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Obstetric and gynecologic disorders

Introduction

Medical care of the obstetric or gynecologic patient reflects a growing interest in improving the quality of health care for women. Skills are needed to assess, counsel, teach, and refer patients, while weighing such relevant factors as the patient's desire to have children, sexual adjustment problems, and self-image. Often, this care is complicated by multiple obstetric and gynecologic abnormalities occurring simultaneously. For example, a patient with dysmenorrhea may also have trichomonal vaginitis, dysuria, and unsuspected infertility. Her condition may be further complicated by associated urologic disorders, due to the proximity of the urinary and reproductive systems. This tendency for multiple and complex disorders is readily understandable upon review of the female genitalia's anatomic structure. (See *External and internal female genitalia.*)

External structures

Female genitalia include the following external structures, collectively known as the *vulva:* mons pubis (or mons veneris), labia majora, labia minora, clitoris, and the vestibule. The perineum is the external region between the vulva and the anus. The size, shape, and color of these structures—as well as pubic hair distribution and skin texture and pigmentation—vary greatly among individuals. Furthermore, these external structures undergo distinct changes during the life cycle.

The mons pubis is the pad of fat over the symphysis pubis (pubic bone), which is usually covered by the base of the inverted triangular patch of

pubic hair that grows over the vulva after puberty.

The labia majora are the two thick, longitudinal folds of fatty tissue that extend from the mons pubis to the posterior aspect of the perineum. The labia majora protect the perineum and contain large sebaceous glands that help maintain lubrication. Virtually absent in the young child, their development is a characteristic sign of puberty's onset. The skin of the more prominent parts of the labia majora is pigmented and darkens after puberty.

The labia minora are the two thin, longitudinal folds of skin that border the vestibule. Firmer than the labia majora, they extend from the clitoris to the fourchette.

The clitoris is the small, protuberant organ located just beneath the arch of the mons pubis. The clitoris contains erectile tissue, venous cavernous spaces, and specialized sensory corpuscles that are stimulated during coitus.

The vestibule is the oval space bordered by the clitoris, labia minora, and fourchette. The urethral meatus is located in the anterior portion of the vestibule; the vaginal meatus, in the posterior portion. The hymen is the elastic membrane that partially obstructs the vaginal meatus in virgins.

Several glands lubricate the vestibule. Skene's glands open on both sides of the urethral meatus; Bartholin's glands, on both sides of the vaginal meatus.

The fourchette is the posterior junction of the labia majora and labia minora. The perineum, which includes the underlying muscles and fascia, is the external surface of the floor of the pelvis, extending from the fourchette to the anus.

Internal structures

The following internal structures are included in the female genitalia: vagina, cervix, uterus, fallopian tubes (or oviducts), and ovaries.

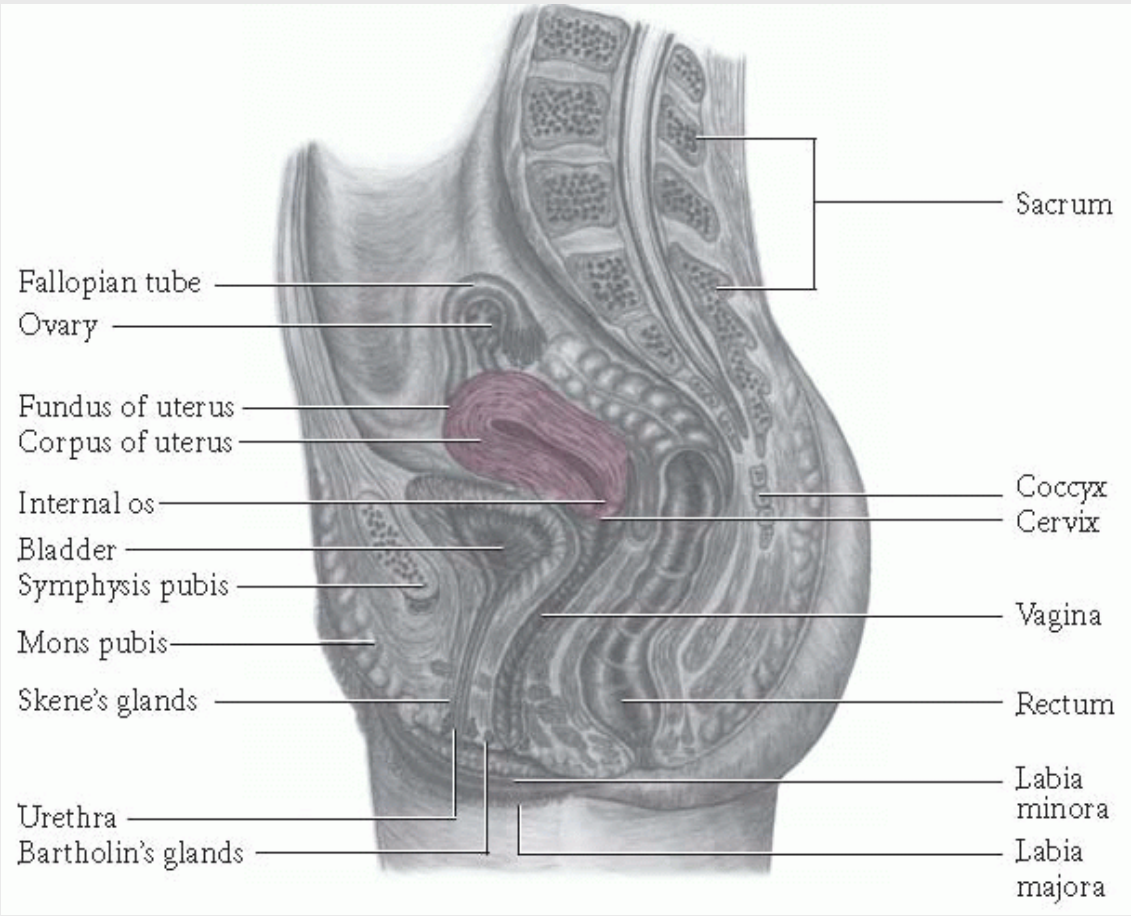
The vagina occupies the space between the bladder and the rectum. A muscular, membranous tube that's about 3″ (7.5 cm) long, the vagina connects the uterus and the vestibule of the external genitalia. It serves

as a passageway for sperm to the fallopian tubes, for the discharge of menstrual fluid, and for childbirth.

The cervix, or neck of the uterus, protrudes at least ¾" (2 cm) into the proximal end of the vagina. A rounded, conical structure, the cervix joins the uterus and the vagina at a 45- to 90-degree angle.

The uterus is the hollow, pear-shaped organ in which the conceptus grows during pregnancy. The part of the uterus above the junction of the fallopian tubes is called the *fundus;* the part below this junction is called the *corpus.* The junction of the corpus and cervix forms the lower uterine segment.

EXTERNAL AND INTERNAL FEMALE GENITALIA



The thick uterine wall consists of mucosal, muscular, and serous layers. The inner mucosal lining—the endometrium—undergoes cyclic changes to

facilitate and maintain pregnancy.

The smooth muscular middle layer—the myometrium—interlaces the uterine and ovarian arteries and veins that circulate blood through the uterus. During pregnancy, this vascular system expands dramatically. After abortion or childbirth, the myometrium contracts to constrict the vasculature and control blood loss.

The outer serous layer—the parietal peritoneum—covers all of the fundus, part of the corpus, but none of the cervix. This incompleteness allows surgical entry into the uterus without incision of the peritoneum, thereby reducing the risk of peritonitis.

The fallopian tubes extend from the sides of the fundus and terminate near the ovaries. Through ciliary and muscular action, these small tubes (3¼″ to 5½″ [8 to 14 cm] long) carry ova from the ovaries to the uterus and facilitate the movement of sperm from the uterus toward the ovaries. Fertilization of the ovum normally occurs in a fallopian tube. The same ciliary and muscular action helps move a zygote (fertilized ovum) down to the uterus, where it implants in the blood-rich inner uterine lining, the endometrium.

The ovaries are two almond-shaped organs, one on either side of the fundus, that are situated behind and below the fallopian tubes. The ovaries produce ova and two primary hormones—estrogen and progesterone—in addition to small amounts of androgen. These hormones, in turn, produce and maintain secondary sex characteristics, prepare the uterus for pregnancy, and stimulate mammary gland development.

The ovaries are connected to the uterus by the utero-ovarian ligament and are divided into two parts: the cortex, which contains primordial and graafian follicles

in various stages of development, and the medulla, which consists primarily of vasculature and loose connective tissue.

A normal female is born with at least 400,000 primordial follicles in her ovaries. At puberty, these ova precursors become graafian follicles, in response to the effects of pituitary gonadotropic hormones—follicle-stimulating hormone (FSH) and luteinizing hormone (LH). In the life

cycle of a female, however, less than 500 ova eventually mature and develop the potential for fertilization.

The menstrual cycle

Maturation of the hypothalamus and the resultant increase in hormone levels initiate puberty. In the young girl, breast development—the first sign of puberty—is followed by the appearance of pubic and axillary hair and the characteristic adolescent growth spurt. The reproductive system begins to undergo a series of hormone-induced changes that result in menarche, onset of menstruation (or menses). In North American females, menarche usually occurs at about age 13 but may occur between ages 9 and 18. Menstrual periods initially are irregular and anovulatory, but after a year or so, they become more regular. (See *Menstrual cycle*.)

The menstrual cycle consists of three different phases: menstrual, proliferative (estrogen-dominated), and secretory (progesterone-dominated). These phases correspond to the phases of ovarian function. The menstrual and proliferative phases correspond to the follicular ovarian phase; the secretory phase corresponds to the luteal ovarian phase.

The *menstrual phase* begins with day 1 of menstruation. During this phase, low estrogen and progesterone levels stimulate the hypothalamus to secrete gonadotropin-releasing hormone (Gn-RH). This substance, in turn, stimulates pituitary secretion of FSH and LH. When the FSH level rises, LH output increases.

The *proliferative (follicular) phase* lasts from cycle day 6 to day 14. During this phase, LH and FSH act on the ovarian follicle, causing estrogen secretion, which in turn stimulates the buildup of the endometrium. Late in this phase, estrogen levels peak, FSH secretion declines, and LH secretion increases, surging at midcycle (around day 14). Then estrogen production decreases, the follicle matures, and ovulation occurs.

During the *secretory phase*, FSH and LH levels drop. Estrogen levels decline initially, then increase along with progesterone levels as the corpus luteum begins functioning. During this phase, the endometrium responds to progesterone stimulation by becoming thick and secretory in

preparation for implantation of a fertilized ovum. About 10 to 12 days after ovulation, the corpus luteum begins to diminish, as do estrogen and progesterone levels, until hormone levels are insufficient to sustain the endometrium in a fully developed secretory state. Then the endometrial lining is shed (menses). Subsequently decreasing estrogen and progesterone levels stimulate the hypothalamus to produce Gn-RH, which in turn begins the cycle again.

In the nonpregnant female, LH controls the secretions of the corpus luteum; in the pregnant female, human chorionic gonadotropin (hCG) controls them. At the end of the secretory phase, the uterine lining is ready to receive and nourish a zygote. If fertilization doesn't occur, increasing estrogen and progesterone levels decrease LH and FSH production. Because LH is necessary to maintain the corpus luteum, a decrease in LH production causes the corpus luteum to atrophy and stop secreting estrogen and progesterone. The thickened uterine lining then begins to slough off, and menstruation begins again.

If fertilization and pregnancy do occur, the endometrium grows even thicker. After implantation of the zygote (about 5 or 6 days after fertilization), the endometrium becomes the decidua. Chorionic villi produce hCG soon after implantation, stimulating the corpus luteum to continue secreting estrogen and progesterone, which prevents further ovulation and menstruation.

HCG continues to stimulate the corpus luteum until the placenta—the vascular organ that develops to transport materials to and from the fetus—forms and starts producing its own estrogen and progesterone.

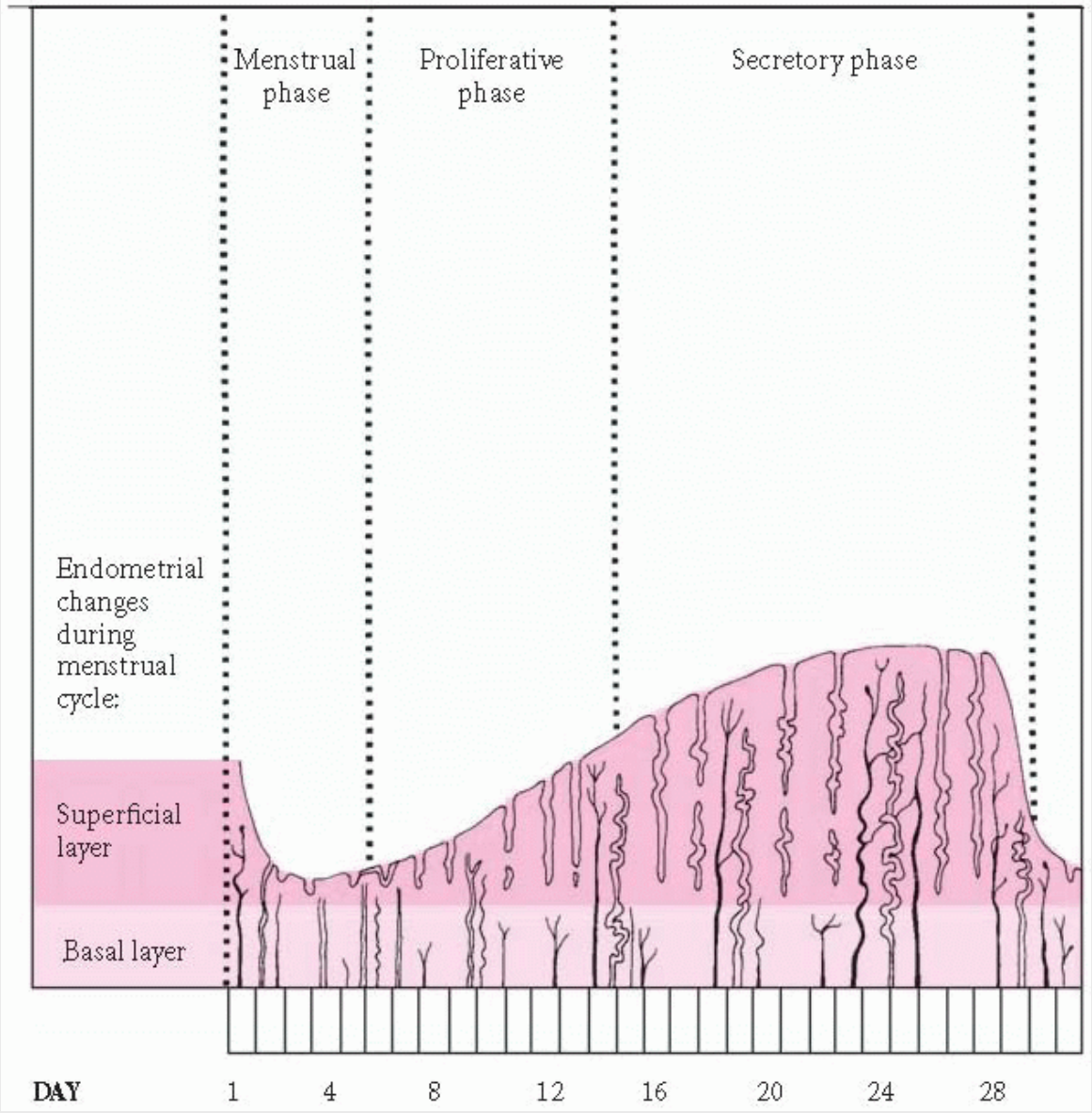
After the placenta takes over hormonal production, secretions of the corpus luteum are no longer needed to maintain the pregnancy, and the corpus luteum gradually decreases its function and begins to degenerate.

MENSTRUAL CYCLE

The menstrual cycle is divided into three distinct phases:

The *menstrual phase* starts on the first day of menstruation. The top layer of the endometrium (the material lining the uterus) breaks down and flows out of

the body. This flow, called the *menses,* consists of blood, mucus, and unneeded tissue.



During *the proliferative phase*, the endometrium begins to thicken, and the estrogen level in the blood rises.

In the *secretory phase*, the endometrium continues to thicken to nourish an embryo should fertilization occur. Without fertilization, the top layer of the endometrium breaks down, and the menstrual phase of the cycle begins again.

Pregnancy

Cell multiplication and differentiation begin in the zygote at the moment of conception. By about 17 days after conception, the placenta has established circulation to what is now an *embryo* (the term used for the

conceptus between the 2nd and 7th weeks of pregnancy). By the end of the embryonic stage, fetal structures are formed. Further development now consists primarily of growth and maturation of already formed structures. From this point until birth, the conceptus is called a *fetus*.

First trimester

Normal pregnancies last an average of 280 days. Although pregnancies vary in duration, they're conveniently divided into three trimesters.

During the first trimester, a female usually experiences physical changes, such as amenorrhea, urinary frequency, nausea and vomiting (more severe in the morning or when the stomach is empty), breast swelling and tenderness, fatigue, increased vaginal secretions, and constipation.

Within 7 to 10 days after conception, pregnancy tests, which detect hCG in the urine and serum, are usually positive. A pelvic examination at this stage can yield various findings, such as Hegar's sign (cervical and uterine softening), Chadwick's sign (a bluish coloration of the vagina and cervix resulting from increased venous circulation), and enlargement of the uterus. A pelvic examination will help estimate gestational age but vaginal sonography is more accurate.

The first trimester is a critical time during pregnancy. Rapid cell differentiation makes the developing embryo or fetus highly susceptible to the teratogenetic effects of viruses, alcohol, cigarettes, caffeine, and other drugs.

Second trimester

From the 13th to the 28th week of pregnancy, uterine and fetal size increase substantially, causing weight gain, a thickening waistline, abdominal enlargement and, possibly, reddish streaks as abdominal skin stretches (striation). In addition, pigment changes may cause skin alterations, such as linea nigra, melasma (mask of pregnancy), and a darkening of the areolae of the nipples.

Other physical changes may include diaphoresis, increased salivation, indigestion, continuing constipation, hemorrhoids, nosebleeds, and some dependent edema. The breasts become larger and heavier, and about 19 weeks after the last menstrual period, they may secrete colostrum. By about the 18th to the 20th week of pregnancy, the fetus is large enough for the mother to feel it move (quickening).

Third trimester

During this period, from 28th week to term, the mother feels Braxton Hicks contractions—sporadic episodes of painless uterine tightening—which help strengthen uterine muscles in preparation for labor. Increasing uterine size may displace pelvic and intestinal structures, causing indigestion, protrusion of the umbilicus, shortness of breath, and insomnia. The mother may experience backaches because she walks with a swaybacked posture to counteract her frontal weight. By lying down, she can help minimize the development of varicose veins, hemorrhoids, and ankle edema.

Labor and delivery

About 2 to 4 weeks before birth, lightening—the descent of the fetal head into the pelvis—shifts the uterine position. This relieves pressure on the diaphragm and enables the mother to breathe more easily.

Onset of labor characteristically produces low back pain and passage of a small amount of bloody “show.” A brownish or blood-tinged plug of cervical mucus may be passed up to 2 weeks before labor. As labor progresses, the cervix becomes soft, then effaces and dilates; the amniotic membranes may rupture spontaneously, causing a gush or leakage of amniotic fluid. Uterine contractions become increasingly regular, frequent, intense, and long.

Labor is usually divided into four stages:

- *Stage I,* the longest stage, lasts from onset of regular contractions until full cervical dilation (4″ [10 cm]). Average duration of this stage is about 12 hours for a primigravida and 6 hours for a multigravida.
- *Stage II* lasts from full cervical dilation until delivery of the infant—about 1 to 3 hours for a primigravida, 30 to 60 minutes for a multigravida.
- *Stage III,* the time between delivery and expulsion of the placenta, usually lasts several

  minutes (duration varies widely) but may last up to 30 minutes.
- *Stage IV* is a period of recovery during which homeostasis is re-established. This final stage lasts 1 to 4 hours after the placenta is expelled.

Sources of pathology

In no other body part do so many interrelated physiologic functions occur so close together as in the area of the female reproductive tract. Besides the internal genitalia, the female pelvis contains the organs of the urinary and the GI systems (bladder, ureters, urethra, sigmoid colon, and rectum). The reproductive tract and its surrounding area are thus the site of urination, defecation, menstruation, ovulation, copulation, impregnation, and parturition. It's easy to understand how an abnormality in one pelvic organ can readily induce abnormality in another.

When conducting a pelvic examination, therefore, you must consider all possible sources of pathology. Remember that some serious abnormalities of the pelvic organs can be asymptomatic. Remember, too, that some abnormal findings in the pelvic area may result from pathologic changes in other organ systems, such as the upper urinary and GI tracts, the endocrine glands, and the neuromusculoskeletal system. Pain symptoms are often associated with the menstrual cycle; therefore, in many common diseases of the female reproductive tract, such pain follows a cyclic pattern. A patient with pelvic inflammatory

disease, for example, may complain of increasing premenstrual pain that's relieved by onset of menstruation.

Pelvic examination

A pelvic examination and a thorough patient history are essential for any patient with symptoms related to the reproductive tract or adjacent body systems. Document any history of pregnancy, miscarriage, and abortion. Ask the patient if she has experienced any recent changes in her urinary habits or menstrual cycle. If she practices birth control, find out what method she uses and whether she has experienced any adverse effects.

Then prepare the patient for the pelvic examination as follows:

- Ask the patient if she has douched within the last 24 hours. Explain that douching washes away cells or organisms that the examination is designed to evaluate.
- Check weight and blood pressure.
- For the patient's comfort, instruct her to empty her bladder before the examination. Provide a urine specimen container if needed.
- To help the patient relax, which is essential for a thorough pelvic examination, explain what the examination entails and why it's necessary.
- If the patient is scheduled for a Papanicolaou (Pap) test, inform her that another smear may have to be taken later if there are abnormal findings with the first test. Reassure her that this is done to confirm the first test's results. If she has never had a Pap test before, tell her it may be uncomfortable.
- After the Pap test, a bimanual examination is performed to assess the size and location of the ovaries and uterus.
- After the examination, offer the patient premoistened tissues to clean the vulva.

Other diagnostic tests

Diagnostic measures for gynecologic disorders also include the following tests, which can be performed in the physician's office:

- wet smear to examine vaginal secretions for specific organisms, such as *Trichomonas vaginalis,* bacterial vaginosis, and *Candida albicans,* or to evaluate semen specimens collected in connection with rape or infertility cases
- endometrial biopsy to assess hormonal secretions of the corpus luteum, to determine whether normal ovulation is occurring, and to check for neoplasia
- dilatation and curettage with hysteroscopy to evaluate atypical bleeding and to detect carcinoma.

Laparoscopy, used to evaluate infertility, dysmenorrhea, and pelvic pain, and as a means of sterilization, is usually performed in a health care facility while the patient is under anesthesia. Increasingly, however, it's being performed as a less invasive procedure under conscious sedation in an office setting using microlaparoscopic technique.

GYNECOLOGIC DISORDERS

Premenstrual syndrome

Also designated “late luteal phase dysphoric disorder” (LLPD) in *the Diagnostic and Statistical Manual of Mental Disorders,* Fourth Edition, Text Revision, premenstrual syndrome (PMS) is characterized by varying symptoms that appear 7 to 14 days before menses and usually subside with its onset. The effects of PMS range from minimal discomfort to severe, disruptive symptoms and can include nervousness, irritability, depression, and multiple somatic complaints.

Causes and incidence

The list of biological theories offered to explain the cause of PMS is impressive. It includes such conditions as a progesterone deficiency in the menstrual cycle's luteal phase and vitamin deficiencies. Although there's no evidence that PMS is hormonally mediated, failure to identify

a specific disorder with a specific mechanism suggests that PMS represents a variety of manifestations triggered by normal physiologic hormonal changes. Researchers believe that 70% to 90% of women experience PMS at some time during their childbearing years, usually between ages 25 and 45.

Complications

- Depression
- Inability to function at home, work, or school

Signs and symptoms

Clinical effects vary widely among patients and may include any combination of the following:

- *behavioral*—mild to severe personality changes, nervousness, hostility, irritability, agitation, sleep disturbances, fatigue, lethargy, and depression
- *somatic*—breast tenderness or swelling, abdominal tenderness or bloating, joint pain, headache, edema, diarrhea or constipation, and exacerbations of skin problems (such as acne or rashes), respiratory problems (such as asthma), or neurologic problems (such as seizures).

PMS may need to be differentiated from premenstrual dysphoric disorder, which is a more severe form of PMS that's marked by severe depression, irritability, and tension before menstruation. (See *Premenstrual dysphoric disorder.*)

Diagnosis

The patient history shows typical symptoms related to the menstrual cycle. To help ensure an accurate history, the patient may be asked to record menstrual symptoms and body temperature on a calendar for 2 to 3 months prior to diagnosis. Estrogen and progesterone blood levels may be evaluated to help rule out hormonal imbalance. A psychological evaluation is also recommended to rule out or detect an underlying psychiatric disorder.

Treatment

Educating and reassuring patients that PMS is a real physiologic syndrome are important parts of treatment. Because treatment is predominantly symptomatic, each patient must learn to cope with her own individual set of symptoms. Treatment may include calcium and magnesium supplementation, vitamins (such as B complex), prostaglandin inhibitors, and nonsteroidal anti-inflammatory drugs. Diuretics may be prescribed for patients who experience significant weight gain due to fluid retention. Psychiatric medications and therapy may be prescribed for women who develop anxiety, irritability, or depression. Hormonal therapy may include a trial on hormonal contraceptives, which may either decrease or increase PMS symptoms. The use of progesterone vaginal suppositories during the second half of the menstrual cycle is still controversial.

For treatment to be effective, the patient may have to maintain a diet that's low in simple sugars, caffeine, and salt.

Special considerations

- Inform the patient that self-help groups exist for women with PMS; help her contact such a group if appropriate.
- Obtain a complete patient history to help identify any emotional problems that

  may contribute to PMS. Refer the patient for psychological counseling if necessary.

PREMENSTRUAL DYSPHORIC DISORDER

Premenstrual dysphoric disorder (PMDD) is a severe form of premenstrual syndrome (PMS) that has a cyclical occurrence of psychiatric symptoms that starts after ovulation (usually the week before the onset of menstruation) and ends within the first day or two of menses. Its underlying cause and pathophysiology remain unclear. However, researchers theorize that

normal cyclic changes in the body cause abnormal responses to neurotransmitters, such as serotonin, resulting in physical and behavioral signs and symptoms.

PMDD affects as many as 1 in 20 American women who have regular menstrual periods. It's unclear why some women are affected while others aren't.

How PMDD and PMS differ

PMDD is characterized by severe monthly mood swings and physical signs and symptoms that interfere with everyday life. Compared to PMS, its signs and symptoms are abnormal and unmanageable. Although depression, anxiety, and sadness are common with PMS, in PMDD, these symptoms are extreme. Some women may feel the urge to hurt or kill themselves or others.

The Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision, sets these criteria for diagnosing PMDD:

- functional impairment
- predominant mood symptoms, with one being affective
- symptoms beginning 1 week before the onset of menstruation
- symptoms that aren't due to any underlying primary mood disorder.

In addition, at least five of the following symptoms must be present:

- Appetite changes
- Decreased interests
- Difficulty concentrating
- Fatigue
- Feelings of being overwhelmed
- Insomnia or hypersomnia

- Irritability
- "Low mood"
- Mood swings
- Physical symptoms
- Tension

- Suggest that the patient seek further medical consultation if symptoms are severe and interfere with her normal lifestyle.

PREVENTION

- *If possible, discuss with your patient ways she can modify her lifestyle, such as making changes in her diet and avoiding stimulants and alcohol.*
- *Encourage your patient to get regular exercise and adequate rest.*

Dysmenorrhea

Dysmenorrhea—painful menstruation—is the most common gynecologic complaint and a leading cause of absenteeism from school (affecting 10% of high school girls each month) and work (causing about 140 million lost work hours annually). Dysmenorrhea can occur as a primary disorder or secondary to an underlying disease. Because primary dysmenorrhea is self-limiting, the prognosis is generally good. The prognosis for secondary dysmenorrhea depends on the underlying disorder.

Causes and incidence

Although primary dysmenorrhea has no known single cause, possible contributing factors include hormonal imbalances and psychogenic factors. The pain of dysmenorrhea probably results from increased prostaglandin secretion, which intensifies normal uterine contractions. (See *Causes of pelvic pain*, page 1160.) Dysmenorrhea may also be secondary to such gynecologic disorders as endometriosis, cervical stenosis, uterine leiomyomas, uterine malposition,

pelvic inflammatory disease, pelvic tumors, or adenomyosis.

CAUSES OF PELVIC PAIN

The characteristic pelvic pain of dysmenorrhea must be distinguished from the acute pain caused by many other disorders, such as:

- *GI disorders:* appendicitis, acute diverticulitis, acute or chronic cholecystitis, chronic cholelithiasis, acute pancreatitis, peptic ulcer perforation, intestinal obstruction
- *pregnancy disorders:* impending abortion (pain and bleeding early in pregnancy), ectopic pregnancy, abruptio placentae, uterine rupture, leiomyoma degeneration, toxemia
- *reproductive disorders:* acute salpingitis, chronic inflammation, degenerating fibroid, ovarian cyst torsion
- *urinary tract disorders:* cystitis, renal calculi.

Other conditions that may mimic dysmenorrhea include ovulation and normal uterine contractions experienced in pregnancy. Emotional conflicts can cause psychogenic (functional) pain.

Because dysmenorrhea almost always follows an ovulatory cycle, both the primary and secondary forms are rare during the anovulatory cycles of menses. After age 20, dysmenorrhea is generally secondary.

Signs and symptoms

Dysmenorrhea produces sharp, intermittent, cramping, lower abdominal pain, which usually radiates to the back, thighs, groin, and vulva. Such pain—sometimes compared to labor pains—typically starts with or immediately before menstrual flow and peaks within 24 hours. Dysmenorrhea may also be associated with the characteristic signs and symptoms of premenstrual syndrome (urinary frequency, nausea,

vomiting, diarrhea, headache, chills, abdominal bloating, painful breasts, depression, and irritability).

Diagnosis

Pelvic examination and a detailed patient history may help suggest the cause of dysmenorrhea.

Primary dysmenorrhea is diagnosed when secondary causes are ruled out. Appropriate tests (such as laparoscopy, dilatation and curettage, and pelvic ultrasound) are used to diagnose underlying disorders in secondary dysmenorrhea.

Treatment

Initial treatment aims to relieve pain. Painrelief measures may include:

- analgesics (such as aspirin) for mild to moderate pain (most effective when taken 24 to 48 hours before onset of menses; are especially effective for treating dysmenorrhea because they also inhibit prostaglandin synthesis; stronger anti-inflammatories may be used.
- opioids if pain is severe (infrequently used)
- prostaglandin inhibitors (such as naproxen and ibuprofen) to relieve pain by decreasing the severity of uterine contractions
- heat applied locally to the lower abdomen (may relieve discomfort in mature women but isn't recommended in young adolescents because appendicitis may mimic dysmenorrhea).

For primary dysmenorrhea, administration of sex steroids is an effective alternative to treatment with antiprostaglandins or analgesics. Such therapy usually consists of hormonal contraceptives to relieve pain by suppressing ovulation. However, patients who are attempting pregnancy should rely on antiprostaglandin therapy instead of hormonal contraceptives to relieve symptoms of primary dysmenorrhea.

Because persistently severe dysmenorrhea may have a psychogenic cause, psychological evaluation and appropriate counseling may be helpful.

In secondary dysmenorrhea, treatment is designed to identify and correct the underlying cause. This may include surgical treatment of underlying disorders, such as endometriosis or uterine leiomyomas.

However, surgical treatment is recommended only after conservative therapy fails.

Special considerations

Effective management of the patient with dysmenorrhea focuses on relief of symptoms, emotional support, and appropriate patient teaching, especially for the adolescent.

- Obtain a complete history, focusing on the patient's gynecologic complaints, including detailed information on any signs and symptoms of pelvic disease, such as excessive bleeding, changes in bleeding pattern, vaginal discharge, and dyspareunia.
- Provide thorough patient teaching. Explain normal female anatomy and physiology to the patient, as well as the nature of dysmenorrhea. This may be a good opportunity, depending on circumstances, to provide the adolescent patient with information on pregnancy and contraception.
- Encourage the patient to keep a detailed record of her menstrual symptoms and to seek medical care if her symptoms persist.
- Instruct the patient on some home care remedies that may be helpful in relieving discomfort, such as applying a heating pad to the lower abdomen, taking warm showers or baths, drinking warm beverages, and performing circular massage with the fingertips around the lower abdomen.
- Encourage the patient to walk or exercise regularly. Recommend pelvic rocking exercises and relaxation techniques, such as meditation or yoga. Let her know that keeping her legs elevated while lying down or lying on her side with her knees bent may also increase comfort.
- Instruct the patient to eat light but frequent meals and to follow a diet rich in foods high in complex carbohydrates, such as whole grains,

fruits, and vegetables. Tell her to avoid alcohol and foods high in salt, sugar, and caffeine.

Vulvovaginitis

Vulvovaginitis is inflammation of the vulva (vulvitis) and vagina (vaginitis). Because of the proximity of these two structures, inflammation of one occasionally causes inflammation of the other. Vulvovaginitis may occur at any age and affects most females at some time. The prognosis is excellent with treatment.

Causes and incidence

Common causes include:

- infection with *Trichomonas vaginalis,* a protozoan flagellate usually transmitted through sexual intercourse
- infection with *Candida albicans,* a fungus that requires glucose for growth. Incidence rises during the menstrual cycle's secretory phase (Such infection occurs twice as often in pregnant females as in nonpregnant females. It also commonly affects users of hormonal contraceptives, patients who are diabetic, and patients receiving systemic therapy with broad-spectrum antibiotics [incidence may reach 75%.])
- infection with *Gardnerella vaginalis,* a gram-negative bacillus
- parasitic infection (*Phthirus pubis* [crab louse])
- trauma (skin breakdown may lead to secondary infection)
- poor personal hygiene
- chemical irritations, or allergic reactions to hygiene sprays, douches, detergents, clothing, or toilet paper
- vulval atrophy in menopausal women due to decreasing estrogen levels
- retention of a foreign body, such as a tampon or diaphragm.

Complications

- Infection
- Perineal inflammation and edema
- Skin breakdown

Signs and symptoms

In trichomonal vaginitis, vaginal discharge is thin, bubbly, green-tinged, and malodorous. This infection causes marked irritation and itching, and urinary symptoms, such as burning and frequency. Candidal vaginitis produces a thick, white, cottage cheese-like discharge and red, edematous mucous membranes, with white flecks adhering to the vaginal wall, and is often accompanied by intense itching. *G. vaginalis* produces a gray, foul, “fishy” smelling discharge.

Acute vulvitis causes a mild to severe inflammatory reaction, including edema, erythema, burning, and pruritus. Severe pain on urination and dyspareunia may necessitate immediate treatment. Herpes infection may cause painful ulceration or vesicle formation during the active phase.

Diagnosis

Diagnosis of vulvovaginitis requires identification of the infectious organism during microscopic examination of vaginal exudate on a wet slide preparation (a drop of vaginal exudate placed in normal saline solution). In some cases, a culture of the vaginal discharge may identify the organism causing the infection.

Diagnosis of vulvitis or suspected venereal disease may require complete blood count, urinalysis, cytology screening, biopsy of chronic lesions to rule out malignancy, culture of exudate from acute lesions, and possible human immunodeficiency virus testing.

Treatment

The cause of vulvovaginitis determines the appropriate treatment. It may include oral or topical antibiotics, antifungal creams, antibacterial

creams, or similar medications. An antihistamine may be prescribed for allergic reactions. Cold compresses or cool sitz baths may provide relief from pruritus in acute vulvitis; severe inflammation may require warm compresses. Other therapy includes avoiding drying soaps, wearing loose clothing to promote air circulation, and applying topical corticosteroids to reduce inflammation. Chronic vulvitis may respond to topical hydrocortisone or antipruritics and good hygiene (especially in elderly or incontinent patients). Topical estrogen ointments may be used to treat atrophic vulvovaginitis. No cure exists for herpes-virus infections; however, oral and topical acyclovir decreases the duration and symptoms of active lesions.

If a sexually transmitted disease (STD) is diagnosed, it's very important that partners also receive treatment, even if there are no symptoms. Failure of partners to receive treatment can lead to continual reinfection, which may eventually lead to infertility and affect the patient's overall health.

Special considerations

- Ask the patient if she has any drug allergies. Stress the importance of taking the medication for the length of time prescribed, even if symptoms subside.
- Teach the patient how to insert vaginal ointments and suppositories. Tell her to remain prone for at least 30 minutes after insertion to promote absorption (insertion at bedtime is ideal). Suggest she wear a pad to prevent staining her underclothing.
- Report notifiable cases of STDs to local public health authorities.
- Tell the patient that persistent, recurring candidiasis may suggest diabetes or undiagnosed pregnancy.

PREVENTION

- *Encourage good hygiene.*
- *Advise the patient with a history of recurrent vulvovaginitis to wear all-cotton underpants. Advise her to avoid wearing tight-fitting pants and panty hose,*

which encourage the growth of the infecting organisms. Removing underpants at night is also helpful.

- *Encourage the use of condoms to avoid contracting sexually transmitted diseases.*
- *Advise the patient to avoid the use of irritants such as scented soaps and feminine hygiene products.*
- *Advise the patient to avoid baths and the use of hot tubs.*

Ovarian cysts

Ovarian cysts are usually nonneoplastic sacs on an ovary that contain fluid or semisolid material. Although these cysts are usually small and produce no symptoms, they generally require thorough investigation as possible sites of malignant change. Common ovarian cysts include follicular cysts, lutein cysts (granulosa-lutein [corpus luteum] and theca-lutein cysts), and polycystic ovarian disease. Ovarian cysts can develop at any time between puberty and menopause, including during pregnancy. Granulosa-lutein cysts occur infrequently, usually during early pregnancy. The prognosis for nonneoplastic ovarian cysts is excellent.

Causes and incidence

Follicular cysts are generally very small and arise from follicles that overdistend. When such cysts persist into menopause, they secrete excessive amounts of estrogen in response to the hypersecretion of follicle-stimulating hormone and luteinizing hormone that normally occurs during menopause. (See *Follicular cyst*.)

Granulosa-lutein cysts, which occur within the corpus luteum, are functional, nonneoplastic enlargements of the ovaries caused by excessive accumulation of blood during the hemorrhagic phase of the menstrual cycle. Theca-lutein cysts are commonly bilateral and filled with clear, straw-colored fluid; they're often associated with

hydatidiform mole, choriocarcinoma, or hormone therapy (with human chorionic gonadotropin [hCG] or clomiphene citrate).

Complications

- Amenorrhea
- Infertility
- Oligomenorrhea
- Secondary dysmenorrhea

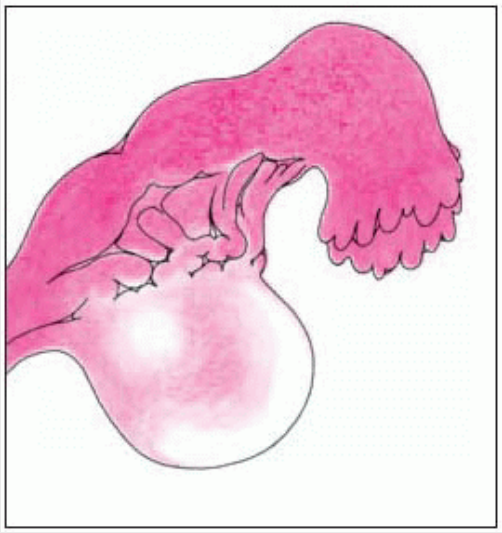
Signs and symptoms

Small ovarian cysts (such as follicular cysts) usually don't produce symptoms unless torsion or rupture causes signs of an acute abdomen (vomiting, abdominal tenderness, distention, and rigidity). Large or multiple cysts may induce mild pelvic discomfort, low back pain, dyspareunia, or abnormal uterine bleeding secondary to a disturbed ovulatory pattern. Ovarian cysts with torsion induce acute abdominal pain similar to that of appendicitis.

Granulosa-lutein cysts that appear early in pregnancy may grow as large as 2″ to 2½″ (5 to 6 cm) in diameter and produce unilateral pelvic discomfort and, if rupture occurs, massive intraperitoneal hemorrhage. In nonpregnant women, these cysts may cause delayed menses, followed by prolonged or irregular bleeding. Polycystic ovarian disease may also produce secondary amenorrhea, oligomenorrhea, or infertility.

FOLLICULAR CYST

A common type of ovarian cyst, a follicular cyst is usually semitransparent and overdistended with watery fluid that's visible through its thin walls.



Diagnosis

Generally, characteristic clinical features suggest ovarian cysts.

CONFIRMING DIAGNOSIS

Visualization of the ovary through ultrasound, computed tomography scan, magnetic resonance imaging, laparoscopy, or surgery (often for another condition) confirms ovarian cysts.

Extremely elevated hCG titers strongly suggest theca-lutein cysts. Pregnancy, including molar pregnancy, must be ruled out.

Treatment

Follicular cysts generally don't require treatment because they tend to disappear spontaneously within 60 to 90 days. However, if they interfere with daily activities, clomiphene citrate by mouth for 5 days or progesterone I.M. (also for 5 days) re-establishes the ovarian hormonal cycle and induces ovulation. Hormonal contraceptives haven't been proven to accelerate involution of functional cysts (including both types of lutein cysts and follicular cysts).

Treatment for granulosa-lutein cysts that occur during pregnancy is aimed at relieving symptoms because these cysts diminish during the third trimester and rarely require surgery. Theca-lutein cysts disappear spontaneously after elimination of the hydatidiform mole, destruction of choriocarcinoma, or discontinuation of hCG or clomiphene citrate therapy.

Surgery, in the form of laparoscopy or exploratory laparotomy with possible ovarian cystectomy or oophorectomy, may become necessary if an ovarian cyst is found to be persistent or suspicious.

Special considerations

Thorough patient teaching is a primary consideration. Carefully explain the cyst's nature, the type of discomfort the patient may experience, and how long the condition is expected to last.

- Preoperatively, watch for signs of cyst rupture, such as increasing abdominal pain, distention, and rigidity. Monitor vital signs for fever, tachypnea, or hypotension, which may indicate peritonitis or intraperitoneal hemorrhage. Administer sedatives, as ordered, to ensure adequate rest before surgery.
- Postoperatively, encourage frequent movement in bed and early ambulation, as ordered. Early ambulation prevents pulmonary embolism.
- Provide emotional support. Offer appropriate reassurance if the patient fears cancer or infertility.
- Before discharge, advise the patient to increase her activities at home gradually—preferably over 4 to 6 weeks. Tell her to abstain from sexual intercourse and not to use tampons and douches during this period.

Polycystic ovary syndrome

Polycystitic ovarian syndrome (PCOS) is a metabolic syndrome characterized by multiple ovarian cysts. It produces a syndrome of hormonal disturbances that includes insulin resistance, obesity, hirsutism, acne, and ovulation and fertility problems. With proper treatment, the prognosis for ovulation and fertility is good.

As with all anovulation syndromes, the pulsaltile release of gonadotropin-releasing hormone is lacking. This allows for the initial ovarian follicle development to be normal, but many small follicles begin to accumulate because there's no selection of a dominant follicle. These follicles then respond abnormally to hormonal stimulation, causing an abnormal estrogen secretion during the menstrual cycle.

Insulin, along with other hormones, plays a role in regulating ovary function. Insulin resistance results in high levels of circulating insulin. The high levels of insulin affect the function of cells, including ovary cells. This can lead to anovulation and infertility.

Causes and incidence

The precise cause of PCOS isn't known. There are several theories regarding the cause including abnormal enzyme activity triggering excess androgen secretion from the ovaries and adrenal glands, as well as endocrine abnormalities or genetic links.

It's most common in women under age 30. In the United States it affects 4% to 12% of women of childbearing age.

Complications

- Hypertension
- Increased risk of breast or endometrial cancer
- Infertility
- Type 2 diabetes mellitus

Signs and symptoms

Signs and symptoms of PCOS include mild pelvic discomfort, lower back pain, and dyspareunia caused by multiple ovarian cysts, abnormal uterine bleeding secondary to disturbed ovulatory pattern, hirsutism and male-pattern hair loss that result from abnormal patterns of estrogen secretion, obesity caused by abnormal hormone regulation, and acne caused by excess sebum production that results from disturbed androgen secretion.

Diagnosis

In polycystic ovarian syndrome, bilaterally enlarged polycystic ovaries can be seen on

physical examination. Tests reveal slight elevation of urinary 17-ketosteroids and anovulation (shown by basal body temperature graphs and endometrial biopsy). Blood testing reveals an elevated LH to FSH ratio (usually 3:1 or greater), and elevated testosterone, androstenedione, and glucose levels. Direct observation must rule out paraovarian cysts of the broad ligaments, salpingitis, endometriosis, and neoplastic cysts. Ultrasonography, abdominal magnetic imaging, or surgery (commonly done for another condition), allows visualization of the ovary, which may confirm the presence of ovarian cysts.

Treatment

Treatment of polycystic ovarian syndrome may include use of such drugs as cloniphene citrate to induce ovulation or medroxyprogesterone acetate for 10 days of every month for the patient who doesn't want to become pregnant. For the patient who needs reliable contraception, a low-dose hormonal contraceptive is used to treat abnormal bleeding. Surgical treatment may involve a hysterectomy with bilateral salpingo-oophorectomy for refractory pain and bleeding.

Drugs such as metformin, pioglitazone, or rosiglitazone can be used to make cells more insulin-sensitive.

Special considerations

Provide appropriate postoperative care including the following:

- Watch for signs of cyst rupture, such as increasing abdominal pain, distention, and rigidity.
- Monitor the patient's vital signs for fever, tachypnea, or hypotension (possibly indicating peritonitis or intraperitoneal hemorrhage).
- Encourage frequent movement in bed and early ambulation, as ordered, to prevent pulmonary embolism.

- Provide emotional support, offering appropriate reassurance if the patient fears cancer or infertility.
- Before discharge, if the patient is overweight, discuss the importance of weight reduction and the role it may play in reducing insulin resistance.

Endometriosis

Endometriosis is the presence of endometrial tissue outside the lining of the uterine cavity. Such ectopic tissue is generally confined to the pelvic area, most commonly around the ovaries, uterovesical peritoneum, uterosacral ligaments, and cul-de-sac, but it can appear anywhere in the body. This ectopic endometrial tissue responds to normal stimulation in the same way that the endometrium does. During menstruation, the ectopic tissue bleeds, which causes inflammation of the surrounding tissues. This inflammation causes fibrosis, leading to adhesions that produce pain and infertility.

Active endometriosis usually occurs between ages 20 and 40; it's uncommon before age 20. Severe symptoms of endometriosis may have an abrupt onset or may develop over many years. This disorder usually becomes progressively severe during the menstrual years; after menopause, it may subside.

Causes and incidence

The mechanisms by which endometriosis causes symptoms, including infertility, are unknown. The main theories to explain this disorder are:

- transtubal regurgitation of endometrial cells and implantation at ectopic sites
- coelomic metaplasia (repeated inflammation may induce metaplasia of mesothelial cells to the endometrial epithelium)
- lymphatic or hematogenous spread to explain extraperitoneal disease.

Endometriosis occurs in 10% of women during the reproductive years. Prevalence may be as high as 25% to 35% among infertile women. A woman with a mother or sister with endometriosis is six times more

likely to develop endometriosis than a woman without this familial history.

Complications

- Anemia
- Infertility
- Spontaneous abortion

Signs and symptoms

The classic symptom of endometriosis is acquired dysmenorrhea, which may produce

constant pain in the lower abdomen and in the vagina, posterior pelvis, and back. This pain usually begins from 5 to 7 days before menses reaches its peak and lasts for 2 to 3 days. It differs from primary dysmenorrheal pain, which is more cramplike and concentrated in the abdominal midline. However, the pain's severity doesn't necessarily indicate the extent of the disease.

Other clinical features depend on the location of the ectopic tissue:

- ovaries and oviducts: infertility and profuse menses
- ovaries or cul-de-sac: deep-thrust dyspareunia
- bladder: suprapubic pain, dysuria, hematuria
- small bowel and appendix: nausea and vomiting, which worsen before menses, and abdominal cramps
- cervix, vagina, and perineum: bleeding from endometrial deposits in these areas during menses.

The primary complications of endometriosis are infertility and chronic pelvic pain.

Diagnosis

Pelvic examination may suggest endometriosis. Palpation may detect multiple tender nodules on uterosacral ligaments or in the rectovaginal septum in one-third of patients. These nodules enlarge and become more tender during menses. Palpation may also uncover ovarian enlargement in the presence of endometrial cysts on the ovaries or thickened, nodular adnexa (as in pelvic inflammatory disease). Laparoscopy must confirm the diagnosis and determine the disease's stage before treatment is started. Endometriosis is classified in stages: Stage I, mild; Stage II, moderate; Stage III, severe; and Stage IV, extensive.

Treatment

Treatment varies according to the disease's stage and the patient's age and desire to have children. Conservative therapy for young women who want to have children includes androgens, such as danazol, which produce a temporary remission in Stages I and II. Progestins and hormonal contraceptives also relieve symptoms. Gonadotropin-releasing hormone agonists, by inducing a pseudomenopause and, thus, a “medical oophorectomy,” may cause a remission of disease and are commonly used. However, medical therapy remains inadequate.

When ovarian masses are present, surgery must rule out cancer. Conservative surgery includes laparoscopic removal of endometrial implants with conventional or laser techniques and presacral neurectomy for severe dysmenorrhea. The treatment of choice for women who don't want to bear children or for extensive disease is a total abdominal hysterectomy with bilateral salpingo-oophorectomy.

Special considerations

- Because infertility is a possible complication, advise the patient who wants children not to postpone childbearing.
- Recommend an annual pelvic examination and Papanicolaou test to all patients.

Uterine leiomyomas

The most common benign tumors in women, uterine leiomyomas, also known as *myomas, fibromyomas,* or *fibroids,* are smooth-muscle tumors. They usually occur in multiples in the uterine corpus, although they may appear on the cervix or on the round or broad ligament. Though uterine leiomyomas are often called *fibroids,* this term is misleading because they consist of muscle cells and not fibrous tissue.

Causes and incidence

The cause of uterine leiomyomas is unknown, but steroid hormones, including estrogen and progesterone, and several growth factors, including epidermal growth factor, have been implicated as regulators of leiomyoma growth. Leiomyomas typically arise after menarche and regress after menopause, implicating estrogen as a promoter of leiomyoma growth.

Uterine leiomyomas occur in 20% to 25% of women of reproductive age and reportedly affect three times as many black women as white women. The tumors become malignant (leiomyosarcoma) in only 0.1% or less of patients.

Complications

- Anemia
- Dystocia
- Infertility
- Intestinal obstruction
- Preterm labor
- Spontaneous abortion

Signs and symptoms

Leiomyomas may be located within the uterine wall or may protrude into the endometrial cavity or from the serosal surface of the uterus. Most leiomyomas produce no symptoms. The most common symptom is

abnormal bleeding, which typically presents clinically as menorrhagia. Uterine leiomyomas probably don't cause pain directly except when associated with torsion of a pedunculated subserous tumor. Pelvic pressure and impingement on adjacent viscera are common indications for treatment. Other symptoms may include urinary retention, constipation, or dyspareunia.

Diagnosis

Clinical findings and patient history may suggest uterine leiomyomas. Bimanual examination may reveal an enlarged, firm, nontender, and irregularly contoured uterus. Ultrasound (transvaginal or pelvic) or magnetic resonance imaging allows accurate assessment of the dimensions, number, and location of tumors. Other diagnostic procedures include hysterosalpingography, dilatation and curettage, endometrial biopsy, and laparoscopy.

Treatment

Treatment depends on the symptoms' severity, the tumors' size and location, and the patient's age, parity, pregnancy status, desire to have children, and general health.

Treatment options include nonsurgical as well as surgical procedures. Nonsurgical methods include taking serial histories and performing physical assessments at clinically indicated intervals and administering gonadotropin-releasing hormone (Gn-RH) analogues, which are capable of rapidly suppressing pituitary gonadotropin release, leading to profound hypoestrogenemia and a 50% reduction in uterine volume. The peak effects of these Gn-RH analogues occur in the 12th week of therapy. The benefits are reduction in tumor size before surgery, reduction in intraoperative blood loss, and an increase in preoperative hematocrit. Gn-RH analogues aren't curative.

Surgical procedures include abdominal, laparoscopic, or hysteroscopic myomectomy—for patients who want to preserve fertility. Myolysis can successfully treat fibroids without hysterectomy or major surgery. Performed on an outpatient basis, this laparoscopic procedure coagulates the fibroids and preserves the uterus and the patient's childbearing potential. Hysterectomy is the definitive treatment for

symptomatic women who have completed childbearing, but uterine artery embolization may be an alternative in some situations.

Special considerations

- Tell the patient to report any abnormal bleeding or pelvic pain immediately.
- If a hysterectomy or oophorectomy is indicated, explain the operation's effects on menstruation, menopause, and sexual activity to the patient.
- Reassure the patient that she most likely won't experience premature menopause if her ovaries are left intact.
- If it's necessary for the patient to have a multiple myomectomy, make sure she understands pregnancy is still possible. Explain that a cesarean delivery may be indicated.

Precocious puberty

In females, precocious puberty is the early onset of pubertal changes: breast development, pubic and axillary hair development, and menarche before age 9. (Normally, the mean age for menarche is 13.) In true precocious puberty, the ovaries mature and pubertal changes progress in an orderly manner. In pseudoprecocious puberty, pubertal changes occur without corresponding ovarian maturation.

Causes and incidence

About 85% of all cases of true precocious puberty in females are constitutional, resulting from early development and activation of the endocrine glands without corresponding

abnormality. Other causes of true precocious puberty are pathologic and include central nervous system (CNS) disorders resulting from tumors, trauma, infection, or other lesions. These CNS disorders include hypothalamic tumors, intracranial tumors (pinealoma, granuloma, hamartoma), hydrocephaly, degenerative encephalopathy, tuberous sclerosis, neurofibromatosis, encephalitis, skull injuries, meningitis, and

peptic arachnoiditis. McCune-Albright's syndrome, Silver's syndrome, and juvenile hypothyroidism are conditions often associated with female precocity.

Pseudoprecocious puberty may result from increased levels of sex hormones due to ovarian and adrenocortical tumors, adrenal cortical virilizing hyperplasia, or ingestion of estrogens or androgens. It may also result from increased end-organ sensitivity to low levels of circulating sex hormones, whereby estrogens promote premature breast development and androgens promote premature pubic and axillary hair growth.

Risk factors include race (more blacks are affected than whites), sex (more girls are affected than boys), and obesity.

Other signs may include underarm hair growth, acne, and adult-type body odor.

Complications

- Polycystic ovary syndrome
- Short stature

Signs and symptoms

The usual pattern of precocious puberty in females is a rapid growth spurt, thelarche (breast development), pubarche (pubic hair development), and menarche—all before age 9. These changes may occur independently or simultaneously.

Diagnosis

Diagnosis requires a complete patient history, a thorough physical examination, and special tests to differentiate between true and pseudoprecocious puberty and to indicate what treatment may be necessary. X-rays of the hands, wrists, knees, and hips determine bone age and possible premature epiphyseal closure. Other tests detect abnormally high hormonal levels for the patient's age: vaginal smear for estrogen secretion, urinary tests for gonadotropic activity and excretion

of 17-ketosteroids, and radioimmunoassay for both luteinizing and follicle-stimulating hormones.

As indicated, ultrasound, laparoscopy, or exploratory laparotomy may verify a suspected abdominal lesion; EEG, ventriculography, pneumoencephalography, computed axial tomography scan, or angiography can detect CNS disorders.

Treatment

Treatment of constitutional true precocious puberty may include medroxyprogesterone to reduce secretion of gonadotropins and prevent menstruation. Other therapy depends on the cause of precocious puberty and its stage of development:

- Adrenogenital syndrome necessitates cortical or adrenocortical steroid replacement.
- Abdominal tumors necessitate surgery to remove ovarian and adrenal tumors. Regression of secondary sex characteristics may follow such surgery, especially in young children.
- Choriocarcinomas require surgery and chemotherapy.
- Hypothyroidism requires thyroid extract or levothyroxine to decrease gonadotropic secretions.
- Drug ingestion requires that the medication be discontinued.

In precocious thelarche and pubarche, no treatment is necessary.

Special considerations

The dramatic physical changes produced by precocious puberty can be upsetting and alarming for the child and her family. Provide a calm, supportive atmosphere, and encourage the patient and her family to express their feelings about these changes. Explain all diagnostic procedures and tell the patient and her family that surgery may be necessary.

- Explain the condition to the child in terms she can understand to prevent feelings of shame and loss of self-esteem. Provide appropriate

sex education, including information on menstruation and related hygiene.

- Tell parents that, although their daughter seems physically mature, she isn't psychologically mature, and the discrepancy between physical appearance and psychological and psychosexual maturation may create problems. Warn them against expecting more of her than they would expect of other children her age.
- Suggest that parents continue to dress their daughter in clothes that are appropriate for her age and that don't call attention to her physical development.
- Reassure parents that precocious puberty doesn't usually precipitate precocious sexual behavior.

Menopause

Menopause is the cessation of menstruation. It results from a complex syndrome of physiologic changes—the climacteric—caused by declining ovarian function. The climacteric produces various body changes, the most dramatic being menopause.

Causes and incidence

- Physiologic menopause, the normal decline in ovarian function due to aging, begins in most women between ages 45 and 55, on average 51, and results in infrequent ovulation, decreased menstrual function and, eventually, cessation of menstruation.
- Pathologic (premature) menopause, the gradual or abrupt cessation of menstruation before age 40, occurs idiopathically in about 1% of women in the United States. However, certain diseases, especially severe infections and reproductive tract tumors, may cause pathologic menopause by seriously impairing ovarian function. Other factors that may precipitate pathologic menopause include malnutrition, chemotherapy, debilitation, extreme emotional stress, excessive radiation exposure, and surgical procedures that impair ovarian blood supply.

Complications

- Atherosclerosis
- Osteoporosis
- Urinary incontinence
- Weight gain

Signs and symptoms

Many menopausal women are asymptomatic but some have severe symptoms. The decline in ovarian function and consequent decreased estrogen level produce menstrual irregularities: a decrease in the amount and duration of menstrual flow, spotting, and episodes of amenorrhea and polymenorrhea (possibly with hypermenorrhea). Irregularities may last a few months or persist for several years before menstruation ceases permanently.

The following body system changes may occur (usually after the permanent cessation of menstruation):

- Reproductive system: Menopause may cause shrinkage of vulval structures and loss of subcutaneous fat, possibly leading to atrophic vulvitis; atrophy of vaginal mucosa and flattening of vaginal rugae, possibly causing bleeding after coitus or douching; vaginal itching and discharge from bacterial invasion; and loss of capillaries in the atrophying vaginal wall, causing the pink, rugal lining to become smooth and white. Menopause may also produce excessive vaginal dryness and dyspareunia due to decreased lubrication from the vaginal walls and decreased secretion from Bartholin's glands; smaller ovaries and oviducts; and progressive pelvic relaxation as the supporting structures lose their tone due to the absence of estrogen.

ELDER TIP

As a woman ages, atrophy causes the vagina to shorten and the mucous lining to become thin, dry, less elastic, and pale as a result of decreased vascularity. In addition, the pH of vaginal secretions increases, making the

vaginal environment more alkaline. The type of flora also changes, increasing the older woman's chance of vaginal infections.

- Urinary system: Atrophic cystitis due to the effects of decreased estrogen levels on bladder mucosa and related structures may cause pyuria, dysuria, and urinary frequency, urgency, and incontinence. Urethral carbuncles from loss of urethral tone and mucosal thinning may cause dysuria, meatal tenderness, and hematuria.
- Mammary system: Breast size decreases.
- Integumentary system: The patient may experience loss of skin elasticity and turgor due to estrogen deprivation, loss of pubic and axillary hair and, occasionally, slight alopecia.
- Autonomic nervous system: The patient may exhibit hot flashes and night sweats (in 75% of women), vertigo, syncope, tachycardia, dyspnea, tinnitus, emotional disturbances (irritability, nervousness, crying spells, fits of anger), and exacerbation of pre-existing depression, anxiety, and compulsive, manic, or schizoid behavior.

Menopause may also induce atherosclerosis, and a decrease in estrogen level contributes to osteoporosis.

Ovarian activity in younger women is believed to provide a protective effect on the cardiovascular system, and the loss of this function at menopause may partly explain the increased death rate from myocardial infarction in older women. Also, estrogen has been found to increase levels of high-density lipoprotein cholesterol.

Diagnosis

Patient history and typical clinical features suggest menopause. A Papanicolaou (Pap) test may show the influence of estrogen deficiency on vaginal mucosa. Radioimmunoassay (RIA) may be performed, but because of the expense involved, it isn't necessary to confirm a diagnosis of menopause. If done, RIA shows the following blood hormone levels:

- estrogen: 0 to 14 ng/dl

- plasma estradiol: 15 to 40 pg/ml
- estrone: 25 to 50 pg/ml.

RIA also shows the following urine values:

- estrogen: 6 to 28 mcg/24 hours
- pregnanediol (urinary secretion of progesterone): 0.3 to 0.9 mg/24 hours.

Follicle-stimulating hormone production may increase as much as 15 times its normal level; luteinizing hormone production, as much as 5 times.

Pelvic examination, endometrial biopsy, and dilatation and curettage may rule out organic disease in patients with abnormal menstrual bleeding.

Treatment

Menopause is a natural process that doesn't require treatment unless menopausal symptoms, such as hot flashes or vaginal dryness, are particularly bothersome. Hormonal agents for patients with a uterus include estrogen with progesterone to prevent endometrial cancer. If the patient doesn't have a uterus, progesterone isn't necessary.

The Women's Health Initiative has led physicians to revise their recommendations regarding hormone replacement therapy (HRT). Health risks (increased incidence of breast cancer, heart attacks, strokes, and blood clots) outweigh the health benefits (decreased osteoporosis) for women taking both estrogen and progesterone. If symptoms are severe, HRT may be considered for short-term use (2 to 4 years) to reduce vaginal dryness, hot flashes, and other symptoms. If this is used, frequent pelvic examinations, Pap smears, physical examinations, breast examinations, and mammograms are indicated to reduce the risks of estrogen replacement therapy while still gaining the treatment's benefits.

Medications may be prescribed to help with mood swings, hot flashes, and other symptoms. These include low doses of antidepressants, such

as paroxetine, venlafaxine, and fluoxetine, or clonidine, which is normally used to control high blood pressure.

Special considerations

- Provide the patient with all the facts about HRT, if used. Make sure she realizes the need for regular monitoring.
- Before HRT begins, have the patient undergo a baseline physical examination, Pap test, and mammogram.
- Advise the patient not to discontinue contraceptive measures until cessation of menstruation has been confirmed.
- Tell the patient to immediately report vaginal bleeding or spotting after menstruation has ceased.
- Discuss alternatives to HRT, which can help with the discomforting symptoms of menopause:

  – Advise the patient to dress lightly and in layers.

  – Tell the patient to avoid caffeine, alcohol, and spicy foods. Encourage soy-based foods.

  – Instruct the patient to practice slow, deep breathing whenever a hot flash starts to come on, or to try other relaxation techniques, such as yoga, tai chi, or meditation. Tell her that acupuncture may also be helpful.

  – If the patient is in a sexually active relationship, tell her to remain sexually active to preserve vaginal elasticity. Water-based lubricants can be used during sexual intercourse to decrease dryness.

  – Instruct the patient that Kegel exercises may be performed daily to strengthen the vaginal and pelvic muscles.

Female infertility

Primary infertility is the inability to conceive after regular intercourse for at least 1 year without contraception. Secondary infertility occurs in couples who have previously been pregnant at least once, but are unable

to achieve another pregnancy. About 30% to 40% of all infertility is attributed to the male, and 40% to 50% to the female; about 10% to 30% is due to a combination of male and female factors. Following extensive investigation and treatment, about 50% of these infertile couples achieve pregnancy. Of the 50% who don't, 10% have no pathologic basis for infertility; the prognosis for this group becomes extremely poor if pregnancy isn't achieved within 3 years.

Causes and incidence

The causes of female infertility may be functional, anatomic, or psychosocial:

- Functional causes: complex hormonal interactions determine the normal function of the female reproductive tract and require an intact hypothalamic-pituitaryovarian axis—the system that stimulates and regulates the hormone production necessary for normal sexual development and function. Any defect or malfunction of this axis can cause infertility due to insufficient gonadotropin secretions (both luteinizing hormone [LH] and follicle-stimulating hormone). The ovary controls, and is controlled by, the hypothalamus through a system of negative and positive feedback mediated by estrogen production. Insufficient gonadotropin levels may result from infections, tumors, or neurologic disease of the hypothalamus or pituitary gland. Hypothyroidism also impairs fertility.
- Anatomic causes include the following:

  – Ovarian factors are related to anovulation and oligo-ovulation (infrequent ovulation) and are a major cause of infertility. Pregnancy or direct visualization provides irrefutable evidence of ovulation. Presumptive signs of ovulation include regular menses, cyclic changes reflected in basal body temperature readings, postovulatory progesterone levels, and endometrial changes due to the presence of progesterone. Absence of presumptive signs suggests anovulation. Ovarian failure, in which no ova are produced by the ovaries, may result from ovarian dysgenesis or premature menopause. Amenorrhea is often associated with ovarian failure. Oligo-ovulation may be due to a mild hormonal imbalance in gonadotropin production and regulation and may be caused by

polycystic disease of the ovary or abnormalities in the adrenal or thyroid gland that adversely affect hypothalamic-pituitary functioning.

– Uterine fibroids or uterine abnormalities rarely cause infertility; however, uterine abnormalities may include congenitally absent uterus, bicornuate or double uterus, leiomyomas, or Asherman's syndrome, in which the anterior and posterior uterine walls adhere because of scar tissue formation.

– Tubal and peritoneal factors are due to faulty tubal transport mechanisms and unfavorable environmental influences affecting the sperm, ova, or recently fertilized ovum. Tubal loss or impairment may occur secondary to ectopic pregnancy.

Frequently, tubal and peritoneal factors result from anatomic abnormalities: bilateral occlusion of the tubes due to salpingitis (resulting from gonorrhea, tuberculosis, or puerperal sepsis), peritubal adhesions (resulting from endometriosis, pelvic inflammatory disease [PID], diverticulosis, or childhood rupture of the appendix), and uterotubal obstruction (due to tubal spasm).

– Cervical factors may include malfunctioning cervix that produces deficient or excessively viscous mucus and is impervious to sperm, preventing entry into the

uterus. In cervical infection, viscous mucus may contain spermicidal macrophages. Cervical antibodies have also been found to immobilize sperm.

- Psychosocial problems probably account for relatively few cases of infertility. Occasionally, ovulation may stop under stress due to failure of LH release. The frequency of intercourse may be related. More often, however, psychosocial problems result from, rather than cause, infertility.

About 10% to 20% of couples will be unable to conceive after 1 year of attempting to become pregnant. Healthy couples who are younger than age 30 and having intercourse regularly only have a 25% to 30% change of getting pregnant each month. A woman's peak fertility is in her early

20s. As a woman ages beyond 35 (and particularly beyond 40), the likelihood of conception is less than 10% per month.

Complication

- Depression

Diagnosis

Inability to achieve pregnancy after having regular intercourse without contraception for at least 1 year suggests infertility. (In women older than age 35, many clinicians use 6 months rather than 1 year as a cutoff point.)

Diagnosis requires a complete physical examination and health history, including specific questions on the patient's reproductive and sexual function, past diseases, mental state, previous surgery, types of contraception used in the past, and family history. Irregular, painless menses may indicate anovulation. A history of PID may suggest fallopian tube blockage. Sometimes PID is silent, and no history may be known.

The following tests assess ovulation:

- Basal body temperature graph shows a sustained elevation in body temperature postovulation until just before the onset of menses, indicating the approximate time of ovulation.
- Endometrial biopsy, done on or about day 26, provides histologic evidence that ovulation has occurred. However, endometrial biopsy is retrospective, which diminishes its utility.
- Progesterone blood levels, measured when they should be highest, can show a luteal phase deficiency or presumptive evidence of ovulation.

The following procedures assess structural integrity of the fallopian tubes, the ovaries, and the uterus:

- Urinary LH kits, available without a prescription, can sensitively detect the LH surge about 24 hours preovulation, allowing couples to time coitus.
- Hysterosalpingography provides radiologic evidence of tubal obstruction and uterine cavity abnormalities by injecting radiopaque

contrast fluid through the cervix.

Male-female interaction studies include the following:

- Postcoital test (Sims'-Huhner test) examines the cervical mucus for motile sperm cells following intercourse that takes place at midcycle (as close to ovulation as possible).
- Immunologic or antibody testing detects spermicidal antibodies in the female's sera.

Treatment

Treatment depends on identifying the underlying abnormality or dysfunction within the hypothalamic-pituitary-ovarian complex. In hyperactivity or hypoactivity of the adrenal or thyroid gland, hormone therapy is necessary; progesterone deficiency requires progesterone replacement. Anovulation necessitates treatment with clomiphene, human menopausal gonadotropins, or human chorionic gonadotropin; ovulation usually occurs several days after such administration. If mucus production decreases (an adverse effect of clomiphene), small doses of estrogen to improve the quality of cervical mucus may be given concomitantly; however, such intervention remains unproven.

Surgical restoration may correct certain anatomic causes of infertility such as fallopian tube obstruction. Surgery may also be necessary to remove tumors located within or near the hypothalamus or pituitary gland. Endometriosis requires drug therapy (danazol or medroxyprogesterone, or noncyclic administration of hormonal contraceptives), surgical removal of areas

of endometriosis, or a combination of both.

PREVENTION

PREVENTING INFERTILITY

Several things can be done to help prevent infertility. Advise your patient about the following:

Sexually transmitted diseases (STDs)

Gonorrhea and Chlamydia are the two most common causes of STD-related causes of infertility. These STDs can lead to ectopic pregnancy and scarring of the fallopian tubes. Practicing safe sex can help prevent STDs.

Endometriosis

The early detection and treatment of endometriosis can eliminate the scarring that leads to infertility.

Lifestyle changes

Advise your patient to get regular moderate exercise, not intense exercise, which can lead to amenorrhea. Advise her to maintain adequate weight. Being underweight or overweight can affect hormones. Advise her to avoid using alcohol and illegal drugs, and if she smokes, suggest that she quit; these substances may have an effect on fertility. Suggest that she discuss with her practitioner the use of prescription over-the-counter drugs and the possible effects they may have on fertility.

Other options, often controversial and involving emotional and financial cost, include surrogate mothering, frozen embryos, or in vitro fertilization (IVF). In view of the good success rate of IVF (about 20%), IVF may be used instead of surgery in many cases.

Special considerations

Management includes providing the infertile couple with emotional support and information about diagnostic and treatment techniques. (See *Preventing infertility.*)

- An infertile couple may suffer loss of self-esteem; they may feel angry, guilty, or inadequate, and the diagnostic procedures for this disorder may intensify their fear and anxiety. You can help by explaining these procedures thoroughly. Above all, encourage the patient and her partner to talk about their feelings, and listen to what they have to say with a nonjudgmental attitude.

- If the patient requires surgery, tell her what to expect postoperatively; this, of course, depends on which procedure is to be performed.

Pelvic inflammatory disease

Pelvic inflammatory disease (PID) is any acute, subacute, recurrent, or chronic infection of the oviducts and ovaries, with adjacent tissue involvement. It includes inflammation of the fallopian tubes (salpingitis) and ovaries (oophoritis), which can extend to the connective tissue lying between the broad ligaments (parametritis). Early diagnosis and treatment prevent damage to the reproductive system. Untreated PID may cause infertility and may lead to potentially fatal septicemia and shock.

Causes and incidence

PID can result from infection with aerobic or anaerobic organisms. The organisms *Neisseria gonorrhoeae* and *Chlamydia trachomatis* are the most common cause because

they most readily penetrate the bacteriostatic barrier of cervical mucus.

Normally, cervical secretions have a protective and defensive function. Therefore, conditions or procedures that alter or destroy cervical mucus impair this bacteriostatic mechanism and allow bacteria present in the cervix or vagina to ascend into the uterine cavity; such procedures include conization or cauterization of the cervix.

Uterine infection can also follow the transfer of contaminated cervical mucus into the endometrial cavity by instrumentation. Consequently, PID can follow insertion of an intrauterine device, use of a biopsy curet or an irrigation catheter, or tubal insufflation. Other predisposing factors include abortion, pelvic surgery, and infection during or after pregnancy.

Bacteria may also enter the uterine cavity through the bloodstream or from drainage from a chronically infected fallopian tube, a pelvic abscess, a ruptured appendix, diverticulitis of the sigmoid colon, or other infectious foci.

Common bacteria found in cervical mucus are staphylococci, streptococci, diphtheroids, chlamydiae, and coliforms, including *Pseudomonas* and *Escherichia coli*. Uterine infection can result from any one or several of these organisms or may follow the multiplication of normally nonpathogenic bacteria in an altered endometrial environment. Bacterial multiplication is most common during parturition because the endometrium is atrophic, quiescent, and not stimulated by estrogen.

In the United States, nearly 1 million people develop PID each year; many cases go undiagnosed. About 1 in 8 active adolescents will develop PID before age 21.

Complications

- Infertility
- Pulmonary emboli
- Septicemia
- Shock

Signs and symptoms

Clinical features of PID vary with the affected area but generally include a profuse, purulent vaginal discharge, sometimes accompanied by low-grade fever and malaise (particularly if gonorrhea is the cause). The patient experiences lower abdomen pain; movement of the cervix or palpation of the adnexa may be extremely painful. Frequent, painful urination is also commonly reported. Additional signs and symptoms include irregular or absent menstruation, dyspareunia, low back pain, and nausea and vomiting.

Diagnosis

Diagnostic tests generally include:

- Gram stain of secretions from the endocervix or cul-de-sac. Culture and sensitivity testing aids selection of the appropriate antibiotic. Urethral and rectal secretions may also be cultured.
- White blood cell count may reveal leukocytosis.

- Pelvic ultrasonography or computed tomography scan to identify an adnexal or uterine mass.

In addition, patient history is significant. In general, PID is associated with recent sexual intercourse, insertion of an intrauterine device, childbirth, abortion, or a sexually transmitted disease.

Treatment

To prevent progression of PID, antibiotic therapy begins immediately after culture specimens are obtained. Such therapy can be re-evaluated as soon as laboratory results are available (usually after 24 to 48 hours). Infection may become chronic if treated inadequately.

Development of a pelvic abscess necessitates adequate drainage. A ruptured abscess is life-threatening. If this complication develops, the patient may need a total abdominal hysterectomy with bilateral salpingo-oophorectomy. Alternatively, laparoscopic drainage with preservation of the ovaries and uterus may be done.

Concurrent treatment of sexual partners and condom use throughout the course of treatment are necessary.

Special considerations

- After establishing that the patient has no drug allergies, administer antibiotics and analgesics, as ordered.
- Check for fever. If it persists, carefully monitor fluid intake and output for signs of dehydration.

- Watch for abdominal rigidity and distention, possible signs of developing peritonitis. Provide frequent perineal care if vaginal drainage occurs.
- Stress the need for the patient's sexual partner to be examined and, if necessary, treated for infection.
- Because PID may cause painful intercourse, advise the patient to consult with her physician about sexual activity.

PREVENTION

- *To prevent a recurrence, explain the nature and seriousness of PID, and encourage the patient to comply with the treatment regimen.*
- *To prevent infection after minor gynecologic procedures such as dilatation and curettage, tell the patient to immediately report fever, increased vaginal discharge or pain, and advise her to avoid douching and intercourse for at least 7 days.*

UTERINE BLEEDING DISORDERS

Amenorrhea

Amenorrhea is the abnormal absence or suppression of menstruation. Primary amenorrhea is the absence of menarche in an adolescent (by age 18). Secondary amenorrhea is the failure of menstruation for at least 3 months after normal onset of menarche.

Causes and incidence

Amenorrhea is normal before puberty, after menopause, or during pregnancy and lactation; it's pathologic at any other time. It usually results from anovulation due to hormonal abnormalities, such as decreased secretion of estrogen, gonadotropins, luteinizing hormone, and follicle-stimulating hormone; lack of ovarian response to gonadotropins; or constant presence of progesterone or other endocrine abnormalities.

Amenorrhea may also result from the absence of a uterus, endometrial damage, or from ovarian, adrenal, or pituitary tumors. It's also linked to emotional disorders and is common in patients with severe disorders, such as depression and anorexia nervosa. Mild emotional disturbances tend merely to distort the ovulatory cycle, while severe psychic trauma may abruptly change the bleeding pattern or may completely suppress one or more full ovulatory cycles. Amenorrhea may also result from malnutrition, obesity, intense exercise, and prolonged hormonal

contraceptive use. The incidence of primary amenorrhea in the United States is less than 0.1%. The incidence of secondary amenorrhea (caused by something other than pregnancy) is about 4%.

Diagnosis

CONFIRMING DIAGNOSIS

A history of failure to menstruate in a female older than age 18 confirms primary amenorrhea.

Secondary amenorrhea can be diagnosed when a change is noted in a previously established menstrual pattern (absence of menstruation for 3 months). A thorough physical and pelvic examination rules out pregnancy, as well as anatomic abnormalities such as cervical stenosis that may cause false amenorrhea (cryptomenorrhea), in which menstruation occurs without external bleeding.

Onset of menstruation within 1 week after administration of pure progestational agents, such as medroxyprogesterone and progesterone, indicates a functioning uterus. If menstruation doesn't occur, special diagnostic studies are appropriate.

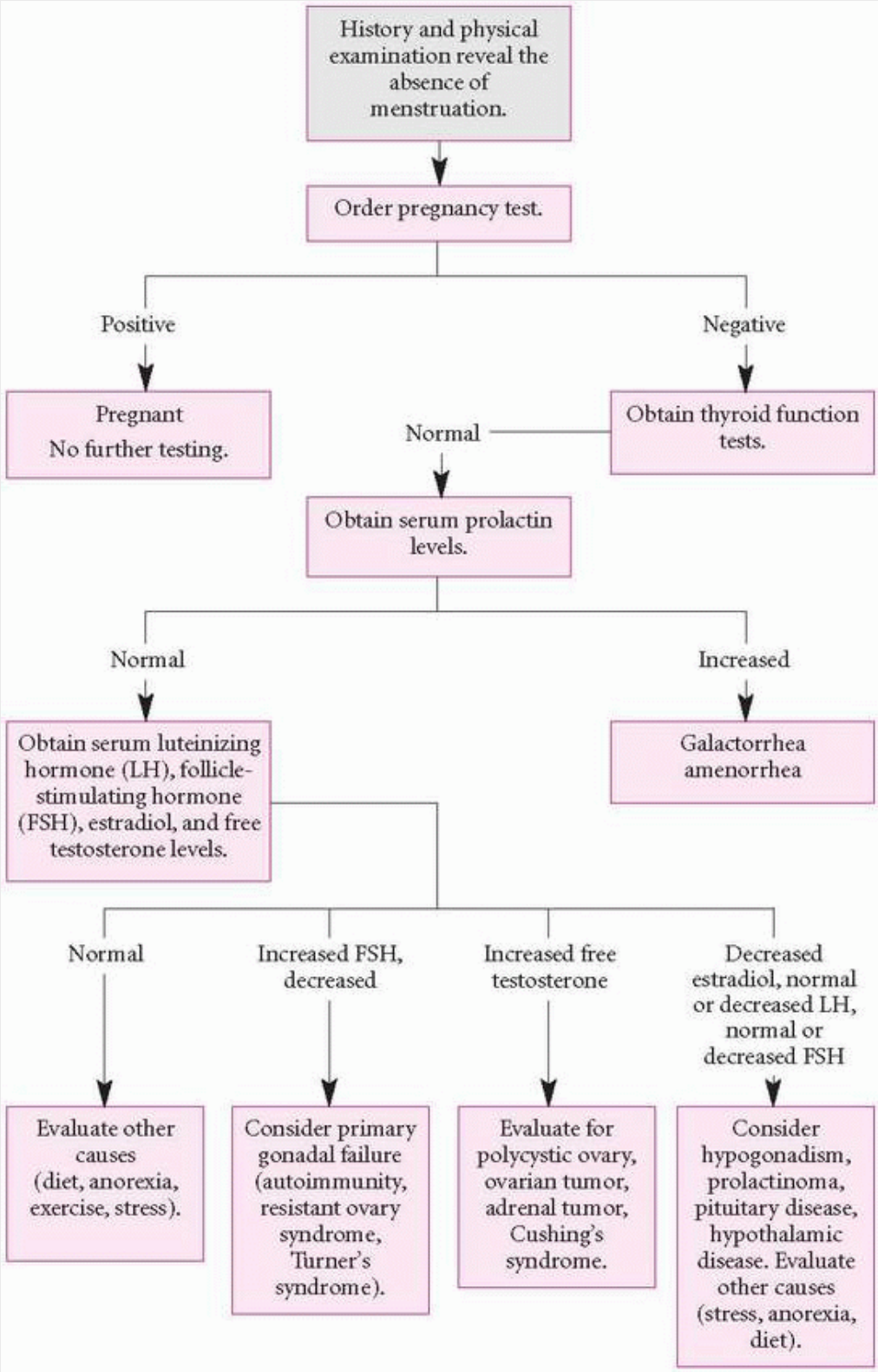
Blood and urine studies may reveal hormonal imbalances, such as lack of ovarian response to gonadotropins (elevated pituitary gonadotropins), failure of gonadotropin secretion (low pituitary gonadotropin levels), and abnormal thyroid levels. Tests for identification of dominant or missing hormones include cervical mucus ferning, vaginal cytologic examinations, basal body temperature, endometrial biopsy (during dilatation and curettage), urinary 17-ketosteroids, and plasma progesterone, testosterone, and androgen levels. A complete medical workup, including X-rays, laparoscopy, and a biopsy, may detect ovarian, adrenal, and pituitary tumors. (See *Diagnosing amenorrhea,* page 1176.)

DIFFERENTIAL DIAGNOSIS

DIAGNOSING AMENORRHEA

The flowchart lists possible diagnostic findings and interpretations to assist with the treatment of the patient

with amenorrhea.



Treatment

Appropriate hormone replacement re-establishes menstruation. Treatment of amenorrhea not related to hormone deficiency depends on the cause. For example, amenorrhea that results from a tumor usually requires surgery.

Special considerations

- Explain all diagnostic procedures.
- Provide reassurance and emotional support. Psychiatric counseling may be necessary if amenorrhea results from emotional disturbances.
- After treatment, teach the patient how to keep an accurate record of her menstrual cycles to aid early detection of recurrent amenorrhea.

Abnormal premenopausal bleeding

Abnormal premenopausal bleeding refers to any bleeding that deviates from the normal menstrual cycle before menopause. These deviations include menstrual bleeding that's abnormally infrequent (oligomenorrhea), abnormally frequent (polymenorrhea), excessive (menorrhagia or hypermenorrhea), deficient (hypomenorrhea), or irregular (metrorrhagia [uterine bleeding between menses]). Rarely, menstrual symptoms aren't accompanied by external bleeding (cryptomenorrhea). Premenopausal bleeding may merely be troublesome or can result in severe hemorrhage; the prognosis depends on the underlying cause. Abnormal bleeding patterns often respond to hormonal or other therapy.

Causes and incidence

Causes of abnormal premenopausal bleeding vary with the type of bleeding:

- Oligomenorrhea and polymenorrhea usually result from anovulation due to an endocrine or systemic disorder.

- Menorrhagia usually results from local lesions, such as uterine leiomyomas, endometrial polyps, and endometrial hyperplasia. It may also result from endometritis, salpingitis, and anovulation.
- Hypomenorrhea results from local, endocrine, or systemic disorders, or from blockage due to partial obstruction by the hymen or to cervical obstruction.
- Cryptomenorrhea may result from an imperforate hymen or cervical stenosis.
- Metrorrhagia usually results from slight physiologic bleeding from the endometrium during ovulation but may also result from local disorders, such as uterine malignancy, cervical erosions, polyps (which tend to bleed after intercourse), or inappropriate estrogen therapy. Complications of pregnancy can also cause premenopausal bleeding. Such bleeding may be as mild as spotting or as severe as menorrhagia. (See *Causes of abnormal premenopausal bleeding*, page 1178.)

Signs and symptoms

Bleeding not associated with abnormal pregnancy is usually painless, but it may be severely painful. When bleeding is associated with abnormal pregnancy, other symptoms include nausea, breast tenderness, bloating, and fluid retention. Severe or prolonged bleeding causes anemia, especially in patients with underlying disease such as blood dyscrasia and in patients receiving anticoagulants.

Diagnosis

The typical clinical picture confirms abnormal premenopausal bleeding. Special tests identify the underlying cause:

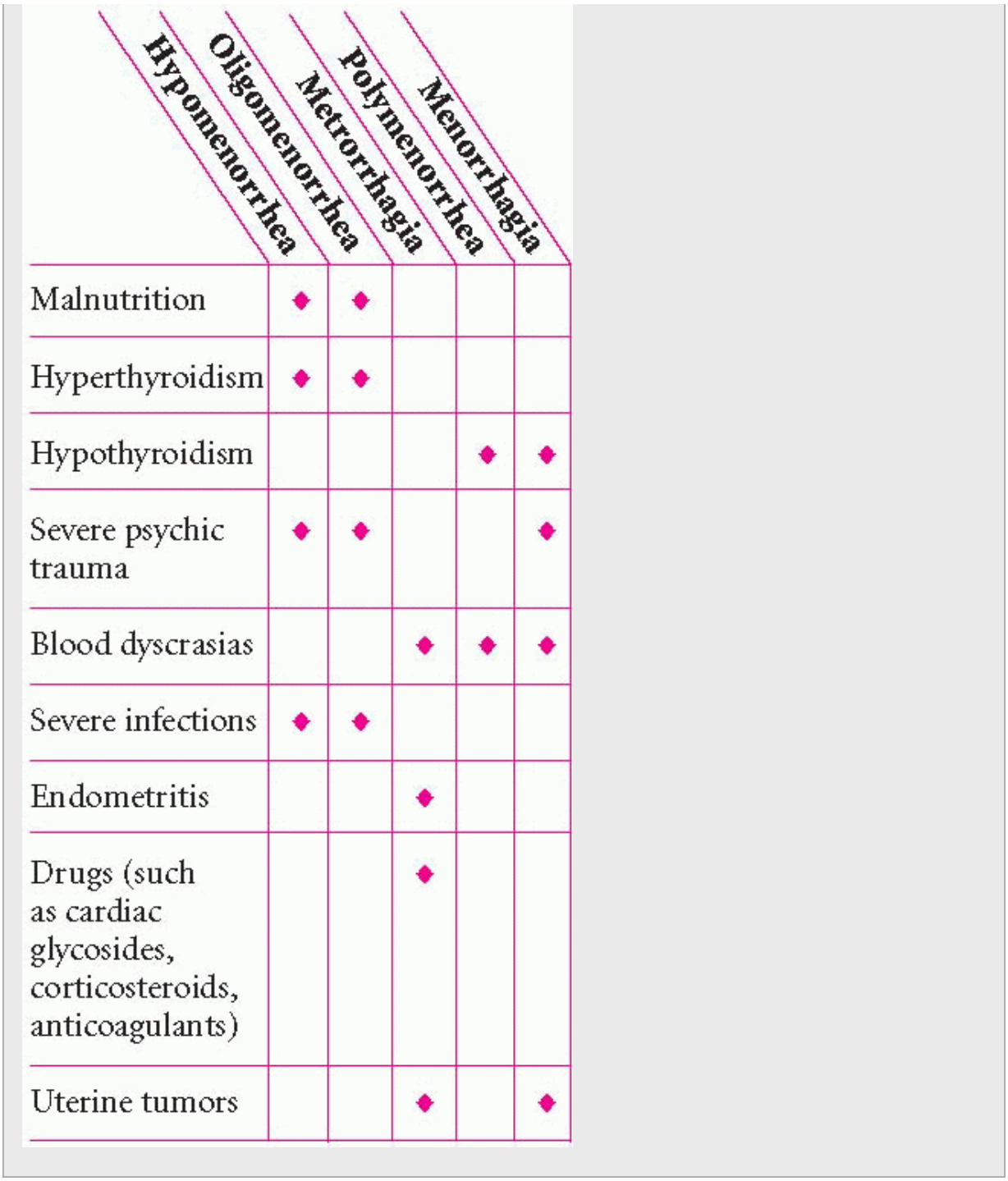
- Serum hormone levels reflect adrenal, pituitary, or thyroid dysfunction.
- Urinary 17-ketosteroids reveal adrenal hyperplasia, hypopituitarism, or polycystic ovarian disease.
- Endometrial sampling rules out malignant tumors and should be performed in all patients with premenopausal bleeding who are older than age 35.

- Pelvic examination, Papanicolaou (Pap) test, and patient history rule out local or malignant causes.
- Complete blood count rules out anemia.

If testing rules out pelvic and hormonal causes of abnormal bleeding, a complete hematologic survey (including platelet count and bleeding time) is appropriate to determine clotting abnormalities.

CAUSES OF ABNORMAL PREMENOPAUSAL BLEEDING

Premenopausal bleeding can take several forms and may be due to a number of causes.



	Hypomenorrhea	Oligomenorrhea	Metrorrhagia	Polymenorrhea	Menorrhagia
Malnutrition	◆	◆			
Hyperthyroidism	◆	◆			
Hypothyroidism				◆	◆
Severe psychic trauma	◆	◆			◆
Blood dyscrasias			◆	◆	◆
Severe infections	◆	◆			
Endometritis			◆		
Drugs (such as cardiac glycosides, corticosteroids, anticoagulants)			◆		
Uterine tumors			◆		◆

Treatment

Treatment depends on the type of bleeding abnormality and its cause. Menstrual irregularity alone may not require therapy unless it interferes with the patient's attempt to achieve or avoid conception or leads to anemia. When it requires treatment, clomiphene induces ovulation.

Electrocautery, chemical cautery, or cryosurgery can remove cervical polyps; dilatation and curettage, uterine polyps. Organic disorders (such as cervical or uterine malignancy) may necessitate hysterectomy, radium or X-ray therapy, or both of these treatments, depending on the disease's site and extent. Of course, anemia and infections require appropriate treatment.

Special considerations

- If the patient complains of abnormal bleeding, tell her to record the dates of the bleeding and the number of tampons or pads used per day. This helps to assess the cyclic pattern and the amount of bleeding.
- Instruct the patient to report abnormal bleeding immediately to help rule out major hemorrhagic disorders, such as occur in abnormal pregnancy.
- Offer reassurance and support. The patient may be particularly anxious about excessive or frequent blood loss and passage of clots. Suggest that she minimize blood flow by avoiding strenuous activity and occasionally lying down with her feet elevated.

PREVENTION

To prevent abnormal bleeding due to organic causes, and for early detection of malignancy, encourage the patient to have a Pap smear and a pelvic examination annually.

Dysfunctional uterine bleeding

Dysfunctional uterine bleeding (DUB) refers to abnormal endometrial bleeding without recognizable organic lesions. The prognosis varies with the cause. DUB is the indication for almost 25% of gynecologic surgical procedures.

Causes and incidence

DUB usually results from an imbalance in the hormonal-endometrial relationship, where persistent and unopposed stimulation of the endometrium by estrogen occurs. Disorders that cause sustained high

estrogen levels are polycystic ovary syndrome, obesity, immaturity of the hypothalamic-pituitary-ovarian mechanism (in postpubertal teenagers), and anovulation (in women in their late 30s or early 40s).

In most cases of DUB, the endometrium shows no pathologic changes. However, in chronic unopposed estrogen stimulation (as from a hormone-producing ovarian tumor), the endometrium may show hyperplastic or malignant changes. DUB occurs in 20% of adolescents and in 40% of women older than age 40.

Complications

- Anemia
- Infertility

Signs and symptoms

DUB usually occurs as metrorrhagia (episodes of vaginal bleeding between menses); it may also occur as hypermenorrhea (heavy or prolonged menses, longer than 8 days) or chronic polymenorrhea (menstrual cycle of less than 18 days). Such bleeding is unpredictable and can cause anemia.

Diagnosis

Diagnostic studies must rule out other causes of excessive vaginal bleeding, such as organic, systemic, psychogenic, and endocrine causes, including certain cancers, polyps, incomplete abortion, pregnancy, and infection.

CONFIRMING DIAGNOSIS

Dilatation and curettage (D & C) and biopsy results confirm the diagnosis by revealing endometrial hyperplasia.

Hemoglobin levels and hematocrit determine the need for blood or iron replacement. Coagulation factors indicate coagulopathies. Human

chorionic gonadotropin is done to determine if there's an ectopic pregnancy or a threatened or incomplete abortion. Thyroid function tests are done to check for hyperthyroidism, which can indicate an ovulation dysfunction. Pelvic ultrasound is done to check the endometrium for fibroids.

Treatment

High-dose estrogen-progestogen combination therapy (hormonal contraceptives), the primary treatment, is designed to control endometrial growth and re-establish a normal cyclic pattern of menstruation. (The patient's age and the cause of bleeding help determine the drug choice and dosage.) In patients older than age 35, endometrial biopsy is necessary before the start of estrogen therapy to rule out endometrial adenocarcinoma. Progestogen therapy is a necessary alternative in some women such as those susceptible to estrogen's adverse effects (thrombophlebitis, for example).

If drug therapy is ineffective, D & C serves as a supplementary treatment, through removal of a large portion of the bleeding endometrium. Also, D & C can help determine the original cause of hormonal imbalance and can aid in planning further therapy. Regardless of the primary treatment, the patient may need iron replacement or transfusions of packed cells or whole blood, as indicated, because of anemia caused by recurrent bleeding.

Special considerations

- Explain the importance of adhering to the prescribed hormonal therapy. If D & C is ordered, explain this procedure and its purpose.
- Stress the need for regular checkups to assess the effectiveness of treatment.

Postmenopausal bleeding

Postmenopausal bleeding is defined as bleeding from the reproductive tract that occurs 1 year or more after cessation of menses. Sites of bleeding include the vulva, vagina, cervix, and endometrium. The prognosis varies with the cause.

Causes and incidence

Postmenopausal bleeding may result from:

- exogenous estrogen, when administration is excessive or prolonged or when small amounts are given in the presence of a hypersensitive endometrium
- endogenous estrogen production, especially when levels are high, as in persons with estrogen-producing ovarian tumor; however, in some persons, even a slight fluctuation in estrogen levels may cause bleeding
- atrophic endometrium due to low estrogen levels
- atrophic vaginitis, usually triggered by trauma during coitus in the absence of estrogen production
- aging, which increases vascular vulnerability by thinning epithelial surfaces, increasing vascular fragility, producing degenerative tissue changes, and decreasing resistance to infections
- cervical or endometrial cancer (more common after age 60)

- adenomatous hyperplasia or atypical adenomatous hyperplasia (usually considered a premalignant lesion).

Signs and symptoms

Vaginal bleeding, the primary symptom, ranges from spotting to outright hemorrhage; its duration also varies. Other symptoms depend on the cause. Excessive estrogen stimulation, for example, may also produce copious cervical mucus; estrogen deficiency may cause vaginal mucosa to atrophy.

Diagnosis

Diagnostic evaluation of the patient with postmenopausal bleeding should include physical examination (especially pelvic examination), a detailed history, standard laboratory tests (such as complete blood

count), and cytologic examination of smears from the cervix and the endocervical canal. An endometrial biopsy or dilatation and curettage (D & C) with hysteroscopy reveals pathologic findings in the endometrium.

Diagnosis must rule out underlying degenerative or systemic disease. For instance, evidence of elevated levels of endogenous estrogen may suggest an ovarian tumor. Before testing for estrogen levels, the patient must stop all sources of exogenous estrogen intake—including face and body creams that contain estrogen—to rule out excessive exogenous estrogen as a cause.

Treatment

Emergency treatment to control massive hemorrhage is rarely necessary, except in advanced cancer. Treatment may include D & C to relieve bleeding. Other therapy varies according to the underlying cause. Estrogen creams and suppositories are usually effective in correcting estrogen deficiency because they're rapidly absorbed. Hysterectomy is indicated for repeated episodes of postmenopausal bleeding from the endometrial cavity. Such bleeding may indicate endometrial cancer.

Special considerations

Obtain a detailed patient history to rule out excessive exogenous estrogen as a cause of bleeding. Ask the patient about use of cosmetics (especially face and body creams), drugs, and other products that may contain estrogen. Discuss the risks and benefits of estrogen replacement therapy with her.

- Provide emotional support. The patient will probably be afraid that the bleeding indicates cancer.

PREVENTION

To prevent disorders that cause postmenopausal bleeding, stress that periodic gynecologic examinations are as important after menopause as they were before.

DISORDERS OF PREGNANCY

Abortion

Abortion is the spontaneous or induced (therapeutic) expulsion of the products of conception from the uterus. Up to 15% of all pregnancies and about 30% of first pregnancies end in spontaneous abortion (miscarriage). At least 75% of miscarriages occur during the first trimester. (See *Types of spontaneous abortion*.)

Causes and incidence

Spontaneous abortion may result from fetal, placental, or maternal factors. Fetal factors, which usually cause such abortions at up to 12 weeks' gestation, include the following:

- defective embryologic development resulting from abnormal chromosome division (most common cause of fetal death)
- faulty implantation of the fertilized ovum
- failure of the endometrium to accept the fertilized ovum.

Placental factors usually cause abortion around the 14th week of gestation, when the placenta takes over the hormone production necessary to maintain the pregnancy. These factors include:

- premature separation of the normally implanted placenta
- abnormal placental implantation.

Maternal factors usually cause abortion between the 11th and 19th week of gestation and include:

- maternal infection, abnormalities of the reproductive organs (especially an incompetent cervix, in which the cervix dilates painlessly in the second trimester)
- endocrine problems, such as thyroid dysfunction or a luteal phase defect
- trauma
- phospholipid antibody disorder

- blood group incompatibility
- drug ingestion (particularly uterotonic agents).

The goal of therapeutic abortion is to preserve the mother's mental or physical health in cases of rape, unplanned pregnancy, or medical conditions such as moderate or severe cardiac dysfunction.

Complications

- Anemia
- Disseminated intravascular coagulation
- Hemorrhage
- Infection

Signs and symptoms

Prodromal signs of spontaneous abortion may include a pink discharge for several days or a scant brown discharge for several weeks before the onset of cramps and increased vaginal bleeding. For a few hours, the cramps intensify and occur more frequently; then the cervix dilates to expel uterine contents. If the entire contents are expelled, cramps and bleeding subside. However, if any contents remain, cramps and bleeding continue.

Diagnosis

Diagnosis of spontaneous abortion is based on clinical evidence of expulsion of uterine contents, pelvic examination, and laboratory studies. Human chorionic gonadotropin (hCG) in the blood or urine confirms pregnancy; decreased hCG levels suggest spontaneous abortion or tubal pregnancy. Pelvic examination determines the uterus' size and whether this size is consistent with the pregnancy's length. Tissue histology indicates evidence of products of conception. Laboratory tests reflect decreased hemoglobin levels and hematocrit due to blood loss. However, blood

loss is rarely excessive in spontaneous abortion. It's critical that ectopic

pregnancy be ruled out in a woman who's pregnant with vaginal bleeding.

TYPES OF SPONTANEOUS ABORTION

- *Threatened abortion*: Bloody vaginal discharge occurs during the first half of pregnancy. Approximately 20% of pregnant women have vaginal spotting or actual bleeding early in pregnancy; of these, about 50% abort.
- *Inevitable abortion:* Membranes rupture and the cervix dilates. As labor continues, the uterus expels the products of conception.
- *Incomplete abortion:* The uterus retains part or all of the placenta. Before the 10th week of gestation, the fetus and placenta usually are expelled together; after the 10th week, separately. Because part of the placenta may adhere to the uterine wall, bleeding continues. Hemorrhage is possible because the uterus doesn't contract and seal the large vessels that fed the placenta.
- *Complete abortion:* The uterus passes all the products of conception. Minimal bleeding usually accompanies complete abortion because the uterus contracts and compresses maternal blood vessels that feed the placenta.
- *Missed abortion:* The uterus retains the products of conception for 2 months or more after the fetus' death. Uterine growth ceases; uterine size may even seem to decrease. Prolonged retention of the dead products of conception may cause coagulation defects, such as disseminated intravascular coagulation, usually after at least 1 month in utero.
- *Habitual abortion:* Spontaneous loss of three or more consecutive pregnancies constitutes habitual abortion.

- *Septic abortion*: Infection accompanies abortion. This may occur with spontaneous abortion but usually results from an illegal abortion.

Treatment

An accurate evaluation of uterine contents is needed before a plan of treatment can be formulated. The progression of spontaneous abortion can't be prevented, except possibly in cases caused by an incompetent cervix. The patient must be hospitalized to control severe hemorrhage. If bleeding is severe, a transfusion with packed red blood cells or whole blood is required. Initially, I.V. administration of oxytocin stimulates uterine contractions (if given above 20 weeks' gestation—receptors are absent before this gestational age). If any remnants remain in the uterus, dilatation and curettage or dilatation and evacuation (D & E) should be performed.

D & E is also performed in first- and second-trimester therapeutic abortions. In second-trimester therapeutic abortions, the insertion of a prostaglandin vaginal suppository induces labor and the expulsion of uterine contents. When performed competently, second-trimester D & E is a very safe procedure and allows for termination of pregnancy without the need for a lengthy induction of labor. Early first-trimester abortion may also be accomplished pharmacologically with mifepristone (RU-486) an antiprogestin, followed by a dose of a prostaglandin analogue 2 days later, or surgically, using vacuum aspiration.

After an abortion, spontaneous or induced, an Rh-negative female with a negative indirect Coombs' test should receive Rh_o(D) immune globulin (human) to prevent future Rh isoimmunization.

In a habitual aborter, spontaneous abortion can result from an incompetent cervix (a clinical retrospective diagnosis suggested by a history of previous second-trimester losses accompanied by membrane rupture or painless cervical dilation). Treatment involves surgical reinforcement of the cervix (cerclage) 12 to 24 weeks after the last menstrual period. A few weeks before the estimated delivery date, the sutures are removed, and the patient awaits the onset of labor. An alternative procedure is to leave the sutures in place and to deliver the

infant by cesarean birth. Cerclage hasn't been shown to be more effective than bed rest.

Special considerations

Elective abortion is a controversial issue. The decision to have an elective abortion is a personal decision that requires competent counseling. Many women believe they can't share their feelings with others, therefore it's important for a woman contemplating an abortion to examine her existing support system and identify those people capable of helping her through a difficult time. A reputable provider or clinic where the woman can obtain adequate counseling regarding all options for pregnancy resolution, have the procedure performed, and obtain support and follow-up care should be identified ahead during the decisionmaking process.

Before possible abortion:

- Be sure to inform the patient who desires an elective abortion of all the available alternatives. She needs to know what the procedure involves, what the risks are, and what to expect during and after the procedure, both emotionally and physically. Be sure to ascertain whether the patient is comfortable with her decision to have an elective abortion.
- The patient *shouldn't* have bathroom privileges, because she may expel uterine contents without knowing it. After she uses the bedpan, inspect the contents carefully for intrauterine material.

After spontaneous or elective abortion:

- Note the amount, color, and odor of vaginal bleeding. Save all the pads the patient uses, for evaluation.
- Administer analgesics and oxytocin, as ordered.
- Provide good perineal care.
- Obtain vital signs as indicated.
- Monitor urine output.

Care of the patient who has had a spontaneous abortion includes emotional support and counseling during the grieving process. Stress to

the patient that she isn't responsible for a spontaneous abortion, as this generally can't be prevented. Encourage the patient and her partner to express their feelings. Some couples may want to talk to a clergy member or, depending on their religion, may wish to have the fetus baptized.

The patient who has had a therapeutic abortion also benefits from support. Encourage her to verbalize her feelings. Remember, she may feel ambivalent about the procedure; intellectual and emotional acceptance of abortion aren't the same. If you identify an inappropriate coping response, refer the patient for professional counseling.

To prepare the patient for discharge:

- Tell the patient to expect vaginal bleeding or spotting and to report bleeding that lasts longer than 8 to 10 days or excessive, bright-red blood immediately.
- Advise the patient to watch for signs of infection, such as a temperature higher than 100.5° F (38° C) and foul-smelling vaginal discharge.
- Encourage the gradual increase of daily activities to include whatever tasks the patient feels comfortable doing, as long as these activities don't increase vaginal bleeding or cause fatigue. Most patients return to work after 24 hours.
- Urge 1 to 2 weeks' abstinence from intercourse, and encourage the use of a contraceptive.
- Instruct the patient to avoid using tampons for 1 to 2 weeks.
- Tell the patient to see her physician in 2 to 4 weeks for a follow-up examination.

PREVENTION

- *To help prevent future elective abortions, make contraceptive information available. An educated patient motivated to use contraception has less need for elective abortion.*

- *To minimize the risk of future spontaneous abortions, emphasize to the pregnant woman the importance of good nutrition and the need to avoid alcohol, cigarettes, and drugs. If the patient has a history of habitual spontaneous abortions, suggest that she and her partner have thorough examinations. For the woman, this includes premenstrual endometrial biopsy, a hormone assessment (estrogen; progesterone; and thyroid, follicle-stimulating, and luteinizing hormones), and hysterosalpingography and laparoscopy to detect anatomic abnormalities. Genetic counseling may also be advised.*

Ectopic pregnancy

Ectopic pregnancy is the implantation of the fertilized ovum outside the uterine cavity. The most common site is the fallopian tube (more than 95% of ectopic implantations occur in the fimbria, ampulla, or isthmus), but other possible sites include the interstitium, tubo-ovarian ligament, ovary, abdominal viscera, and internal cervical os. (See *Implantation sites of ectopic pregnancy*, page 1184.) The prognosis is good with prompt diagnosis, appropriate surgical intervention, and control of bleeding; rarely, in cases of abdominal implantation, the fetus may survive to term. Usually, a subsequent intrauterine pregnancy is achieved.

Causes and incidence

Conditions that prevent or retard the fertilized ovum's passage through the fallopian tube and into the uterine cavity include:

- diverticula, the formation of blind pouches that cause tubal abnormalities
- endometriosis, the presence of endometrial tissue outside the lining of the uterine cavity
- endosalpingitis, an inflammatory reaction that causes folds of the tubal mucosa to agglutinate, narrowing the tube

- pelvic inflammatory disease (PID), an infection of the oviducts and ovaries with adjacent tissue involvement
- previous surgery (tubal ligation or resection, or adhesions from previous abdominal or pelvic surgery)
- tumors pressing against the tube.

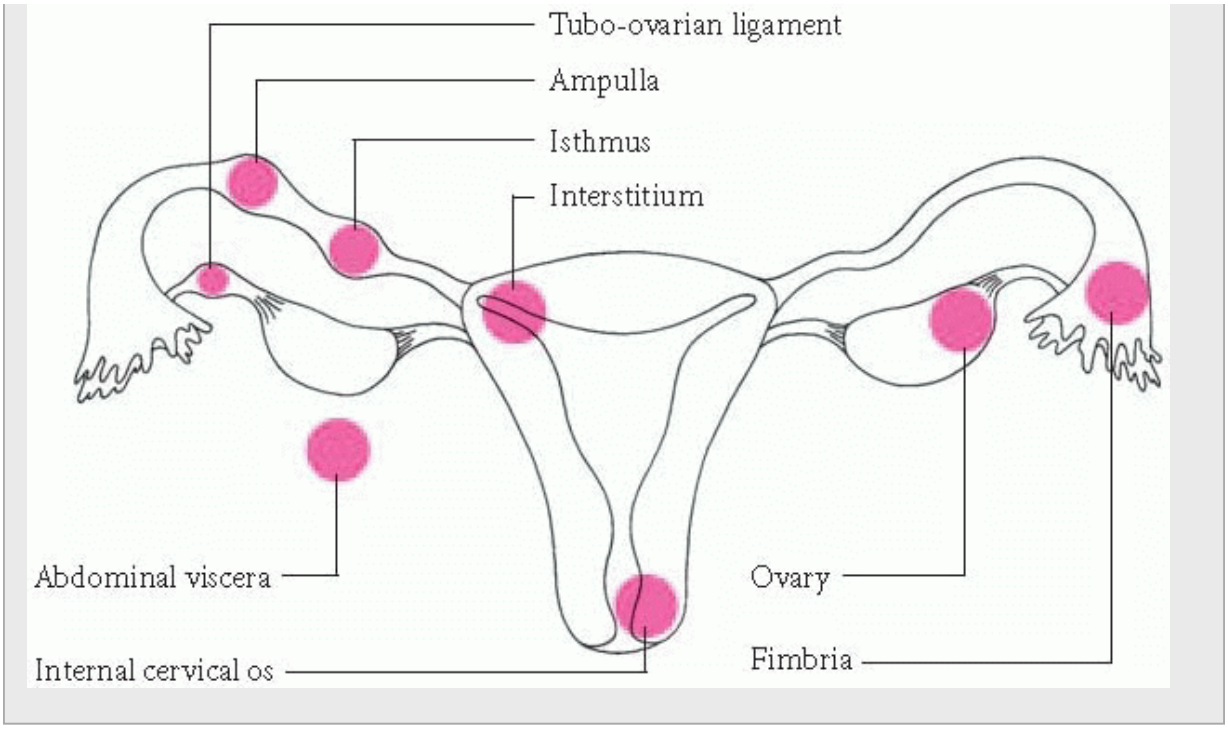
Ectopic pregnancy may result from congenital defects in the reproductive tract or ectopic endometrial implants in the tubal mucosa. The increased prevalence of sexually transmitted tubal infection may also be a factor. Other risk factors include intrauterine device use, multiple previous elective abortions, smoking, and advanced age. It occurs in 1 in 40 to 100 pregnancies.

Complications

- Hemorrhage
- Infertility
- Peritonitis
- Shock

IMPLANTATION SITES OF ECTOPIC PREGNANCY

In 90% of patients with ectopic pregnancy, the ovum implants in the fallopian tube, either in the fimbria, ampulla, or isthmus. Other possible sites include the interstitium, tubo-ovarian ligament, ovary, abdominal viscera, and internal cervical os.



Signs and symptoms

Ectopic pregnancy sometimes produces symptoms of normal pregnancy or no symptoms other than mild abdominal pain, making diagnosis difficult. Characteristic clinical effects after fallopian tube implantation include amenorrhea or abnormal menses, followed by slight vaginal bleeding, and unilateral pelvic pain over the mass. Rupture of the tube causes life-threatening complications, including hemorrhage, shock, and peritonitis. The patient experiences sharp lower abdominal pain, possibly radiating to the shoulders and neck, often precipitated by activities that increase abdominal pressure, such as a bowel movement; she feels extreme pain upon motion of the cervix and palpation of the adnexa during a pelvic examination.

Diagnosis

Clinical features, patient history, and the results of a pelvic examination suggest ectopic pregnancy. The following tests help confirm it:

- Serum pregnancy test shows presence of human chorionic gonadotropin.

- Real time ultrasonography determines extrauterine pregnancy (performed if serum pregnancy test is positive). Magnetic resonance imaging is a useful adjunct to ultrasound if an unusual ectopic location is suspected.
- In culdocentesis, fluid is aspirated from the pouch of Douglas through the posterior vaginal fornix to detect free or nonclotting blood in the peritoneum (sometimes performed if ultrasonography fails to detect a gestational sac in the uterus).
- Laparoscopy or laparotomy is used to diagnose as well as to treat an ectopic pregnancy by either removal of the tube (salpingectomy) or removal of the pregnancy with preservation of the tube (salpingostomy).

Decreased hemoglobin levels and hematocrit due to blood loss support the diagnosis. Differential diagnosis must rule out uterine abortion, appendicitis, ruptured corpus luteum cyst, salpingitis, and torsion of the ovary.

Treatment

If culdocentesis is positive or the patient has peritoneal signs consistent with a surgical abdomen, laparoscopy and laparotomy are indicated. The ovary is preserved as a rule; however, ovarian pregnancy may necessitate oophorectomy. Interstitial pregnancy may rarely require hysterectomy; abdominal pregnancy requires a laparotomy to remove the fetus, except in rare cases, when the fetus survives to term or calcifies undetected in the abdominal cavity.

Supportive treatment includes transfusion with whole blood or packed red cells to replace excessive blood loss, administration of broad-spectrum antibiotics I.V. for septic infection, and administration of supplemental iron by mouth or I.M.

Methotrexate I.M. is also a therapeutic option in stable patients, avoiding surgery in most cases.

Special considerations

Patient care measures include careful monitoring and assessment of vital signs and vaginal bleeding, preparing the patient with excessive blood loss for emergency surgery, and providing blood replacement and emotional support and reassurance.

- Record the pain's location and character, and administer analgesics as ordered. (Remember, however, that analgesics may mask the symptoms of intraperitoneal rupture of the ectopic pregnancy.)
- Check the amount, color, and odor of vaginal bleeding. Ask the patient the date of her last menstrual period and to describe this period's character.
- Observe for signs of pregnancy (enlarged breasts, soft cervix).
- Provide a quiet, relaxing environment, and encourage the patient to freely express her feelings of fear, loss, and grief.

PREVENTION

- *To prevent diseases of the fallopian tube, advise prompt treatment of pelvic infections. Tell patients who have undergone surgery involving the fallopian tubes or those with confirmed PID that they're at increased risk for ectopic pregnancy.*
- *Tell the patient who's vulnerable to ectopic pregnancy to delay using an intrauterine device until she decides not to have more children.*

Hyperemesis gravidarum

Unlike the transient nausea and vomiting normally experienced between the 6th and 12th weeks of pregnancy, hyperemesis gravidarum is severe and unremitting nausea and vomiting that persists after the first trimester. If untreated, it produces substantial weight loss; starvation; dehydration, with subsequent fluid and electrolyte imbalance (hypokalemia); and acid-base disturbances (acidosis and alkalosis). This syndrome occurs in about 1 in 200 pregnancies. The prognosis is good with appropriate treatment.

Causes and incidence

Although its cause is unknown, hyperemesis gravidarum often affects pregnant females with conditions that produce high levels of human chorionic gonadotropin, such as hydatidiform mole or multiple pregnancies. Its other possible causes include pancreatitis (elevated serum amylase levels are common), biliary tract disease, drug toxicity, inflammatory obstructive bowel disease, and vitamin deficiency (especially of B_6). In some patients, it may be related to psychological factors such as ambivalence toward pregnancy.

This disorder occurs in 1 of every 200 pregnancies. The incidence increases in molar and multiple pregnancies.

Complications

- Acid-base disturbances
- Dehydration
- Hypokalemia
- Weight loss

Signs and symptoms

The cardinal symptoms of hyperemesis gravidarum are unremitting nausea and vomiting. The vomitus initially contains undigested food and mucus as well as small amounts of bile; later, only bile and mucus; and finally, blood and material that resembles coffee grounds. Persistent vomiting causes substantial weight loss and eventual emaciation. Associated effects may include pale, dry, waxy, and possibly jaundiced skin; subnormal or elevated temperature; rapid pulse; a fetid, fruity breath odor from acidosis; and central nervous system symptoms, such as confusion, delirium, headache, lassitude, stupor and, possibly, coma.

Diagnosis

Diagnosis depends on a history of uncontrolled nausea and vomiting that persists beyond the first trimester, evidence of substantial weight loss, and other characteristic clinical features. Serum analysis shows decreased protein, chloride, sodium, and potassium levels, and increased blood urea nitrogen levels. Other laboratory tests reveal ketonuria, slight proteinuria, and elevated hemoglobin and white blood cell levels. Diagnosis must rule out other conditions with similar clinical effects.

Treatment

Hyperemesis gravidarum may necessitate hospitalization to correct electrolyte imbalance and prevent starvation. I.V. infusions maintain nutrition until the patient can tolerate oral feedings. She progresses slowly to a clear liquid diet, then a full liquid diet and, finally, small, frequent meals of high-protein solid foods. A midnight snack helps stabilize blood glucose levels; vitamin B supplements help correct vitamin deficiency.

When vomiting stops and electrolyte balance has been restored, the pregnancy usually continues without recurrence of hyperemesis gravidarum. Most patients feel better as they begin to regain normal weight, but some continue to vomit throughout the pregnancy, requiring extended treatment. If appropriate, some patients may benefit from consultations with clinical nurse specialists, psychologists, or psychiatrists.

Special considerations

- Encourage the patient to eat. Suggest dry foods and decreased liquid intake during meals.
- Instruct the patient to remain upright for 45 minutes after eating to decrease reflux.
- Provide reassurance and a calm, restful atmosphere. Encourage the patient to discuss her feelings regarding her pregnancy.
- Before discharge, provide good nutritional counseling.

Gestational hypertension

Gestational hypertension, also known as *pregnancy-induced hypertension,* is a potentially life-threatening disorder that usually develops late in the second trimester or in the third trimester. *Preeclampsia,* the nonconvulsive form of gestational hypertension, may be mild or severe. *Eclampsia* is the convulsive form of gestational hypertension.

Causes and incidence

The cause of gestational hypertension isn't known, but geographic, ethnic, racial, nutritional, immunologic, and familial factors and pre-existing vascular disease may contribute to its development. Age is also a factor. Primiparas who are older than age 35 are at higher risk for preeclampsia.

Preeclampsia develops in about 7% of pregnancies. Incidence is significantly higher in low socioeconomic groups. About 5% of females with preeclampsia develop eclampsia; of these, about 15% die from eclampsia or its complications. Fetal mortality is high because of the increased incidence of premature delivery and uteroplacental insufficiency.

Complications

- Abruptio placentae
- Intrauterine growth retardation
- Placental infarcts

Signs and symptoms

Mild preeclampsia generally produces the following clinical effects: hypertension, proteinuria (less than 5 g/24 hours), generalized edema, and sudden weight gain of more than 3 lb (1.4 kg) per week during the second trimester or more than 1 lb (0.5 kg) a week during the third trimester.

Severe preeclampsia is marked by increased hypertension and proteinuria, eventually leading to the development of oliguria. Hemolysis, elevated liver enzymes, and low platelets (the HELLP

syndrome) is a severe variant. Other symptoms that may indicate worsening preeclampsia include blurred vision due to retinal arteriolar spasms, epigastric pain or heartburn, and severe frontal headache.

In eclampsia, all the clinical manifestations of preeclampsia are magnified and are associated with seizures and, possibly, coma, premature labor, stillbirth, renal failure, and hepatic damage.

Diagnosis

The following findings suggest preeclampsia:

- elevated blood pressure readings: 140 mm Hg systolic, measured on two occasions, 6 hours apart; 90 mm Hg diastolic, measured on two occasions, 6 hours apart
- proteinuria: at least 300 mg/24 hours.

The following findings suggest severe preeclampsia:

- elevated blood pressure readings: 160/110 mm Hg or higher on two occasions, 6 hours apart, on bed rest
- increased proteinuria: 5 g/24 hours or more
- presence of pulmonary edema
- ultrasound: may reveal oligohydraminos
- oliguria: urine output less than or equal to 400 ml/24 hours.

Seizures strongly suggest eclampsia. Rarely, ophthalmoscopic examination may reveal vascular spasm, papilledema, retinal edema or detachment, and arteriovenous nicking or hemorrhage.

Real-time ultrasonography, stress and nonstress tests, and biophysical profiles evaluate fetal status. In the stress test, oxytocin stimulates contractions; fetal heart tones are then monitored electronically. In the nonstress test, fetal heart tones are monitored electronically during periods of fetal activity, without oxytocin stimulation. Electronic monitoring reveals stable or increased fetal heart tones during periods of fetal activity.

Ultrasonography aids evaluation of fetal health by assessing fetal breathing movements, gross body movements, fetal tone, reactive fetal heart rate, and qualitative amniotic fluid volume.

Treatment

Therapy for preeclampsia is designed to halt the disorder's progress—specifically, the early effects of eclampsia, such as seizures, residual hypertension, and renal shutdown—and to ensure fetal survival. Some physicians advocate the prompt induction of labor, especially if the patient is near term; others follow a more conservative approach. Therapy may include complete bed rest to increase placental perfusion, reduce hypertension, and evaluate response to therapy. Antihypertensive therapy doesn't alter the potential for developing eclampsia. Diuretics aren't appropriate during pregnancy.

If the patient's blood pressure fails to respond to bed rest and sedation and persistently rises above 160/100 mm Hg, or if central nervous system irritability increases, magnesium sulfate may produce general sedation, promote diuresis, and prevent seizures. Cesarean birth or oxytocin induction may be required to terminate the pregnancy.

ALERT

Emergency treatment of eclamptic seizures consists of immediate administration of magnesium sulfate (I.V. drip), oxygen administration, and electronic fetal monitoring.

After the seizures subside and the patient's condition stabilizes, delivery should proceed with induction of labor or cesarean birth, depending upon the circumstances.

Adequate nutrition, good prenatal care, and control of pre-existing hypertension during pregnancy decrease the incidence and severity of preeclampsia. Early recognition and prompt treatment of preeclampsia can prevent progression to eclampsia.

Special considerations

- Monitor regularly for changes in blood pressure, pulse rate, respiration, fetal heart tones, vision, level of consciousness, and deep tendon reflexes and for headache unrelieved by medication. Report changes immediately. Assess these signs before administering medications. Absence of patellar reflexes may indicate magnesium sulfate toxicity.
- Assess fluid balance by measuring intake and output and by checking daily weight.
- Observe for signs of fetal hypoxemia by closely monitoring the results of stress and nonstress tests.
- Instruct the patient to lie in a left lateral position to increase venous return, cardiac output, and renal blood flow.

ALERT

Keep emergency resuscitative equipment and drugs available in case of seizures and cardiac or respiratory arrest. Also keep calcium gluconate at the bedside because it counteracts magnesium sulfate's toxic effects.

- To protect the patient from injury, maintain seizure precautions. Don't leave an unstable patient unattended.
- Assist with emergency medical treatment for the convulsive patient. Provide a quiet, darkened room until the patient's condition stabilizes, and enforce absolute bed rest. Monitor administration of magnesium sulfate; give oxygen as ordered. Don't administer anything by mouth. Insert an indwelling urinary catheter for accurate measurement of intake and output.
- Inform the patient about the tests that are done to evaluate fetal status. The baby's welfare is of prime concern to the parents.
- Provide emotional support for the patient and her family. If the patient's condition necessitates premature delivery, point out that infants of mothers with gestational hypertension are usually small for gestational age but sometimes fare better than other premature

babies of the same weight, possibly because they have developed adaptive responses to stress in utero.

Hydatidiform mole

Hydatidiform mole is an uncommon chorionic tumor of the placenta. Its early signs—amenorrhea and uterine enlargement—mimic normal pregnancy; however, it eventually causes vaginal bleeding. With prompt diagnosis and appropriate treatment, the prognosis is excellent; however, about 15% of patients with hydatidiform mole develop gestational trophoblastic neoplasm. Recurrence is possible in about 2% of cases.

Causes and incidence

The cause of hydatidiform mole is unknown. Abnormal genetic events upon fusion of the oocyte with one or more sperm play a role. It occurs in 1 in 1,500 to 2,000 pregnancies, most commonly in women older than age 45. Incidence is highest in Asian women.

Complications

- Anemia
- Hemorrhage
- Infection
- Spontaneous abortion
- Uterine rupture

Signs and symptoms

The early stages of a pregnancy in which a hydatidiform mole develops typically seem normal, except that the uterus may grow more rapidly than usual. The first obvious signs of trouble—absence of fetal heart tones, vaginal bleeding (from spotting to hemorrhage), and lower abdominal cramps—mimic those of spontaneous abortion. The blood may contain hydatid vesicles; hyperemesis is possible, and signs and symptoms of preeclampsia are also possible. Other complications of

hydatidiform mole may include anemia, infection, trophoblast embolism, uterine rupture, and choriocarcinoma.

Diagnosis

Persistent bleeding and an abnormally enlarged uterus suggest hydatidiform mole. Diagnosis is based on a typical sonographic "snowstorm" pattern. Confirmation of hydatidiform mole requires dilatation and curettage.

The following findings also support a diagnosis of hydatidiform mole:

- absence of a gestational sac, by ultrasound assessment, or absence of fetal heart tones, by Doppler, after 12 weeks
- pregnancy test showing elevated human chorionic gonadotropin (hCG) serum levels greater than 100,000 IU
- development of preeclampsia prior to 20 weeks
- uterine size greater than estimated gestational size
- vaginal bleeding.

Treatment

Hydatidiform mole necessitates uterine evacuation via suction curettage. Oxytocin I.V. may be used to promote uterine contractions.

Postoperative treatment varies, depending on the amount of blood lost and complications. If no complications develop, hospitalization is usually brief, and normal

activities can be resumed quickly, as tolerated.

Because of the possibility of choriocarcinoma development following hydatidiform mole, scrupulous follow-up care is essential. HCG levels initially are checked on a weekly basis, until they're repeatedly negative; then on a monthly basis for one year. A baseline chest X-ray is used to rule out pulmonary involvement, and a head computed tomography scan may be performed to rule out cranial metastases.

Another pregnancy should be postponed until at least 1 year after hCG levels return to normal.

Special considerations

- Preoperatively, observe for signs of complications, such as hemorrhage and uterine infection, and vaginal passage of hydatid vesicles. Save any expelled tissue for laboratory analysis.
- Postoperatively, monitor vital signs, especially blood pressure, and check blood loss.
- Provide teaching and emotional support to the patient and her family. Encourage the patient to express her feelings, and help her through the grieving process for her lost infant.
- Instruct the patient to promptly report new symptoms (for example, hemoptysis, cough, suspected pregnancy, nausea, vomiting, and vaginal bleeding).
- Stress the need for regular follow-up by hCG and chest X-ray monitoring, for early detection of possible malignant changes.
- Explain to the patient that she must use contraceptives to prevent pregnancy for at least 1 year after hCG levels return to normal, and regular ovulation and menstrual cycles are re-established.

Placenta previa

In placenta previa, the placenta is implanted in the lower uterine segment, where it encroaches on the internal cervical os. Placenta previa with life-threatening maternal bleeding typically necessitates termination of the pregnancy. Maternal prognosis is good if hemorrhage can be controlled; fetal prognosis depends on gestational age and amount of blood lost. Anemia may be managed by blood transfusion to permit the pregnancy to continue in utero.

Causes and incidence

In placenta previa, the placenta may cover all (total, complete, or central), part (partial or incomplete), or a fraction (marginal or low-lying) of the internal cervical os. The degree of placenta previa depends

largely on the extent of cervical dilation at the time of examination because the dilating cervix gradually uncovers the placenta. (See *Three types of placenta previa,* page 1190.) Although the specific cause of placenta previa is unknown, factors that may affect the site of the placenta's attachment to the uterine wall include:

- defective vascularization of the decidua
- multiple pregnancy (the placenta requires a larger surface for attachment)
- previous uterine surgery
- multiparity
- advanced maternal age.

In placenta previa, the uterus' lower segment fails to provide as much nourishment as the fundus. The placenta tends to spread out, seeking the blood supply it needs, and becomes larger and thinner than normal. Eccentric insertion of the umbilical cord often develops, for unknown reasons. Hemorrhage occurs as the internal cervical os effaces and dilates, tearing the uterine vessels.

This disorder, one of the most common causes of bleeding during the second half of pregnancy, occurs in about 1 in 500 pregnancies, and more commonly in multigravidas than in primigravidas.

Complications

- Hemorrhage
- Infection
- Premature birth
- Shock
- Thromboembolism

Signs and symptoms

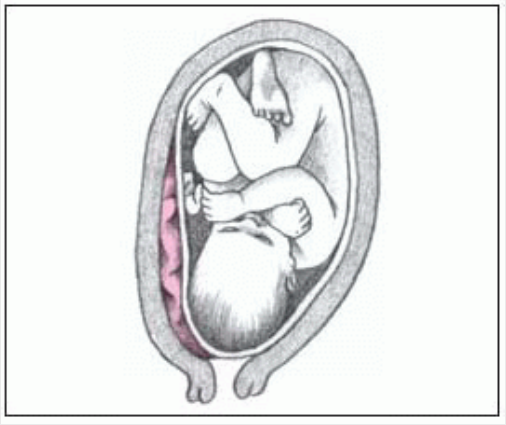
Placenta previa usually produces painless third-trimester bleeding (often the first complaint). Various malpresentations occur because of the placenta's location and interfere with proper descent of the fetal head.

(The fetus remains active, however, with good heart tones.)
Complications of

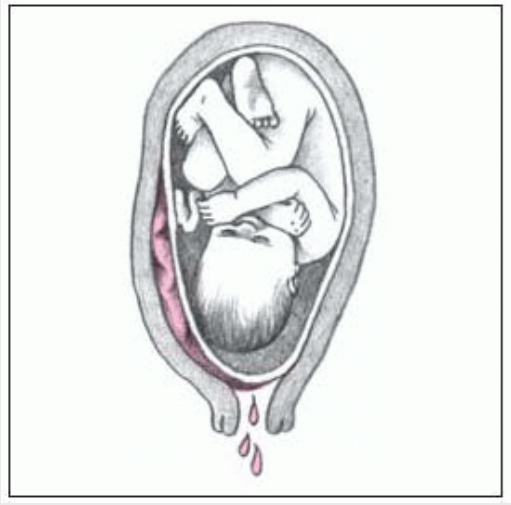
placenta previa include shock or maternal and fetal death.

THREE TYPES OF PLACENTA PREVIA

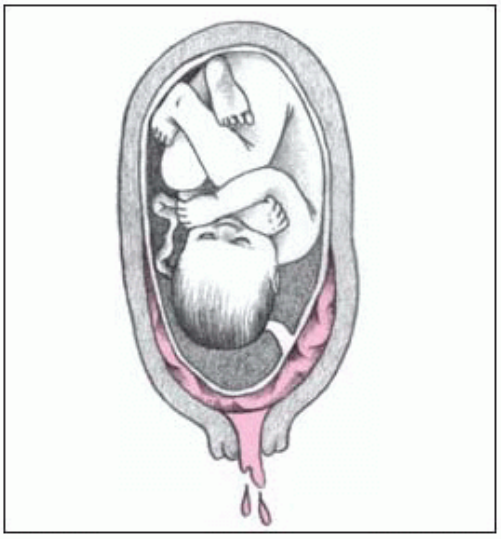
Low marginal implantation—A small placental edge can be felt through the internal os.



Partial placenta previa—The placenta partially caps the internal os.



Total placenta previa—The internal os is covered entirely.



Diagnosis

Special diagnostic measures that confirm placenta previa include:

- transvaginal ultrasound scanning for placental position
- pelvic examination (under a double setup because of the likelihood of hemorrhage), performed only immediately before delivery to confirm the diagnosis. In most cases, only the cervix is visualized.

Digital examination should be deferred in any pregnant woman in the third trimester with vaginal bleeding, until ultrasound rules out placenta previa.

Treatment

Treatment of placenta previa is designed to assess, control, and restore blood loss; to deliver a viable infant; and to prevent coagulation disorders. Immediate therapy includes starting an I.V. line using a large-bore catheter; drawing blood for hemoglobin levels and hematocrit as well as type and crossmatching; initiating external electronic fetal monitoring; monitoring maternal blood pressure, pulse rate, and respirations; and assessing the amount of vaginal bleeding.

If the fetus is premature, following determination of the degree of placenta previa and necessary fluid and blood replacement, treatment consists of careful observation to allow the fetus more time to mature.

If clinical evaluation confirms complete placenta previa, the patient may be hospitalized because of the increased risk of hemorrhage. As soon as the fetus is sufficiently mature, or in case of intervening severe hemorrhage, immediate delivery by cesarean birth may be necessary. Vaginal delivery is considered only when the bleeding is minimal and the placenta previa is marginal, or when the labor is rapid. Because of the possibility of fetal blood loss through the placenta, a pediatric team should be on hand during such delivery to immediately assess and treat neonatal shock, blood loss, and hypoxia.

Complications of placenta previa necessitate appropriate and immediate intervention.

Special considerations

- If the patient shows active bleeding because of placenta previa, a primary nurse should be assigned for continuous monitoring of maternal blood pressure, pulse rate, respirations, central venous pressure, intake and output, amount of vaginal bleeding, and fetal heart tones. Electronic monitoring of fetal heart tones is recommended.
- Prepare the patient and her family for a possible cesarean birth and the birth of a premature infant. Thoroughly explain postpartum care, so the patient and her family know what measures to expect.
- Provide emotional support during labor. Because of the infant's prematurity, the patient may not be given analgesics, so labor pain may be intense. Reassure her of her progress throughout labor and keep her informed of the fetus' condition. Although neonatal death is a possibility, continued monitoring and prompt management reduce this prospect.
- Placenta previa, especially in women who have had one or more cesarean births, is associated with placenta accreta, a dangerous condition in which the placenta grows into the myometrium.

Abruptio placentae

In abruptio placentae, also called *placental abruption,* the placenta separates from the uterine wall prematurely, usually after the 20th week of gestation, producing hemorrhage. Abruptio placentae is a common cause of bleeding during the second half of pregnancy. Firm diagnosis, in the presence of heavy maternal bleeding, may necessitate termination of pregnancy. Fetal prognosis depends on the gestational age and amount of blood lost; maternal prognosis is good if hemorrhage can be controlled.

Causes and incidence

The cause of abruptio placentae is often unknown. Predisposing factors include trauma, such as a direct blow to the uterus, placental site bleeding from a needle puncture during amniocentesis, diabetes, chronic or gestational hypertension (which raises pressure on the placenta's maternal side), multiparity, smoking, and cocaine abuse. If abruptio placentae occurred in previous pregnancies, the chance for recurrence is 10% to 20%.

In abruptio placentae, blood vessels at the placental bed rupture spontaneously owing to a lack of resiliency or to abnormal changes in uterine vasculature. Hypertension complicates the situation, as does an enlarged uterus, which can't contract sufficiently to seal off the torn vessels. Consequently, bleeding continues unchecked, possibly shearing off the placenta partially or completely. Typically, such bleeding is external or marginal (in about 80% of patients) if a peripheral portion of the placenta separates from the uterine wall; it is internal or concealed (in about 20%) if the central portion of the placenta becomes detached and the still-intact peripheral portions trap the blood. As blood enters the muscle fibers, complete relaxation of the uterus becomes impossible, increasing uterine tone and irritability. If bleeding into the muscle fibers is profuse, the uterus turns blue or purple, and the accumulated blood prevents its normal contractions after delivery (Couvelaire uterus, or uteroplacental apoplexy).

Complications

- Disseminated intravascular coagulopathy
- Fetal hypoxia

- Shock

Signs and symptoms

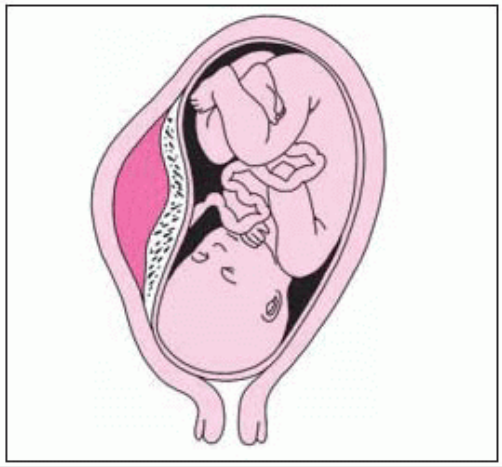
Abruptio placentae produces a wide range of clinical effects, depending on the extent of placental separation and the amount of blood lost from maternal circulation. (See *Degrees of placental separation in abruptio placentae,* page 1192.) Mild abruptio placentae (marginal separation) develops gradually and produces mild to moderate bleeding, vague lower abdominal discomfort, mild to moderate abdominal tenderness, and uterine irritability. Fetal heart tones remain strong and regular.

Moderate abruptio placentae (about 50% placental separation) may develop gradually or abruptly and produces continuous abdominal pain, moderate dark red vaginal bleeding, a tender uterus that remains

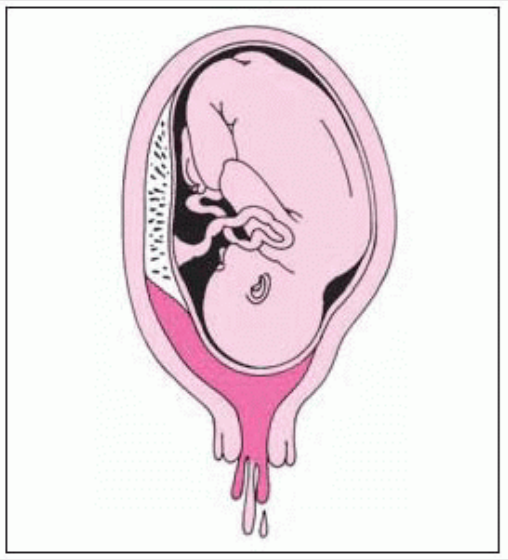
firm between contractions, barely audible or irregular and bradycardiac fetal heart tones and, possibly, signs of shock. Labor usually starts within 2 hours and often proceeds rapidly.

DEGREES OF PLACENTAL SEPARATION IN ABRUPTIO PLACENTAE

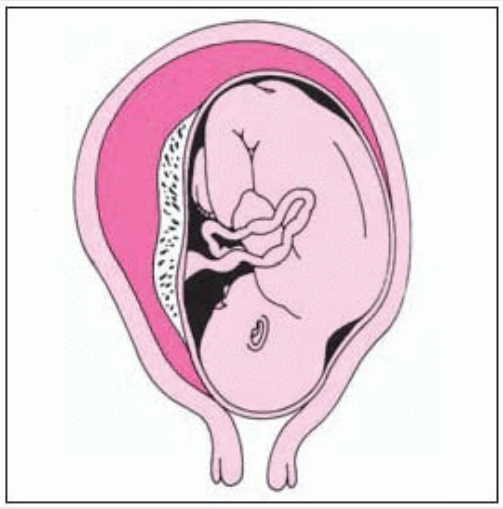
Mild separation with internal bleeding between the placenta and uterine wall.



Moderate separation with external hemorrhage through the vagina.



Severe separation.



Severe abruptio placentae (70% placental separation) develops abruptly and causes agonizing, unremitting uterine pain (described as tearing or knifelike); a boardlike, tender uterus; moderate vaginal bleeding; rapidly progressive shock; and absence of fetal heart tones.

In addition to hemorrhage and shock, complications of abruptio placentae may include renal failure, disseminated intravascular coagulation (DIC), and maternal and fetal death.

Diagnosis

Diagnostic measures for abruptio placentae include observation of clinical features, speculum examination, and ultrasonography to rule out placenta previa. Decreased hemoglobin (Hb) levels and platelet counts support the diagnosis. Periodic assays for fibrin split products aid in monitoring the progression of abruptio placentae and detect the development of DIC.

Treatment

Treatment of abruptio placentae is designed to assess, control, and restore the amount of blood lost; to deliver a viable infant; and to prevent coagulation disorders. Immediate measures for abruptio placentae include starting I.V. infusion (via large-bore catheter) of appropriate fluids (lactated Ringer's solution) to combat hypovolemia; placement of a central venous pressure line and urinary catheter to monitor fluid status; drawing blood for Hb levels and hematocrit determination, for coagulation studies, and for type and crossmatching; external electronic fetal monitoring; and monitoring of maternal vital signs and vaginal bleeding.

ALERT

After determination of the severity of abruption and appropriate fluid and blood replacement, prompt delivery by cesarean birth is necessary if the fetus is in distress.

If the fetus isn't in distress, monitoring continues; delivery is usually performed at the first sign of fetal distress. Because of possible fetal blood loss through the placenta, a pediatric team should be ready at delivery to assess and treat the neonate for shock, blood loss, and hypoxia. If placental separation is severe and there are no signs of fetal life, vaginal delivery may be performed unless uncontrolled hemorrhage or other complications contraindicate it.

Complications of abruptio placentae require appropriate treatment. For example, DIC requires immediate intervention with heparin, platelets, and whole blood to prevent exsanguination.

Special considerations

- Check maternal blood pressure, pulse rate, respirations, central venous pressure, intake and output, and amount of vaginal bleeding every 10 to 15 minutes. Monitor fetal heart tones electronically.
- Prepare the patient and her family for cesarean birth. Thoroughly explain postpartum care so the patient and her family will know what to expect.
- If vaginal delivery is elected, provide emotional support during labor. Because of the infant's prematurity, the mother may not receive analgesics during labor and may experience intense pain. Reassure the patient of her progress through labor and keep her informed of the fetus' condition.
- Provide emotional support. In the case of fetal demise, encourage the patient to seek counseling as appropriate.

PREVENTION

- *Encourage the patient to participate in prenatal care throughout her pregnancy, especially if she's diabetic or hypertensive.*
- *Discuss the importance of avoiding alcohol and illegal drugs during pregnancy.*

Cardiovascular disease in pregnancy

Cardiovascular disease ranks fourth (after infection, gestational hypertension, and hemorrhage) among the leading causes of maternal death. The physiologic stress of pregnancy and delivery is often more than a compromised heart can tolerate and often leads to maternal and fetal mortality.

The prognosis for the pregnant patient with cardiovascular disease is good, with careful management. Decompensation is the leading cause of maternal death. Infant mortality increases with decompensation because uterine congestion, insufficient oxygenation, and the elevated

carbon dioxide content of the blood not only compromise the fetus, but also commonly cause premature labor and delivery.

Causes and incidence

About 1% to 2% of pregnant females have cardiac disease, but the incidence is rising because medical treatment today allows more females with rheumatic heart disease (present in more than 80% of patients who develop cardiovascular complications) and congenital defects (present in 10% to 15% of patients) to reach childbearing age. Coronary artery disease accounts for about 2% of cardiovascular complications.

The diseased heart is sometimes unable to meet the normal demands of pregnancy: 25% increase in cardiac output, 40% to 50% increase in plasma volume, increased oxygen requirements, retention of salt and water, weight gain, and alterations in hemodynamics during delivery. This physiologic stress often leads to the heart's failure to maintain adequate circulation (decompensation). The degree of decompensation depends on the patient's age, the duration of cardiac disease, and the heart's functional capacity at the pregnancy's outset.

Complications

- Endocarditis
- Miscarriage
- Preterm delivery
- Stillbirth

Signs and symptoms

Typical clinical features of cardiovascular disease during pregnancy include distended jugular veins, diastolic murmurs, moist basilar pulmonary crackles, cardiac enlargement (discernible on percussion or as a cardiac shadow on chest X-ray), and cardiac arrhythmias (other than sinus or paroxysmal atrial tachycardia). Other characteristic abnormalities may include cyanosis, pericardial friction rub, pulse delay, and pulsus alternans.

Decompensation may develop suddenly or gradually, with persistent crackles at the lung bases. As it progresses, edema, increasing dyspnea on exertion, palpitations, a smothering sensation, and hemoptysis may occur.

Diagnosis

A diastolic murmur, cardiac enlargement, a systolic murmur of grade 3/6 intensity, and severe arrhythmia suggest cardiovascular disease. Determination of the disease's extent and cause may necessitate electrocardiography, echocardiography (for valvular disorders such as rheumatic heart disease), or other studies. X-rays show cardiac enlargement and pulmonary congestion. Cardiac catheterization should be postponed until after delivery, unless surgery is necessary.

Treatment

The goal of antepartum management is to prevent complications and minimize the strain on the mother's heart, primarily through rest. This may require periodic hospitalization for patients with moderate cardiac dysfunction or with symptoms of decompensation, toxemia, or infection. Older women or those with previous decompensation may require hospitalization and bed rest throughout the pregnancy.

Drug therapy is often necessary and should always include the safest possible drug in the lowest possible dosage to minimize harmful effects to the fetus. Diuretics and drugs that increase blood pressure, blood volume, or cardiac output should be used with extreme caution. If an anticoagulant is needed, heparin is the drug of choice. Cardiac glycosides and common antiarrhythmics, such as quinidine and procainamide, are often required. The prophylactic use of antibiotics is reserved for patients who are susceptible to endocarditis.

A therapeutic abortion should be considered for patients with severe cardiac dysfunction, especially if decompensation occurs during the first trimester. Patients hospitalized with heart failure usually follow a regimen of cardiac glycosides, oxygen, rest, sedation, diuretics, and restricted intake of sodium and fluids. Patients in whom symptoms of

heart failure don't improve after treatment with bed rest and cardiac glycosides may require cardiac surgery, such as valvotomy and commissurotomy. During labor, the patient may require oxygen and an analgesic, such as meperidine or morphine, for relief of pain and apprehension without undue depression of the fetus or herself. Depending on which procedure promises to be less stressful for the patient's heart, delivery may be vaginal or by cesarean birth. Forceps may augment vaginal delivery to minimize the need to push, which strains the heart.

Bed rest and medications already instituted should continue for at least 1 week after delivery because of a high incidence of decompensation, cardiovascular collapse, and maternal death during the early puerperal period. These complications may result from the sudden release of intra-abdominal pressure at delivery and the mobilization of extracellular fluid for excretion, which increase the strain on the heart, especially if excessive interstitial fluid has accumulated. Breast-feeding is undesirable for patients with severely compromised cardiac dysfunction because it increases fluid and metabolic demands on the heart.

Special considerations

- During pregnancy, stress the importance of rest and weight control to decrease the strain on the heart. Suggest a diet of limited fluid and sodium intake to prevent vascular congestion. Encourage the patient to take supplementary folic acid and iron to prevent anemia.

ALERT

During labor, watch for signs of decompensation, such as dyspnea and palpitations. Monitor pulse rate, respirations, and blood pressure.

Auscultate for crackles every 30 minutes during the first phase of labor and every 10 minutes during the active and transition phases. Check carefully for edema and cyanosis, and assess intake and output. Administer oxygen for respiratory difficulty.

- Use electronic fetal monitoring to watch for the earliest signs of fetal distress.

- Keep the patient in a semirecumbent position. Limit her efforts to bear down during labor, which significantly raise blood pressure and stress the heart.

- After delivery, provide reassurance and encourage the patient to adhere to her program of treatment. Emphasize the need to rest during her hospital stay.

PREVENTION

To help prevent complications during pregnancy, teach the patient with cardiovascular disease the following:

- *Advise her to follow the practitioner's recommendations for rest, which may include bed rest.*
- *If appropriate, encourage a practitionerapproved exercise regime.*
- *Advise her to avoid stress during pregnancy by becoming informed about what to expect during pregnancy.*
- *Suggest that she maintain a healthy diet and avoid gaining more weight than is recommended. Extra weight may place added stress on the heart.*
- *Advise her to avoid heat and humidity, which can cause vasodilation and divert blood from the uterus.*
- *Have her report signs of infection to the practitioner, and advise that she avoid persons with upper respiratory infections.*
- *Tell her to change positions frequently and avoid crossing her legs. If on bedrest, have her wear anti-embolism stockings to prevent blood clots.*

Adolescent pregnancy

Adolescent pregnancy can pose a major health risk to both the mother and the child because up to 70% of adolescents who become pregnant don't receive adequate prenatal care. Pregnant adolescents develop special problems and are known to have a significantly higher incidence of anemia, pregnancy-induced hypertension, and perinatal mortality. For example, they're more likely to have babies who are premature or of low birth weight, with a higher neonatal mortality, and who are predisposed to injury at birth, childhood illness, and retardation or other neurologic defects. These risks are aggravated if the mother has abused drugs during the pregnancy. (See *How maternal drug use affects infants,* page 1196.)

As a rule, the younger the mother, the greater the health risk for both mother and infant. Adolescents account for one-third of all abortions performed in the United States.

Causes and incidence

Adolescent pregnancy is prevalent in all socioeconomic levels, and its contributing factors vary. Such factors may include ignorance about sexuality and contraception, increasing sexual activity at a young age, rebellion against parental influence, and a desire to escape an unhappy family situation and to fulfill emotional needs unmet by the family.

In the United States, an estimated 1 million adolescents become pregnant each year.

Complications

- Anemia
- Gestational hypertension
- Intrauterine growth retardation
- Low birth weight
- Placentae previa
- Prematurity
- Toxemia

Signs and symptoms

Clinical manifestations of adolescent pregnancy are the same as those of adult pregnancy (amenorrhea, nausea, vomiting, breast tenderness, fatigue). However, the pregnant adolescent is much more likely to develop complications, such as poor weight gain during pregnancy, premature labor, and pregnancy-induced hypertension. In addition, the neonate is more likely to be of low birth weight. Some of these complications are related to the pregnant adolescent's physical immaturity, rapid growth, interest in fad diets, and generally poor nutrition; other complications may stem from the adolescent's need to deny her condition or to her ignorance of early signs of pregnancy, which often delays initiation of prenatal care.

Diagnosis

CONFIRMING DIAGNOSIS

A positive pregnancy test that shows human chorionic gonadotropin in the blood or urine and a pelvic examination confirm pregnancy.

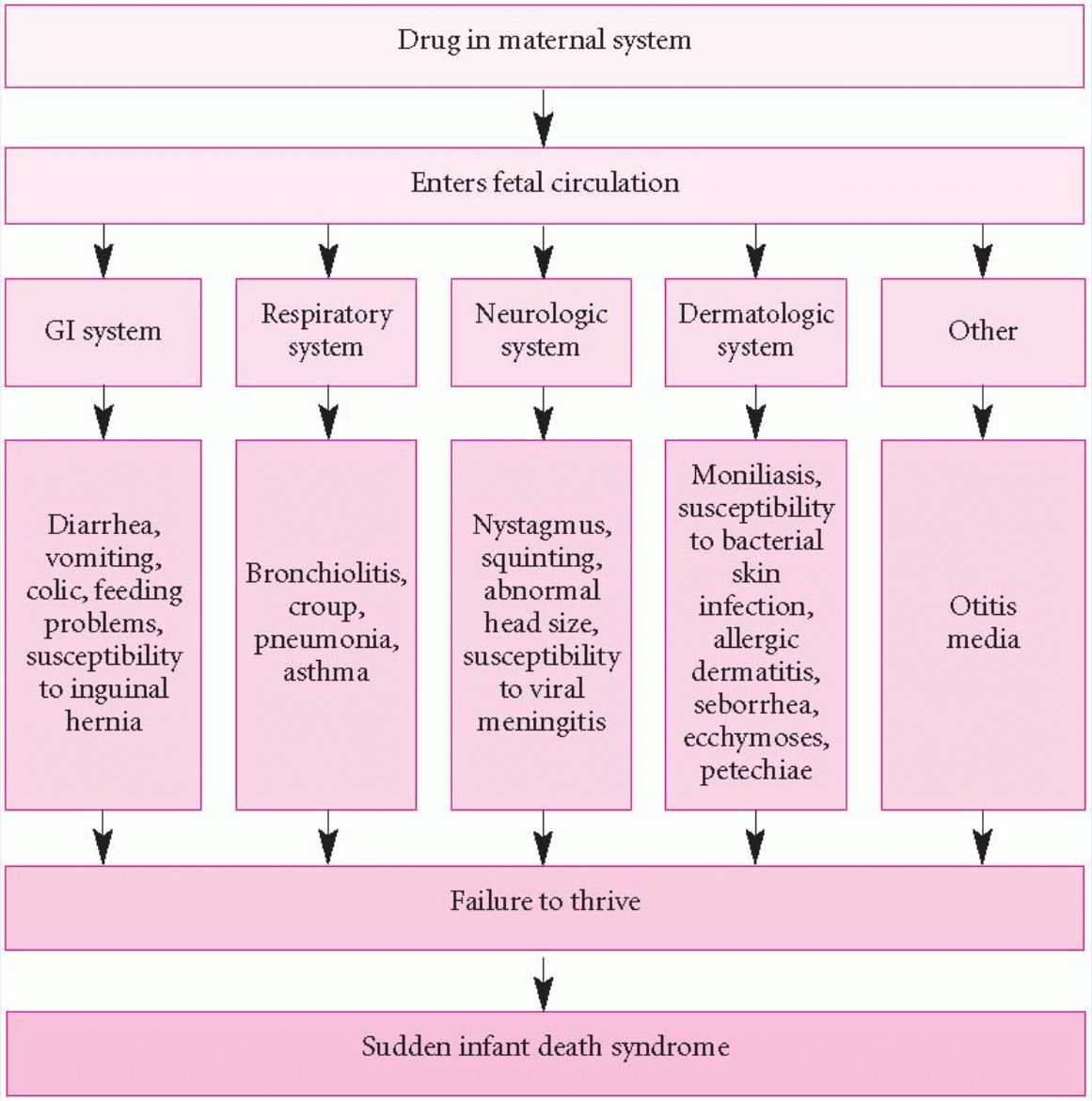
PATHOPHYSIOLOGY

HOW MATERNAL DRUG USE AFFECTS INFANTS

An infant born to a drug-dependent mother risks developing certain medical problems during the first 8 months of life. These problems range from mild to severe withdrawal symptoms, to a host of complications that affect virtually every body system.

The onset and severity of adverse reactions vary with the type and amount of drugs the mother has been taking and for how long. However, *any* drug the mother takes, including over-the-counter products, alcohol, and illicit drugs, may cross the placenta and enter the fetal circulation, where its concentration is 50% to 100%

higher than the maternal drug concentration. This chart lists fetal body systems affected by drugs and some common adverse reactions that may result.



Auscultation of fetal heart sounds with a Doppler ultrasonic flowmeter or fetoscope and ultrasonography assess fetal gestational age.

Treatment

The pregnant adolescent requires the standard prenatal care that's appropriate for an adult. However, she also needs psychological

support and close observation for signs of complications.

Special considerations

- Because you may be the first health care professional the pregnant adolescent encounters, you must help motivate her to follow sound medical advice without being judgmental, condescending, or threatening. Emphasize the importance of adhering to the prescribed diet, getting plenty of rest, and taking prescribed vitamin and iron supplements. Your understanding and support can ensure proper health care during the pregnancy for both mother and infant. Encourage her to ask questions and to express her feelings about the pregnancy. Answer her questions fully. It's also important to ascertain if the patient wishes to place the child up for adoption or is considering abortion. Any decision should be accepted in a nonjudgmental way.
- Try to help the pregnant adolescent identify her own strengths and support systems for coping with pregnancy, birth, and parenting.
- Prepare the patient and her partner for the physical and psychological process of labor and birth. Encourage attendance at prenatal classes: The use of educational films, tours of the health care facility, and role-playing techniques will facilitate her cooperation with care providers.
- Following birth, encourage the patient to set realistic goals for the future. If she opts for adoption, make sure she clearly understands her legal rights and responsibilities. Allow the patient to care for the infant, as she desires.
- If the patient decides to raise the infant, help her make the transition from pregnancy to parenthood during the postpartum period. Facilitate bonding. Help her establish a realistic plan regarding childcare, parenting, returning to school or work, and her relationship with the infant's father.
- In the event of stillbirth or the neonate's death, help the patient through the grieving process.

- Before the patient is discharged, provide information on contraception.

Diabetic complications during pregnancy

Pregnancy places special demands on carbohydrate metabolism and causes the insulin requirement to increase, even in a healthy female. Consequently, pregnancy may lead to a prediabetic state, to the conversion of an asymptomatic subclinical diabetic state to a clinical one (gestational diabetes occurs in about 1% to 2% of all pregnancies), or to complications in a previously stable diabetic state.

The incidence of diabetes mellitus increases with age. Maternal and fetal prognoses can be equivalent to those in nondiabetic females if maternal blood glucose is well controlled and ketosis and other complications are prevented. Infant morbidity and mortality depend on recognizing and successfully controlling hypoglycemia, which may develop within hours after delivery. Preconceptual counseling is helpful in optimizing pregnancy outcomes.

Causes and incidence

In diabetes mellitus, glucose is inadequately utilized either because insulin isn't synthesized or because tissues are resistant to the hormonal action of endogenous insulin. During pregnancy, the fetus relies on maternal glucose as a primary fuel source. Pregnancy triggers protective mechanisms that have anti-insulin effects: increased hormone production (placental lactogen, estrogen, and progesterone), which antagonizes insulin's effects; degradation of insulin by the placenta; and prolonged elevation of stress hormones (cortisol, epinephrine, and glucagon), which raise blood glucose levels.

In a normal pregnancy, an increase in anti-insulin factors is counterbalanced by an increase in insulin production to maintain normal blood glucose levels. However, females who are prediabetic or diabetic are unable to produce sufficient insulin to overcome the insulin antagonist mechanisms of pregnancy, or their tissues are insulin-resistant. As insulin requirements rise toward term, the patient who's prediabetic may develop gestational diabetes, necessitating dietary management and, possibly, exogenous insulin to achieve glycemic

control, whereas the patient who's insulin-dependent may need increased insulin dosage.

Complications

- Fetal anomalies
- Neonatal hypoglycemia, hypocalcemia, hyperbilirubinemia, respiratory distress syndrome
- Neonatal size inappropriate for gestational age (too large or too small)
- Preterm delivery
- Stillbirth

Signs and symptoms

Indications for diagnostic screening for maternal diabetes mellitus during pregnancy include obesity, excessive weight gain, excessive hunger or thirst, polyuria, recurrent monilial infections, glycosuria, previous delivery of a large neonate, polyhydramnios, maternal hypertension, and a family history of diabetes.

Diagnosis

The prevalence of gestational diabetes makes careful screening for hyperglycemia appropriate in all pregnancies. A screening 50-gram 1-hour glucose tolerance test is normally performed at 24 to 28 weeks. In addition, women with a history of fetal macrosomia or who may have nongestational diabetes should be formally tested for diabetes with a 3-hour glucose tolerance test.

CONFIRMING DIAGNOSIS

A 100-gram 3-hour glucose tolerance test confirms diabetes mellitus when two or more values are above normal.

Procedures to assess fetal status include stress and nonstress tests; ultrasonography to determine fetal age and growth; measurement of

phosphatidyl-glycerol; and determination of the lecithin-sphingomyelin (L/S) ratio from amniotic fluid to predict pulmonary maturity. The L/S ratio is less useful in diabetic pregnancies and generally requires a ratio of 3.5:1 to confirm fetal lung maturity.

Treatment

Treatment of both the newly diagnosed and the established diabetic is designed to maintain blood glucose levels within acceptable limits through dietary management and insulin administration. Many females with overt diabetes mellitus require hospitalization at the beginning of pregnancy to assess physical status, check for cardiac and renal disease, and regulate diabetes.

For pregnant patients with diabetes, therapy includes:

- bimonthly visits to the obstetrician and the internist during the first 6 months of pregnancy; weekly visits may be necessary during the third trimester
- maintenance of fasting blood glucose levels at or below 100 mg/dl and 2-hour postprandial blood glucose levels at or below 120 mg/dl during the pregnancy
- frequent monitoring for glycosuria and ketonuria (ketosis presents a grave threat to the fetal central nervous system)
- weight control (gain not to exceed 3 to 3½ lb [1.4 to 1.6 kg] per month during the last 6 months of pregnancy)
- high-protein diet of 2 g/day/kg of body weight, or a minimum of 80 g/day during the second half of pregnancy; daily calorie intake of 30 to 40 calories/kg of body weight; daily carbohydrate intake of 200 g; and enough fat to provide 36% of total calories (however, vigorous calorie restriction can cause starvation ketosis)
- exogenous insulin if diet doesn't control blood glucose levels. Be alert for changes in insulin requirements from one trimester to the next and immediately postpartum. Oral antidiabetic drugs are contraindicated during pregnancy because they may cause fetal hypoglycemia and congenital anomalies.

Generally, the optimal time for delivery is between 37 and 39 weeks' gestation, although with reassuring antenatal testing and no evidence of macrosomia, 40 weeks or later is also feasible. The insulin-dependent diabetic may require hospitalization before delivery for frequent monitoring of blood glucose levels and prompt intervention if complications develop.

Depending on fetal status and maternal history, the obstetrician may induce labor or perform a cesarean delivery. During labor and delivery, the patient with diabetes should receive continuous I.V. infusion of dextrose with regular insulin in water. Maternal

and fetal status must be monitored closely throughout labor. The patient may benefit from half her prepregnancy dosage of insulin before a cesarean delivery. Her insulin requirement will fall markedly after delivery.

Special considerations

- Teach the newly diagnosed patient about diabetes, including dietary management, insulin administration, home monitoring of blood glucose or urine testing for glucose and ketones, and skin and foot care. Instruct her to report ketonuria immediately.
- Evaluate the patient who's diabetic for her knowledge about this disease and provide supplementary patient teaching, as she requires. Inform the patient that frequent monitoring and adjustment of insulin dosage are necessary throughout the course of her pregnancy.
- Give reassurance that strict compliance to prescribed therapy should ensure a favorable outcome.
- Refer the patient to appropriate social service agencies if financial assistance is necessary because of prolonged hospitalization.
- Encourage medical counseling regarding the prognosis of future pregnancies.

ABNORMALITIES OF PARTURITION

Preterm labor

Preterm labor, also called *premature labor,* is the onset of rhythmic uterine contractions that produce cervical change after fetal viability but before fetal maturity. It usually occurs between the 20th and 37th weeks of gestation. About 5% to 10% of pregnancies end preterm; about 75% of neonatal deaths and a great many birth defects stem from this disorder. Fetal prognosis depends on birth weight and length of gestation: Neonates weighing less than 1 lb 10 oz (737 g) and of less than 26 weeks' gestation have a survival rate of 40% to 50%; neonates weighing 1 lb 10 oz to 2 lb 3 oz (737 to 992 g) and of 27 to 28 weeks' gestation have a survival rate of 70% to 80%; those weighing 2 lb 3 oz to 2 lb 11 oz (992 to 1,219 g) and of 28 weeks' gestation have an 85% to 97% survival rate.

Causes and incidence

The possible causes of premature labor are many; they may include premature rupture of the membranes (occurs in 30% to 50% of preterm labors), preeclampsia, chronic hypertensive vascular disease, hydramnios, multiple pregnancy, placenta previa, abruptio placentae, incompetent cervix, abdominal surgery, trauma, structural anomalies of the uterus, infections (such as rubella or toxoplasmosis), congenital adrenal hyperplasia, and fetal death.

Other important provocative factors include:

- Fetal stimulation: Genetically imprinted information tells the fetus that nutrition is inadequate and that a change in environment is required for well-being; this provokes onset of labor.
- Oxytocin sensitivity: Labor begins because the myometrium becomes hypersensitive to oxytocin, the hormone that normally induces uterine contractions.
- Myometrial oxygen deficiency: The fetus becomes increasingly proficient in obtaining oxygen, depriving the myometrium of the oxygen and energy it needs to function normally, thus making the myometrium irritable.

- Maternal genetics: A genetic defect in the mother shortens gestation and precipitates premature labor.

Complications

- Cerebral palsy
- Intracranial bleeding
- Infection
- Visual impairment

Signs and symptoms

Like labor at term, preterm labor produces rhythmic uterine contractions, cervical dilation and effacement, possible rupture of the membranes, expulsion of the cervical mucus plug, and a bloody discharge.

Diagnosis

Preterm labor is confirmed by the combined results of prenatal history, physical examination, presenting signs and symptoms, and ultrasonography (if available) showing the fetus' position in relation to the mother's pelvis. Vaginal examination confirms progressive cervical effacement and dilation.

Treatment

Treatment is intended to suppress preterm labor when tests show immature fetal pulmonary development, cervical dilation is less than 1½″ (4 cm), and the absence of factors that contraindicate continuation of pregnancy. Such treatment consists of bed rest and, when necessary, drug therapy, but neither has been proven beneficial in all patients.

The following pharmacologic agents can suppress preterm labor for up to 48 hours:

- Beta-adrenergic stimulants (terbutaline, isoxsuprine, or ritodrine): Stimulation of the $beta_2$-adrenergic receptors inhibits contractility of uterine smooth muscle. Adverse effects include maternal tachycardia and hypotension, and fetal tachycardia.
- Magnesium sulfate: Direct action on the myometrium relaxes the muscle. It also produces maternal adverse effects, such as drowsiness, slurred speech, flushing, decreased reflexes, decreased GI motility, and decreased respirations. Fetal and neonatal adverse effects may include central nervous system (CNS) depression, decreased respirations, and decreased sucking reflex.

Maternal factors that jeopardize the fetus, making preterm delivery the lesser risk, include intrauterine infection, abruptio placentae, placental insufficiency, and severe preeclampsia. Among the fetal problems that become more perilous as pregnancy nears term are severe isoimmunization and congenital anomalies.

Ideally, treatment for active premature labor should take place in a regional perinatal intensive care center, where the staff is specially trained to handle this situation. In such settings, the neonate can remain close to his parents. (Community health care facilities commonly lack the equipment necessary for special neonatal care and transfer the neonate alone to a perinatal center.)

Treatment and delivery require an intensive team effort, focusing on:

- continuous assessment of the neonate's health through fetal monitoring
- administration of antenatal steroids to assist fetal lung development, unless contraindicated
- maintenance of adequate hydration through I.V. fluids.

Special considerations

A patient in preterm labor requires close observation for signs of fetal or maternal distress, and comprehensive supportive care.

- During attempts to suppress preterm labor, maintain bed rest and administer medications, as ordered. Give sedatives and analgesics sparingly, because they can have potentially harmful effects on the

fetus. Minimize the need for these drugs by providing comfort measures, such as frequent repositioning and good perineal and back care.

- When administering beta-adrenergic stimulants, sedatives, and opioids, monitor blood pressure, pulse rate, respirations, fetal heart rate, and uterine contraction pattern. Minimize adverse effects by keeping the patient in a lateral recumbent position as much as possible. Provide adequate hydration.
- Tocolytic therapy has never been shown to benefit fetal morbidity and mortality; it's best used to delay delivery for 48 hours while allowing antenatal steroids to effect lung development in the fetus.
- When administering magnesium sulfate, monitor neurologic reflexes. Watch the neonate for signs of magnesium toxicity, including neuromuscular and respiratory depression.
- Offer emotional support to the patient and her family. Encourage the parents to express their fears concerning the neonate's survival and health.
- During active preterm labor, remember that the premature neonate has a lower tolerance for the stress of labor and is much more likely to become hypoxic than the term neonate. If necessary, administer oxygen to the patient through a nasal cannula.

Encourage the patient to lie on her left side or sit up during labor; this position prevents caval compression, which can cause supine hypotension and subsequent fetal hypoxia. Observe fetal response to labor through continuous fetal monitoring. Prevent maternal hyperventilation; a rebreathing bag may be necessary. Continually reassure the patient throughout labor to help ease her anxiety.

ALERT

Help the patient get through labor with as little analgesic medication and anesthetic as possible. To minimize fetal CNS depression, avoid administering analgesics when delivery seems imminent. Monitor fetal and maternal response to local and regional anesthetics.

- Explain all procedures. Throughout labor, keep the patient informed of her progress and the fetus' condition. If the father is present during labor, allow the parents some time together to share their feelings.
- A prepared resuscitation team, consisting of a physician, nurse, respiratory therapist, and an anesthesiologist or anesthetist, should be in attendance to take care of the neonate immediately. Have resuscitative equipment available in case of neonatal respiratory distress.
- Inform the parents of their child's condition. Describe his appearance and explain the purpose of any supportive equipment. Help them gain confidence in their ability to care for their child. Provide privacy and encourage them to hold and feed the neonate, when possible.
- As necessary, before the parents leave the facility with the neonate, refer them to a community health nurse who can help them adjust to caring for a premature neonate.

PREVENTION

- *Advise the patient to get good prenatal care, adequate nutrition, and proper rest.*
- *Insertion of a purse-string suture (cerclage) to reinforce an incompetent cervix at 14 to 18 weeks' gestation may prevent preterm labor in patients with histories of this disorder. However, this can be dangerous if an incompetent cervix is misdiagnosed and preterm labor is the true cause.*

Premature rupture of membranes

Premature rupture of membranes (PROM) is a spontaneous break or tear in the amniochorial sac before onset of regular contractions, resulting in progressive cervical dilation. Labor usually starts within 24 hours; more than 80% of these neonates are mature. The latent period (between membrane rupture and onset of labor) is generally brief when the membranes rupture near term; when the neonate is premature, this period is prolonged, which increases the risk of mortality from maternal

infection (amnionitis, endometritis), fetal infection (pneumonia, septicemia), and prematurity.

Causes and incidence

Although the cause of PROM is unknown, malpresentation and contracted pelvis commonly accompany the rupture. Predisposing factors may include:

- poor nutrition and hygiene, and lack of proper prenatal care
- incompetent cervix (perhaps as a result of abortions)
- increased intrauterine tension due to hydramnios or multiple pregnancies
- defects in the amniochorial membranes' tensile strength
- uterine infection.

PROM occurs in nearly 10% of all pregnancies over 20 weeks' gestation.

Complications

Maternal

- Amnionitis
- Cesarean delivery
- Endometritis

Neonatal

- Asphyxia
- Congenital anomalies
- Cord prolapse
- Fetal distress
- Malpresentation
- Pulmonary hypoplasia

- Respiratory distress syndrome

Signs and symptoms

Typically, PROM causes blood-tinged amniotic fluid containing vernix particles to

gush or leak from the vagina. Maternal fever, fetal tachycardia, and foul-smelling vaginal discharge indicate infection.

Diagnosis

Characteristic passage of amniotic fluid confirms PROM. Physical examination shows amniotic fluid in the vagina. Examination of this fluid helps determine appropriate management. For example, aerobic and anaerobic cultures and a Gram stain from the cervix reveal pathogenic organisms and indicate uterine or systemic infection.

CONFIRMING DIAGNOSIS

Alkaline pH of fluid collected from the posterior fornix turns Nitrazine paper deep blue. (The presence of blood can give a false-positive result.) If a smear of fluid is placed on a slide and allowed to dry, it takes on a fernlike pattern due to the high sodium and protein content of amniotic fluid.

Staining the fluid with Nile blue sulfate reveals two categories of cell bodies. Bluestained bodies represent shed fetal epithelial cells, while orange-stained bodies originate in sebaceous glands. Incidence of prematurity is low when more than 20% of cells stain orange.

Physical examination also determines the presence of multiple pregnancies. Fetal presentation and size should be assessed by abdominal palpation (Leopold's maneuvers).

Other data determine the fetus's gestational age:

- historical: date of last menstrual period, quickening

- physical: initial detection of unamplified fetal heart sound, measurement of fundal height above the symphysis, ultrasound measurements of fetal biparietal diameter
- chemical: tests on amniotic fluid, such as the lecithin-sphingomyelin (L/S) ratio (an L/S ratio greater than 2 indicates pulmonary maturity); foam stability (shake test) also indicates fetal pulmonary maturity. Presence of phosphatidylglycerol (PG) in the fluid indicates that respiratory distress is unlikely.

Treatment

Treatment for PROM depends on fetal age and the risk of infection. In a term pregnancy, if spontaneous labor and vaginal delivery aren't achieved within a relatively short time (usually within 24 hours after the membranes rupture), induction of labor with oxytocin is usually required; if induction fails, cesarean delivery is usually necessary. Cesarean hysterectomy is recommended with gross uterine infection.

Management of a preterm pregnancy of less than 34 weeks is controversial. However, with advances in technology, a conservative approach to PROM has now been proven effective. With a preterm pregnancy of 28 to 34 weeks, treatment includes hospitalization and observation for signs of infection (maternal leukocytosis or fever, and fetal tachycardia) while awaiting fetal maturation. If clinical status suggests infection, baseline cultures and sensitivity tests are appropriate. If these tests confirm infection, labor must be induced, followed by I.V. administration of antibiotics. A culture should also be made of gastric aspirate or a swabbing from the neonate's ear because antibiotic therapy may be indicated for him as well. At such delivery, have resuscitative equipment available to treat neonatal distress.

Special considerations

- Teach the patient in the early stages of pregnancy how to recognize PROM. Make sure she understands that amniotic fluid doesn't always gush; it may leak slowly.
- Stress that the patient *must* report PROM immediately because prompt treatment may prevent dangerous infection.

- Warn the patient not to engage in sexual intercourse or to douche after the membranes rupture.
- Before physical examination in suspected PROM, explain all diagnostic tests and clarify any misunderstandings the patient may have. During the examination, stay with the patient and provide reassurance. Such examination requires sterile gloves and sterile lubricating jelly. Don't use iodophor antiseptic solution, because it discolors Nitrazine paper and makes pH determination impossible.
- After the examination, provide proper perineal care. Send fluid samples to the laboratory promptly because bacteriologic studies need immediate evaluation to be valid. If labor starts, observe the mother's contractions and monitor vital signs every 2 hours. Watch for signs of maternal infection (fever, abdominal tenderness, and changes in amniotic fluid, such as foul odor or purulence) and fetal tachycardia. (Fetal tachycardia may precede maternal fever.) Report such signs immediately.
- The L/S ratio isn't useful if obtained from a vaginal pool of amniotic fluid, because vaginal epithelium secretes lecithin. The PG level is accurate, however.

Cesarean birth

Cesarean birth, also known as *cesarean section,* is delivery of a neonate by surgical incision through the abdomen and uterus. It can be performed as elective surgery or as an emergency procedure when conditions prohibit vaginal delivery.

Causes and incidence

The most common reasons for cesarean birth are malpresentation (such as shoulder or face presentation), fetal intolerance of labor distress, cephalopelvic disproportion ([CPD] the pelvis is too small to accommodate the fetal head), certain cases of toxemia, previous cesarean birth, and inadequate progress in labor (failure of induction).

Conditions causing fetal distress that indicate a need for cesarean birth include prolapsed cord with a live fetus, fetal hypoxia, abnormal fetal heart rate patterns, unfavorable intrauterine environment (from infection), and moderate to severe Rh isoimmunization. Less common maternal conditions that may necessitate cesarean birth include complete placenta previa, abruptio placentae, placenta accreta, malignant tumors, and chronic diseases in which delivery is indicated before term.

Cesarean birth may also be necessary if induction is contraindicated or difficult or if advanced labor increases the risk of morbidity and mortality.

In the case of a previous cesarean delivery, some physicians allow a subsequent vaginal delivery if the cesarean wasn't classic or if the original reason for the cesarean no longer exists. However, vaginal delivery risks uterine rupture if the uterus is scarred.

The rising incidence of cesarean birth coincides with recent medical and technologic advances in fetal and placental surveillance and care. In the United States, 9% to 16% of all pregnancies terminate in cesarean births, rising to 17% to 25% in perinatal centers that handle high-risk deliveries.

Diagnosis

Special tests and monitoring procedures provide early indications of the need for cesarean birth:

- Magnetic resonance imaging or clinical pelvimetry reveals CPD and malpresentation.
- Ultrasonography shows pelvic masses that interfere with vaginal delivery and fetal position.
- Auscultation of fetal heart rate (by fetoscope, Doppler unit, or electronic fetal monitor) determines acute fetal intolerance of labor.

Treatment

The most common type of cesarean birth is the *lower segment cesarean*, in which a transverse incision across the lower abdomen opens the

visceral peritoneum over the uterus. The lower anterior uterine wall is then incised (transversely or longitudinally) behind the bladder.

The *classic cesarean*—in which a longitudinal incision is made into the body of the uterus, extending into the fundus and opening the top of the uterus—is rarely performed because it exaggerates the risk of infection and of uterine rupture in subsequent pregnancies. *Cesarean hysterectomy* removes the entire uterus and is reserved for such cases as malignant tumors, severe infection, and placenta accreta.

Patients may have general or regional anesthetic for surgery, depending on the extent of maternal or fetal distress. Possible maternal complications of cesarean delivery include respiratory tract infection, wound dehiscence, thromboembolism, paralytic ileus, hemorrhage, and genitourinary tract infection.

Special considerations

Before cesarean delivery:

- Explain cesarean birth to the patient and her partner, and answer any questions they may have. Provide reassurance and emotional support. Cesarean birth is often performed after hours of labor have exhausted the patient.
- Administer preoperative medications as ordered.
- Prepare the patient by shaving her from below the breasts to the pubic region and the upper quarter of the anterior thighs. Make sure her bladder is empty, using an indwelling urinary catheter as ordered. Insert an I.V. line for fluid replacement therapy as ordered. Assess maternal temperature, pulse rate, respirations, and blood pressure and fetal heart rate.
- In the operating room, place the patient in a slight lateral position. Use of a 15-degree wedge reduces caval compression (supine hypotension) and subsequent fetal hypoxia.

After cesarean delivery:

- Check vital signs every 15 minutes until they stabilize. Maintain a patent airway. If general anesthetic was used, remain with the patient until she's responsive. If regional anesthetic was used, monitor the return of sensation to the legs.
- Encourage parent-infant bonding as soon as practical.
- Gently assess the fundus. Check the incision and lochia for signs of infection such as a foul odor. Check frequently for bleeding and report it immediately. Keep the incision clean and dry.
- Observe the neonate for signs of respiratory distress (tachypnea, retractions, and cyanosis) until there's evidence of physiologic stability. Keep resuscitative equipment available.
- Assess intake and output (some patients have indwelling urinary catheters in place up to 48 hours postoperative). Observe the patient closely for indications of bladder fullness or urinary tract infection.
- Administer pain medication, as ordered, and provide comfort measures for breast engorgement as appropriate. Offer reassurance and reduce anxiety by answering any questions. If the mother wishes to breast-feed, offer encouragement and help. Recognize afterpains in multiparas.
- Promote early ambulation to prevent cardiovascular and pulmonary complications.
- Provide psychological support. If the patient seems anxious about having had a cesarean delivery, encourage her to share her feelings with you. If appropriate, suggest that she participate in a cesarean birth sharing group. Encourage support from her family members as well.

POSTPARTUM DISORDERS

Puerperal infection

A common cause of childbirth-related death, puerperal infection is a postpartum infection of the uterus and higher structures, with a characteristic fever pattern. It can result in endometritis, parametritis,

pelvic and femoral thrombophlebitis, and peritonitis. The prognosis is good with treatment.

Causes and incidence

Microorganisms that commonly cause puerperal infection include group B streptococci, coagulase-negative staphylococci, *Clostridium perfringens, Bacteroides fragilis,* and *Escherichia coli*. Most of these organisms are considered normal vaginal flora but are known to cause puerperal infection in the presence of certain predisposing factors:

- prolonged and premature rupture of the membranes
- prolonged (more than 24 hours) labor
- frequent or unsanitary vaginal examinations or unsanitary delivery
- retained products of conception
- hemorrhage
- maternal conditions, such as anemia or debilitation from malnutrition
- cesarean birth (20-fold increase in risk for puerperal infection).

In the United States, puerperal infection develops in about 6% of maternity patients.

Complications

- Infertility
- Pulmonary embolism
- Septic shock
- Stroke

Signs and symptoms

A characteristic sign of puerperal infection is fever (at least 100.4° F [38° C]) that occurs in the first 24 hours in the first 9 days postpartum. This fever can spike as high as 105° F (40.6° C) and is commonly

associated with chills, headache, malaise, restlessness, and anxiety. Abortion or miscarriage isn't usually associated with this infection and fever.

Accompanying signs and symptoms depend on the infection's extent and site and may include:

- endometritis: heavy, sometimes foul-smelling lochia; tender, enlarged uterus; backache; severe uterine contractions persisting after childbirth
- parametritis (pelvic cellulitis): vaginal tenderness and abdominal pain and tenderness (pain may become more intense as infection spreads).

The inflammation may remain localized, may lead to abscess formation, or may spread through the blood or lymphatic system. Widespread inflammation may cause:

- pelvic thrombophlebitis: severe, repeated chills and dramatic swings in body temperature; lower abdominal or flank pain; and, possibly, a palpable tender mass over the affected area, which usually develops near the second postpartum week
- femoral thrombophlebitis: pain, stiffness, or swelling in a leg or the groin; inflammation or shiny, white appearance of the affected leg; malaise; fever; and chills, usually beginning 10 to 20 days postpartum (these signs may precipitate pulmonary embolism)
- peritonitis: body temperature usually elevated, accompanied by tachycardia (greater than 140 beats/minute), weak pulse, hiccups, nausea, vomiting, and diarrhea; constant and possibly excruciating abdominal pain.

Diagnosis

Development of the typical clinical features, especially fever within 48 hours after delivery, suggests a diagnosis of puerperal infection. Uterine tenderness is also highly suggestive.

A culture of lochia, incisional exudate (from cesarean incision or episiotomy), uterine tissue, or material collected from the vaginal cuff that reveals the causative organism may confirm the diagnosis, but such

cultures are generally contaminated with vaginal flora and aren't considered helpful.

Within 36 to 48 hours, white blood cell count usually demonstrates leukocytosis (15,000 to 30,000/µl).

Typical clinical features usually suffice for diagnosis of endometritis and peritonitis. In parametritis, pelvic examination shows induration without purulent discharge.

Diagnosis of pelvic or femoral thrombophlebitis is suggested by characteristic clinical signs, venography, Doppler ultrasonography, palpable veins inside the thigh and calf, pain in the calf when pressure is applied on the inside of the foot, and pain on passive dorsiflexion of the foot with the knee extended (Homans' sign). Homans' sign should be elicited passively by asking the patient to dorsiflex the foot. Active dorsiflexion could lead to embolization of a clot.

Treatment

Treatment of puerperal infection usually begins with I.V. infusion of a broad-spectrum antibiotic to control the infection and prevent its spread while awaiting culture results. After identification of the infecting organism, a more specific antibiotic should be administered. (An oral antibiotic may be prescribed after hospital discharge.)

Ancillary measures include analgesics for pain; antiseptics for local lesions; and antiemetics for nausea and vomiting from peritonitis. Isolation or transfer from the maternity unit generally isn't appropriate.

Supportive care includes bed rest, adequate fluid intake, I.V. fluids when necessary, and measures to reduce fever. Sitz baths and heat lamps may relieve discomfort from local lesions.

Surgery may be necessary to remove any remaining products of conception or to drain local lesions such as an abscess in parametritis.

Management of septic pelvic thrombophlebitis consists of heparinization for about 10 days in conjunction with broad-spectrum antibiotic therapy.

Special considerations

- Monitor vital signs every 4 hours (more frequently if peritonitis has developed) and intake and output. Enforce strict bed rest.
- Frequently inspect the perineum. Assess the fundus and palpate for tenderness (subinvolution may indicate endometritis). Note the amount, color, and odor of vaginal drainage, and document your observations.
- Administer antibiotics and analgesics, as ordered. Assess and document the type, degree, and location of pain as well as the patient's response to analgesics. Give the patient an antiemetic to relieve nausea and vomiting, as necessary.
- Provide sitz baths and a heat lamp for local lesions. Change bed linen and perineal pads and under pads frequently. Keep the patient warm.
- Elevate the thrombophlebitic leg about 30 degrees. Provide warm soaks. Watch for signs of pulmonary embolism, such as cyanosis, dyspnea, and chest pain.

ALERT

Don't rub or manipulate the thrombophlebitic leg or compress it with bed linen.

- Offer reassurance and emotional support. Thoroughly explain all procedures to the patient and her family.
- If the mother is separated from her neonate, provide her with frequent reassurance about his progress. Encourage the father to reassure the mother about the neonate's condition as well.

PREVENTION

- *Maintain sterile technique when performing a vaginal examination. Limit the number of vaginal examinations done during labor. Wash your hands thoroughly after each patient contact.*
- *Tell a pregnant patient to call her practitioner immediately when her membranes rupture. Warn her*

to avoid intercourse after rupture or leaking of the amniotic sac.

- Keep the episiotomy site clean and teach the patient how to maintain good perineal hygiene.
- Screen personnel and visitors to keep persons with active infections away from maternity patients.

Mastitis and breast engorgement

Mastitis (parenchymatous inflammation of the mammary glands) and breast engorgement (congestion) are disorders that may affect lactating females. The prognosis for both disorders is good.

Causes and incidence

Mastitis develops when a pathogen that typically originates in the nursing infant's nose or pharynx invades breast tissue through a fissured or cracked nipple and disrupts normal lactation. The most common pathogen of this type is *Staphylococcus aureus;* less frequently, it's *S. epidermidis* or beta-hemolytic streptococci. Rarely, mastitis may result from disseminated tuberculosis or the mumps virus. Predisposing factors include a fissure or abrasion on the nipple; blocked milk ducts; and an incomplete let-down reflex, usually due to emotional trauma. Blocked milk ducts can result from a tight bra or prolonged intervals between breast-feedings. Causes of breast engorgement include venous and lymphatic stasis, and alveolar milk accumulation. (See *Physiology of lactation.*)

Mastitis occurs postpartum in about 1% of pregnant women, mainly in primiparas who are breast-feeding. It occurs occasionally in nonlactating females and rarely in males. All breast-feeding mothers develop some degree of engorgement, which isn't an infectious process.

Complication

- Abscess

Signs and symptoms

Mastitis may develop anytime during lactation but usually begins 1 to 2 weeks postpartum with fever (101° F [38.3° C] or higher in acute mastitis), malaise, and flulike symptoms. The breast (or, occasionally, both breasts) becomes tender, hard, swollen, and warm. Unless mastitis is treated adequately, it may progress to breast abscess.

Breast engorgement generally starts with onset of lactation (day 2 to day 5 postpartum). The breasts undergo changes similar to those in mastitis, and body temperature

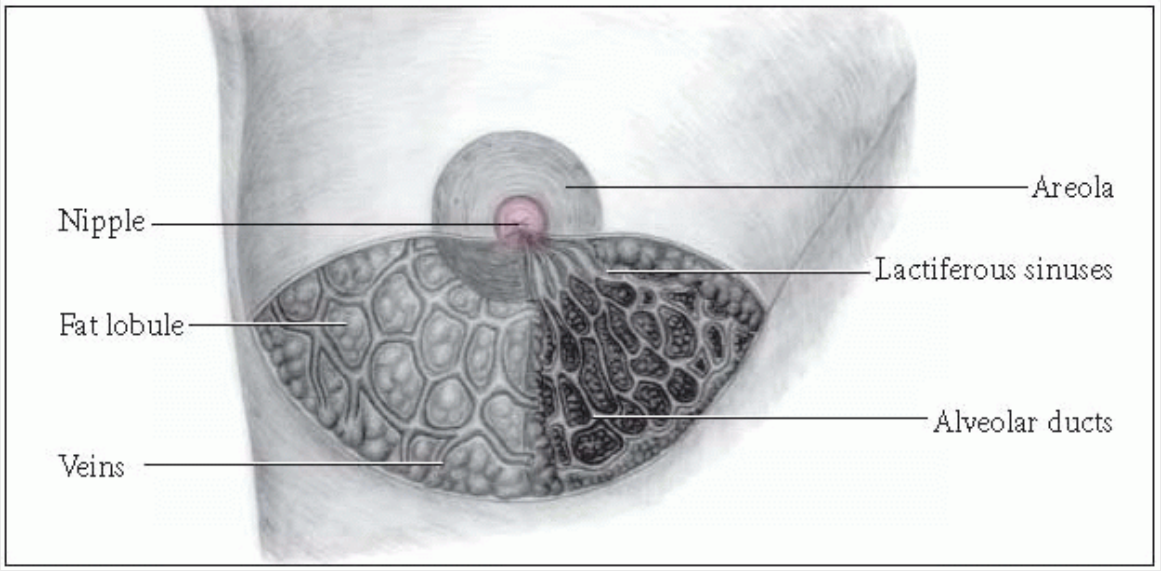
may be elevated. Engorgement may be mild, causing only slight discomfort, or severe, causing considerable pain. A severely engorged breast can interfere with the infant's capacity to feed because of his inability to position his mouth properly on the swollen, rigid breast.

PHYSIOLOGY OF LACTATION

During pregnancy, progesterone and estrogen normally interact to suppress milk secretion while developing the breasts for lactation. Estrogen causes the breasts to grow by increasing their fat content; progesterone causes lobule growth and develops the alveolar cells' secretory capacity.

After childbirth, the mother's anterior pituitary gland secretes prolactin (suppressed during pregnancy), which helps the alveolar epithelium produce and release colostrum. Usually, within 3 days of prolactin release, the breasts secrete large amounts of milk rather than colostrum. The infant's sucking stimulates nerve endings at the nipple, initiating the let-down reflex that allows the expression of milk from the mother's breasts. Sucking also stimulates the release of another pituitary hormone, oxytocin, into the mother's bloodstream. This hormone causes alveolar contraction, which forces milk into the

ducts and the lactiferous sinuses beneath the alveolar surface, making milk available to the infant. (It also promotes normal involution of the uterus.)



The infant's suckling provides the stimulus for both milk production and milk expression. As a result, the more the infant breast-feeds, the more milk the breast produces. Conversely, the less sucking stimulation the breast receives, the less milk it produces.

Diagnosis

CONFIRMING DIAGNOSIS

Diagnosis is usually easily made if pus is expressed from the nipple; culture may be helpful in confirming mastitis.

Treatment

Antibiotic therapy, the primary treatment for mastitis, generally consists of oral cephalosporins, cloxacillin, or dicloxacillin to combat staphylococcus; azithromycin may be used in patients allergic to penicillin. Although symptoms usually subside 2 to 3 days after treatment begins, antibiotic therapy should continue for 10 days. Other appropriate measures include analgesics for pain and, rarely, when

antibiotics fail to control the infection and mastitis progresses to breast abscess, incision and drainage of the abscess.

PREVENTION

PREVENTING MASTITIS

To help your patient prevent mastitis from recurring, teach her the following:

- Stress the importance of emptying the breasts completely, because milk stasis can cause infection and mastitis.
- Have her alternate feeding positions and rotate pressure areas on the nipples.
- Remind her to position the infant properly on the breast with the entire areola in his mouth.
- Advise her to expose sore nipples to the air as often as possible.
- Teach her proper hand-washing techniques and personal hygiene.
- Tell her to get plenty of rest and to consume sufficient fluids and a balanced diet to enhance breast-feeding.
- Suggest that she apply a warm, wet towel to the breast that was affected or that she take a warm shower to relax and improve breast-feeding.

The goal of treatment of breast engorgement is to relieve discomfort and control swelling, and may include analgesics to alleviate pain, and ice packs and an uplift support bra to minimize edema. Rarely, oxytocin nasal spray may be necessary to release milk from the alveoli into the ducts. To facilitate breast-feeding, the mother may manually express excess milk before a feeding so the infant can grasp the nipple properly.

Special considerations

If the patient has mastitis:

- Isolate the patient and her infant to prevent the spread of infection to other nursing mothers. Explain mastitis to the patient and why isolation is necessary.
- Obtain a complete patient history, including a drug history, especially allergy to penicillin.
- Assess and record the cause and amount of discomfort. Give analgesics as needed.
- Reassure the mother that breast-feeding during mastitis won't harm her infant, because he's the source of the infection. However, if an open abscess develops, she must stop breast-feeding with this breast and use a breast pump until the abscess heals. She should continue to breast-feed on the unaffected side.
- Instruct the patient to combat fever by getting plenty of rest, drinking sufficient fluids, and following the prescribed antibiotic therapy.

If the patient has breast engorgement:

- Assess and record the level of discomfort. Give analgesics, and apply ice packs and a compression binder, as needed.
- Teach the patient how to express excess breast milk manually. She should do this just before nursing to enable the infant to get the swollen areola into his mouth. Caution against excessive milk expression between feedings because this stimulates milk production and prolongs engorgement.
- Explain that because breast engorgement is caused by the physiologic processes of lactation, breast-feeding is the best remedy for engorgement. Suggest breast-feeding every 2 to 3 hours and at least once during the night.
- Ensure that the mother wears a well-fitted nursing bra, usually a size larger than she normally wears. (See *Preventing mastitis.*)

Galactorrhea

Galactorrhea, also known as *hyperprolactinemia,* is inappropriate breast milk secretion. It generally occurs 3 to 6 months after the

discontinuation of breast-feeding (usually after a first delivery). It may also follow an abortion or may develop in a female who hasn't been pregnant; it rarely occurs in males.

Causes and incidence

Galactorrhea usually develops in a person with increased prolactin secretion from the anterior pituitary gland, with possible abnormal patterns of secretion of growth, thyroid, and adrenocorticotropic hormones. However, increased prolactin serum concentration doesn't always cause galactorrhea.

Additional factors that may precipitate this disorder include the following:

- endogenous: pituitary (high incidence with chromophobe adenoma), ovarian, or adrenal tumors and hypothyroidism; in males, pituitary, testicular, or pineal gland tumors
- idiopathic: possibly from stress or anxiety, which causes neurogenic depression of the prolactin-inhibiting factor
- exogenous: breast stimulation, genital stimulation, or drugs (such as hormonal contraceptives, meprobamate, and phenothiazines).

Complications

- Central nervous system disturbances
- Increased intracranial pressure
- Visual field disturbances

Signs and symptoms

In the female with galactorrhea, milk continues to flow after the 21-day period that's normal after weaning. Galactorrhea may also be spontaneous and unrelated to normal lactation, or it may be caused by manual expression. Such abnormal flow is usually bilateral and may be accompanied by amenorrhea.

Diagnosis

Characteristic clinical features and the patient history (including drug and sex histories) confirm galactorrhea.

Laboratory tests to help determine the cause include measurement of serum levels of prolactin, cortisol, thyroid-stimulating hormone, triiodothyronine, and thyroxine. A computed tomography scan and, possibly, mammography may also be indicated.

Treatment

Treatment varies according to the underlying cause and ranges from simple avoidance of precipitating exogenous factors such as drugs to treatment of tumors with surgery, radiation, or chemotherapy.

Therapy for idiopathic galactorrhea depends on whether the patient plans to have more children. If she does, treatment usually consists of bromocriptine or cabergoline (Dostinex); if she doesn't, oral estrogens such as ethinyl estradiol and progestins such as progesterone effectively treat this disorder. Idiopathic galactorrhea may recur after discontinuation of drug therapy.

Special considerations

- Watch for central nervous system abnormalities, such as headache, failing vision, and dizziness.
- Maintain adequate fluid intake, especially if the patient has a fever. However, advise the patient to avoid tea, coffee, and certain tranquilizers that may aggravate engorgement.
- Instruct the patient to keep her breasts and nipples clean.
- Tell the patient who's taking bromocriptine to report nausea, vomiting, dyspepsia, appetite loss, dizziness, fatigue, numbness, and hypotension. To prevent GI upset, advise her to eat small meals frequently and to take this drug with dry toast or crackers. After treatment with bromocriptine, milk secretion usually stops in 1 to 2 months, and menstruation recurs after 6 to 24 weeks.

HEMOLYTIC DISEASES OF THE NEONATE

Hyperbilirubinemia

Hyperbilirubinemia, also called *neonatal jaundice,* is the result of hemolytic processes in the neonate. It's marked by elevated serum bilirubin levels and mild jaundice and can be physiologic (with jaundice the only symptom) or pathologic (resulting from an underlying disease). Physiologic jaundice tends to be more common and more severe in certain ethnic groups (Chinese, Japanese, Koreans, Native Americans), whose mean peak of unconjugated bilirubin is about twice that of the rest of the population. Physiologic jaundice is self-limiting; the prognosis for pathologic jaundice varies, depending on the cause.

Untreated, severe hyperbilirubinemia may result in kernicterus, a neurologic syndrome resulting from deposition of unconjugated bilirubin in the brain cells and characterized by severe neural symptoms. Survivors may develop cerebral palsy, epilepsy, or mental retardation or have only minor sequelae, such as perceptualmotor handicaps and learning disorders.

Causes and incidence

As erythrocytes break down at the end of their neonatal life cycle, hemoglobin (Hb) separates into globin (protein) and heme (iron) fragments. Heme fragments form unconjugated (indirect) bilirubin, which binds with albumin for transport to liver cells to conjugate with glucuronide, forming direct bilirubin. Because unconjugated bilirubin is fat-soluble and can't be excreted in the urine or bile, it may escape to extravascular tissue, especially fatty tissue and the brain, resulting in hyperbilirubinemia.

This pathophysiologic process may develop when:

- certain factors disrupt conjugation and usurp albumin-binding sites, including drugs (such as aspirin, tranquilizers, and sulfonamides) and

conditions (such as hypothermia, anoxia, hypoglycemia, and hypoalbuminemia)

- decreased hepatic function results in reduced bilirubin conjugation
- increased erythrocyte production or breakdown results from hemolytic disorders, or Rh or ABO incompatibility
- biliary obstruction or hepatitis results in blockage of normal bile flow
- maternal enzymes present in breast milk inhibit the neonate's glucuronyltransferase conjugating activity. (See *Causes of hyperbilirubinemia*.)

Complications

- Cerebral palsy
- Deafness
- Kernicterus

Signs and symptoms

The primary sign of hyperbilirubinemia is jaundice, which doesn't become clinically apparent until serum bilirubin levels reach about 7 mg/dl. Physiologic jaundice develops 24 hours after delivery in 50% of term neonates (usually day 2 to day 3) and 48 hours after delivery in 80% of premature neonates (usually day 3 to day 5). It generally disappears by day 7 in term neonates and by day 10 in premature neonates. Throughout physiologic jaundice, serum unconjugated bilirubin levels don't exceed 12 mg/dl. Pathologic jaundice may appear anytime after the first day of life and persists beyond 7 days with serum bilirubin levels greater than 12 mg/dl in a term neonate, 15 mg/dl in a premature neonate, or increasing more than 5 mg/dl in 24 hours.

Diagnosis

CONFIRMING DIAGNOSIS

Jaundice and elevated levels of serum bilirubin confirm the diagnosis of hyperbilirubinemia.

Inspection of the neonate in a well-lit room (without yellow or gold lighting) reveals yellowish skin coloration, particularly in the sclerae. Jaundice can be verified by pressing the skin on the cheek or abdomen lightly with one finger, then releasing pressure and observing skin color immediately. Signs of jaundice necessitate measuring and charting serum bilirubin levels every 4 hours. Testing may include direct and indirect bilirubin levels, particularly for pathologic jaundice. Bilirubin levels that are excessively elevated or vary daily suggest a pathologic process.

Identifying the underlying cause of hyperbilirubinemia requires a detailed patient history (including prenatal history), family history (paternal Rh factor, inherited red cell defects), present neonate status (immaturity, infection), and blood testing of the neonate and mother (blood group incompatibilities, Hb levels, direct Coombs' test, hematocrit).

Treatment

Depending on the underlying cause, treatment may include phototherapy, exchange transfusions, albumin infusion and, possibly,

drug therapy. Phototherapy is the treatment of choice for physiologic jaundice, and pathologic jaundice due to erythroblastosis fetalis (after the initial exchange transfusion). Phototherapy uses fluorescent light to decompose bilirubin in the skin by oxidation and is usually discontinued after bilirubin levels fall below 10 mg/dl and continue to decrease for 24 hours. However, phototherapy is rarely the only treatment for jaundice due to a pathologic cause.

CAUSES OF HYPERBILIRUBINEMIA

The neonate's age at onset of hyperbilirubinemia may provide clues to the sources of this jaundice-causing disorder.

Day 1

- Blood type incompatibility (Rh, ABO, other minor blood groups)

- Intrauterine infection (rubella, cytomegalic inclusion body disease, toxoplasmosis, syphilis and, occasionally, bacteria such as *Escherichia coli, Staphylococcus*, *Pseudomonas, Klebsiella, Proteus,* and *Streptococcus*)

Day 2 or 3

- Infection (usually from gram-negative bacteria)
- Polycythemia
- Enclosed hemorrhage (skin bruises, subdural hematoma)
- Respiratory distress syndrome (hyaline membrane disease)
- Heinz body anemia from drugs and toxins (vitamin K3, sodium nitrate)
- Transient neonatal hyperbilirubinemia
- Abnormal red blood cell morphology
- Red cell enzyme deficiencies (glucose-6-phosphate dehydrogenase, hexokinase)
- Physiologic jaundice
- Blood group incompatibilities

Days 4 and 5

- Breast-feeding, respiratory distress syndrome, maternal diabetes
- Crigler-Najjar syndrome (congenital nonhemolytic icterus)
- Gilbert syndrome

Day 7 and later

- Herpes simplex
- Pyloric stenosis
- Hypothyroidism
- Neonatal giant cell hepatitis

- Infection (usually acquired in neonatal period)
- Bile duct atresia
- Galactosemia
- Choledochal cyst

An exchange transfusion replaces the neonate's blood with fresh blood (less than 48 hours old), removing some of the unconjugated bilirubin in serum. Possible indications for exchange transfusions include hydrops fetalis, polycythemia, erythroblastosis fetalis, marked reticulocytosis, drug toxicity, and jaundice that develops within the first 6 hours after birth.

Other therapy for excessive bilirubin levels may include albumin administration (1 g/kg of 25% salt-poor albumin), which provides additional albumin for binding unconjugated bilirubin. This may be done 1 to 2 hours before exchange or as a substitute for a portion of the plasma in the transfused blood.

Drug therapy, which is rare, usually consists of phenobarbital administered to the mother before delivery and to the neonate several days after delivery. This drug stimulates the hepatic glucuronide-conjugating system.

Special considerations

- Assess and record the neonate's jaundice, and note the time it began. Report the jaundice and serum bilirubin levels immediately.
- Reassure parents that most neonates experience some degree of jaundice. Explain

  hyperbilirubinemia, its causes, diagnostic tests, and treatment. Also, explain that the neonate's stool contains some bile and may be greenish.

For the neonate receiving phototherapy:

- Keep a record of how long each bilirubin light bulb is in use because these bulbs require frequent changing for optimum effectiveness.

- Undress the neonate, so his entire body surface is exposed to the light rays. Keep him 18″ to 30″ (46 to 76 cm) from the light source. Protect his eyes with shields that filter the light.
- Monitor and maintain the neonate's body temperature; high and low temperatures predispose him to kernicterus. Remove the neonate from the light source every 3 to 4 hours and take off the eye shields. Allow his parents to visit and feed him.
- The neonate usually shows a decrease in serum bilirubin level 1 to 12 hours after the start of phototherapy. When the neonate's bilirubin level is less than 10 mg/dl and has been decreasing for 24 hours, discontinue phototherapy as ordered. Resume therapy, as ordered, if serum bilirubin increases several milligrams per deciliter, as it often does because of a rebound effect.

For the exchange transfusions:

- Prepare the neonate warmer and tray before the transfusion. Try to keep the neonate quiet. Give him nothing by mouth for 3 to 4 hours before the procedure.
- Check the blood to be used for the exchange—type, Rh, age. Keep emergency equipment (resuscitative and intubation equipment, and oxygen) available. During the procedure, monitor respiratory and heart rates every 15 minutes; check the neonate's temperature every 30 minutes. Continue to monitor vital signs every 15 to 30 minutes for 2 hours.
- Measure intake and output. Observe for cord bleeding and complications, such as hemorrhage, hypocalcemia, sepsis, and shock. Report serum bilirubin and Hb levels. Bilirubin levels may rise, as a result of a rebound effect, within 30 minutes after transfusion, necessitating repeat transfusions.

PREVENTION

- *Advise your patient to maintain oral intake and to not skip meals, because fasting stimulates the conversion of heme to bilirubin.*
- *Administer Rh_o(D) immunoglobulin (human), as indicated, to an Rh-negative mother after*

amniocentesis, or—to prevent hemolytic disease in subsequent children—to an Rh-negative mother during the third trimester, after the birth of an Rh-positive neonate, or after spontaneous or elective abortion.

Erythroblastosis fetalis

Erythroblastosis fetalis, a hemolytic disease of the fetus and neonate, stems from an incompatibility of fetal and maternal blood, resulting in maternal antibody activity against fetal red cells. Intrauterine transfusions can save 40% of fetuses with erythroblastosis. However, in severe, untreated erythroblastosis fetalis, the prognosis is poor, especially if kernicterus develops. About 70% of these neonates die, usually within the first week of life; survivors inevitably develop pronounced neurologic damage—sensory impairment, mental deficiencies, and cerebral palsy. Severely affected fetuses that develop hydrops fetalis (the most severe form of this disorder, associated with profound anemia and edema) are commonly stillborn; even if they're delivered live, they rarely survive longer than a few hours.

Causes and incidence

Although more than 60 red cell antigens can stimulate antibody formation, erythroblastosis fetalis usually results from Rh isoimmunization—a condition that develops in about 7% of all pregnancies in the United States. Before the development of Rho(D) immuneglobulin, this condition was an important cause of kernicterus and neonatal death. (See *ABO incompatibility*.)

During her first pregnancy, an Rh-negative female becomes sensitized (during delivery or abortion) by exposure to Rh-positive fetal blood antigens inherited from the father. A female may also become sensitized from receiving blood transfusions

with alien Rh antigens, causing agglutinins to develop; from inadequate doses of Rh_o(D); or from failure to receive Rh_o(D) after significant fetal-maternal leakage from abruptio placentae. Subsequent pregnancy with an Rh-positive fetus provokes increasing amounts of maternal agglutinating antibodies to cross the placental barrier, attach to Rh-positive cells in the fetus, and cause hemolysis and anemia. To compensate for this, the fetus steps up the production of red blood cells (RBCs), and erythroblasts (immature RBCs) appear in the fetal circulation. Extensive hemolysis results in the release of large amounts of unconjugated bilirubin, which the liver is unable to conjugate and excrete, causing hyperbilirubinemia and hemolytic anemia. (See *What happens in Rh isoimmunization,* page 1214.)

ABO INCOMPATIBILITY

ABO incompatibility, a form of fetomaternal incompatibility, occurs between mother and fetus in about 25% of all pregnancies, with highest incidence among blacks. In about 1% of this number, it leads to hemolytic disease of the neonate. Although ABO incompatibility is more common than Rh isoimmunization, it's less severe. Low antigenicity of fetal or neonatal ABO factors may account for the milder clinical effects.

Blood group	Antigens on RBCs	Antibodies in serum	Most common incompatible groups
A	A	Anti-B	Mother A, neonate B or AB
B	B	Anti-A	Mother B, neonate A or AB
AB	A and B	No antibodies	Mother AB, neonate (no incompatibility)
O	No antigens	Anti-A and anti-B	Mother O, neonate A or B

Each blood group has specific antigens on red blood cells (RBCs) and specific antibodies in serum. Maternal antibodies form against fetal cells when blood groups differ. Neonates with group A blood, born of group O mothers, account for approximately 50% of all ABO incompatibilities. Unlike Rh isoimmunization, which always follows sensitization in a previous pregnancy, ABO incompatibility is likely to develop in a firstborn neonate.

Clinical effects of ABO incompatibility include jaundice, which usually appears in the neonate in 24 to 48 hours, mild anemia, and mild hepatosplenomegaly.

Diagnosis is based on clinical symptoms in the neonate, the presence of ABO incompatibility, a weak to moderate positive Coombs' test, and elevated serum bilirubin levels. Cord hemoglobin and indirect bilirubin levels indicate the need for an exchange transfusion. This type of transfusion is done with blood of the same group and Rh type as that of the mother. Because infants with ABO incompatibility respond so well to phototherapy, an exchange transfusion is seldom necessary.

Complications

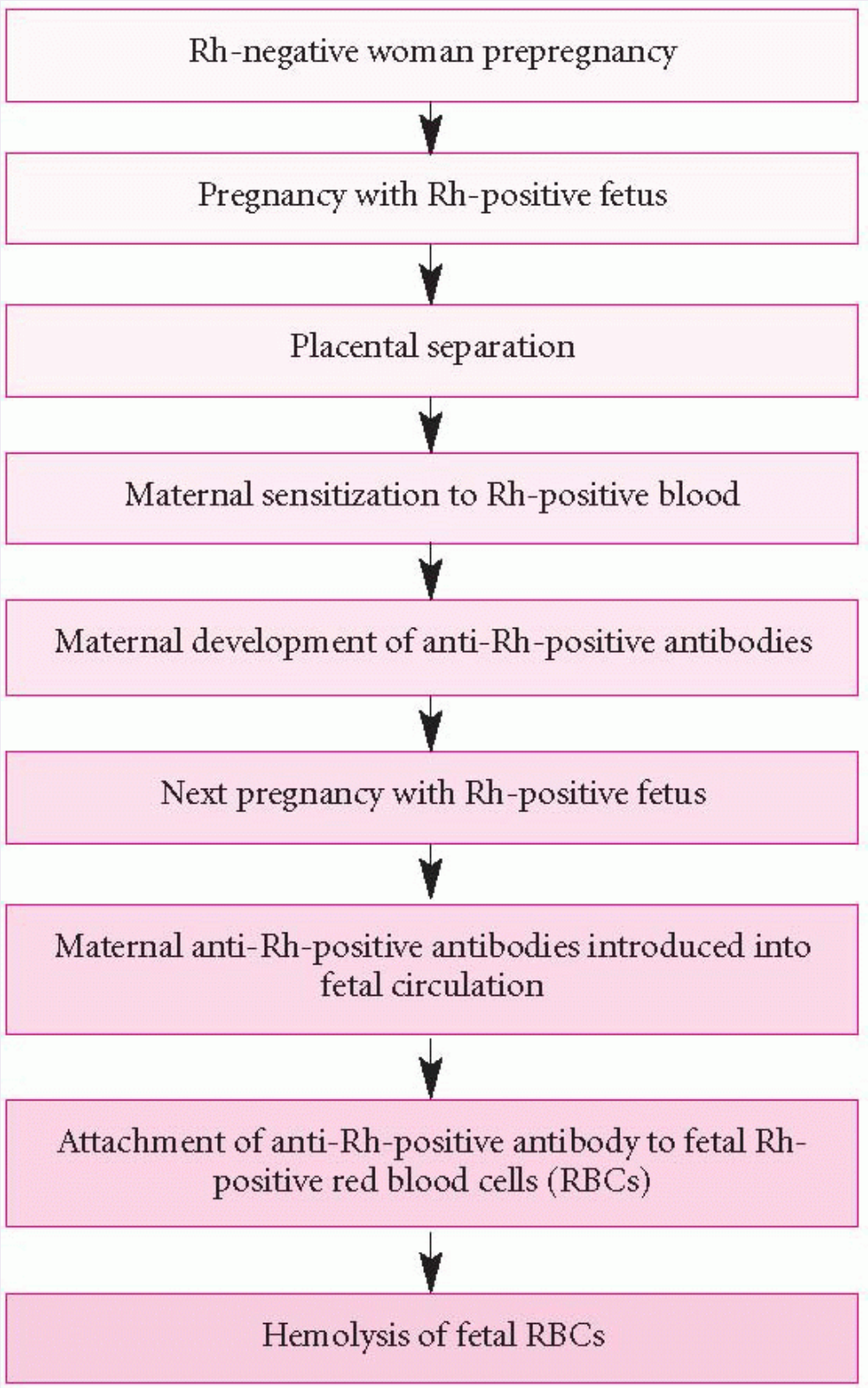
- Cerebral palsy
- Kernicterus
- Mental deficiencies
- Sensory impairment

PATHOPHYSIOLOGY

WHAT HAPPENS IN RH ISOIMMUNIZATION

Rh isoimmunization occurs when a pregnant woman who's Rh-negative develops antibodies against an RH-

positive fetus, as shown at right.



Signs and symptoms

Jaundice usually isn't present at birth but may appear as soon as 30 minutes later or within 24 hours. The mildly affected neonate shows mild to moderate hepatosplenomegaly and pallor. In severely affected neonates who survive birth, erythroblastosis fetalis usually produces pallor, edema, petechiae, hepatosplenomegaly, grunting respirations, pulmonary crackles, poor muscle tone, neurologic unresponsiveness, possible heart murmurs, a bilestained umbilical cord, and yellow or meconium-stained amniotic fluid. About 10% of untreated neonates develop kernicterus from hemolytic disease and show symptoms such as anemia, lethargy, poor sucking ability, retracted head, stiff limbs, squinting, a high-pitched cry, and seizures.

Hydrops fetalis causes extreme hemolysis, fetal hypoxia, heart failure (with possible pericardial effusion and circulatory collapse), edema (ranging from mild peripheral edema to anasarca), peritoneal and pleural effusions (with dyspnea and pulmonary crackles), and green- or browntinged amniotic fluid (usually indicating a stillbirth).

Other distinctive characteristics of the neonate with hydrops fetalis include enlarged placenta, marked pallor, hepatosplenomegaly, cardiomegaly, and ascites. Petechiae and widespread ecchymoses are present in severe cases, indicating concurrent disseminated intravascular coagulation. This disorder retards intrauterine growth, so the neonate's lungs, kidneys, brain, and thymus are small, and despite edema, his body size is smaller than that of neonates of comparable gestational age.

Diagnosis

Diagnostic evaluation takes into account both prenatal and neonatal findings:

- maternal history (for erythroblastotic stillbirths, abortions, previously affected children, previous anti-Rh titers)

- blood typing and screening (titers should be taken frequently to determine changes in the degree of maternal immunization)
- paternal blood test (for Rh, blood group, and Rh zygosity)
- history of blood transfusion.

In addition, amniotic fluid analysis may show an increase in bilirubin (indicating possible hemolysis) and anti-Rh titers. Radiologic studies may show edema and, in hydrops fetalis, the halo sign (edematous, elevated, subcutaneous fat layers) and the Buddha position (fetus' legs are crossed).

Neonatal findings indicating erythroblastosis fetalis include:

- direct Coombs' test of umbilical cord blood to measure RBC (Rh-positive) antibodies in the neonate (positive only when the mother is Rh-negative and the fetus is Rh-positive)
- decreased cord hemoglobin (Hb) level (less than 10 g), signaling severe disease
- many nucleated peripheral RBCs.

Treatment

Treatment depends on the degree of maternal sensitization and hemolytic disease's effects on the fetus or neonate.

- Percutaneous umbilical cord sampling allows for assessment of fetal well-being and direct transfusion, if necessary.
- An intrauterine-intraperitoneal transfusion is performed when amniotic fluid analysis suggests the fetus is severely affected and delivery is inappropriate because of fetal immaturity. A transabdominal puncture under fluoroscopy into the fetal peritoneal cavity allows infusion of group O, Rh-negative blood. This may be repeated every 2 weeks until the fetus is mature enough for delivery. Some facilities can perform percutaneous umbilical blood sampling to provide transfusion.
- Planned delivery is usually done 2 to 4 weeks before term date, depending on maternal history, serologic tests, and amniocentesis; labor may be induced from the 34th to 38th week of gestation. During

labor, the fetus should be monitored electronically; capillary blood scalp sampling determines acid-base balance. Any indication of fetal distress necessitates immediate cesarean delivery.

- An exchange transfusion removes anti-body-coated RBCs and prevents hyperbilirubinemia through removal of the neonate's blood and replacement with fresh group O, Rh-negative blood.
- Albumin infusion aids in the binding of bilirubin, reducing the chances of hyperbilirubinemia.
- Phototherapy by exposure to ultraviolet light reduces bilirubin levels.

Neonatal therapy for hydrops fetalis consists of maintaining ventilation by intubation, oxygenation, and mechanical assistance, when necessary; and removal of excess fluid to relieve severe ascites and respiratory distress. Other appropriate measures include an exchange transfusion and maintenance of the neonate's body temperature.

Gamma globulin that contains anti-Rhpositive antibody (Rh_o[D]) can provide passive immunization, which prevents maternal Rh isoimmunization in Rh-negative females. However, it's ineffective if sensitization has already resulted from a previous pregnancy, abortion, or transfusion. (See *Prevention of Rh isoimmunization,* page 1216.)

Special considerations

Structure the care plan around close maternal and fetal observation, explanations of diagnostic tests and therapeutic measures, and emotional support.

PREVENTION

PREVENTION OF RH ISOIMMUNIZATION

Administering Rh_o (D) immune human globulin to an unsensitized Rh-negative mother immediately after the birth of an Rh-positive neonate or after a spontaneous or elective abortion prevents complications in later pregnancies.

The following patients should be screened for Rh isoimmunization or irregular antibodies:

- all Rh-negative mothers during their first prenatal visit, and at 28 weeks' gestation
- all Rh-positive mothers with histories of transfusion, a jaundiced baby, stillbirth, cesarean birth, induced abortion, placenta previa, or abruptio placentae.

- Reassure the parents that they aren't to blame for having a child with erythroblastosis fetalis. Encourage them to express their fears concerning possible complications of treatment.
- Before intrauterine transfusion, explain the procedure and its purpose. Before the transfusion, obtain a baseline fetal heart rate through electronic monitoring. Afterward, carefully observe the mother for uterine contractions and fluid leakage from the puncture site. Monitor fetal heart rate for tachycardia or bradycardia.
- During exchange transfusion, maintain the neonate's body temperature by placing him under a heat lamp or overhead radiant warmer. Keep resuscitative and monitoring equipment handy and warm blood before transfusion.
- Watch for complications of transfusion, such as lethargy, muscular twitching, seizures, dark urine, edema, and change in vital signs. Watch for postexchange serum bilirubin levels that are usually 50% of preexchange levels (although these levels may rise to 70% to 80% of pre-exchange levels due to rebound effect). Within 30 minutes of transfusion, bilirubin may rebound, requiring repeat exchange transfusions.
- Measure intake and output. Observe for cord bleeding and complications, such as hemorrhage, hypocalcemia, sepsis, and shock. Report serum bilirubin and Hb levels.
- To promote normal parental bonding, encourage parents to visit and to help care for the neonate as often as possible.
- To prevent hemolytic disease in the neonate, evaluate all pregnant females for possible Rh incompatibility. Administer Rh_o(D) I.M., as