to confer with the radiologist. The best time for you to sonographically determine hyperplasia is just after the menstrual phase when the stripe should be around 4 mm in diameter. Anything measured between 4 and 15 mm is considered normal.

In *postmenopausal* women who are asymptomatic and who are *not* taking hormones, you should see and demonstrate the stripe to be thin and atrophic at all times. In these women, hyperplasia is suggested if stripe thickness exceeds 4 or 5 mm. You should note and document women on unopposed estrogen and continuous estrogen and progesterone administrations (daily); they also will have thin stripes, with hyperplasia suggested for any thickening above 5 mm to 8 mm. Ensure that you pay strict attention to the medical history of the patient relating to hormone therapy and correctly present the information to the radiologist.

For *peri-* and *postmenopausal* women on sequential hormone therapy, the stripe should be less than 5 mm just after the normal bleeding from hormone administration and up to 15 mm during the estrogen phase. 8 mm is considered the upper limit of normal during any part of the progesterone phase. However, the greatest sonographic determinant of hyperplastic endometrium in the sequential HRT patient is *unchanging* endometrial thickness throughout the cycle.

Endometrial polyps

On sonography, you should see the endometrial polyp to be round and echogenic, located within the cavity of the uterus. In premenopausal women, this appearance is readily seen during the proliferative or menstrual phase of the cycle, when the strip is thin. In the secretory phase, the echogenicity of the endometrium sometimes matches that of the polyp and can obscure it. It is best to examine the endometrium during the proliferative phase of the cycle for better visualization of polyps. Occasionally, polyps will have cysts within them. Use color Doppler to demonstrate blood vessels that course toward the polyps.

To measure the endometrial polyp, treat it as you would any other mass and use all three dimensions. Also, if visualization of a suspected polyp is difficult, the use of administering saline fluid into the uterine cavity (hystosonography or sonohistography) can outline the shape of a polyp. Sonohistography can help determine if a lesion has a stalk or if it is attached to the endometrial wall.

Sonohistography is normally a procedure that should only be done in coordination with established local procedures and with the radiologist.

Endometrial carcinoma

The sonographic appearance of endometrial carcinoma is usually of an abnormally thickened endometrium, which is virtually indistinguishable from hyperplasia and polyps. However, in suspected cases, look for certain clues that may help clarify the suggestion of malignancy. For instance, note the interface or border between the endometrium and myometrium, which may be blurred and irregular instead of smooth and sharp as with hyperplasia. The echotexture is also more heterogeneous than in the hyperplasia. Cysts also appear, but this is more prevalent for hyperplasia and polyps. Endometrial carcinoma may obstruct the genital canal causing a back up of fluid (hydrometra) and blood (hematometra).

Attempt to measure the thickness of the endometrium as much as possible. This may be exceedingly difficult given the ill-defined borders. Sonography is particularly helpful for documenting the extent of malignant invasion into the myometrium. Careful scanning should be performed to detect invasive spread. Not all invasion will demonstrate ill-defined borders, and you may see echogenic endometrium extending fingerlike projections deep into the myometrium.

Endometritis and IUDs

Recall that endometritis is associated with PID. An inflamed endometrium may also occur after dilation and curettage or postpartum. In these cases, look for the endometrium to be thickened, but not as much as with hyperplastic changes. The most outstanding feature of this abnormality for the sonographer is the presence of gas within the cavity. Be sure to demonstrate the gas by imaging the distal shadowing produced from *within* the canal. Pus with debris and strands (pyometra) may be present as well.

Intrauterine contraceptive devices (IUD; sometimes referred to by the acronym IUCD) are easy for you to see if located in or near the endometrial cavity. They are extremely hyperechoic linear structures of varying shapes and sizes. On sonography, you will usually see two bright, parallel lines that tend to shadow posteriorly, an appearance that you should document. The goal is for you to demonstrate position, possible perforation, or partial removal of the device. If any part of an IUD is located within the myometrium, perforation is highly suggested.

Cervix and vagina

Cysts of the cervix and vagina are occasionally detected with endovaginal sonography. The abnormality you are likely to see is the benign nabothian cyst. Most are a few millimeters in size yet can reach sizes of over 3 cm. You may find some with internal echoes from hemorrhage or infection, but most will be anechoic. Gartner cysts also may appear in the vaginal tract, which are more readily apparent if you withdraw the endovaginal probe a few centimeters away from the cervix.

Solid masses are usually seen in the cervix and not the vagina, the most common being leiomyoma. Cervical carcinoma has a similar appearance to a leiomyoma and cannot be readily distinguished on sonography. If you see an isoechoic to hypoechoic mass that displaces and interrupts the normal contour of the cervix, note that this is suggestive either of a benign or malignant lesion. Measure it in three dimensions as you would any other mass in the body.

Self-Test Questions

After you complete these questions, you may check your answers at the end of the unit.

403. General pelvic imaging

1. What are the advantages for the use of endovaginal probes during pelvic sonography?

2. List key information to be obtained from a pelvic sonography patient before the start of the exam.

3. What are the standard sonographic images of the pelvis transabdominally?

4. What are the standard sonographic images of the pelvis endovaginally?

5. Apart from thickness, what is the difference between the appearance of the endometrial stripe in the late proliferative phase and in the secretory phase?

6. List the normal variants of the sonographic uterine appearances?

7. Which congenital malformation is associated with infertility and spontaneous abortions?

404. Pelvic pathology

1. List the four infections which make up PID.

2. What are the organisms most responsible for PID?

3. What is hydrosalpinx?

4. How does TOA cause GI problems?

5. What is the most common ovarian malignancy?

6. List the major locations for leiomyomas in the uterus. ?

405. Imaging pelvic inflammation and torsion

1. How does pyosalpinx and hydrosalpinx sonographically differ?

2. Describe the sonographic appearance of TOA?

406. Imaging ovarian abnormalities

1. What happens to the sonographic appearance of a hemorrhagic cyst as it ages?

2. Why will follicular cysts change in sonographic appearance from cycle to cycle?

3. What will happen when you place color Doppler over a corpus luteum cyst?

4. What is the sonographic appearance of theca-lutein cysts?

5. What is the best way to distinguish the paraovarian cyst from a functional cyst?

6. What is the sonographic appearance of polycystic ovaries?

7. How would you distinguish the differences between endometrial cysts and hemorrhagic cysts?

8. List sonographic appearances of the adnexa that suggest malignancy.

9. Describe the classic sonographic appearances of mature cystic teratomas.

10. What sonographically makes the fibromas echotexture similar to a uterine fibroid?

407. Imaging uterine, cervical, and vaginal abnormalities

1. In the premenopausal patient, what is the criterion that suggests endometrial hyperplasia?

2. What's the normal limit for stripe thickness during the estrogen phase in postmenopausal women who use sequential hormone therapy?

Answers to Self-Test Questions

401

1. The floor muscles support pelvic organs, provide resistance to the downward force of intraabdominal pressure from actions such as coughing or defecation, and provide for contraction and relaxation of the rectum, urethra, and vagina.
2. $V = d_1 \times d_2 \times d_3 \times 0.523$.
3. Just beneath the germinal epithelium covering the ovary is a dense layer of connective tissue called tunica albuginea. The tissues directly beneath this tunica are functional cells, or parenchyma, of the ovary, composed of two layers: the cortex and medulla.
4. The basal layer is a thin layer that connects the deep endometrium to the myometrium, and the functional layer is a relatively thicker layer that contains glands and capillaries supported by loose connective tissue (stroma).
5. 7–8 cm in length, 3–5 cm in width, and 3–4 cm in height (AP diameter).
6. Serosa.
7. The right ovarian vein drains directly into the anterior IVC. Conversely, the left ovarian vein continues superiorly and drains into the left renal vein.

402

1. Estradiol matures and maintains the reproductive organs and sex characteristics as well as prepares the uterus for implantation. Progesterone plays a major role in preparing the uterus for implantation.
2. The excessive amount of estrogen production acts as a feedback on the pituitary and causes the reduction of FSH.
3. The proliferative phase (ovarian follicular) and the secretory (ovarian luteal).
4. In the postmenopausal woman, the supply of ovarian follicles is depleted, creating a lack of response to FSH. Thus, the sizes of the ovaries decrease significantly. Also, the endometrium becomes a thin, inactive layer.

403

1. Better resolution of details in pelvic organs or masses; close viewing of the cervix without bladder compression distorting its true structure; better viewing of a retroverted or retroflexed uterus; and increased ability to detect fetal heart motion and details of first trimester anatomy sooner than with the transabdominal approach.
2. (1) Age (related to menstrual status).
   (2) 1st day of the LMP or LNMP.
   (3) Gravidity and parity.
   (4) Current complaint.
   (5) Previous problems.
   (6) Surgery.
3. Longitudinal views in the midline (with/without measurements of the length from fundus to cervix, and AP of endometrium); right and left lateral portions of the uterus; transverse views of vagina, cervix, inferior portion of uterus, midline with and without width measurements, and superior fundus; longitudinal and endovaginal views of the ovaries with and without measurements and or adnexa.
4. Sagittal midline view with measurements of length from the fundus to the cervix and the AP of the widest portion of the body of the uterus; the endometrial stripe is also measured in this view; left and right portions of the sagittal uterus; coronal images of the cervix, isthmus, inferior, midline, and superior are acquired in successive order; midline image of the uterus with width measurements at the widest portion; sagittal and coronal (transaxial) views and length, AP, and width measurements of the ovaries.
5. In the proliferative phase, the endometrium is hypoechoic; in the secretory phase the endometrium is hyeperechoic.
6. (1) Anteversion.
   (2) Anteflexion.

(3) Lateral versions.

(4) Retroversion.

(5) Retroflexion.

7. Uterus septus/subseptus.

404

1. Endometritis, salpingitis, tubo-ovaritis, and peritonitis.
2. *Neisseria gonorrhoeae* and *Chlamydia trachomatis.*
3. Clear serous fluid in the uterine tube, usually caused by chronic PID.
4. Adhesions frequently generate from a TOA and attach to surrounding peritoneal structures, such as the outer surface of bowel loops. These adhesions freeze bowel walls, preventing normal peristalsis.
5. Serous cystadenocarcinoma.
6. Intercavitary, submucosal, intramural, subserosal.

405

1. Hydrosalpinx shows an anechoic lumen; pyosalpinx has internal echoes, making it hypoechoic.
2. Heterogeneous mass with no recognizable ovary is present in the adnexa or the cul-de-sac; also present with identifiable ovary, yet the tissue around it is disorganized, moving with the ovary as a unit.

406

1. The blood begins to congeal and clot, which changes the sonographic appearance. The old blood will be displayed on sonography as a heterogeneous mass with extremely thin hyperechoic strands in a reticular or branching pattern throughout the cyst. Older blood will progressively become more anechoic as time goes on. Occasionally, fluid-fluid levels will be seen, with the low-level echoes of old heavier blood cells settling according to gravity and the more anechoic fluid component resting above.
2. They are responsive to the hormonal cycle.
3. The corpus luteum cyst usually has an extremely hypervascular rim, notable by placing color Doppler over the cyst, revealing bright colors relative to the surrounding tissue.
4. Bilateral, large, multiloculated cysts resembling a cluster of grapes are seen to obscure the ovaries.
5. Prove that it is unconnected to the ovary.
6. Bilaterally enlarged and rounded ovaries, usually twice the normal size, with a number of small follicles seen at the periphery or scattered throughout the ovary. In most cases there is central, echogenic ovarian stroma or parenchyma.
7. By noting the absence of septations and fluid-fluid levels usually seen within hemorrhagic cysts. Also, the endometrioma does not regress with the cycle.
8. (1) Complex mass (mostly cystic or multiloculated cysts with solid elements such as mural nodules, thick septations, and irregular wall thickening).

   (2) Solid masses (may be with poorly defined margins, signifying spread to nearby structures).

   (3) Doppler flow demonstrates new blood vessel formation (neovascularization) in the center of the mass (color Doppler may reveal tortuous vessels and spectral Doppler may reveal low resistance to incoming flow to the tumor, with a resistive index of 0.40 or less and a high diastolic signal).

   (4) Ascites in the pelvis and abdomen (sometimes called malignant ascites in stages III and IV, especially if solid masses are seen attached to bowel loops or other surfaces, which is called peritoneal metastases).
9. They have a range from completely anechoic to complex to completely hyperechoic. Typical appearances of a dermoid mainly are of a fluid-fat level seen with one half of the tumor echogenic and homogeneous (fat) and the other side anechoic or hypoechoic (fluid). Occasionally the echogenic area will be anterior to the anechoic area, despite gravity; suggesting the presence of a dermoid rather than a hemorrhagic cyst, where the echogenicity corresponds to heavy blood clot that settles with gravity. Most dermoids are also accompanied by an echogenic mural nodule protruding from the wall of the tumor toward the center. This solid material is called either a *dermoid plug* or a *Rokitansky nodule*. Some will have bright hyperechoic structures which shadow, which are typically mixtures of hair, fat, and sometimes bone. Frequently, a bright echogenic line will lie along the fluid-fat interface level. This represents hair.

10. Multiple edge shadows distally and hetergenous attenuation throughout the mass.

407

1. Endometrial thickness greater than 15 mm.
2. Up to 15 mm.

Complete the unit review exercises before going to the next unit.

Unit Review Exercises

Note to Student: Consider all choices carefully, select the *best* answer to each question, and *circle* the corresponding letter. When you have completed all unit review exercises, transfer your answers to ECI (AFIADL) Form 34, Field Scoring Answer Sheet.

Do not return your answer sheet to AFIADL.

1. (401) What is the purpose of using volume measurements of ovaries in children?
   a. Better reproducibility.
   b. Less interference from uterus.
   c. Larger ovaries in children relative to pelvis.
   d. Compression of ovaries from lower pole of kidneys.

2. (401) What is the difference between anteversion and retroversion in the pelvis?
   a. Anteversion is forward tilt of the fundus; retroversion is backward tilt of the uterus.
   b. Anteversion is forward tilt of the cervix; retroversion is backward tilt of the uterus.
   c. Anteversion is forward tilt of the uterus; retroversion is backward tilt of the uterus.
   d. Anteversion is forward tilt of the uterus; retroversion is backward tilt of the cervix.

3. (401) Which ligament does the ovarian artery follow on its course to the ovary?
   a. Broad.
   b. Ovarian.
   c. Suspensory.
   d. Mesovarium.

4. (402) The *primary* function of the ovaries is to produce
   a. gametes.
   b. estrogen.
   c. progesterone.
   d. leutinizing hormone.

5. (402) What happens to the thickness of the endometrium as estrogen levels rise during the proliferative phase?
   a. Increased.
   b. Oscillates.
   c. Decreased.
   d. Stays the same.

6. (403) What is the *primary* advantage for using a transabdominal technique for pelvic sonography?
   a. Wide field-of-view.
   b. Detailed view of ovary.
   c. Narrow view of endometrium.
   d. Closer view of retroverted uterus.

7. (403) Which method is valuable for locating ovaries situated high in the pelvis?
   a. Transaxial.
   b. Endovaginal.
   c. Longitudinal.
   d. Transabdominal.

8. (404) What is the difference between salpingitis and pyosalpinx?
   a. Salpingitis is infection in the uterine tube; pyosalpinx is also an infected tube.
   b. Salpingitis is infection in the uterine tube; pyosalpinx is an infected tube with pus.
   c. Salpingitis is infection in the endometrium; pyosalpinx is an infected tube with pus.
   d. Salpingitis is infection in the endometrium; pyosalpinx is an infected tube without pus.

9. (404) What forms from adhesions and inflammatory pus extending from a uterine tube and attached ovary?
   a. Pelvic abscess.
   b. Tubo-ovaritis.
   c. Tubo-ovarian abscess.
   d. Tubo-ovarian complex.

10. (404) What normally happens to the corpus luteum late in the luteal phase?
   a. Divides.
   b. Septates.
   c. Expands.
   d. Regresses.

11. (404) What is the *primary* reason ovarian carcinoma is the *main* cause of death in gynecological cancers/?
   a. It is undetectable.
   b. It tends to regress.
   c. It is detected too late.
   d. It tends to spontaneously dissolve.

12. (404) What characteristic of the immature ovarian teratoma distinguishes it from a mature cystic teratoma?
   a. It is larger.
   b. It contains cysts.
   c. It occurs primarily in children.
   d. It contains a Rokitansky nodule.

13. (404) Which feature of uterine adenomyosis is a highly suggestive way to distinguish it from endometriosis?
   a. Nulliparous women over 60.
   b. Multiparous women over 40.
   c. Nulliparous women under 30.
   d. Multiparous women under 20.

14. (405) What is the purpose for placing color Doppler over any tubular structure in the pelvis?
   a. To determine if the structure is an abscess.
   b. Doppler is a standard requirement for all endometritis cases.
   c. To determine if the structure is a blood vessel or distended tube.
   d. Doppler is a standard requirement for all pelvic inflammatory disease (PID) cases.

15. (405) The purpose for color Doppler over an adnexal mass in suspected torsion is to detect
   a. an endometrioma.
   b. a corpus luteum cyst.
   c. blood flow in a spiral pattern.
   d. blood flow in a reticular pattern.

16. (406) Which feature of a serous cystadenocarcinoma distinguishes it from a serous cystadenoma?
    a. Large size.
    b. Thinner walls.
    c. Fully developed papillary projections.
    d. Occurs in women before the age of 50.

17. (407) What is the greatest sonographic determinant of endometrial hyperplasia in the sequential hormone therapy regimen (HRT) patient?
    a. Unchanging endometrial thickness.
    b. Decreased endometrial thickness.
    c. Increased endometrial thickness.
    d. Absent endometrial thickness.

18. (407) Which sonographic feature of endometrial carcinoma distinguishes it from endometrial hyperplasia?
    a. Sharp endometrial border.
    b. Blurred endometrial border.
    c. Thicker endometrial border.
    d. Thinner endometrial border.

Please read the unit menu for unit 2 and continue. ➔

Student Notes

Unit 2. Obstetrical Sonography: The First Trimester

FROM THE moment of fertilization, a woman's body begins a number of changes to prepare for pregnancy. The uterus, especially the inner cavity, changes in preparation for the implantation of a fertilized ovum. The ovaries continue hormonal support, and the pelvis bones and muscles begin to adjust their positions in anticipation of pregnancy. During this time, the sonographer is frequently called on to assist with pregnancy management. The usefulness of sonography lies in its primary and singular capability to directly visualize the pelvic, uterine, and amniotic environment that surrounds the growing embryo and fetus. Such access affords the physician with knowledge needed to manage or even modify the pregnancy up until the birth event.

In this unit, we will discuss the embryo and its environment during the first trimester of pregnancy. We will briefly touch on uterine anatomy and physiology only as it is affected by the presence of a fertilized ovum. We will then list the rapid landmark events of embryonic and early fetal development before moving on to standard imaging performed during a first trimester pregnancy.

2–1. Uterine, Embryonal, and Early Fetal First Trimester Anatomy and Physiology

In this section, we will cover the changes to the endometrium upon fertilization of an ovum and subsequent implantation. We will also take a look at some of the developments in the embryo as it progresses into a fetus. An awareness of the changing structures in the uterus from the moment of conception until the end of the first trimester will prepare you to accurately obtain corresponding sonographic images and to distinguish abnormal from normal development. As we look at the highlights of embryonic development, realize that only a few will be visible through sonography.

408. Fertilization affect on uterine anatomy and physiology

Pregnancy begins with fertilization and the most accurate age of the embryo can be counted from that point. Obstetricians and radiologists refer to this method of dating variously as gestational age (other terms are embryonal age, fetal age, or conceptional age). Unless assisted fertilization or ovulation induction is involved, most women are unaware of the date of conception. Because the date of this event is unknown in many women, most physicians use another method, which has proven more consistent with data obtained sonographically.

The first trimester (3 months or first third) of pregnancy is conveniently thought to begin on the first day of the last normal menstrual period (LNMP) and extend up until 12 weeks but no more than 13 weeks later. Thus, the duration of the first trimester is based on *menstrual age* (MA). Gestational age (technically two weeks younger if the fertilization occurs day 15 of the cycle) and menstrual age are used interchangeably by most radiologists and in most of this course.

Fertilization to implantation

When an ovary releases an oocyte (ovum) into the peritoneal cavity (ovulation on day 14 of the menstrual cycle), the infundibular fimbrial ends of the uterine tube capture it. The cilia and fluid within the tube move the ovum along. Fertilization is the process of a sperm cell (spermatozoon) penetrating into an oocyte, usually within the ampulla of the uterine tube (day 15). At this point, the

sperm cell and oocyte fuse into one cell called a *zygote*. Tubal cilia and fluid currents move the zygote along the tube toward the uterus from days 16 to 19. As this occurs, the cells of the zygote divide in stages of 2, 4, 8, and finally 16 cells or *blastomeres*. The spherical cluster of 16 blastomeres is called the *morula*. From day 19 until day 20, as the morula enters the endometrial cavity, fluid from within the cavity enters into the morula, separating the blastomeres into two layers. The morula with fluid is now called a *blastocyst*, which remains in the endometrial cavity for a few days before attaching to the endometrial lining, usually about day 22–23 (fig. 2–1).

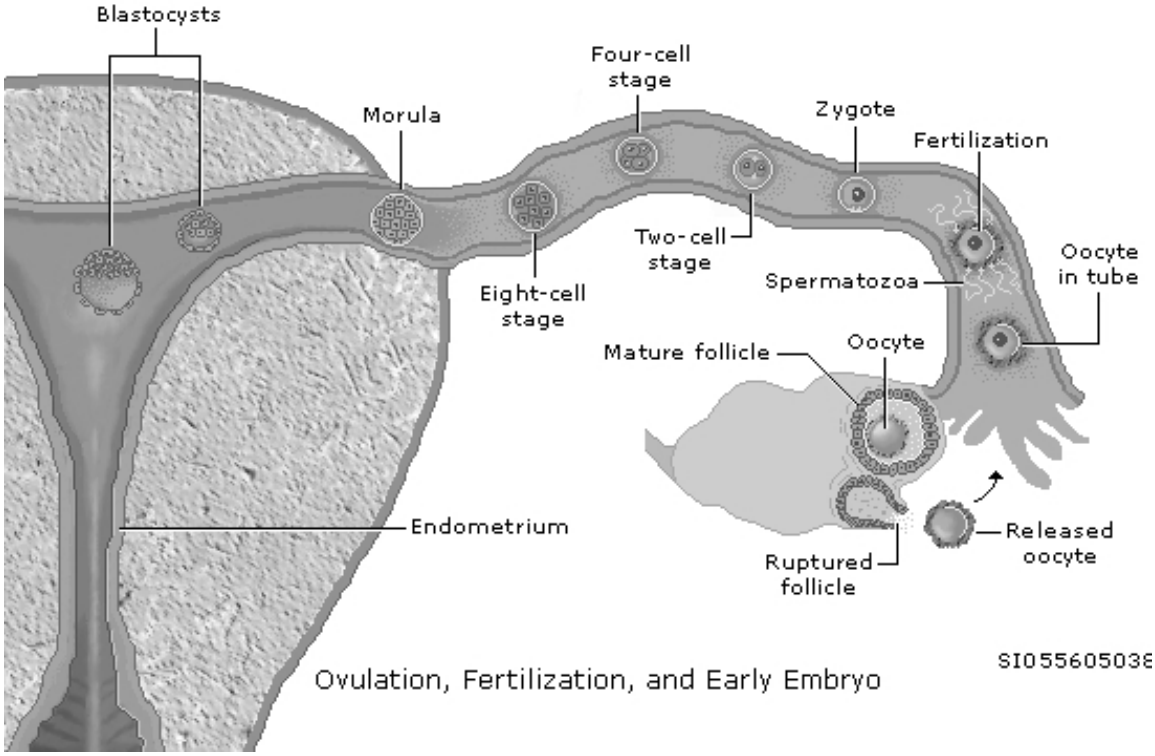

Figure 2–1. Ovulation and fertilization.

The outer layer of cells that make up the blastocysts are trophoblastic cells, specialized cells whose job is to anchor the blastocyst to the endometrium and to form the early placenta.The inner layer of cells becomes a mass that will later develop into a gestational sac. This sac will contain embryo, yolk sac (primary and secondary), amnion, and cord. The trophoblasts invade and merge with the endometrium, changing it into cells that can provide nourishment to the developing implantation (decidual reaction).

At the point the implantation is completely submerged (day 23) the trophoblast begins to produce a hormone called human chorionic gonadotrophin (hCG). This substance is produced in quantities that can be detected by laboratory analysis. The blastocyst begins to expand and becomes the gestational sac.

Gestational sac and yolk sac

Approximately four days later (day 27), the primary structures of the pregnancy have developed. The primary yolk sac has been pinched off and regressed, leaving the large secondary yolk sac; the primitive embryo (shaped like a disc or plate) has formed between the secondary yolk sac and the amnion; and the early placenta with circulation has been established through fingerlike projections called chorionic villi. At this point, the developing mass (now sometimes called the *conceptus*) is approximately 2 to 3 mm in size. The outer layer of the gestational sac is chorionic membrane, which expands outward continuously.

Beyond day 28, menses has not occurred. The fifth week has produced a 5 mm gestational sac with an embryo that has transformed into a trilaminar disk, made of three primitive layers: the endoderm, the mesoderm, and the ectoderm. These embryonal layers will become the various parts of the body in later development. Meanwhile, the yolk sac manufactures much of the embryonal blood cells (hematopoiesis or hemopoiesis), and contributes cells that make up elements of the reproductive and gastrointestinal system. As the gestational sac expands, the outer chorionic membrane fuses with the endometrium, while simultaneously the inner amnion also continues to expand. The gestational sac has obtained a size that usually can be detected by endovaginal sonography. In between the chorionic membrane and the amnion is a space of fluid called the chorionic cavity, which houses the secondary yolk sac.

Multiple gestations

When two separate ova are fertilized, the result is two zygotes (dizygotic, fraternal twins). If one ovum is fertilized and subsequently divides, the result is monozygotic twins (identical twins). All dizygotic twins form their own blastocyst and implantation, complete with separate chorions and amnions. Monozygotic twins come in three types, depending on the time of zygote division.

- Dichorionic diamniotic (up to 3 days after fertilization).
- Monochorionic diamniotic (4–8 days, most common type).
- Monochorionic monoamniotic (more than 8 days, rare/more than 13 days will form conjoined twins, even rarer).

Remember that the amnion forms after the chorion. Thus, all dichorionic membranes will always be diamniotic. However, one chorionic cavity can have one or two amnions, depending on when zygote division occurs.

Infertility

Problems that can interfere with the fertilization process, and thus hinder pregnancy, are numerous. For instance, congenital anomalies, chronic infection, tumors, and hormonal abnormalities are common sources of infertility. Whatever the original source, the main cause of infertility (inability to produce offspring) is the interruption of fertilization or implantation. Most of the interruptions come from obstruction or damage (scarring) of the inner lining of the uterine tubes and endometrium. Any process that prevents the ovary from developing and releasing an ovum is also a major obstacle to fertilization.

In many women with infertility problems, assistance is needed to facilitate pregnancy, from assisted fertilization to assisted implantation or both. Many methods have been developed in recent years to reverse the process of infertility. One method is ovulation induction, which is a method of stimulating the follicles to develop and to ovulate. The primary way to do this is by administering gonadotropins (FSH [follicle-stimulating hormone] and LH [luteinizing hormone]).

Another method used with infertility is through assisted reproductive technology (ART). These are methods possessing the common goal of retrieving or extracting oocytes and combining them with sperm cells. Some of the ART are in vitro fertilization (IVF), gamete intrafallopian transfer (GIFT), and zygote intrafallopian transfer (ZIFT). IVF involves aspirating follicular fluid, incubating the retrieved oocyte with sperm cells, and transferring the zygote into the uterine fundus for implantation, bypassing damaged or nonfunctional uterine tubes. For GIFT and ZIFT procedures catheters are used to transfer ova, sperm or zygotes into the distal uterine tube. GIFT and ZIFT procedures are generally used for women with cervical or unknown causes of infertility.

409. Embryological development

By the sixth week MA, the embryo develops the full complement of structures that will grow to maturity. Most of these early structures can be seen with sonography by the late first trimester. However some structures, such as genitourinary and lungs, are not seen until the second trimester.

Cardiac development is rapid. Twin cardiac tubes begin beating sometime in the fifth week and beginning of the sixth week. The tubes fuse into one tube, which transforms into a tiny final adult form by the end of week 8 and no later than 12 weeks.

The primitive gut also forms beginning in the sixth week, divided into foregut (organs such as stomach and liver), midgut (small intestine and most of the colon), and hindgut (the distal colon and rectum). In the eighth week the midgut herniates into the base of the umbilical cord and stays there until about the end of the first trimester, when it will rotate and descend back into a changed abdominal cavity. The change is due to urinary and genital structures separating and moving in the cavity, which is easily done without the presence of the midgut. The primitive kidneys move out of the pelvis and ascend into the abdomen to their adult location by week 11.

Skeletal features such as extremities, fingers, and toes are also developed during week 8, usually in a bud formation. The limbs usually begin movement in the tenth week. Most skeletal mineralization has developed by the end of the first trimester, particularly the skull.

The early brain and spinal cord, called a neurotube, is separated into open anterior and posterior portions. The anterior portion will have divided up into three segments by the beginning of the sixth week: the forebrain (prosencephalon), midbrain (mesencephalon), and hindbrain (rhombencephalon). These segments will become the brain by the end of the sixth week when the anterior neurotube completely closes. The simultaneous closure of the posterior neurotube begins the development of spinal cord and spine with calcification or mineralization of the spinal elements by the beginning of week 8.

Self-Test Questions

After you complete these questions, you may check your answers at the end of the unit.

408. Fertilization affect on uterine anatomy and physiology

1. What is the outer layer of cells called that make up the blastocyst, and what is its function?

2. What are the three primitive layers of the embryonal trilaminar disk?

409. Embryological development

1. When does the embryonic heart begin beating?

2. List the three segments that make up the anterior neuropore.

2–2. Imaging in the First Trimester

Sonographic imaging of the first trimester pregnancy can be quite challenging. However, with basic knowledge of the routine appearance of normal structures, you should be prepared to accurately identify and document abnormalities. In this section we will consider some of the information that you must understand surrounding the first trimester sonogram. We will also briefly look at some of the problems that develop from interruptions to normal pregnancy and their sonographic appearances.

410. First trimester pathology considerations

Abnormalities in the first trimester usually involve the uterine environment. The conditions in the uterus may prevent the early pregnancy from developing or progressing normally. Although abnormalities also involve the embryo, these are usually detected after insult to the uterine environment. This lesson will briefly discuss some of the uterine abnormalities and one extrauterine abnormality.

General pathology in the first trimester

A useful way to approach the first trimester pregnancy is to systematically confirm or rule out certain criteria. Thus, the first reasonable thing to establish is the presence of a gestational sac that corresponds to a given level of beta-human chorionic gonadotropin (β-hCG). If a pregnancy test is positive at approximately 23 days MA and the uterus appears normal on sonography with no sign of an intrauterine pregnancy (IUP), the possibility increases that the pregnancy is early (normal or abnormal). Each institution has dating criteria that correspond to threshold levels of β-hCG, above or below which will cause physicians to presume an early embryonic demise or even an ectopic. β-hCG levels tend to be very low if a pregnancy was established and subsequently aborted completely before your examination. For ectopics, the levels rise but frequently do so at a slower rate than normal. A key thing to remember is that β-hCG levels tend to double every two days beyond 23 days MA.

If a woman is spotting blood when presenting during weeks 6 and 7, it is likely due to blood from the trophoblastic activity of the conceptus interacting with the myometrium, which is normal. However, if the bleeding is heavy in the early first trimester, this may be sign of complete or incomplete abortion. In these cases β-hCG levels fall slowly and plateau, largely because of the continued activity of trophoblastic cells.

Gestational sac abnormalities, fetal heart rate aberrations, and yolk sac irregularities will have negative effects on a first trimester pregnancy. For the gestational sac, the condition of the amnion membrane and the size of the sac play a profound part on whether an embryo appears at all. For the embryonic heart, the sonographic absence of cardiac activity beyond the end of the sixth week MA is a sure sign of failed pregnancy. A slow heart rate (bradycardia) in the early first trimester is also an ominous sign. Yolk sac size and shape tend to suggest an abnormal embryologic development, particularly if the yolk sac is extremely large or if it is calcified.

Another particularly deadly occurrence is hemorrhage. This occurs when the early placenta ruptures either partially or completely, separating from the myometrium. This produces bleeding outside of the gestational sac, just beneath the chorionic membrane, which is loosely fused to the endometrium, specifically the decidua parietalis (vera). Sometimes only a pocket of blood, called a hematoma, at the margins of the placenta is produced; at other times, most of the sac is surrounded in intrauterine blood.

Finally, the pregnancy itself may be abnormal. Occasionally a sperm cell will fertilize an empty ovum. The resulting cell divides with chromosomes only from the father. The trophoblast of the implanted conceptus is abnormally hypertrophic and develops villous swelling that fills with fluid, producing multiple cystic spaces. This trophoblastic proliferation is called a *complete hydatidiform mole (CHM)*. It is complete because the pregnancy is completely replaced by trophoblastic tissue (a mole). Patients usually present with vaginal bleeding and significantly high levels of β-hCG. CHM can produce massive ovarian theca lutein cysts, which may cause painful torsion.

Ectopic pregnancy

The leading cause of maternal death in the first trimester is from ectopic pregnancies. When a pregnancy is implanted outside of the endometrium, it is considered ectopic. The most common location is the ampullary portion of the uterine tube. Other locations of the tube are less common. However, an interstitial implantation is the more serious ectopic, as this can cause severe hemorrhage from the major network of blood vessels in this location. Other more rare locations are ovaries, pelvic cavity, and cervix. The major cause of ectopic pregnancies is usually due to scarred uterine tubes that prevent the passage of fertilized ova. Prior ectopics, tubal ligations, and infertility treatments (largely necessary because of previous tubal damage), are the main sources of the risk for ectopics. Use of intrauterine devices (IUD) as well as exposure to certain chemical substances also increase the chances of ectopics. Even women who have had cesarean sections (C-sections) are at increased risk for an implantation in the uterine scar. This last type of ectopic can be confused with the sonographic appearance of an incomplete spontaneous abortion.

Less than half of women with ectopic pregnancies present clinically with classic symptoms: the triad of pelvic pain, vaginal bleeding, and a mass felt (palpable) by the clinician in the area of the adnexa. Laboratory tests are positive with β-hCG levels indicating pregnancy. Again, each laboratory has its own standard and discriminatory level at which the β-hCG levels correspond to a certain age of pregnancy. Find out what your facility uses, and compare the MA indicated by the lab values with what you should see sonographically in the normal pregnancy.

411. Imaging the first trimester pregnancy

Sonography of the first trimester pregnancy is similar to routine pelvic sonography. Both use the endovaginal probe with at least 6 MHz in frequency, and both evaluate uterine contents. The difference lies in the appearance of the endometrial stripe and the developing embryo within it. Everything about the female patient changes with pregnancy to include hormonal effects. Thus, the ovaries are different as well. The changes can bring on abnormalities that may prompt the physician to order a sonogram for diagnosis. Several reasons for performing first trimester sonography exist and most of those reasons center around basic questions in need of answers concerning the pregnancy.

Indications and patient history

Common indications or reasons for first trimester sonography are:

- Confirm the existence of an IUP.
- To confirm cardiac activity.
- To estimate MA; also called gestational age.
- To diagnose multiple gestations.
- To evaluate possible causes of pelvic pain, vaginal bleeding.
- To locate or confirm a suspected ectopic pregnancy.
- To evaluate a suspected hydatidiform mole.

The questions that many of these indications answer are:

- Is there a pregnancy?
- How many embryos are present?
- Is the embryo alive (is there cardiac activity)?
- Where is the early placenta in relation to the cervix?
- What is the gestational age?
- Is there anything inside or outside the uterus that can potentially interfere with the pregnancy?

Answering these questions are the main goals for sonography in this period of pregnancy, all of which are legitimate medical reasons. Further, maternal illness may be another reason for first trimester sonography in known pregnancies.

Laboratory values

For you to properly perform the first trimester sonogram, you will need to check the laboratory values. β-hCG, a subunit of human chorionic gonadotropin, is easier to distinguish in laboratory analysis from other gonadotropins produced by the pituitary such as FSH and LH. Trophoblastic cells from the implanted blastocyst or conceptus begin to produce β-hCG, and certain amounts of this substance in the serum of the mother indicate the presence of an IUP. β-hCG ranges also correspond to MA of the developing embryo, which in general should correspond to sonographic measurements at each week of age. Because standards vary from laboratory to laboratory, use the standard for your facility when checking for β-hCG values. Common standards are the First International Reference Preparation (FIRP) and the Third International Standard (TIS). The level of β-hCG established by your facility at which IUPs are always detected at a given MA (which should correspond sonographically) is called the discriminatory level.

Sonographic imaging of first trimester uterine environment and embryo

Patient prep is identical to that of pelvic examination. The first day of the LNMP is even more critical here. By asking the patient for the LNMP, you may learn information that can affect the calculation of MA. For instance if the MA indicated by the patient-provided LNMP does not match the LNMP calculated from your sonographic measurements, this could be an indication of an abnormality, such as early growth restriction. Careful questioning of the mother can reveal clinically relevant information such as past pregnancies, failed pregnancies, cancer, or surgeries that may affect what you see on the sonogram.

Your equipment should be standard and the same as that used for pelvic sonography. For endovaginal work, most measurement data published assumes the sonographer is using transducer frequencies of 6 MHz or more. If available for your unit, pay particular attention to thermal and mechanical indexes (TI and MI, respectively). The TI gives you an indication of the risk for a rise in temperature (heat) of tissue exposed to the energy in the ultrasound beam. MI is the indication of bubble formation (cavitation) in the fluid components of cells as a result of the pressure activity in an ultrasound beam. Keep the TI and MI at acceptable manufacturer and department protocol levels for any given examination and especially during obstetrical work.

As with pelvic sonograms, the use of a latex or latex free endovaginal probe cover (depending on the latex tolerance of the patient) is important to keep from transmitting body fluids or pathogens. Also, ensure that you disinfect the endovaginal probe both before and after the examination according to the specifications of the manufacturer. This is for the safety of both patients and staff handlers.

Standard views

Transabdominal as well as endovaginal scanning should be performed for a comprehensive coverage of the contents of the entire pelvis. Standard structures that should be documented during the first trimester are the uterus, the adnexa, and cul-de-sac. The ovaries should be imaged as you would for a routine pelvic sonogram. The presence of the corpus luteum should be documented if seen. A good way to prove to your radiologist that it is most likely a corpus luteum is to capture an image of the ovary with a color Doppler box overlaying it. The image should display the classic color bloom of blood flow around the periphery of the corpus luteum. Use power Doppler if you do not get a color signal.

Within the uterus, the presence of a gestational sac, yolk sac, embryo, and measurements of these structures should be documented. The cardiac activity should be sought and confirmed through the use of M-mode. Ensure you magnify the image of the embryologic heart rate (sometimes called fetal heart rate, which is incorrect until after the eighth week when the embryo is considered a fetus) before

attempting the M-mode image. This will provide the radiologist with strong, clear evidence of cardiac rhythms. Document the number of amnions and chorions if multiple gestational sacs are seen. Finally, the presence, location, and size of any intra- or extrauterine masses are important.

Gestational sac and yolk sac

The gestational sac sonographically appears to be a round fluid collection surrounded by an echogenic rim, which represents the decidual reaction. Normally, another deciduous, echogenic, concentric circle surrounds this one, with one side nearly against the inner decidual reaction. Both circles are called the double decidual sign. The inner ring of the gestational sac is composed of chorion (frondosum on the thicker placental side and laeve on the smooth thin side), and the outer ring is composed of the decidual endometrial lining (decidua capsularis against the laeve, parietalis, and basalis behind the frondosum). Both rings are at least 2 mm thick and noticeably echogenic.

Endovaginally, the gestational sac can be seen as early as 4 weeks MA but consistently is seen at 5 weeks. It is the first sonographic sign of pregnancy. Usually, nothing else is seen in the sac at this time. Measure the gestational sac by obtaining the inner wall diameters of the sac's length, AP (height), and width. Adding these dimensions and dividing the sum by 3 will give you the mean sac diameter (MSD). Many ultrasound units will provide obstetrics packages that automatically calculate the MSD for you, as well as the likely estimated MA. The MSD is usually 5 mm at 5 weeks and grows at the rate of approximately 1 mm per day thereafter up until 10 weeks.

By 5.5 weeks, the first structure seen inside the gestational sac is usually the yolk sac. Endovaginally the yolk sac should be seen by an MSD of 8 mm or more (20 mm or more if using transabdominal). The yolk sac itself should be measured in the AP dimension to give you a diameter that should never exceed 5.6 mm between 5 and 10 weeks.

Occasionally, at this time, the amnion will briefly appear against the yolk sac, with the two structures looking like the shape of the number '8.' This temporary appearance is called the double bleb sign. The amnion disappears and does not reappear until after the embryo is seen. By 6 weeks a heartbeat can be seen next to the yolk sac, and you may be able to see enough of an embryo to measure, which is usually about 2 mm to 3 mm. An embryo is seen if the MSD is 16 mm or more (25 mm or more by transabdominal). Ensure that you use the M-mode to document the heart rate. By week 7 the embryo and the yolk sac will begin to move away from each other (fig. 2–2).

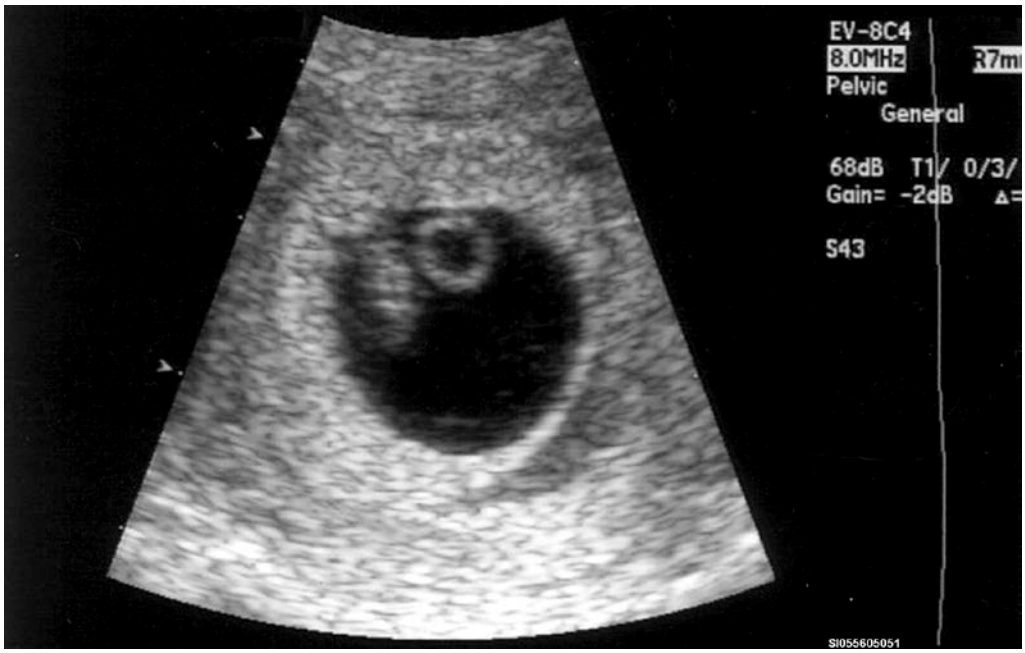

Figure 2–2. Embryo and yolk sac.

After 8 or 9 weeks the amniotic sac is readily visible, containing anechoic amniotic fluid. The fluid outside the amnion, the chorion fluid contains low-level echoes. If seen before chorionic fusion, the fluid outside the chorionic membrane represents the blood in the emdometrial cavity (commonly called the subchorionic space), which is even more echogenic than the chorionic space.

For multiple gestations, the process is similar to singleton. However, you must attempt to determine the type of multiple pregnancies. This requires demonstrating chorionicity and amnionicity, as well as the symmetry between the two fetuses. To determine chorionicity count the gestational sacs. To determine amnionicity count the yolk sacs (fig. 2–3).

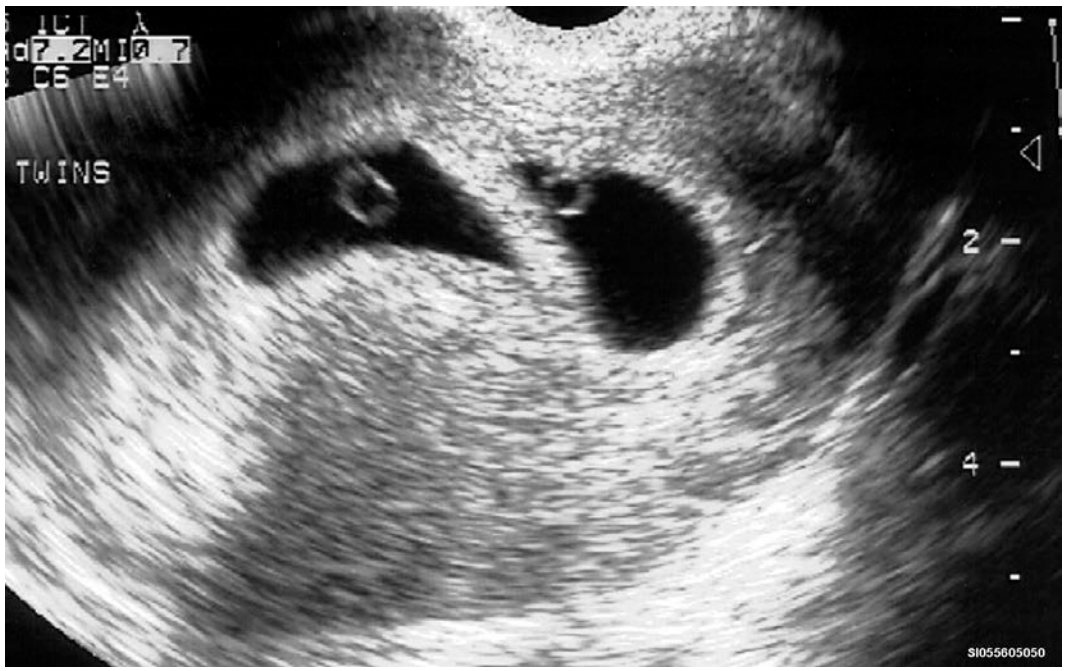

Figure 2–3. Multiple gestations.

Crown-rump length

To measure the embryo, use the crown rump length (CRL) method. To do this, place the caliper at one end of the embryo (increasing age will demonstrate the head structure) and another caliper at the other end (fig. 2–4). This will give you a length that can be compared with CRL growth charts in use in your facility as well as the electronic database used in your ultrasound unit's obstetric package. At 6.5 weeks the CRL is generally around 6 mm and a heart rate of no more than 125 beats per minute (bpm) should be visible. By the eighth week, the CRL has extended to 16 mm with visible amniotic sac, fetal movement, and a normal heart rate of no more than 175 bpm. The CRL taken between weeks 6 and 10 are the most accurate measurements for MA taken at anytime during pregnancy.

Normal physiologic changes

From 6 to 8 weeks, you may see a cystic structure at one end of the embryonal pole. This is the normal appearance of the rhombencephalon or hindbrain. The structures inside the fetal brain continue to develop, increasing in echogenicity, so that by week twelve you should see an echogenic line separating two echogenic structures. These represent the lateral ventricles of the brain filled with choroid plexus.

Also, beginning at 8 weeks, herniated bowel appears at the base of the umbilical cord. Between week 10 and 12, the bowel rotates 90 degrees counterclockwise and descends back into the abdomen, rotating as it goes. This process is normal rotation of the midgut.

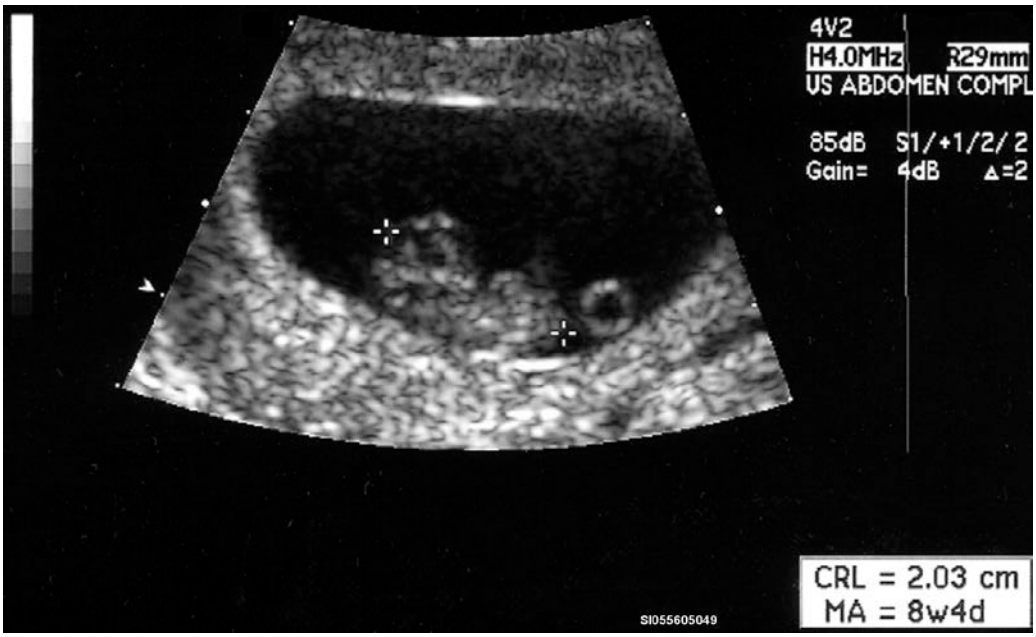

Figure 2–4. Crown rump length.

Imaging first trimester abnormalities

Most women who come to the sonography department during the first trimester are sent for early dating confirmation or to verify, sonographically, the existence of an IUP. Pregnancy tests usually center on the presence of β-hCG in the maternal serum or urine, and not every elevation of this substance signifies a normal pregnancy. A certain percentage of first trimester pregnancies are sent for evaluation due to bleeding or even unusually low β-hCG levels when the *clinically* estimated MA is nearly certain. Your job is to not only confirm the presence of a living embryo or embryos, it is also to evaluate the status of the pregnancy and look for signs of impending pregnancy failure.

Early pregnancy failure and demise of the embryo

If the endometrium shows echogenic thickening and irregularity, this could be the material (retained products of conception) from of an incomplete spontaneous abortion. Intrauterine blood, particularly if it's new and in significant quantities, also is quite echogenic and usually signals pregnancy failure.

If a gestational sac is seen, it is reasonable to expect certain characteristics of the sac at weeks 6–7. You should note its location. The sac should be in the central endometrial cavity but as near the fundus as possible. If it is located in the lower endometrial cavity, closer to the isthmus, it is likely abnormal. However, this finding by itself (that is, it is nonspecific) does not confirm a failed pregnancy. The one finding that is specific for pregnancy failure is of a gestational sac seen at a certain size without yolk sac or embryo (8 mm and 16 mm, respectively). The terms *blighted ovum*, *missed abortion*, and *anembryonic pregnancy* have been used to describe this state, and you should use the term your radiologist determines fits the description. However, if you need to say anything at all about it to the patient (which should be exceptionally rare), a useful way to put it might be: *unsuccessful pregnancy*. Be aware that a pseudogestational sac, a fluid collection within the endometrium that does not have a decidual ring, can be confused with gestational sac. The pseudogestational sac is normally associated with ectopic pregnancy, and β-hCG levels should be consulted to match the appearance and size of the fluid collection.

Another indication of anembryonic pregnancy is the appearance at any time (usually after 7 weeks) of the amnion but no embryo. The amnion should never be seen without the embryo beyond the temporary appearance of the double bleb sign early in the trimester.

If an embryo is seen endovaginally at more than 5 mm CRL without a heartbeat, it is usually nonviable. However, to make such a determination requires looking at other factors such as β-hCG levels, patient history, and the MSD diameter. Also, you should use the M-mode in the presence of another medical observer, preferably the radiologist, before categorizing embryonic demise from an absent embryonic heart rate.

If you see gestational sac, yolk sac, and an embryo with a beating heart, the pregnancy is a confirmed living IUP. However, certain parameters about these features may predict an abnormal outcome or early pregnancy failure. The first would have to be an abnormal heart rate. If the embryonic heart rate in an embryo with a CRL of less than 5 mm is less than 100 bpm, it is suspicious for impending demise. This abnormally slow heart rate is called bradycardia. Because most embryos between 5 and 9 mm in CRL have at least 120 bpm heart rates (mean heart rate: 140 bpm), any rate below 110 bpm suggest a poor outcome.

An irregular appearance of the yolk sac is another sign of an embryo at risk. Large yolk sacs (above 5.6 mm), thick or extremely thin walls, irregular walls, and an echogenic or calcified lumen are all some of the irregularities that hint at poor outcome. However, except for large or calcified yolk sacs, the above wall appearances can be seen in pregnancies that go on to deliver successfully.

Subchorionic hemorrhage or hematoma is another ominous sign. The most important sonographic finding that is predictive of the outcome is whether the blood is located behind the early placenta, as this type has a poor prognosis.

Finally, the classic sonographic appearance of the CHM is of a uterus filled with cystic spaces frequently referred to as a "snowstorm." No embryo or fetus is present. The combination of the massive β-hCG levels with this snowstorm appearance is highly suggestive of molar pregnancy. Normally, the tissue is surgically removed through curettage to ensure that all pieces are eliminated. If any piece remains, the likelihood of it becoming malignant is increased.

Ectopic pregnancy

After 5 weeks MA up until 10 weeks, you should see an intrauterine gestational sac with double decidual borders. If the uterus is empty and β-hCG levels are steadily (but more slowly than normal) climbing, suspect an ectopic pregnancy. The presence of a gestational sac, particularly a yolk sac and an embryo with a heartbeat, nearly excludes further possibility of an ectopic pregnancy. However, some infertility patients who have undergone ovulation induction or in vitro fertilization are at increased risk for pregnancies simultaneously inside and outside the uterus (heterotopic). Also a rounded fluid collection in the uterus, centrally located with a diameter that exceeds 13 to 16 mm, should not be mistaken for a gestational sac. The absence of a double decidual sign is also an indicator that the fluid collection is a false sac (pseudogestational sac). Measure the pseudogestational sac in all three planes.

After documenting the uterine environment suggestive of ectopic, you should scan the adnexa for the presence of a gestational sac. The appearance of the sac should be nearly identical to that of a normal IUP with or without an embryo. The ring around the sac will be echogenic with a decidual cast. This can be difficult, particularly if the woman is extremely tender to probe pressure at the adnexa. You may have to use the transabdominal transducer to obtain the necessary images of an adnexal mass. With either method, make sure you demonstrate the ectopic's relationship to the ovary. Using color Doppler around the mass, you should see continuous high velocity flow around the periphery, similar to that located around the corpus luteum of the ovary. However, ectopics are rarely located in the ovaries, distinguishing them from the corpus luteum.

Finally, locate, measure in three dimensions (as much as possible), and document any excessive fluid in the cul-de-sac. If the fluid is complex it is likely blood from a continuously bleeding tubal ectopic. The possibility of fluid traveling to the flanks and up into the hepatorenal space (pouch of Morrison) is increased the more severe the bleeding. Moderate to high amounts of fluid in the cul-de-sac tends

to prompt radiologists to recommend immediate surgery. Make sure that you scan throughout the pelvis for pockets of fluid.

You should realize that some ectopics are impossible to visualize sonographically. Nonvisualization does not mean exclude the presence of an ectopic pregnancy, especially at certain β-hCG levels and an empty uterus.

Nuchal translucency thickness

A useful measurement that some radiologists may require and should be attempted between 10 and 14 weeks is measurement of the nuchal translucency (NT). This is a thin membrane that lies over the posterior neck of the fetus. It is best seen away from any thin amniotic membrane with the fetus in a sagittal position. The neck should be in a neutral position, neither extended nor flexed. By magnifying up to ensure proper caliper placement, you will reduce errors. Place the calipers at the fetal skin and within the thin membrane at the neck. The resulting measurement should be less than 3 mm in normal fetuses. Any measurement at or above 3 mm is abnormal and suggestive of genetic abnormality, the most common being Down syndrome (trisomy 21), Edwards syndrome (trisomy 18), and Turner syndrome (45/XO).

Self-Test Questions

After you complete these questions, you may check your answers at the end of the unit.

410. First trimester pathology considerations

1. What are some abnormalities that have negative effects on a first trimester pregnancy?

2. What is the major cause of ectopic pregnancies?

411. Imaging the first trimester pregnancy

1. Trophoblastic cells from the implanted blastocyst or conceptus begin to produce what substance?

2. Endovaginally, what is the first sonographic sign of pregnancy?

3. What does the sonographic appearance of the amnion but no embryo beyond 7 weeks signify?

4. Describe the adnexal sonographic appearance of an ectopic pregnancy.

Answers to Self-Test Questions

408

1. Trophoblastic cells, specialized cells whose job is to anchor the blastocyst to the endometrium and to form the early placenta.
2. Endoderm, mesoderm, and ectoderm.

409

1. Sometime in the 5th week and beginning of the 6th week.
2. The forebrain (prosencephalon), midbrain (mesencephalon), and hindbrain (rhombencephalon).

410

1. Gestational sac abnormalities, fetal heart rate aberrations, and yolk sac irregularities, and hemorrhage.
2. Scarred uterine tubes that prevent the passage of fertilized ova.

411

1. β-hCG.
2. The gestational sac.
3. Anembryonic pregnancy.
4. Nearly identical to that of a normal IUP with or without an embryo. The ring around the sac will be echogenic with a decidual cast. Using color Doppler around the mass, you should see continuous high velocity flow around the periphery.

Complete the unit review exercises before going to the next unit.

Unit Review Exercises

Note to Student: Consider all choices carefully, select the *best* answer to each question, and *circle* the corresponding letter.

19. (408) The *difference* between a blastocyst and a morula is that a blastocyst contains
    a. pus.
    b. fluid.
    c. blood.
    d. cysts.

20. (408) The *most* common monozygotic twin is the monochorionic
    a. diamniotic.
    b. dichorionic.
    c. monoamniotic.
    d. monochorionic.

21. (408) The purpose for administering gonadotropins to infertility patients is to
    a. fight infections.
    b. induce ovulation.
    c. cause fertilization.
    d. cause implantation.

22. (409) What forms in the embryo after the complete closure of the anterior neurotube?
    a. The brain.
    b. The spine.
    c. The heart.
    d. The lungs.

23. (410) If a woman is bleeding heavily in the first trimester, what abnormality may be indicated?
    a. Ectopic pregnancy.
    b. Incomplete abortion.
    c. Anembryonic pregnancy.
    d. Complete hydatidiform mole.

24. (411) A common reason for first trimester sonography is to confirm
    a. fetal gender.
    b. cardiac activity.
    c. oligohydramnios.
    d. fetal lung maturity.

25. (411) The purpose for asking the first trimester patient for the first day of the last normal menstrual period (LNMP) is to learn information that may affect the
    a. menstrual age (MA) calculation.
    b. estimated fetal weight (EFW) calculation.
    c. abdominal circumference (AC) parameter.
    d. head circumference/femur length (HC/FL) ratio.

26. (411) The purpose for using M-mode during the first trimester is to confirm
    a. fetal gender.
    b. cardiac activity.
    c. oligohydramnios.
    d. fetal lung maturity.

27. (411) To determine amnionicity in first trimester multiple gestations you should count the
    a. embryos.
    b. placentas.
    c. yolk sacs.
    d. gestational sacs.

28. (411) What method should you use to measure the embryo?
    a. Mean sac diameter (MSD).
    b. Crown rump length (CRL).
    c. Beta-human chorionic gonadotropin (β-hCG)
    d. First International Reference Preparation (FIRP)

29. (411) What should you use in the presence of a medical observer before categorizing embryonic demise?
    a. M-mode.
    b. B-mode.
    c. Doppler.
    d. Gain.

30. (411) The significance of seeing gestational sac, yolk sac, and an embryo with a beating heart is that it confirms
    a. an impending fetal demise.
    b. a living intrauterine pregnancy (IUP).
    c. the absence of chromosomal abnormalities.
    d. the presence of chromosomal abnormalities.

31. (411) What distinguishes the ectopic pregnancy from the corpus luteum?
    a. Corpus luteum has flow around the periphery.
    b. Ectopic pregnancy is usually inside of the ovary.
    c. Ectopic pregnancy is usually outside of the ovary.
    d. Corpus luteum causes excessive fluid in the cul-de-sac.

Please read the unit menu for unit 3 and continue. ➔

Student Notes

Unit 3. Obstetrical Sonography: The Second and Third Trimesters

SONOGRAPHY becomes increasingly more challenging as the embryo develops into a fetus and continues to grow. If a fetus reaches the second and third trimesters, the chances for a successful delivery are increased. However, not all fetuses make it to term and complications, either fetal or maternal, may develop. Also congenital abnormalities of the fetus itself may manifest in the second trimester. Thus, the sonographer should be familiar with most of these processes.

In this unit, we will broadly cover most of the critical areas in obstetrical sonography. We will briefly touch on some of the anatomy and physiology or functions of the uterine environment as well as some of the fetal anatomy changes. We will then take a general overview of acquiring sonographic images in the second trimester—from routine methods used to estimate gestational age and growth to describing some of the complications that may arise in pregnancies at high risk for termination. We will then round out your knowledge of obstetric sonography by providing brief summaries of the most common fetal abnormalities and co-existing anomalies.

3–1. Anatomy and Physiology of the Second and Third Trimester Uterus and Fetus

The intrauterine environment grows rapidly throughout the first trimester. This growth slows but continues in the second and third trimesters. The fetus itself also continues to grow, and it is within the second trimester that most of the developing body systems are recognized with sonography. In this section we will list the anatomical and functional highlights of the two biggest changes since the first trimester, the placenta and amniotic fluid environment. We then will look at the major fetal anatomy changes, mostly due to growth, that can readily be seen through sonography.

412. Uterine environment

The uterus itself grows to a size that fills the much of the pelvis and abdomen during the second and third trimesters. Organs such as bowel and ovaries literally shift out of the way of the expanding mass. Inside the uterus, the walls become thin as they stretch to accommodate the new growth. The chorionic and amnionic membranes remain much as they were in the first trimester, with the exception that the amnionic membrane is now fused to the inside of the chorion laeve and surface of the chorion frondosum, the site of the new placenta. Thus, the secondary yolk sac that is so prominent in the first trimester between the chorion and amnion is greatly diminished or reabsorbed by the middle second trimester.

Placenta

The placenta is composed of elements of the chorionic membrane and the endometrium's basalis decidua layer. The chorion that is attached to the basal portion, the frondosom, is made of many tiny chorionic villi, fingerlike projections that extend toward the basal endometrial layer. The space in between each villus projection (intervillous space) is filled with maternal blood. On the maternal side, the basalis of the endometrium, arterioles feed arterial blood into the chorionic intervillous spaces, while venules transport venous blood away from the chorionic spaces. Inside each villous extension are fetal capillaries, which are connected to major fetal blood vessels. Those fetal vessels converge and penetrate up onto the chorionic surface (plate) that is fused with amnionic membrane. Beneath the amniotic layer but above the chorionic plate, all of the fetal vessels converge toward the single umbilical cord that connects to the fetus. In the umbilical cord a single umbilical vein carries *oxygenated* blood from the placenta toward the fetus. Wrapped around the single vein, usually in a tight spiral, are two umbilical arteries that carry *deoxygenated* blood away from the fetus toward the placenta. The naming of the vein and arteries of the cord is based on the direction of blood flow in relation to the *fetal* heart. The placenta grows from 1 to 4 cm thick and approximately 18 cm in diameter, but this varies considerably from woman to woman.

The placenta's main functions are to provide the fetal blood with maternal oxygen and nutrients and to take away fetal waste and carbon dioxide. Another important function is the manufacturing of a number of hormones, the most important being beta-human chorionic gonadotropin (β-hCG) early in the second trimester, with estrogen and progesterone production in the placenta increasing between the twentieth and twenty-fourth week. β-hCG, which reaches peak production around the eighth week in the first trimester, stimulates the maternal ovarian corpus luteum to continue secreting progesterone and estrogen, which maintain the placenta. The levels of the β-hCG produced by the placenta begin to drop rapidly at the beginning of the second trimester and remain low until delivery. Starting at the twentieth week, the placenta takes over the production of progesterone and estrogen.

The placenta's other function is to store nutrients such as carbohydrates, protein, calcium, and iron, which are released into the fetal circulation at certain rates. No blood is exchanged between mother and fetus at the villi level. Instead the cell walls are thin enough to allow the exchange of substances, such as nutrients and oxygen from the mother and waste and carbon dioxide from the fetus. This arrangement forms a protective barrier between the fetal environment and the maternal envrironment. The barrier is a good defense against most organisms; however, a host of viruses can pass through such as HIV (human immunodeficiency virus), measles, and chickenpox. Chemical substances including drugs and alcohol can also easily pass through into the fetal circulation.

Amniotic fluid

In the early first trimester, the amniotic fluid is generated largely from the maternal water and other substances that are allowed to pass freely through the chorioamnionic membranes. However beginning weeks 9 to 11, the fetal kidneys begin secreting urine, which slightly increases the volume of amniotic fluid. By the sixteenth week, the fetal kidneys are the major source of amniotic fluid production. The fluid is also composed of fetal cells that are sloughed off into the fluid. Those cells floating in the amnion are sometimes of interest to a clinician. A procedure called *amniocentesis*, allows physicians to puncture the amnionic sac and extract fluid with a needle, retrieving the fetal cells for genetic analysis.

Fetal swallowing and fluid passing through the amnionic membrane maintain amniotic fluid volume. Balanced against this, the fetal skin, lungs, and kidneys put fluid back into the cavity. Fetal skin remains thin enough to allow fluid exchange until near the end of the second trimester. The quantity of amniotic fluid increases steadily until around the middle of the third trimester, where it begins to decline.

The main functions of the amniotic fluid are to:

- Serve as a cushion for the fetus, preventing shockTEST 412.
- Prevent adhesions.
- Provide enough space that permits the fetus to grow symmetrically and allow movement, which further develops muscle tone.
- Maintain amniotic temperature favorable to the fetus.
- Serve as an aid to the development of the lungs and gastrointestinal system.

413. Fetal anatomy and function

As the second trimester begins, certain body structures in the fetus begin to assume the forms they will have in adult life. Other body structures change dramatically and usually this is a matter of increased size.

Central nervous system

The anatomy of the fetal head continues to develop. For example, the lateral ventricles undergo a change in shape even as the atria remain unchanged in size. The choroid plexus fills most of the lateral ventricle throughout the second and third trimesters. The occipital and frontal horns remain devoid of choroid. Most fetal brain structures are largely in place by the beginning of the second trimester and remain unchanged except for size.

However, a few structures start developing at 15 weeks. For instance the cerebrum, the largest most superior component of the brain starts at 15 weeks and continues to expand throughout pregnancy, folding and twisting into folds called gyri. Brain cells that are originally located against the lateral ventricles (called the germinal matrix) begin to migrate toward the cortex or outer layer of cerebrum, forming the gray matter. The vermis, or central portion, of the cerebellum is not completely formed until the eighteenth week. Also, the corpus callosum develops between the fifteenth and eighteenth week and forms the portions of the cavum septum pellucidi.

Important for the sonographer is the development of the lateral ventricles and the choroid plexus. The choroid secretes cerebral spinal fluid (CSF), which bathes the brain and spinal cord. Through the ventricles CSF is transported where it can regulate pressure on the brain within the cranial vault and also pressure in the spinal column. The fluid itself has nutrients (protein and glucose) that are provided for the brain cells.

Thoracic

Fetal lungs continue their development throughout fetal life. However, this is largely at the terminal alveoli (air sac) level, where future gas exchange will take place. All lung bronchi have formed by weeks 16 to 20. The alveoli will have fully formed by 26 weeks. The lungs mature completely at different times in the third trimester, but generally are thought to mature by at least 36 weeks. Filled with fluid, they do not, of course, exchange carbon dioxide for air filled with oxygen. That doesn’t happen until after birth. However, amniotic fluid is passed into and out of the bronchial tubes and trachea through the mouth in the third trimester. Prominent in this process of moving fluid into and out of the lungs is the movement of fetal diaphragm.

The dominant structure of the fetal thoracic cavity is the heart. All four chambers continue growing in size, with the atrial chambers symmetric and the ventricular chambers equal in size. The position of the heart remains constant throughout pregnancy, with most of it on the left side of the thoracic cavity. The heart fills a major portion of the thoracic cavity, with the right ventricle resting against the anterior wall of the thorax. The apex of the heart (the most anterior point where the two ventricles meet) is turned roughly 45 degrees to the left of the fetal midplane.

The fetal heart performs the same functions as adult hearts: it pumps blood. However, there are slight variations in the process, largely due to the presence of umbilical circulation. For instance, between

the two atria is a flap of cardiac tissue called the foramen ovale, which allows oxygenated and deoxygenated blood to pass back and forth, mixing the two.

Abdomen

At the beginning of the second trimester, the abdominal organs are in place. The gastrointestinal organs, esophagus, stomach, gallbladder, liver, and bowel, are located in their adult configurations. However, the liver is proportionally larger in the fetal abdomen compared to the amount of space it fills in the adult. After entering the fetus, the vein of the umbilical cord, courses within the abdomen anteriorly before diving into the liver toward the spine, where it meets the umbilical portion of the portal vein and the ductus venosus. Most of the blood from the placenta enters the ductus, which is connected to the inferior vena cava.

By the beginning of the second trimester, the fetal kidneys are in the final location in the abdomen: to either side of the spine. They grow steadily throughout the remainder of pregnancy. As the kidneys excrete urine, the bladder fills up and empties approximately every 30 to 45 minutes, particularly in the late second trimester.

Extremities (skeletal)

At the start of the second trimester, most of the skeletal structures have begun or completed ossification, except in the laminae (posterior bodies) within the thoracicolumbar spine and certain foot and pelvic bones. The spinal structures ossify between 20 and 24 weeks and some of the foot and pelvic bones do not complete ossification until 20 weeks.

Self-Test Questions

After you complete these questions, you may check your answers at the end of the unit.

412. Uterine environment

1. Describe the functions of the placenta.

2. List the main functions of the amniotic fluid.

413. Fetal anatomy and function

1. What structure does the choroid plexus mostly fill throughout the second and third trimesters?

2. Describe the course of the umbilical vein after it enters the fetus.

3–2. Imaging in the Second and Third Trimesters

Although the sonographer is to keep a sharp eye for any fetal abnormality, the main bulk of obstetric work focuses on evaluating for menstrual age (MA) and proper growth of the fetus. In women with

high risks for complications, the emphasis on looking for abnormalities increases, but mostly in the uterine environment that may possibly threaten the successful development of the fetus.

414. Second and third trimester sonographic considerations

The second trimester is the second three-month period of pregnancy, roughly the thirteenth or fourteenth week and ending in the twenty-fourth or twenty-fifth week. The third trimester is the third period of approximately three months that spans week 26 until week 40. The sonographic appearances of the fetus and the uterine environment change significantly throughout this period. Thus, standard approaches to second and third should be used.

Standard imaging of second and third trimester obstetrics

As with first trimester sonography, obstetric sonography in the second or third trimester should not be performed without a valid medical reason. Some of the common indications for sonography during this period are:

- Estimation of MA.
- Evaluation for size discrepancies.
- Maternal complaints, such as vaginal bleeding, abdominal or pelvic pain, palpable pelvic mass, abnormal biochemical markers.
- Evaluate for complications.
- Follow-up for fetal anomaly or resolution of unconfirmed abnormalities.
- Evaluation of multiple pregnancies.

The transducer used should mostly be transabdominal with as high a frequency as possible without sacrificing the ability of the sound beam to penetrate. A suitable frequency is between 3 and 5 MHz for most second and third trimester examinations. Endovaginal probes should never be used during this period unless attempting to determine the presence of the placenta lying near or over the cervix (previa), particularly in the late third trimester, or when determining the length and character of a possible incompetent cervix.

A standard examination of the fetus in the second trimester and third trimesters should include evaluation and documentation of the way the fetus is oriented within the uterus (fetal presentation), cardiac activity (fetal heart rate), the amount of amniotic fluid, the location of the placenta in relation to the internal os, the measurements of the fetus, and a survey of the basic anatomy. When performing the survey of a structure, such as the brain, make sure this involves sweeping the transducer through the area in search of subtle abnormalities. Also cervix measurements are considered extremely helpful to obstetricians and may be mandatory in your facility. Cervical measurements are performed most accurately with an endovaginal probe, which can improve the ability to place the calipers at both the internal and external os for a true cervical length. This method may or may not be allowed in your facility until the transabdominal view of the cervix with a full bladder indicates an abnormality. Normally, a cervix measurement less than 3 cm in length is suggestive of an incompetent cervix. Also, a funnel-shaped fluid collection contiguous with the amniotic cavity encroaching into the cervix is usually a good reason to perform the endovaginal cervical assessment.

The standard second trimester examination should have images of the head (mostly axial views) to include: the lateral ventricles with the choroid plexus, the midline falx that separates the two hemispheres, the cisterna magna and cerebellum at the posterior fossa, and the cavum septum pellucidi. Most radiologists will require more than just a representative image of these structures. They will probably want you to measure them. For instance, an anteroposterior (AP) measurement at the atria of the lateral ventricles is helpful. Try to obtain a length measurement of the axial view the cerebellum. Perpendicular to the length measurement of the cerebellum, measure the diameter of the cisterna magna from the vermis to the skin. Other radiologists may require images of the fetal facial structures or the bones that support the face. For instance, many departments consider images of the

lips and nose to be standard for the purpose of detecting cleft lip or cleft palate. Other sites require these images only if seen during the routine sweep through the entire head. The cleft lip is normally seen with a coronal view of the fetal face, while a more axial view of the nasal area will display the roof of the mouth for a possible cleft palate.

To document the fetal thoracic cavity, obtain a four-chamber view of the heart (fig. 3–1). This is best seen axially through the fetal chest, with the echogenic lungs against the heart. While not standard for a basic examination, some radiologists may require coronal and sagittal views of the thoracic cavity to demonstrate the lungs in relation to the diaphragm.

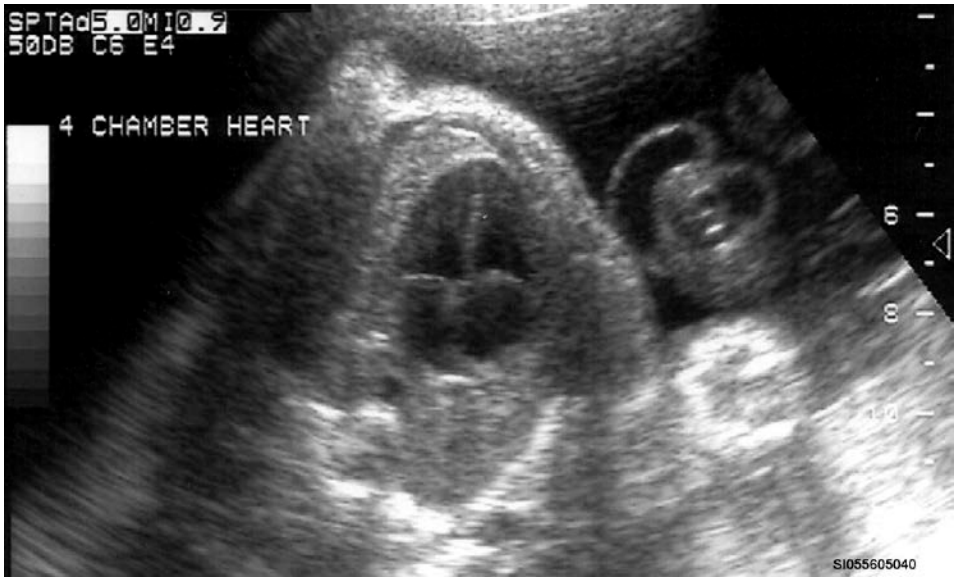

Figure 3–1. Four-chamber heart.

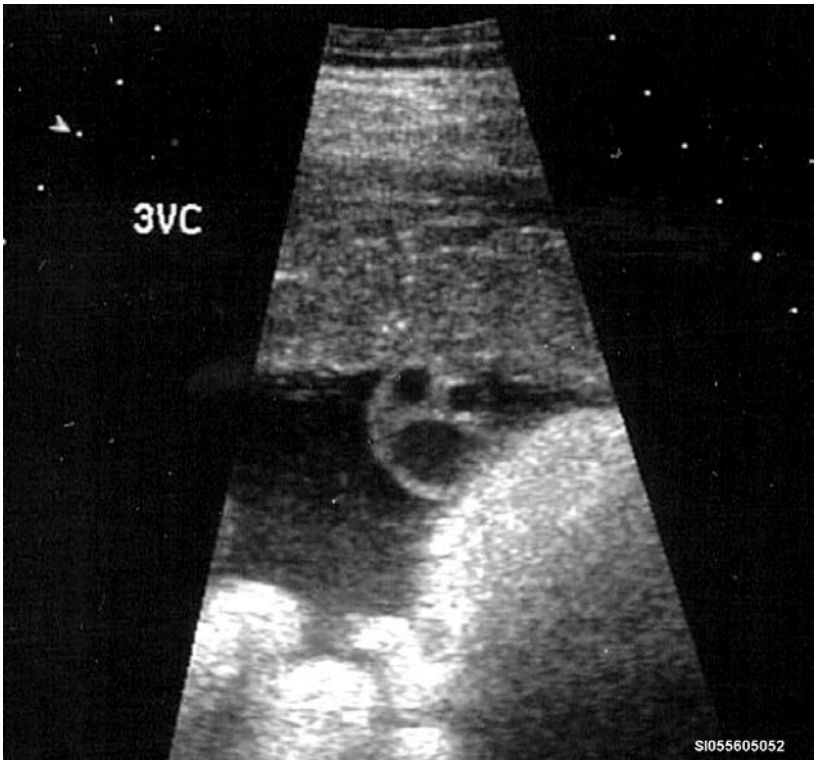

Figure 3–2. 3-vessel cord.

The abdominal organs should be documented with axial images of the stomach, kidneys, bladder, and the insertion site of the umbilical cord into the abdomen. Color Doppler, briefly, is helpful in properly demonstrating cord insert. Also, Doppler at the bladder may demonstrate two hypogastric arteries on either side of the bladder, indirectly indicating the presence of the two arteries in a 3-vessel umbilical cord. Vessel number of the cord should also be established through an axial image of a cord segment that is away from other structures, preferably surrounded by amniotic fluid (fig. 3–2).

Document the fetal spine to include the neck region (cervical), thoracic, lumbar, and sacral area. You should attempt to obtain as much of the spine on one image as possible. The spinal images should be obtained sagittal with posterior and anterior elements in the same plane. Also sweep through and obtain representative axial images of the spine at the cervical, thoracic, lumbar, and iliac segments.

Finally, document the presence or absence of all four extremities. It will be helpful to have some part of the body attached to the extremity in the image. Also, if possible, obtain fetal gender in multiple gestations. While commonly requested, seen, and performed, determination of the fetal gender is not part of a standard obstetric exam in some departments, and it may even be prohibited.

415. Assessment of menstrual age and fetal growth

Assessment of menstrual age is most accurately obtained with a *well-performed* first trimester crown rump length (CRL). Once a first trimester MA has been established, it should never be adjusted. The second trimester begins the thirteenth week of menstrual age and spans to week 25. Many women have routine obstetrical (OB) sonograms at least once during the second trimester, usually around the middle of the period (weeks 18–20). A significant portion of women in the second trimester have not had menstrual age established *sonographically*. You will find that much of the confusion surrounding certain MA cases is based on substandard second trimester measurements not matching first trimester measurements (which may or may not be substandard) or not matching *clinically* established dates (which can vary widely from obstetricians using multiple methods, including clinic sonography).

Apart from using first trimester CRL for establishing the MA, the next best method is the *biparietal diameter* (BPD) measurement of the head, usually before 20 weeks. The addition of the *femur length* (FL) before 20 weeks can also give reliable MA measurements after 14 weeks; however, the FL is not as precise as the BPD. Although in many departments the *abdominal circumference* (AC) is added to an overall MA calculation, the primary usefulness for the AC is fetal growth. Any of the parameters used for MA beyond 20 weeks begin to show decreases in precision from ± 2 weeks for the late second trimester to ± 3 and 4 weeks in the third trimester.

Menstrual age parameters

BPD, using a frozen axial image with the falx, thalamus, and cavum septum pellucidum centrally located within the cranium, is obtained by placing electronic calipers to the outer edge of one parietal bone in the near field to the inner edge of the opposite parietal bone. If obtained before 20 weeks, the measurement is accurate to within 1 week, comparable to the CRL.

The BPD measurement assumes a normal oval shape of the head axially. If the head shape appears narrow or round, the BPD may not be trustworthy for accurate measurement. To determine if the shape of the head will cause the BPD measurement to under- or overestimate the MA, it is helpful to use a *cephalic index* (CI). To obtain this quantity (unless your ultrasound unit performs this automatically), use the same frozen BPD image to place calipers at the frontal bone and at the occipital bone, the fronto-occipital diameter (FOD). Use both the BPD and FOD measurements to calculate the CI thus:

$$\frac{(BPD + FOD) \times 2}{100}$$

The CI formula will give you a normal value range of 70 to 86, with the mean set at 78. If the value you calculate is above or below the range, the BPD is unreliable for MA. Thus, one of two methods

must be used to achieve better precision: either a corrected BPD (BPDc) or a head circumference measurement must be obtained. For the BPDc, use this formula based on the CI of 78:

$$BPDc = \frac{(BPD \times FOD)}{1.265}$$

For the head circumference (which usually is required in most departments as a mandatory parameter), either use an electronic trace tool around the periphery of the same BPD image or manually calculate the formula for circle circumference:

$$\frac{(BPD + FOD) \times \pi}{2} = (BPD + FOD) \times 1.57$$

The FL is used to augment the head measurements for MA but is not as accurate. The long axis of the femur should be as perpendicular to the center of the ultrasound beam direction as possible. This is critical for accurate measurement as the unossified epiphyseal cartilages may be accidentally included in the measurement. Most charts of femur length and MA assume calipers placed on the ends of a horizontal diaphysis excluding the epiphyseals. FL measurements can be particularly useful in late-entry-to-care, third trimester cases when BPD and head circumference (HC) are unreliable for dating.

You will find it common in most departments to add abdominal circumference measurements to the BPD, HC, and FL parameters. This is usually based on a reasonable assumption that a composite range of precision is better than reliance on any one parameter with a potential for error. However, not all radiologists feel this way, so be aware that some may not require the AC measurement when the objective is to determine MA in the second or third trimester. Usually, the AC is a growth parameter and is used as a method to determine the estimated fetal weight (EFW) and as part of an HC/AC ratio for identifying growth problems.

One of two methods is used for AC measurements: the electronic trace calculations performed by the ultrasound package or by using two outer to outer edge measurements (anterior to posterior and side to side of the abdomen) and manually calculating the circumference of a circle. The level to measure is at the widest point of the abdomen that *also contains the landmarks of the umbilical portion of the left portal vein* and the stomach. As with head circumference, the formula for AC is:

$$\frac{(D1 + D2) \times \pi}{2} = (D1 + D2) \times 1.57$$

where D1 represents the anterior to posterior diameter and D2 represents the side diameter perpendicular to it.

A standard of primary significance to second trimester and third trimester sonography is to measure the amount of amniotic fluid. Too much or too little amniotic fluid can have profound consequences on the development of the fetus. With experience, you can subjectively determine from sight alone if enough amniotic fluid is present. However, the experience levels among sonographers vary wildly. Thus, several methods are available to determine fluid volume: the *amniotic fluid index* (AFI) and the deepest single-pocket (maximum vertical pocket). For the single deepest pocket, the procedure is to measure the deepest pocket of amniotic fluid AP free from cord or extremities in the fluid. Proposals of any amount less than 1–5 cm have been offered to indicate abnormally low amounts of amniotic fluid (oligohydramnios).

The AFI method is more rigorous and more widely used than the single-pocket method. For the fluid index, divide the uterus into four imaginary quadrants and measure the deepest vertical pocket in each quadrant. The resulting measurements are added together and the sum is compared to a chart based on MA. The range of normal is usually between 7 cm and 20 cm. The AFI can prove extremely helpful, particularly when coupled with an experienced sonographer making a visual (qualitative) assessment.

Fetal growth assessment

AC and AFI are both used for and are integral parts of evaluating fetal growth. Once a valid MA is established, either from first trimester sonography or from second trimester corrected BPDc or HC parameters, a composite of other common measurements are obtained. Thus AC and FL are additionally considered. The three parameters (normally AC, BPDc/HC, and FL) are then used in various formulas, which yield EFW. The EFW is then compared to weight percentile charts based on MA.

The abnormally restricted growth of the fetus, *intrauterine growth restriction* (IUGR), can be detected on sonography. If the abdominal circumference is abnormally small while the head size remains at appropriate size for MA, the IUGR is said to be asymmetric. When the entire fetus is small, it is considered symmetric IUGR. HC/AC ratios may be helpful in determining fetal symmetry while attempting to estimate fetal weight. The combination of ratios outside certain ranges (standard deviations) as published in numerous HC/AC charts, and EFW of less than 10 percent for MA, is highly suggestive of IUGR.

416. General obstetric abnormalities

Abnormalities or pathology for obstetrics can affect both the mother and the fetus. Maternal pathology acquired before or during the pregnancy can have a profound impact on the development of the fetus. However, not every maternal illness or pathology affects the fetus; instead, it puts the pregnancy at risk. Some mothers do not have any abnormalities, and only the fetus manifests pathology. In this lesson, we will look at both maternal and fetal abnormalities.

Fetal growth abnormalities

Fetal weight values that fall below the tenth percentile for a given menstrual age are considered growth restricted. Because this is 10 percent of fetuses, growth restriction is not all that common. Naturally small fetuses (termed small for gestational age or SGA) are also considered if the weight is less than the tenth percentile for gestational age. The difference between growth restricted and SGA fetuses, however, is the prognosis for growth restriction is poor.

In the course of routine examinations, you should be aware of the parameters and their relations to one another. This is because growth restricted fetuses have a poor prognosis. IUGR is caused primarily by placental insufficiency, usually as a result of maternal factors, such as hypertension, drugs, and diabetes. Placental insufficiency simply indicates that the placenta is unable to support the pregnancy, usually as a result of some vascular abnormality. Early insult from viral infections and genetic abnormalities can cause early first trimester IUGR.

On the opposite end of the growth evaluation spectrum are fetuses excessively large for gestational age (LGA). The condition for an abnormally large sized fetal body is called *macrosomia*, which, when applied around the delivery date (perinatally), is commonly an EFW of more than 4,000 grams (g). LGA and EFW are largely found with diabetic mothers. The LGA fetus, technically one with an EFW above the ninetieth percentile for MA, and the macrosomic fetus are both at risk for increased disease or death. Because these complications can occur with a large fetus during delivery, most obstetricians opt for a cesarean section (C-section).

High risk pregnancies

Maternal illness or abnormalities place a pregnancy at risk for significant interruption in fetal development. Some of the more common abnormalities that obstetricians consider high risk are:

- Diabetes.
- Hypertension.
- Blood group incompatibility and sensitivity.

Other events that are quite normal can place a pregnancy into the high risk category, such as maternal age (typically greater than 35) and multiple gestations.

Diabetic mothers

Insulin dependent diabetic or gestational diabetic mothers (those who acquire diabetes only during pregnancy) have increased risk for carrying fetuses with macrosomia or LGA. The poor glucose control contributes to the increased growth of the fetus. Diabetes is a primary cause of increased and excessive amniotic fluid production, or *polyhydramnios*.

Polyhydramnios can cause the amniotic membranes to rupture prematurely (premature rupture of the membranes, or PROM), which in turn can lead to leakage of amniotic fluid before term, and may even start preterm labor. The risk is for amniotic infection and a premature fetal birth.

A small percentage of diabetic mothers will also have fetuses that display a certain skeletal abnormality called *caudal dysplasia sequence* (also known as caudal regression syndrome). This is a spectrum of abnormalities that constitute the abnormal development (hypoplasia) or absence (agenesis) of the fetal sacrum, parts of the lower spine, and lower limbs. Frequently, renal and gastrointestinal (GI) abnormalities accompany this sequence. Do not confuse this sequence with sirenomelia sequence, another skeletal sequence with a nearly identical spectrum of problems. However, caudal dysplasia is associated with polyhydramnios, while sirenomelia is featured with severe oligohydraminios. While rare in diabetic mothers, caudal dysplasia is most likely to be found associated *only* with diabetic mothers.

Hypertensive patients

Mothers who have high blood pressure before pregnancy (chronic hypertension, usually diagnosed before 20 weeks) may go on to carry fetuses with placental insufficiency. The main cause is vascular insult, which results in abnormal vascular supply to the placenta. The poor development of the placenta results in a measurement of less than 2 cm from chorion plate to basal layer. The thin placenta will be unable to support the fetus, which in turn becomes vulnerable to IUGR, morbidity, or mortality.

Mothers, who acquire hypertension during pregnancy, particularly after 20 weeks, are said to have *preeclampsia*. Preeclampsia is pregnancy-induced hypertension accompanied by protein in the maternal urine (proteinuria) and general swelling (edema). If the hypertensive state is not treated, preeclampsia increases in severity and results in outright eclampsia (one or more convulsions that can cause coma or death).

Another severe abnormality often found in either chronic hypertension or preeclampsia is *placental abruption* (abruptio placentae), a premature detachment of the placenta from the myometrium. Such an occurrence can cause excessive bleeding, intensive maternal pain, or even the onset of preterm labor. Immediate delivery is usually necessary for abruption patients.

Do not confuse painless vaginal bleeding in the second and third trimesters with placental abruption. Bleeding without pain is normally caused by placenta previa, the covering of the internal os of the cervix by the placenta.

Maternal causes for immune and nonimmune fetal hydrops

When maternal blood type (negative Rh factor [Rhesus factor]) is incompatible with fetal blood type (positive), a danger exists if the fetal blood cells enter the maternal circulation or if the mother is already sensitive to the positive Rh factor from blood entry of previous childbirth. Other reasons for fetal blood entry into maternal circulation are amniocentesis, abruption, and placental hemorrhage. If the incompatible fetal cells gain entry, the maternal immune system may kick in and manufacture antibodies small enough to cross the blood barrier of the placenta and destroy the fetal blood cells. The result is fetal anemia, which can cause the fetus to increase production of red blood cells. This overproduction, in turn leads to general edema as well as other abnormalities such as ascites and

polyhydramnios. The placenta will also thicken abnormally. The term for this abnormality is called *immune fetal hydrops* or erythroblastosis fetalis.

An identical series of abnormalities can also occur where there is no maternal/fetal blood type incompatibility. This *nonimmune fetal hydrops* can be caused by severe maternal diabetes and anemia. Also, infections and twin-twin transfusion syndrome in multiple pregnancies (more on that below) are other maternal risk factors associated with nonimmune hydrops. Other fetal causes can be found for nonimmune hydrops such as chromosomal, cardiovascular, and other malformations.

Multiple gestations

The presence of multiple gestations is inherently high-risk because the chance of a poor outcome is increased. Thus, these pregnancies are carefully watched. Some of the things that can go wrong in multifetal pregnancies:

- Spontaneous abortion or preterm birth.
- IUGR.
- Congenital anomalies.
- Fetal demise.

One problem with multiple gestations is with monoamniotic pregnancies. If two fetuses share one amniotic sac and one placenta, the two may get the cords tangled. The danger is in twisted cord cutting off circulation. With diamniotic gestations each fetus will have an individual sac but still can share the same placenta.

Monochorionic *diamniotic* pregnancies pose a risk for twin-to-twin transfusion syndrome. This is an abnormality of the placental blood supply, due to a connection of arteriovenous shunting of blood from one twin (donor), to the other twin (recipient). The donor twin, because of placental insufficiency soon becomes growth restricted and anemic, with oligohydramnios resulting from malfunctioning kidneys unable to produce urine. Thus, the donor sac shrinks and the amnion is wrapped around the fetus, pulling it toward the uterine wall. This process is often termed *stuck twin*. Conversely, the recipient twin is receiving twice the blood flow and is consequentially growing in size with kidneys overproducing urine. Eventual overwork of blood flow can lead to fetal heart failure and the perfusion of serous fluid and blood plasma into the fetal tissues.

417. Common obstetric abnormalities

Fetal abnormalities that can occur are literally in the hundreds. The differences between many of them are barely detectable with sonography. We will concentrate on only the most common that you are likely to encounter during the obstetric sonogram.

Fetal head

Fetal head pathology can be divided into those abnormalities involving the skull or cranium and those abnormalities involving brain tissues. Frequently, skull abnormalities will coexist with brain anomalies. We will take a look at a few of each type.

Anencephaly and acrania

The most common neural tube defect abnormality is *anencephaly*, which is the absence of cerebral brain and skull above the level of the orbits. It occurs in one out of every 1,000 births and is caused by the failure of the anterior end of the embryological neural tube (rostral neuropore) to close properly. The remaining midbrain and posterior fossa elements are necrotic and covered by a thin vascular membrane. The forebrain is sometimes replaced by a small, flat mass of vascular tissue called the *area cerebrovasculosa*. Levels of alpha-fetoprotein (a fetal protein that is used as an indicator of open neural tube defects) are elevated in the maternal blood serum (MSAFP). Anencephaly is universally fatal.

Maternal diabetes mellitus and exposure to certain chemicals increases the risk for anencephaly. Occasionally, early rupture of the amniotic membranes (amniotic rupture sequence, which is also know as amniotic band syndrome) can cause anencephaly, but is usually accompanied by other similar defects and even amputations.

Acrania is the absence of the cranium. What remains uncovered is the brain, a condition called *exencephaly*. The failure of the cranium to develop is due to improper embryologic processes. Acrania may be a precursor to anencephaly.

Microcephaly

An abnormally small fetal head is called *microcephaly*. A poorly developed brain that is small is the source of the small skull. A host of problems can cause it from genetic to chemical, radiation, and viral exposure.

Cephaloceles

Cephaloceles are intracranial contents that herniate out through cranial holes or defects. The protrusion of the meninges (membranous covering of the brain) with CSF through a defect in the cranium is called a *meningocele*. If brain matter protrudes through the defect, it is called an *encephalocele* or *meningoencephalocele*. These are neural tube defects that cause elevations in MSAFP. The cephaloceles mostly occur along the midline of the occipital bone, but some can be seen at the frontal bone or along the sides.

Intracranial abnormalities

Compared with the neural tube defects just listed, the abnormalities of the brain are relatively rare. Most intracranial abnormalities are the result of cerebral hemisphere formation. These developmental errors give rise to a host of accompanying facial abnormalities. Other intracranial abnormalities become evident only because of underlying causes or are isolated, acquired occurrences. We will list only the abnormalities you are likely to encounter.

Ventriculomegaly

Excessive lateral ventricular size is called *ventriculomegaly*. It is the most common abnormality of the fetal brain. Ventriculomegaly is caused by the obstruction of normal CSF flow. The most common obstruction is located in the nearby aqueduct of Sylvius below the third ventricle (aqueductal stenosis). Causes of obstruction are encephaloceles, dilation of the fourth ventricle, and chromosomal disorders, and aqueductal stenosis.

Choroid plexus cysts

Frequently, the choroid plexus will demonstrate small cysts, unilateral or bilateral. They are usually normal and are reabsorbed by the twenty-sixth week MA. Large cysts, however, tend to be associated with a chromosomal abnormality, specifically trisomy 18.

Less common abnormalities

Rarely will you encounter an instance of *holoprosencephaly*, failure of the cerebral hemisphere development. Of the three types (lobar, semilobar, and alobar) that signify the level of hemisphere separation and development, alobar is the most severe. The alobar variety demonstrates on sonography as a single ventricle and brain tissue surrounding it, with thalami fusion. The falx is completely absent. Facial abnormalities are common with this abnormality.

Dandy-Walker complex is seen on sonography as incomplete fusion of the hemispheres of the cerebellum, cysts in the posterior fossa, and an enlarged cisterna magna beyond 10 mm. Variations of each of these is common within the complex.

Fetal spine

A defect in the posterior laminae of fetal vertebral bodies is called *spina bifida*. This is a neural tube defect in which elements of the spinal cord are uncovered by unfused spinal laminae. Spina bifida in which nothing is covering the exposed cord is called spina bifida *aperta* or *cystica*. Spina bifida with skin covering the open cord is called spina bifida *occulta*.

Associated with spina bifida is *Arnold-Chiari malformation, type II*. The second of two types, this malformation displaces portions of the cerebellum through the foramen magnum, distorting the cerebellar shape.

Fetal chest

Occasionally, problems will manifest in the fetal chest or thorax. From masses to fluid, fetal chest abnormalities can be quite dramatic or barely noticeable on sonography. For that reason, it will help you to be familiar with some of the most common problems of fetal chest pathology.

Congenital diaphragmatic hernia

The failure of the diaphragm to completely develop by 10 to 14 weeks can cause the appearance of defects or holes through which abdominal contents can herniate into the thoracic cavity. This occurrence is called *congenital diaphragmatic hernia* (CDH), the most common thoracic abnormality outside of the heart. The herniation occurs mostly on the left side through a posterolateral opening called the foramen of Bochdalek. Bilateral hernias are rare. The principal organs that pass through are the stomach, intestine, liver, and spleen. The causes are mostly sporadic but approximately a fourth of CDH cases are chromosomal, particularly trisomy 18. The presence of the organs within the thoracic cavity obviously places enormous pressure on the lungs. The consequences of this compression are impaired development of the lung tissue, or pulmonary hypoplasia.

Congenital cystic adenomatoid malformation

Slightly less common than CDH is the congenital cystic adenomatoid malformation (CCAM) of the lungs. This abnormality is the proliferation of pulmonary tissue that far exceeds the development of surrounding tissue (harmatomatous), causing the formation of cystic spaces. Three types exist based on cyst size:

- Type I—macrocystic, large cysts or cyst 2–10 cm in diameter (the most common).
- Type II—medium-sized cystic, multiple cysts up to 2 cm in diameter.
- Type III—microcystic, cysts less than 0.5 cm giving collection of cysts a solid appearance.

Less common lung abnormalities

Another mass that may appear in the lung is the *bronchopulmonary sequestration*. This is a mass of nonfunctional pulmonary tissue that is formed mostly within the base of the lung. Two types, intralobar and extralobar, exist with intralobar being found in adults. The extralobar variety is called an extrapulmonary sequestration. The mass is separate from lung tissue by its own pleural covering.

Rare *bronchogenic cysts* can be seen that normally do not cause a shift of the mediastinum, as with CCAM. They are not associated with fetal anomalies and usually do not cause problems unless very large.

Pleural effusion (called *hydrothorax*) is fluid in the space surrounding the lungs, or the pleura. It is thought to be an early sign of hydrops. The pressure placed on the lung can prevent normal fetal lung development. Unilateral effusions have a better prognosis than bilateral appearances.

Fetal abdominal wall

Disruption of the anterior skin surface of the fetal abdomen causes elevations in MSAFP, just as with neural tube defects.

Omphalocele

The most common fetal abdominal wall defect is the *omphalocele*, the presence of abdominal contents in the base of the umbilical cord. Large omphaloceles usually contain liver, bowel and ascites, and are thought to be due to the failure of the abdominal wall to develop properly early in the first trimester. Small omphaloceles filled with bowel and ascites are considered to be the failure of normal physiologic herniation to completely resolve. Omphaloceles are frequently associated with fetal anomalies, half of which are related to the heart. A series of syndromes and disorders are associated with omphalocele, principally the Pentalogy of Cantrell (heart outside of the chest, ectopia cordis) and Beckwith-Wiedemann syndrome (macrosomia and macroglossia, an enlargement of the tongue). Chromosomal abnormalities are also seen, commonly trisomies 13 and 18. Maternal age is a risk factor for omphalocele.

Gastroschisis

A small defect of a few centimeters, located in the abdominal wall beside the umbilical cord insertion, allows the bowel to herniate freely into the amniotic fluid. This condition is called *gastroschisis*. The defect usually appears on the right of a normal cord insert. Gastroschisis is not associated with genetic abnormalities and the only fetal anomalies that occur with it are linked to the floating bowel. For instance, bowel ischemia can develop leading to atresia. Unlike omphalocele, the bowel is exposed to amniotic fluid because of the absence of a membranous covering. Thus a fibrous coating develops on the outer walls of the floating bowel loops.

Other wall defects

Pentalogy of Cantrell and *limb-body wall complex* (LBWC, or sometimes body-stalk anomaly) are two disorders that are each spectrums of abnormalities centered on defects in the thoracic and abdominal wall. The presence of heart outside the chest wall, body ascites, and an omphalocele are hallmarks of pentalogy of Cantrell. The word *pentalogy* refers to the five wall defects that are present.

LBWC is more extensive with neural tube defects, *pentalogy of Cantrell*, omphalocele, and defects of the extremities. Amniotic bands are occasionally seen, which can lash the fetal face and cause craniofacial abnormalities. Another LBWC characteristic is severe scoliosis of the spine. Usually the prognosis is poor for any fetus with the heart located outside of the body and exposed to the amnion.

Fetal gastrointestinal system

Obstruction of the fetal GI track is an abnormality that is more common in the esophagus and duodenum. Other GI abnormalities occur such as liver masses and meconium peritonitis, but are not as common as the atresias.

Esophageal atresia

When a fetus swallows, the amniotic fluid passes through the esophagus to the stomach. Also fluid is drawn into the trachea of the fetus and on into the lungs where the amniotic fluid pressure keeps the bronchioles open for optimal development. Occasionally, a fetus' esophagus will fail to develop and instead is closed off, which prevents the swallowed amniotic fluid from reaching the stomach. This terminal pouch is called esophageal atresia. Five types exist, of which the relatively rare pure form just described will present with an absent fetal stomach and polyhydramnios on sonography. The most common form, however, contains an alternate pathway for amniotic fluid to reach the stomach, the distal tracheoesophageal fistula. The fistula is a communicative channel that allows fluid to pass through. In this case the distal esophagus that enters the stomach is attached directly to the trachea, allowing small amounts of amniotic fluid to leak into the stomach.

Esophageal atresia is associated with IUGR in less than half of cases, with polyhydramnios, mostly seen after 24 weeks MA. Associations with cardiac abnormalities and other GI abnormalities are present with esophageal atresia, as is the association with trisomy 18.

Duodenal atresia

The most common of fetal *intestinal* obstructions, duodenal atresia is caused by blockage of the proximal duodenum by a web or membrane. Half of the cases of duodenal atresia are accompanied by polyhydramnios. Anomalies associated with duodenal atresia are IUGR, cardiac, and vertebral abnormalities. Chromosomal association is mostly from trisomy 21.

Fetal urinary system

An important part of the fetal system is the urinary system, which also plays a significant role in the maintenance of amniotic fluid. Abnormalities of the urinary system can be divided into obstructional anomalies and structural anomalies. The obstruction of the fetal urinary tract causes dilation. The level of obstruction characterizes the structure dilated (usually proximal) and type. For instance:

- Ureteropelvic junction obstruction (renal pelvis obstruction: renal hydronephrosis).
- Ureterovesical obstruction (obstruction at bladder entrance: ureterovesical junction stenosis, megaureter, and ectopic ureterocele).
- Uretheral obstruction (obstruction at the posterior urethral valves in males: posterior urethral valves, megacystis).

Hydronephrosis

Hydronephrosis is the most common fetal anomaly. It is the dilation of the renal pelvis as a result of obstruction at one of three levels: the *ureteropelvic junction* (UPJ), the *ureterovesical junction* (UVJ), and *posterior urethral valves* (PUV). Of the three, the most common cause of hydronephrosis is UPJ obstruction. The least common cause is UVJ stenosis. The causes of UPJ obstruction are unknown, but probably are related to the anatomy of the junction of renal pelvis and ureter, specifically the presence of abnormal valves.

PUV is caused by abnormal posterior urethral valves, which are only in males. Females can also have rare forms of bladder outlet obstruction, but these are usually chromosomal anomalies such as cloacal malformation (a failure of embryological bladder to naturally separate from the rectum). PUV causes massive dilation of the bladder, which fills nearly the entire abdomen of a fetus. This abnormality is usually accompanied by oligohydramnios, megaureters, and hydronephrosis.

Renal agenesis

Renal agenesis is the absence of the kidneys. *Bilateral renal agenesis* leads to certain fetal demise. Without the kidneys to produce urine, the bladder is unfilled and the fetus is unable to begin contributing to amniotic fluid volume. Severe oligohydramnios is the result but usually only after 16 weeks when the fetal kidneys would normally take over fluid production. Oligohydramnios begins the *Potter sequence*; that is, fetal compression from lack of amniotic fluid causes further abnormalities such as abnormal development of the fetal lungs (pulmonary hypoplasia), respiratory distress, blood instability, edema, and facial abnormalities (Potter facies). This is why bilateral renal agenesis is commonly referred to synonymously with Potter syndrome or sequence. A *syndrome* is a pattern of multiple abnormalities with a common cause; a *sequence* is the process that describes a single abnormality (whatever the cause) developing further abnormalities. Thus, bilateral renal agenesis causes oligohydramnios, which triggers a Potter sequence lethal to the fetus.

Unilateral renal agenesis is the absence of one kidney only. It is four times as common as the bilateral variety. Because the fetus still has another kidney and bladder function, amniotic fluid production is normal. The kidney compensates for the function of a missing kidney, increasing in size as it works twice as hard as normal (compensatory hypertrophy).

Polycystic renal disease

Polycystic renal disease is an inherited disease that is passed on between generations in two ways: through dominant chromosomes (autosomes) or recessive autosomes. *Autosomal-dominant polycystic*

kidney disease (ADPKD) normally is established in adults (with true cysts) but can also be seen in neonates and infants. Thus, you may hear the term, "*adult* polycystic kidney disease," being associated exclusively for adults. Be aware that it can appear in the fetus as well.

Autosomal-recessive polycystic kidney disease (ARPKD) is usually established in the fetus and young children. You may commonly hear it referred to as *infantile* polycystic kidney disease. Although rare, this disease is more common in the fetus than ADPKD. In ARPKD the renal tubules are dilated (tubular ectasia). These multiple dilated tubules (hence, *polycystic*) progressively affect the function of the kidneys. Also, the size of the kidneys dramatically increases, often filling the fetal abdomen. By the end of the second trimester or early third trimester, the kidneys cannot produce adequately to maintain amniotic fluid volume and oligohydramnios sets in.

Multicystic dysplasic kidney

The total replacement of the fetal kidney by cysts is called *multicystic dysplastic kidney* (MCDK). It is caused by the complete obstruction (atresia) of the ureters and renal pelvis during the period of embryologic renal development. The MCDK kidney is nonfunctional. The disease is usually unilateral with less than a fourth manifesting bilaterally. Bilateral MCDK is fatal to the fetus because of the absence of normal renal tissue and subsequent oligohydramnios with pulmonary hypoplasia.

However, the prognosis is improved with the presence of a contralateral (opposite side) kidney, which provides normal bladder filling and amniotic fluid volume. The absence of a contralateral kidney (agenesis) with MCDK is virtually synonymous with bilateral renal agenesis; both cases are lethal.

Skeletal abnormalities

Skeletal dysplasia is the abnormal development of the bone and cartilage, some of which are lethal. Despite the high number of types (over 200), skeletal dysplasias are relatively rare when compared with the incidence of other abnormalities of the second and third trimester fetal abnormalities.

418. Genetic obstetric abnormalities

A significant portion of fetal abnormalities develop as result of maternal disease or illness, maternal and first trimester exposure to chemicals, drugs, infections, embryological dysplasia or hypoplasia, and trauma. Some fetal abnormalities are inherited. Genetic or chromosomal disorders constitute most of the fetal *syndromes, malformations, associations, and sequences* occasionally seen in sonography.

A syndrome is a pattern of different abnormalities with a common cause (eitiology) in a single individual. A malformation is a structural defect in an organ or body caused by an abnormal development process. An association is an apparently more than coincidental occurrence of two or more abnormalities together for which no common cause is known. A sequence is a process where an abnormality (whatever the cause) prompts the development of further abnormalities.

Common genetic disorders

Often, maternal biochemical laboratory screening will prompt suspicions for chromosomal abnormalities. These patients are then referred to the sonography department for evaluation. The goal is to rule out genetic abnormalities. Most genetic disorders are rare. A few are common enough to potentially occur when you scan.

Trisomy 21

Trisomy 21, Down syndrome, is the most common of fetal chromosomal abnormalities. The cause is usually an extra chromosome number 21. Most are born with moderate mental retardation.

Trisomy 18

Trisomy 18, Edwards syndrome, is the second most common chromosomal abnormality, with an extra chromosome 18. This trisomy is much more lethal than trisomy 21, as most cases end in death two to three years after birth. Severe mental retardation is expected with these cases.

Trisomy 13

Trisomy 13, Patau syndrome, is less common than either 18 or 21. The cause is an extra chromosome 13 that is fatal within two years after birth.

Turner syndrome

Several types of *Turner syndrome* exist. Half of the cases will have a chromosome count of 45 (instead of the normal 23) and the absence of an X or Y sex chromosome, leaving a single X. The fetus may also be hydropic, with pleural effusion and ascites. Cardiac abnormalities are common.

Gestational trophoblastic disease

Partial hydatidiform moles are triploid genetic disorders; that is, two sets of paternal chromosomes are inherited along with one set from the mother. Significantly different from the complete hydatidiform mole is the presence of a fetus alongside partial moles.

When a mole begins to invade the myometrium, it is called invasive mole. If it penetrates across the myometrium, the condition is called *placenta percreta*, and heavy bleeding is the result.

Half of molar pregnancies subsequently develop into malignant tumors called *choriocarcinoma*. The choriocarcinoma is known to metastasize to the lungs, liver, or brain.

419. Imaging of second and third trimester pregnancies

Some women begin pregnancy at risk for complications; that is, they possess certain characteristics that automatically place their pregnancies in jeopardy. Others acquire high risks as pregnancy proceeds. You should be aware of some of the problems that can abruptly manifest during the second and third trimesters.

Recall that the standard for routine obstetrical sonography is to document fetal heart rate, AFV, fetal presentation, placental position, fetal measurements, and to perform an anatomic survey. The purpose for performing the anatomic survey is to detect structural fetal abnormalities. There are literally hundreds of fetal abnormalities, with many various subtypes. Thus, in this final section, we will briefly touch on only the most common that you may encounter.

High risk pregnancies

Pregnancies that have a tendency to develop into abnormalities are termed high risk. Usually, the risk is incurred because of maternal problems or because of advanced maternal age. For the maternal problems, a broad range of events can negatively affect a pregnancy, from viral infections to drug use. Frequently, the problems are disease related or beyond the control of the mother. Below, we will briefly touch on the general sonographic approaches to three of the most common categories of high risk pregnancies.

Diabetic mothers

If you find on sonography that the fetus seems to be completely surrounded by fluid (which becomes more unusual after 21 weeks), and that your AFI, EFW, and even AC/HC ratio measurements all exceed the highest normal ranges, polyhydramnios with macrosomia or LGA is highly suggested. Also, diabetic mothers may have a fetus with structural defects located at the caudal or tail end of the spine.

Based on your measurements, your ultrasound equipment should be able to calculate the EFW. It may be helpful to know which EFW chart your equipment uses to calculate fetal weight. Ensure that all of your measurements, particularly the abdominal circumference, are as accurate as possible. If you see the estimated fetal weight is above 4,000 grams, you should inform the radiologist that this is a strong indication of macrosomia. Remember that this is more the case for *diabetic* mothers. If your measurements yield fetal weights higher than 90 percent of fetuses for a given MA, the likelihood that the fetus is LGA is increased.

Hypertensive patients

On sonography, some pregnancies of hypertensive mothers will display a hypoechoic to anechoic area thicker than 1 to 2 cm in between the placenta and the myometrium (retroplacental bleed or hematoma), or at the edge of the placenta (marginal bleed). This has the effect, quite noticeable on sonography, of pulling the placenta away from the wall of the uterus. In the second trimester, the situation can be dangerous if close to half the placenta is pulled away.

Use color Doppler to ensure that the retroplacental area is not vascular or is not a mass (leiomyomas or other tumors behind the placenta will usually display some vascularity). The hematoma should be measured in three dimensions, which will be important for subsequent sonograms that will evaluate the appearance of this area.

Multiple gestations

One way to determine if the placenta is shared or if it is merely two placentas fused together is to look for a flare of placental tissue up into the area between the sacs. This tiny triangle of tissue, the "twin peak," is a good indication (no matter how thin the tissue separating the sacs) that the pregnancy is dichorionic. Incidentally, same gender fetuses will never be dichorionic, so check the sex.

Recall that for stuck twins, the donor sac shrinks. On sonography, the fetus appears stuck to the uterine wall. Also recall, the recipient twin receives twice the blood flow and is consequentially growing in size with kidneys overproducing urine. This leads to the recipient twin's sac being polyhydramniotic. You may also note a general edema and ascities (nonimmune hydrops).

If twin-to-twin transfusion is not an issue with a multiple gestation pregnancy, the standard approach is to evaluate each fetus as if it were a singleton pregnancy. Be sure to establish location within the uterus and use labels annotating this. Note the locations of each placental insertion of the umbilical cords. Perhaps the most important measurement for multifetal sonography is the EFW of each. A comparison of the weights should help determine the presence or absence of growth discordancy. A difference in weight of 20 to 25 percent is commonly thought to suggest discordancy.

Fetal head

The most common of congenital defects, central nervous system (CNS) abnormalities are usually obvious on sonography. However, some can prove extremely subtle or are impossible to see during a certain menstrual age. The optimum time for examining most of the following CNS abnormalities is between 18 and 22 weeks.

Recall that standard head surveys require evaluation of the lateral ventricles with the choroid plexus, the midline falx that separates the two hemispheres, the cisterna magna and cerebellum at the posterior fossa, and the cavum septum pellucidi in central anterior portion of the midbrain. However, some abnormalities of the head involve the skull instead of the brain.

Several abnormalities of the cranium can usually be identified by sonography. While attempting to locate structures for standard imaging and for BPD/HC measurements, the first two of the following should be obvious, while the last two may be subtle depending on your carefulness.

Anencephaly and acrania

For suspected anencephaly, a sagittal or coronal view of the fetal head will display no skull or brain superior to the orbits. A small mass of midlevel echoes may be seen in the area where the cerebral hemispheres would be. This small mass is the area cerebrovasculosa. Other sonographic abnormalities may be seen in the fetus. Also, anencephaly is associated with polyhydramnios, which may be a response to swallowing impairment, continuous urination, or from the likelihood of maternal diabetes.

Except for the appearance of disorganized brain tissue with prominent sulci, acrania is identical in appearance sonographically to anencephaly.

Microcephaly

On sonography, microcephaly is usually discovered by the unusually small appearance of the head and the disorganized appearance of the brain. BPD and HC measurements sometimes can indicate the small head size, but this is usually only in the presence of certain dates. The criterion used for head measurements in most departments is two or three standard deviations below the mean HC or BPD for menstrual age. Additional clues to the presence of microcephaly are the presence of calcifications within the brain tissue and possibly enlarged ventricles (ventriculomegaly).

Cephaloceles

If you see a cystic structure attached to surface of the cranium, it is likely to be a cephalocele. It may be helpful if you measure the diameter of the protrusion. Frequently, other abnormalities of the face, spine, and extremities are present with cephaloceles. Therefore, an extremely careful look into the brain and along the spine is appropriate for these abnormalities.

Ventriculomegaly

On sonography, the anechoic lateral ventricles are expanded to a noticeable size with moderate ventriculomegaly. Massive ventriculomegaly will demonstrate the choroid literally hanging or dangling down into the ventricle from the near walls. The most common method to confirm ventriculomegaly, however, is to measure the atria of the lateral ventricle. Use an AP measurement on the inner edge to inner edge of the point where the parallel lateral horn walls turns slightly in direction to become the walls of the occipital horn. Atrial measurements in the normal ventricle are around 7 mm. If the measurement you obtain is over 10 mm, ventriculomegaly is highly suggested.

Choroid plexus cysts

If you see cysts within the echogenic choroid plexus of the lateral ventricles, measure them as you would any other. You should try to magnify the axial view at the level of the atria so that only the fetal head fills the display screen. This will allow for more accurate placement of your electronic calipers. For multiple cysts, measure AP and width diameters for the largest cyst. Some radiologist may have you measure all cysts.

Fetal spine

A meningocele can protrude through the defect with a sonographically cystic appearance, identical to that found in the cranium. If you see solid elements of various echogenicities, these are likely to be portions of the spinal cord accompanying the meninges and CSF. This complex cyst is called a meningomyelocele. Measure any of the spina bifida abnormalities. Spina bifida should prompt an even more careful evaluation of each vertebral body in both sagittal and transverse planes beyond the original survey. MSAFP values should be elevated. Occasionally a large mass, called a sacro-coccygeal teratoma, will protrude from the sacrum. Do not confuse this with a meningomyelocele. The teratoma is mostly solid and heterogeneous, while the meningomyelocele is largely cystic.

For a case of suspected Arnold-Chiari malformation, be sure to demonstrate on the image the round lobes of the cerebellum compressed enough so that you should see the structure shaped like a banana. The cisterna magna also should be displayed as closed off and unseen on sonography.

Fetal chest

Much of the basic chest imaging performed in many centers concerns the maturity of lung tissue in the third trimester. However, a standard anatomic survey of the chest is largely concerned with a four-chamber view of the fetal heart. Many abnormalities of the heart can be detected with just an attempt to obtain a four-chamber view. Lung masses, cystic, complex, or solid, are also of concern and we will touch on a few.

Congenital diaphragmatic hernia

To demonstrate congenital diaphragmatic hernia on sonography, the classic four-chamber view of the heart will provide the proper level to show the shift of the heart and mediastinum to the right or to the left for right-sided hernias. You should generally see an anechoic stomach on the left side beside the four-chamber heart. Ensure that the chest is at a true axial plane. To do this, make sure the flare of the thoracic ribs to either side of the vertebral body is symmetric in shape. To confirm bowel in the chest, look for peristalsis next to the heart on real time. The AC will show an extremely small size due to the absent contents. You may see polyhydramnios.

Congenital cystic adenomatoid malformation

Sonography usually can detect these masses beginning at 16 weeks. The appearances of macrocystic CCAM can mimic stomach or bowel CDH. To help distinguish between the two, waiting for peristalsis characteristic of bowel is helpful. The outcome, favorable or otherwise, depends on the size of the cysts, the amount of shift they cause on the mediastinum, and the presence of polyhydramnios and hydrops. The presence of polyhydramnios and hydrops is particularly unfavorable. Because some CCAMs are associated with chromosomal abnormalities, you should make a careful search of the fetus for other problems.

Less common lung abnormalities

The bronchopulmonary sequestration is a mass of varying sizes normally seen within the base of the lung. You should demonstrate the presence of an echogenic wedge that is homogeneous in echotexture. Use color Doppler to reveal a feeding vessel branching from the aorta to the sequestration. The extrapulmonary sequestration is an echogenic mass seen in the *lower angles* of the lungs. Both types of sequestration may be confused with CCAM, types I and II.

Pleural effusion can be seen on sonography as anechoic slivers or crescents when there is only a small amount of fluid. However, with increased effusion, the appearance can become more rounded. The appearance of fluid around the lungs or heart is readily apparent and should be thoroughly imaged. Be sure to measure any fluid collections in three dimensions.

Fetal abdominal wall

The sonographic evidence of a thoracicoabdominal wall defect is the presence of fetal organs protruding outside of the body. This would seem to be an obvious appearance, easy to detect sonographically, but this may not always be the case. Particularly in cases of low amniotic fluid, you may be confused by what you see.

Omphalocele

The sonographic appearance of an omphalocele will typically show you a cystic mass in the base of the fetal umbilical cord insertion. The contents are complex with bowel or liver protruding. The entire mass is covered with a thin membrane made of peritoneum and amnion. The cord itself inserts at the top or apex of small omphaloceles, while the cord may insert on the sides of larger omphaloceles. Be sure to image all of these qualities, in separate images if necessary, to strengthen your suspicion of an omphalocele. Measure the mass and use color Doppler to trace the path of the umbilical vessels as they enter the fetus. You may notice the presence of polyhydramnios, which you should confirm with an AFI.

Gastroschisis

Exposed to the amniotic fluid, the fibrous coating that develops on the outer walls of floating bowel loops displays a thickness readily apparent on sonography. With gastroschisis, referred because of sharp increases of MSAFP, locating the cord insert is critical, preferably with color Doppler. The insertion site should be clear of other body parts. If a perforation of one of the loops occurs, usually after massive dilation from compressed segments, meconium can spill out into the amniotic fluid. You should then notice echogenic debris floating freely within the amnion. Some radiologists may

ask that you measure the entire mass of bowel or even the thickness of the bowel walls. Images of the insertion site with herniation in the axial view of the fetus should be accompanied with a sagittal view of the herniation site.

Other wall defects

Both pentalogy of Cantrell and the LBWC are extremely difficult to document with sonography. A helpful thing to remember is to give the radiologist landmarks. For instance, document the pentalogy of Cantrell by showing the ectopia cordis in relation to the fetal spine on an axial view. For the spinal scoliosis of the LBWC, you might include coronal, sagittal and axial segments of the spine. This approach will also help you understand the other abnormalities of these complex disorders. Fetal GI system

The gastrointestinal tube inside the fetus is filled with amniotic fluid, but not so much that the anechoic fluid can be seen, the two major exceptions being the fluid visible in the stomach and the GI tube dilated from obstruction.

Esophageal atresia

Fetuses with distal tracheoesophageal fistulas typically have extremely small stomachs; however, the stomach can still be seen with sonography, making this type of esophageal atresia exceptionally difficult to detect. The normal fetal stomach usually fills to a point where the anechoic appearance in the abdomen is sharply defined. Thus, in the presence of a small fetal stomach, keep an eye on the stomach over a period of at least 45 minutes as you scan the rest of the fetus. If the stomach remains smaller than usual and faint, suspect an esophageal atresia with a distal tracheoesophageal fistula.

Duodenal atresia

The dilation of the proximal duodenum and stomach are seen on sonography as two connected anechoic circles in midabdomen on abdominal circumference views (fig. 3–3). This classic “double bubble” sign occurs largely in the late second and early third trimesters.

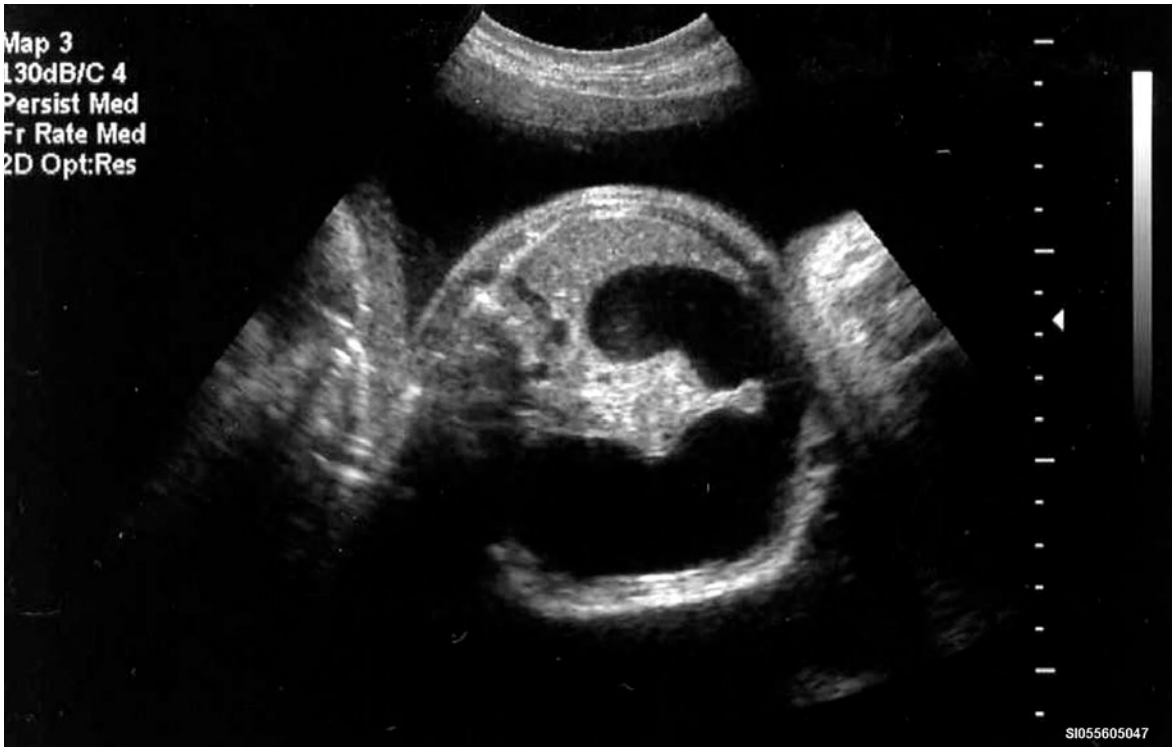

Figure 3–3. Duodenal atresia.

Fetal urinary system

The sonographic approach to the fetal urinary system requires carefulness but need not be overly painstaking. The kidneys generally appear as isoechoic to hypoechoic oval structures to either side of the spine. Renal fat represented by an echogenic border and the echogenic fat of the renal sinus does not generally appear until the late second and third trimesters. The bladder is seen sporadically by the end of the first trimester and consistently thereafter to term. Normal bladder empties and fills every 25 to 45 minutes.

Hydronephrosis

On sonography, dilation of the kidney beyond the renal pelvis is obviously categorized as hydronephrosis. The difficulty is in determining obstruction from just the pelvis itself. To obtain measurements of the renal pelvis, ensure that the transverse view of the fetus demonstrates the kidneys up closer to the transducer. Sonographic criteria for determining a hydronephrotic kidney are abundant and controversial. Most departments follow a protocol that hovers between a range of >4 mm or >5 mm in AP diameter in the second trimester as indicative of hydronephrosis. In the third trimester, pelvis diameters of >7 mm or >10 mm are suggestive of hydronephrosis.

UVJ stenosis is normally not visualized directly on sonography. Indirect evidence is a normal bladder but dilated and tortuous ureters (megaureters). This condition can also be caused by reflux of urine back into the ureters and thus is nonspecific for UVJ stenosis.

The outpouching distal to the PUV bladder represents a dilated proximal urethra, the “keyhole” sign.

Renal agenesis

On sonography, you may have difficulty discerning the fetal anatomy because of the lack of amniotic fluid. Classic sonographic criteria for bilateral renal agenesis are absent kidneys and bladder with severe oligohydramnios. You should attempt a search for other abnormalities common to this lethal disorder, such as skeletal, cardiac, GI, and central nervous system abnormalities.

When sonographically examining the fetus with unilateral renal agenesis, you may notice nothing abnormal aside from the presence of one kidney. However, closer inspection of the single kidney will reveal, particularly if you are required to measure the renal kidney lengths, the large size of the kidney. In place of a kidney, you may see the adrenal gland, close to the spine as two echogenic parallel lines, the “lying down” sign. The adrenal could be large enough to appear oval and may mimic a kidney shape.

Polycystic renal disease

Sonographic appearance of ARPKD is usually unimpressive until the twenty-fourth week. The kidneys will be massively enlarged and echogenic. Despite the enormous sizes, you should be able to detect the smooth borders of the kidneys. The disease is normally bilateral with significant oligohydramnios. Usually you will have extreme difficulty identifying the fetal bladder.

Multicystic dysplastic kidney

On sonography, you should see an enlarged kidney with multiple anechoic cysts of variable sizes in cases of multicystic dysplastic kidney. Measure the largest cyst. In between the cysts, you may see echogenic to hyperechoic tissue, which represents stroma or connective tissue and not normal renal parenchyma. The disease may not manifest until the middle of the second trimester. Document the presence or absence of the fetal bladder and amniotic fluid, positive signs that the contralateral kidney is functioning normally. The inevitable outcome of the multicystic dysplastic kidney is shrinking followed by disappearance (involution). This may occur just before term or right after birth.

Skeletal abnormalities

Although routine sonographic imaging of the skeletal structures is normally limited to what is seen of the bones with required images, specifically FL measurements, your anatomic surveys may

incidentally reveal certain patterns in the long bones, chest, head, and spine that indicate the presence of a general skeletal dysplasia.

Sonographic Patterns Indicative of Skeletal Dysplasia	
Area	***Sonographic Dysplastic Pattern***
Long bones	Extremity shortening: • Rhizomelia: femur and humerus • Mesomelia: radius/ulna and tibula/fibula • Acromelia: hands and feet • Micromelia: entire extremity (mild, bowed, and severe) Bowing, fractures, and decreased brightness (demineralization)
Chest	Four-chamber heart seems to take up most of the chest space (narrow circumference) Bell shape thorax on coronal view Demineralization
Head	Abnormal skull shapes (frontal bossing, cloverleaf shape, lemon shape) Demineralization Facial abnormalities (cleft lip/cleft palate, abnormal eye spacing)
Spine	Abnormal curvature Demineralization

If you encounter any of the general sonographic skeletal patterns, intensive scrutiny is required. A coexisting abnormality may be able to narrow down the possibility of a specific skeletal dysplasia.

During a routine scan, you may also incidentally discover a hand and foot deformity. Some of the more common ones are listed below:

- Polydactyly: more than 5 digits; usually ulnar side; tiny.
- Syndactyly: digits or skin fused; 2nd and 3rd aligned and 4th and 5th aligned.
- Clinodactyly: hypoplastic middle phalanx; causes sharp inversion of digit.
- Ectrodactyly: missing digits and large separation between (lobster-claw).
- Persistent clenched hand: with overlapping finger is usually associated with trisomy 18.
- Congenital talipes equinovarus: fixed lateral or medial inversion of a flexed foot (club foot).
- Rocker bottom foot: prominent heel.

Ultrasound guided amniocentesis

Some institutions use ultrasound to assist with obtaining genetic clues to potential fetal abnormalities. Amniocentesis is an invasive procedure that introduces a needle into the amniotic cavity through the abdominal wall. Fluid is extracted, fetal cells are analyzed, and a karyotype (or chromosomal characteristics) is determined. Other factors are determined such as maternal/fetal blood group compatibility, fetal lung maturity, and alpha-fetoprotein (α-fetoprotein) levels. The procedure is normally accomplished around 15 to 17 weeks. Ultrasound guidance is transabdominal while the procedure is performed. Primary reason for amniocentesis is late maternal age of 35 or more. Other reasons are previous offspring with genetic abnormalities or parental genetic disorders.

In the first trimester, patients may opt for biopsy of the chorion frondosum, located meshed with endometrial basal layer. Some patients undergo chorionic villus sampling (CVS) of the placenta in the second trimester at about 14 weeks or beyond. Also guided with ultrasound, CVS is an option for patients who want an earlier genetic report than received from amniocentesis. Also the CVS needle and catheter never break the amnionic membranes, and mothers need not worry about the fetus in the needle's path. Instead CVS is performed endovaginally or transabdominally through the cervix or myometrium only.

Common genetic disorders

Some of the more common chromosomal disorders display general characteristics detectable through sonography. You should understand that there is considerable overlap in the features of genetic disorders with nongenetic abnormalities. For instance, not all heart defects seen are a sign of trisomy 13; cardiac abnormalities are also seen in trisomy 21. Nevertheless, we will briefly look at a few of the more outstanding sonographic features of each.

Trisomy 21

A sonographic indicator is the presence of a thickened fold of skin at the back of the fetal head called the nuchal fold. You will know if you are in the correct plane for imaging this fold if you have the cerebellum and cisterna magna displayed as you would for standard imaging. Nuchal fold thickness greater than 5 mm is a strong indicator of Down syndrome. Other sonographic signs include brachycephaly, heart defects, duodenal atresias, brightly echogenic bowel, rhizomelia, and clinodactyly.

Trisomy 18

In trisomy 18, the head will have a shape resembling a strawberry on sonography. A string of other sonographic abnormalities occur with trisomy 18 such as choroid plexus cyst, enlarged cisterna magna, micrognathia, heart defects, diaphragmatic hernia, esophageal atresia, bowel-only omphalocele, single umbilical artery, renal defects, myelomeningocele, IUGR, overlapping fingers, general limb shortening, club foot and rocker bottom foot. Each one of the abnormalities should be imaged, preferably with magnified views for clarity.

Trisomy 13

Sonography for trisomy 13 typically reveals cleft lip or palate, midline ventricular and facial deformity, micrognathia, polydactyly, heart defects, fetal convulsions, renal abnormalities, umbilical herniation, and malrotation of the intestines.

Turner syndrome

45,X is the type of Turner syndrome you are likely to see on sonography. Turner syndrome will demonstrate a massive cystic hygroma, a sac of anechoic lymphatic fluid at the back of the fetal neck containing septations that give it a webbed appearance. Measure the hygroma width in the fetal axial view of the posterior fossa of the head.

Gestational trophoblastic disease

While *complete hydatidiform* moles usually are seen in the first trimester, the *partial hydatidiform mole* is typically noticed on sonography at 18 to 22 weeks. Although complete moles can extend into the first trimester, usually they are discovered and removed before this is the case. The partial mole is seen as an enlarged placenta at least 4 cm thick with numerous cystic spaces. The main sonographic feature that distinguishes the partial from the complete mole is the presence of a fetus.Remember that complete hydatidiform pregnancies do not have any fetal or placental tissue.

On sonography, partial molar pregnancies can easily be mistaken for a normal pregnancy and overlooked if you do not notice placental thickness while you scan. However, the fetus will usually have some sort of sonographic evidence of developmental abnormality. You may note that the fetus presents sonographic evidence of IUGR. Also, your scanning many reveal evidence of oligohydramnios. If you demonstrate the sonographic signs of IUGR coupled with an AP measurement of an abnormally thickened placenta, then you will greatly assist the radiologist in making a determination of partial molar pregnancy.

A small percentage of either complete or partial molar pregnancies will develop into invasive moles, which can be difficult for you to determine on sonography. You usually will see islands of echogenicity within the myometrium. Rarely, invasive moles extend through to uterine surfaces

(percreta), which you may be able to see. You may also notice cystic spaces within the islands of echogenicity, which represents blood from myometrial hemorrhage.

An even smaller percentage of molar pregnancies will transform into malignant choriocarcinomas, which you will normally see located in the placenta, where it can hemorrhage and become necrotic. Thus, its appearance is echogenic and solid, but with bleeding or necrosis cystic elements may appear. Color Doppler is also helpful to distinguish this mass from a placental collection of venous blood (subchorionic hematoma). As a malignant neoplasm, the choriocarcinoma should have one or more blood vessels that feed arterial blood to it. This situation should be readily apparent to you as you scan, given the normal sonographic appearance of surrounding placental tissue.

As with all such abnormalities, measure all manifestations of gestational trophoblastic disease in three dimensions. Also, investigate the adnexal space for sonographic signs of multicystic ovaries (theca-lutein cysts), which can appear in a significant number of molar pregnancies. Be sure to check the laboratory values of β-hCG.

Self-Test Questions

After you complete these questions, you may check your answers at the end of the unit.

414. Second and third trimester sonographic considerations

1. With what view of the fetal face is a cleft lip normally seen?

2. What abdominal structures are to be documented with axial images?

415. Assessment of menstrual age and fetal growth

1. What is the best method for establishing the MA in the second trimester?

2. What is the normal range for the cephalic index?

3. Describe how to perform the AFI and the normal range of values obtained.

416. General obstetric abnormalities

1. Which fetal weight values are considered growth restricted?

2. What is a primary cause of polyhydramnios?

3. Mothers, who acquire hypertension during pregnancy, particularly after 20 weeks, are said to have which abnormality?

417. Common obstetric abnormalities

1. What problems increase the risk for anencephaly?

2. Describe holoprosencephaly.

3. CDH occurs mostly on which side of the fetal body?

418. Genetic obstetric abnormalities

1. What is the most common of fetal chromosomal abnormalities?

2. What malignant tumor follows half of molar pregnancies?

419. Imaging of second and third trimester pregnancies

1. A nuchal fold thickness greater than 5 mm is a strong indicator of which fetal chromosomal abnormality?

2. What is the sonographic description of an omphalocele?

Answers to Self-Test Questions

412

1. To provide the fetal blood with maternal oxygen and nutrients and to take away fetal waste and carbon dioxide; to manufacture a number of hormones, the most important being β-hCG early in the second trimester, with estrogen and progesterone production in the placenta increasing between the 20^{th} and 24^{th} week; starting at the 20^{th} week, the placenta takes over the production of progesterone and estrogen, as a way to maintain itself; to store nutrients such as carbohydrates, protein, calcium, and iron, which are released into the fetal circulation at certain rates; forms a protective barrier between the fetal environment and the maternal environment.
2. (1) Serve as a cushion for the fetus, preventing shock.
   (2) To prevent adhesions.
   (3) To provide enough space that permits the fetus to grow symmetrically and allow movement, which further develops muscle tone.
   (4) To maintain amniotic temperature favorable to the fetus.
   (5) Serve as an aid to the development of the lungs and gastrointestinal system.

413

1. Lateral ventricle.

2. Courses within the abdomen anteriorly before diving into the liver toward the spine, where it meets the umbilical portion of the portal vein and the ductus venosus. Most of the blood from the placenta enters the ductus, which is connected to the inferior vena cava.

414

1. Coronal.
2. Stomach, kidneys, bladder, and the insertion site of the umbilical cord into the abdomen.

415

1. Biparietal diameter (BPD) measurement of the head, usually before 20 weeks.
2. 70-86.
3. Divide the uterus into four imaginary quadrants and measure the deepest vertical pocket in each quadrant. The resulting measurements are added together and the sum is compared to a chart based on MA. The range of normal is usually between 7 cm and 20 cm.

416

1. Values that fall below the 10^{th} percentile for a given menstrual age.
2. Diabetes.
3. Preeclampsia.

417

1. Maternal diabetes mellitus and exposure to certain chemicals.
2. Failure of the cerebral hemisphere development. Of the three types (lobar, semilobar, and alobar) that signify the level of hemisphere separation and development only alobar is the most severe. The alobar variety demonstrates on sonography as a single ventricle and brain tissue surrounding it, with thalmai fusion. The falx is completely absent. Facial abnormalities are common with this abnormality.
3. Left.

418

1. Trisomy 21, Down syndrome.
2. Choriocarcinoma.

419

1. Trisomy 21, Down syndrome.
2. A cystic mass in the base of the fetal umbilical cord insertion is seen. The contents are complex with bowel or liver protruding. The entire mass is covered with a thin membrane made of peritoneum and amnion. The cord itself inserts at the top or apex of small omphaloceles, while the cord may insert on the sides of larger omphaloceles. You may notice the presence of polyhydramnios.

Complete the Unit Review Exercise.

Unit Review Exercises

Note to Student: Consider all choices carefully, select the *best* answer to each question, and *circle* the corresponding letter. When you have completed all unit review exercises, transfer your answers to ECI (AFIADL) Form 34, Field Scoring Answer Sheet.

Do not return your answer sheet to AFIADL.

32. (412) The main function of the placenta is to provide the
    a. fetus with oxygen and nutrients.
    b. mother with oxygen and nutrients.
    c. fetus with fetal blood cell antibodies.
    d. mother with a means to form fetal blood.

33. (412) By week 16, the *major* source of amniotic fluid production is from the fetal
    a. lungs.
    b. bowel.
    c. kidneys.
    d. stomach.

34. (413) The function of the choroid found in the fetal lateral ventricles of the brain is to secrete what kind of fluid?
    a. cerebral spinal.
    b. endometrial.
    c. lymphatic.
    d. amniotic.

35. (413) What fetal structure plays a *prominent* role in the process of moving fluid into and out of the lungs?
    a. Diaphragm.
    b. Kidneys.
    c. Bowel.
    d. Heart.

36. (413) By the beginning of the second trimester, where is the final location of the fetal kidneys in relation to the spine?
    a. In the pelvis and away from the spine.
    b. To either side of the spine.
    c. To one side of the spine.
    d. Anterior to the spine.

37. (414) In the second and third trimesters, what type of transducer should you mostly use?
    a. Endovaginal.
    b. Endocervical.
    c. Transluminal.
    d. Transabdominal.

38. (414) For standard sonography of the second trimester, to which structure would you document the relation of the placental location?
    a. Internal os of the cervix.
    b. External os of the cervix.
    c. Cord insert.
    d. Fetus.

39. (414) What image should you obtain to document the fetal thoracic cavity?
    a. The abdominal circumference (AC).
    b. The crown-rump length (CRL).
    c. Four-chamber heart.
    d. The thoracic spine.

40. (414) To indirectly indicate the presence of a 3-vessel umbilical cord you would use color Doppler over the
    a. bladder.
    b. insert.
    c. cord.
    d. fetus.

41. (415) The level that contains the umbilical portion of the left portal vein and the stomach is a landmark for which fetal measurement?
    a. Abdominal circumference (AC).
    b. Biparietal diameter (BPD).
    c. Head circumference (HC).
    d. Femur length (FL).

42. (415) What type of intrauterine growth restriction (IUGR) describes an abnormally small abdominal circumference (AC) while the head size remains at appropriate size for menstrual age (MA)?
    a. Symmetric.
    b. Asymmetric.
    c. Large for gestational age.
    d. Small for gestational age.

43. (416) The condition for an abnormally large sized fetal body is called
    a. microsomia.
    b. macrosomia.
    c. diabetes mellitus.
    d. polyhdramnios.

44. (417) What is the *most* common neural tube defect abnormality?
    a. Acrania.
    b. Anencephaly
    c. Microcephaly.
    d. Encephalocele.

45. (417) What is the difference between spina bifida occulta and spina bifida aperta?
    a. Occulta is covered spinal cord; aperta cord is exposed.
    b. Occulta is exposed spinal cord; aperta cord is exposed.
    c. Occulta is covered spinal cord; aperta cord is covered.
    d. Occulta is exposed spinal cord; aperta cord is exposed

46. (417) What fetal obstruction is the *most* common cause of hydronephrosis?
    a. Ureteropelvic junction (UPJ).
    b. Cloacal malformation.
    c. Posterior urethral valves (PUV).
    d. Ureterovesical junction stenosis.

47. (417) What fetal structure would you document to prove normal functioning of a contralateral kidney in multicystic dysplastic kidney?
    a. Bowel.
    b. Bladder.
    c. Stomach.
    d. Esophagus.

48. (418) The *most* common fetal chromosomal abnormality is trisomy
    a. 13.
    b. 18.
    c. 21.
    d. 45.

49. (418) The condition that describes an invasive mole that penetrates across the myometrium is the placenta
    a. percreta.
    b. previa.
    c. acreta.
    d. occulta.

50. (419) What is suggested by the fetus being completely surrounded with amniotic fluid and having an abnormally high estimated fetal weight (EFW)?
    a. Fetal hydrops.
    b. Polyhydramnios with marcrosomia.
    c. Oligohydramnios with marcrosomia.
    d. Polyhydramnios with non-immune hydrops.

51. (419) How should you determine the presence of a shared placenta or two placentas in second and third trimester multiple gestation cases?
    a. Look for the twin peak sign.
    b. Count the gestational sacs.
    c. Look for the yolk sacs.
    d. Count the yolk sacs.

52. (419) What sonographic view would you use to demonstrate the shift of the fetal heart and mediastinum in a case of congenital diaphragmatic hernia?
    a. Four-chamber heart view.
    b. Coronal view of the spine.
    c. Midsagittal chest view.
    d. Sagittal chest view.

53. (419) *Normally* amniocentesis is occurs during what range of weeks?
    a. 6–10.
    b. 15–17.
    c. 22–30.
    d. 37–40.

54. (419) What is sonographically seen with a partial mole but *not* with a complete mole during pregnancy?
    a. Percreta.
    b. Previa.
    c. Fetus.
    d. Cord.

Glossary

Terms

amniocentesis – A procedure that allows physicians to puncture the amniotic sac and extract fluid with a needle, retrieving the fetal cells for genetic analysis.

anastomosing – Connecting.

anteflexion – The uterus is bent forward at the isthmus with the cervix.

anteversion – The tilting of the entire uterus and cervix forward.

atrophy – Degenerate.

congenital – Appearing at birth.

dextroversion – The body of uterus and cervix deviate to the right in relation to the midline orientation of the vaginal tube.

dizygotic twins – Fraternal twins.

dysmenorrhea – Painful menstruation.

ectoderm – Nervous tissue and skin.

edema – General swelling.

endoderm – The epithelial lining of the gastrointestinal system, glands, and lungs.

endometrium – The innermost layer of the uterus. It is a mucous membrane lining.

follicular cyst – Mature or dominant follicles that fail to ovulate.

ischemia – Localized tissue anemia due to obstruction of the inflow of blood.

lateroversion – The tilting of the entire uterus and cervix to the side of midline.

levoversion – The body of uterus and cervix deviate to the left in relation to the midline orientation of the vaginal tube.

nulliparious – A woman who has never been pregnant.

mennorrhagia – Heavy menses.

mesoderm – Connective tissue.

monozygotic – Identical twins.

myometrium – A layer of the uterus beneath the serosa. It is made of three layers of smooth muscle and connective tissue. Overall the myometrium is thickest at the fundus and thinnest at the level of the cervix.

oocytes – Sex cells released during ovulation.

ovulation – The discharge of ovum or eggs.

parous – A woman who has had one or more pregnancies.

pelvic inflammatory disease – A spectrum of consecutively or concurrently inflamed endometrium, uterine tubes, ovary, and pelvic peritoneum caused mostly from sexually transmitted bacterial infection

postmenopausal – The period after menses has stopped, usually around 50 years of age.

preeclampsia – It is pregnancy-induced hypertension accompanied by protein in the maternal urine (proteinuria) and general swelling (edema). If the hypertensive state is not treated, preeclampsia increases in severity and results in outright eclampsia (one or more convulsions that can cause coma or death).

premenopausal – The period when menses occur cyclically at regular four-week intervals. This period is often referred to as the reproductive period.

prepubertal – Before puberty (can occur anywhere from 8 to 16 years of age).

retroflexion – The uterus is bent backward at the isthmus with the cervix.

retroversion – The tilting of the entire uterus and cervix backward away from the vaginal tube and toward the posterior pelvis and rectum.

serosa – The outermost coat or layer of the uterus, made of peritoneum and fibrous connective tissue.

stroma – Dense connective tissue.

torsion – Twisting of the ovarian pedicle.

Abbreviations and Acronyms

AC	abdominal circumference
ADPKD	autosomal-dominant polycystic kidney disease
AFI	amniotic fluid index
AFP	alpha fetoprotein
AP	anteroposterior
ARPKD	autosomal-recessive polycystic kidney disease
ART	assisted reproductive technology
β-hCG	beta-human chorionic gonadotropin
BPD	biparietal diameter
bpm	beats per minute
CCAM	congenital cystic adenomatoid malformation
CDH	congenital diaphragmatic hernia
CHM	complete hydatidiform mole
CI	cephalic index
cm	centimeters
CNS	central nervous system
CRL	crown rump length
CSF	cerebral spinal fluid
CT	computed tomography
CVS	chronic villus sampling
EFW	estimated fetal weight
FIRP	First International Reference Preparation

FL	femur length
FOD	fronto-occipital diameter
FSH	follicle-stimulating hormone
GI	gastrointestinal
GIFT	gamete intrafallopian transfer
GnRH	gonadotropin-releasing hormone
HC	head circumference
hCG	human chorionic gonadotropins
HIV	human immunodeficiency virus
HRT	hormone replacement therapy or hormone regimen therapy
IUCD	intrauterine contraceptive device
IUD	intrauterine device
IUGR	intrauterine growth restriction
IUP	intrauterine pregnancy
IVC	inferior vena cava
IVF	in vitro fertilization
LBWC	limb-body wall complex
LGA	large for gestational age
LH	luteinizing hormone
LMP	last menstrual period
LNMP	last *normal* menstrual period
MA	menstrual age
MCDK	multicystic dysplastic kidney
MHz	megahertz
MI	mechanical index
MRI	magnetic resonance imaging
MSAFP	alpha-fetoprotein in the maternal serum
MSD	mean sac diameter
NT	nuchal translucency
OB	obstetrical
OB/GYN	obstetrics/gynecology
PCOS	polycystic ovarian syndrome
PID	pelvic inflammatory disease
PROM	premature rupture of the membranes
PUV	posterior urethral valves
RI	Resistive Index

SGA	small for gestational age
TI	thermal index
TIS	Third International Standard
TOA	tubo-ovarian abscess
UPJ	ureteropelvic junction
UVJ	ureterovesical junction
ZIFT	zygote intrafallopian transfer

Student Notes

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