Complications of Pregnancy

Objectives

After studying this chapter, you should be able to:

1. Describe the hemorrhagic conditions of early pregnancy, including spontaneous abortion, ectopic pregnancy, and gestational trophoblastic disease.
2. Explain disorders of the placenta, such as placenta previa and abruptio placentae, that may result in hemorrhagic conditions of late pregnancy.
3. Discuss the effects and management of hyperemesis gravidarum.
4. Describe the development and management of hypertensive disorders of pregnancy.
5. Compare etiology, fetal and neonatal complications, and management of Rh and ABO incompatibility.
6. Explain nursing considerations for each complication of pregnancy.

Go to your Student CD-ROM for Review Questions keyed to these Objectives.

Definitions

Abortion  Pregnancy that ends before 20 weeks’ gestation, either spontaneously or electively. Miscarriage is a lay term for a spontaneous abortion that is frequently used by professionals.

Abruptio Placentae  Premature separation of a normally implanted placenta.

Antiphospholipid Antibodies  Autoimmune antibodies that are directed against phospholipids in cell membranes. It is associated with recurrent spontaneous abortion, fetal loss, and severe preeclampsia.

Bicornuate (Bicornate) Uterus  Malformed uterus having two horns.

Cerclage  Encircling the cervix with suture to prevent recurrent spontaneous abortion caused by early cervical dilation.

Dilation and Curettage (D&C)  Stretching the cervical os to permit suctioning or scraping of the walls of the uterus. The procedure is performed in abortion, to obtain samples of uterine lining tissue for laboratory examination, and during the postpartum period to remove retained fragments of placenta.

Dilation and Evacuation (D&E)  Wide cervical dilation followed by mechanical destruction and removal of fetal parts from the uterus. After complete removal of the fetus, a vacuum curet is used to remove the placenta and remaining products of conception.

Eclampsia  Form of hypertension of pregnancy complicated by generalized (grand mal) seizures.

Ectopic Pregnancy  Implantation of a fertilized ovum in any area other than the uterus; the most common site is the fallopian tube.

Erythroblastosis Fetalis  Agglutination and hemolysis of fetal erythrocytes resulting from incompatibility between maternal and fetal blood. In most cases the fetus is Rh-positive and the mother is Rh-negative.

Gestational Trophoblastic Disease  Spectrum of diseases that includes both benign hydatidiform mole and gestational trophoblastic tumors, such as invasive moles and choriocarcinoma.

Hydatidiform Mole  Abnormal pregnancy resulting from proliferation of chorionic villi that give rise to multiple cysts and rapid growth of the uterus.

Hypovolemic Shock  Acute peripheral circulatory failure resulting from loss of circulating blood volume.

Kernicterus  Staining of brain tissue caused by accumulation of unconjugated bilirubin in the brain. Bilirubin...
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DEFINITIONS—cont'd

encephalopathy is the brain damage that results from these deposits.
Laparoscopy Insertion of an illuminated tube into the abdominal cavity to visualize contents, locate bleeding, and perform surgical procedures.
Linear Salpingostomy Incision along the length of a fallopian tube to remove an ectopic pregnancy and preserve the tube.
Maceration Discoloration and softening of tissues and eventual disintegration of a fetus that is retained in the uterus after its death.
Perinatologist Physician who specializes in the care of the mother, fetus, and infant during the perinatal period (from about the twentieth week of pregnancy to 4 weeks after childbirth).
Preeclampsia A hypertensive disorder of pregnancy characterized by hypertension and proteinuria.
Salpingectomy Surgical removal of a fallopian tube.
Vacuum Curettage (Vacuum Aspiration) Removal of the uterine contents by application of a vacuum through a hollow curet or cannula introduced into the uterus.
Vasoconstriction Narrowing of the lumen of blood vessels.

Although childbearing is a normal process, numerous maternal and fetal adaptations must occur in an orderly sequence. If problems develop in these physiologic processes, complications may arise that threaten the well-being of the expectant mother, the fetus, or both. A nurse-midwife or family physician may manage some mild conditions, or the woman may be referred to an obstetrician or a perinatologist for management of severe complications.

Nurses who work at a primary care site or perinatal center frequently fill the role of case manager or coordinator of services provided for the woman. Often the nurse is the only consistent provider involved in the woman’s care and therefore is the person on whom the woman relies to guide her through the system.

Conditions that complicate pregnancy are divided into two broad categories: (1) those that are related to pregnancy and are not seen at other times and (2) those that could occur at any time but when they occur concurrently with pregnancy may complicate its course. Concurrent conditions that affect pregnancy are covered in Chapter 26.

The most common pregnancy-related complications are hemorrhagic conditions that occur in early pregnancy, hemorrhagic complications of the placenta in late pregnancy, hyperemesis gravidarum (HEG), hypertensive disorders of pregnancy, and blood incompatibilities.

HEMORRHAGIC CONDITIONS OF EARLY PREGNANCY

The three most common causes of hemorrhage during the first half of pregnancy are abortion, ectopic pregnancy, and gestational trophoblastic disease.

Abortion

Abortion is the loss of pregnancy before the fetus is viable, or capable of living outside the uterus. The medical consensus today is that a fetus of less than 20 weeks’ gestation or one weighing less than 500 g is not viable. Ending of pregnancy before this time is considered an abortion. Abortion may be either spontaneous or induced. Abortion is an accepted medical term for either a spontaneous or induced ending of pregnancy, although the lay term miscarriage is becoming an accepted medical term to denote spontaneous abortion. Induced abortion is described in Chapter 33.

SPONTANEOUS ABORTION

Spontaneous abortion is a termination of pregnancy without action taken by the woman or another person.

INCIDENCE AND ETIOLOGY. Determining the exact incidence of spontaneous abortion is difficult because many unrecognized losses occur in early pregnancy, but it averages about 15% to 20% with any pregnancy. The incidence of spontaneous abortion increases with parental age. The incidence is 12% for women younger than 20 years, rising to 26% for women older than 40 years. Paternal age younger than 20 years is associated with a spontaneous abortion rate of 12%, rising to 20% for fathers older than 40 years. Most spontaneous abortions occur in the first 12 weeks of pregnancy, with the rate declining rapidly thereafter (Cunningham et al., 2001).

The most common cause of spontaneous abortion is severe congenital abnormalities that are often incompatible with life. Chromosomal abnormalities account for about 50% to 60% of early spontaneous abortions. Additional causes include maternal infections such as syphilis, listeriosis, toxoplasmosis, brucellosis, rubella, and cytomegalic inclusion disease. Intraabdominal infections also increase the risk. Maternal endocrine disorders such as hypothyroidism and abnormalities of the reproductive organs have also been implicated. Still other women who have repeated early pregnancy losses appear to have immunologic factors that play a role in their higher-than-expected spontaneous abortion incidence. Anatomic defects of the uterus or cervix may contribute to pregnancy loss at any gestational age (Branch & Scott, 2003; Cunningham et al., 2001).

Spontaneous abortion is divided into six subgroups: threatened, inevitable, incomplete, complete, missed, and recurrent. Figure 25-1 illustrates threatened, inevitable, and incomplete abortion.

THREATENED ABORTION

Clinical Manifestations. The first sign of threatened abortion is vaginal bleeding, which is rather common during early pregnancy. One third of pregnant women experi-
ence “spotting” or bleeding in early pregnancy, and up to 50% of these pregnancies end in spontaneous abortion. Pregnancies complicated by early bleeding that do not end with a spontaneous abortion are more likely to have further complications during late pregnancy such as prematurity, a small-for-gestational age infant, abnormal presentation, or perinatal asphyxia (Branch & Scott, 2003; Cunningham, et al., 2001; Lu & Hobel, 2004).

Vaginal bleeding may be followed by rhythmic uterine cramping, persistent backache, or feelings of pelvic pressure. These symptoms increase the chance that the threatened abortion will progress to inevitable abortion.

Therapeutic Management. Bleeding during the first half of pregnancy should be considered a threatened abortion, and women must be advised to notify their physician or nurse-midwife if brownish or red vaginal bleeding is noted. When a woman reports bleeding in early pregnancy, the nurse obtains a detailed history that includes length of gestation (or first day of her last menstrual period) and the onset, duration, and amount of vaginal bleeding. Any accompanying discomfort, such as cramping, backache, or abdominal pain also is noted. Ultrasound examination helps to verify if the embryo or fetus is present and alive and the approximate gestational age. Determining if the woman’s chorionic gonadotropin (β-hCG) levels are normal for the estimated gestational age provides added information about whether the pregnancy is likely to continue.

Vaginal ultrasound examination is performed to determine whether a fetus is present and, if so, whether it is alive. Maternal serum β-hCG and progesterone levels provide added information about the viability of the pregnancy. No evidence exists to support physical activity restrictions to stop spontaneous abortion. The woman may be advised to limit sexual activity until bleeding has ceased. The woman is instructed to count the number of perineal pads used and to note the quantity and color of blood on the pads. She should also look for evidence of tissue passage, which would indicate progression beyond a threatened abortion.

Therapeutic Management. Natural expulsion of uterine contents is common in inevitable abortion. Vacuum curettage is used to clean out the uterus if the natural process is ineffective or incomplete. If the pregnancy is more advanced or if bleeding is excessive, a dilation and curettage (D&C) may be needed. Intravenous (IV) sedation or other anesthesia provides pain management for the procedure.
INCOMPLETE ABORTION

Clinical Manifestations. Incomplete abortion occurs when some but not all of the products of conception are expelled from the uterus. The major manifestations are active uterine bleeding and severe abdominal cramping. The cervix is open, and fetal and placental tissue is passed. The products of conception may have been expelled from the uterus but remain in the vagina because of their small size, often no larger than a ping-pong ball if the gestation is very early.

Therapeutic Management. The retained tissue prevents the uterus from contracting firmly, thereby allowing profuse bleeding from uterine blood vessels. Initial treatment should focus on stabilizing the woman cardiovascularly. A blood specimen is drawn for blood type and screen or cross-match, and an IV line is inserted for fluid replacement. When the woman's condition is stable, a D&C usually is performed to remove the remaining tissue. This procedure may be followed by IV administration of oxytocin (Pitocin) or intramuscular administration of methylergonovine (Methergine) to contract the uterus and control bleeding.

A D&C may not be performed if the pregnancy has advanced beyond 14 weeks because of the danger of excessive bleeding. In this case, oxytocin or prostaglandin is administered to stimulate uterine contractions until all products of conception (fetus, membranes, placenta, and amniotic fluid) are expelled.

COMPLETE ABORTION

Clinical Manifestations. Complete abortion occurs when all products of conception are expelled from the uterus. After passage of all products of conception, uterine contractions and bleeding subside and the cervix closes. The uterus feels smaller than the length of gestation would suggest. The symptoms of pregnancy are no longer present, and the pregnancy test becomes negative as hormone levels fall.

Therapeutic Management. Once complete abortion is confirmed, no additional intervention is required unless excessive bleeding or infection develops. The woman should be advised to rest and to watch for further bleeding, pain, or fever. She should not have intercourse until after a follow-up visit with her health care provider. Contraception is discussed at the follow-up visit if she wishes to prevent pregnancy.

MISSED ABORTION

Clinical Manifestations. Missed abortion occurs when the fetus dies during the first half of pregnancy but is retained in the uterus. When the fetus dies, the early symptoms of pregnancy (nausea, breast tenderness, urinary frequency) disappear. The uterus stops growing and decreases in size, reflecting the absorption of amniotic fluid and maceration of the fetus. Vaginal bleeding of a red or brownish color may or may not occur.

Therapeutic Management. An ultrasound examination confirms fetal death by identifying a gestational sac or fetus that is too small for the presumed gestational age. No fetal heart activity can be found. Pregnancy tests for hCG should show a decline in placental hormone production.

In most cases, the woman would expel the contents of the uterus spontaneously, but this is emotionally difficult once she knows her fetus is not living. Therefore her uterus usually is emptied by the most appropriate method for the size when the diagnosis of missed abortion is made. For a first-trimester missed abortion, a D&C can usually be done. If the missed abortion occurs during the second trimester, when the fetus is larger, a D&E may be done or vaginal prostaglandin E2 (PGE2) or misoprostol (Cytotec) may be needed to induce uterine contractions that expel the fetus.

Two major complications of missed abortion are infection and disseminated intravascular coagulation (DIC). If signs exist of uterine infection, such as elevation in temperature, vaginal discharge with a foul odor, or abdominal pain, evacuation of the uterus will be delayed until cultures are obtained and antimicrobial therapy is initiated.

RECURRENT SPONTANEOUS ABORTION

Clinical Manifestations. Recurrent spontaneous abortion usually is defined as three or more spontaneous abortions, although some authorities now use two or more pregnancy losses as the definition. The primary causes of recurrent abortion are believed to be genetic or chromosomal abnormalities and anomalies of the reproductive tract, such as bicornuate uterus or incompetent cervix. Additional causes include an inadequate luteal phase with insufficient secretion of progesterone and immunologic factors that involve increased sharing of human leukocyte antigens by the sperm and ovum of the man and woman who conceived. The theory is that because of this sharing the woman’s immunologic system is not stimulated to produce blocking antibodies that protect the embryo from maternal immune cells or other damaging antibodies. Systemic diseases such as lupus erythematosus and diabetes mellitus have been implicated in recurrent abortions. Reproductive infections and some sexually transmitted diseases are also associated with recurrent abortions.

Therapeutic Management. The first step in management of recurrent spontaneous abortion is a thorough examination of the reproductive system to determine whether anatomic defects are the cause. If the cervix and uterus are normal, the woman and her partner are usually referred for genetic screening to identify chromosomal factors that would increase the possibility of recurrent abortions.

Additional therapeutic management of recurrent pregnancy loss depends on the cause. For instance, treatment may involve assisting the woman to develop a regimen to maintain normal blood glucose if diabetes mellitus is a factor. Supplemental hormones may be given if her progesterone or other hormone levels are lower than normal. Additional therapeutic management of recurrent pregnancy loss depends on the cause. For example, antimicrobials are prescribed for the woman with infection, or hormone-related drugs may be prescribed if imbalance preventing normal fetal implantation and support is found.

Recurrent spontaneous abortion may be caused by cervical incompetence, an anatomic defect that results in painless dilation of the cervix in the second trimester. In this sit-
uation the cervix may be sutured to keep it from opening in a cerclage procedure. The cerclage is most likely to be successful if done before much cervical dilation or bulging of the membranes through the cervix has occurred. Sutures may be removed near term in preparation for vaginal delivery, or they may be left in place if a cesarean birth is planned. Prophylactic antimicrobials are ordered if the woman is at increased risk for infection.

Rh immune globulin (RhoGAM) is given to the unsensitized Rh (D)-negative woman to prevent development of anti-Rh antibodies (see p. 654). A microdose (50 mcg) is given to the woman whose fetus is less than 13 weeks’ gestational age at the time of the abortion.

NURSING CONSIDERATIONS. Nurses must consider the psychological needs of the woman experiencing spontaneous abortion. Vaginal bleeding is frightening, and waiting and watching are often difficult (although possibly the only treatment recommended). Many women and their families feel an acute sense of loss and grief with spontaneous abortion. Grief often includes feelings of guilt and speculation about whether the woman could have done something to prevent the loss. Nurses may help by emphasizing that abortions usually occur as the result of factors or abnormalities that could not be avoided.

Anger, disappointment, and sadness are commonly experienced emotions, although the intensity of the feelings may vary. For many people the fetus has not yet taken on specific physical characteristics, but they grieve for their fantasies of the unseen, unborn child. The couple may want to express their feelings of sadness but may feel that family, friends, and often health personnel are uncomfortable or unable to provide emotional support after early pregnancy loss.

Recognizing the meaning of the loss to each woman and her significant others is important. Nurses must listen carefully to what the woman says and observe how she behaves. Nurses must convey acceptance of the feelings expressed or demonstrated. A couple should remain together as much as possible. Providing information and simple brief explanations of what has occurred and what will be done facilitates the family’s ability to grieve.

The family should realize that grief may last from 6 months to a year, or even longer. Family support, knowledge of the grief process, spiritual counselors, and the support of other bereaved couples may provide needed assistance during this time. Chapter 24 provides additional information about pregnancy loss and grief.

DISSEMINATED INTRAVASCULAR COAGULATION

DIC, also called consumptive coagulopathy, is a life-threatening defect in coagulation that may occur with several complications of pregnancy. DIC is not limited to obstetric conditions. While anticoagulation is occurring, inappropriate coagulation also is occurring in the microcirculation. The second result of DIC is that tiny clots form in the tiny blood vessels, blocking blood flow to the organs and causing ischemia.

Something in one of these disease processes initiates clotting mechanisms inappropriately. The first result is a consumption of plasma factors including platelets, fibrinogen, prothrombin, factor V, and factor VIII. When these plasma factors are consumed, the circulating blood is then deficient in clotting factors and unable to clot. Fibrin degradation products accumulate and further interfere with coagulation.

Diseases that cause DIC fall into three major groups:

- Infusion of tissue thromboplastin into the circulation, which consumes, or “uses up,” other clotting factors such as fibrinogen and platelets. Abruptio placentae and prolonged retention of a dead fetus cause this because the placenta is a rich source of thromboplastin.
- Conditions characterized by endothelial damage. Severe preeclampsia and the HELLP syndrome (p. 653) are characterized by endothelial damage.
- Nonspecific effects of some diseases. Diseases such as maternal sepsis or amniotic fluid embolism (see Chapter 27) are in this category.

DIC allows excess bleeding to occur from any vulnerable area, such as IV sites, incisions, or the gums or nose, and from expected sites such as the site of placental attachment during the postpartum period.

Laboratory studies help establish a diagnosis. Fibrinogen and platelets usually are decreased, prothrombin time (PT) and activated partial thromboplastin time (aPTT) may be prolonged, and fibrin degradation products, the most sensitive measurement, are increased. A newer test, the D-dimer study, which normally has negative results, confirms fibrin split products (FSP) and is presumptive for DIC when results are positive.

The priority in treatment of DIC is to correct the cause. In the case of a missed abortion, delivery of the fetus and placenta ends production of thromboplastin, which is fueling the process. Blood replacement products, such as whole blood, packed red blood cells, and cryoprecipitate, are administered as needed to maintain the circulating volume and to transport oxygen to body cells.

NURSING CONSIDERATIONS

When caring for a woman who has any of the disorders that increase her risk for having DIC, the nurse should observe for bleeding from unexpected sites. Sites for IV insertion or lab work, nosebleeds, or spontaneous bruising may be early indicators of DIC and should be reported. Also, if her coagulation studies are severely abnormal, an epidural block may be contraindicated because of possible bleeding into the spinal canal, so other types of labor pain management should be anticipated.

CHECK YOUR READING

1. What are the signs of threatened abortion, and how do they differ from those of inevitable abortion?
2. What are the major causes of recurrent spontaneous abortion?
3. How can nurses intervene for the grief families experiencing as a result of early pregnancy loss?
4. What is DIC?
Ectopic Pregnancy

Ectopic pregnancy is an implantation of a fertilized ovum in an area outside the uterine cavity. Although implantation can occur in the abdomen or cervix, more than 98% of ectopic pregnancies are in the fallopian tube (Heard & Buster, 2003). Figure 25-2 illustrates common sites of ectopic implantation.

Ectopic pregnancy has been called “a disaster of reproduction” for two reasons:

- It remains a significant cause of maternal death from hemorrhage.
- It reduces the woman’s chance of subsequent pregnancies because of damage or destruction of a fallopian tube.

INCIDENCE AND ETIOLOGY

Ectopic pregnancies have increased in the United States since 1970 from a rate of 4.5 per 1000 pregnancies to 19.7 per 1000 pregnancies. The increase in incidence is attributed to the growing number of women of childbearing age who experience scarring of the fallopian tubes because of pelvic infection, inflammation, or surgery. Additionally, sensitive tests that identify pregnancy earlier and transvaginal ultrasound allow diagnosis of some pregnancies within the fallopian tube that previously might have resolved spontaneously before diagnosis (Heard & Buster, 2003).

Pelvic infection often is caused by Chlamydia or Neisseria gonorrhoeae. A failed tubal ligation, even if undergone many years before, and a history of previous ectopic pregnancy also increase the risk for an ectopic pregnancy that implants in the fallopian tube. Greater incidences of ectopic pregnancies occur in women who conceived with assisted reproduction, most likely related to the tubal factors that contributed to infertility. Contraception such as intrauterine contraceptive devices or low-dose progesterone agents is associated with increased risk of ectopic pregnancy (Cunningham et al., 2001; Heard & Buster, 2003).

Additional causes of ectopic pregnancy are delayed or premature ovulation, with the tendency of the fertilized ovum to implant before arrival in the uterus, and altered tubal motility in response to changes in estrogen and progesterone levels. Multiple induced abortions increase the risk for tubal pregnancy, possibly because of salpingitis (infection of the fallopian tube) that has occurred after induced abortion (Box 25-1). Regardless of the cause of tubal pregnancy, the effect is that transport of the fertilized ovum through the fallopian tube is hampered.

CLINICAL MANIFESTATIONS

The classic signs of ectopic pregnancy include the following:

- Missed menstrual period
- Abdominal pain
- Vaginal “spotting”

More subtle signs and symptoms depend on the site of implantation. If implantation occurs in the distal end of the fallopian tube, the signs and symptoms might not be recognized early. One sign of distal tubal pregnancy is that the menstrual period is missed, but the pain and vaginal bleeding might be minor or absent. The pain often is described as cramping and is felt in the lower abdomen. If the pregnancy is near the entrance of the tube, abdominal pain can be mild or even absent.

Additional signs and symptoms of tubal pregnancy include:

- Pelvic or shoulder pain
- Faintness
- Nausea and vomiting
- Hematometra (blood in the uterus)
- Hemoperitoneum (blood in the abdominal cavity)

If the ectopic pregnancy is ruptured, there might be an acute abdomen with hypotension, tachycardia, and shock. The treatment for ectopic pregnancy is surgical removal of the pregnancy, followed by medical or surgical treatment of the underlying cause.
fallopian tube, which can contain the growing embryo longer, the woman may at first exhibit the usual early signs of pregnancy and consider herself to be normally pregnant. Several weeks into the pregnancy, intermittent abdominal pain and small amounts of vaginal bleeding occur that initially could be mistaken for threatened abortion. Because routine ultrasound examination in early pregnancy is common, however, it is not unusual to diagnose an ectopic pregnancy before onset of symptoms.

If implantation has occurred in the proximal end of the fallopian tube, rupture of the tube may occur within 2 to 3 weeks of the missed period because the tube is narrow in this area. Symptoms include sudden, severe pain in one of the lower quadrants of the abdomen as the tube tears open and the embryo is expelled into the pelvic cavity, often with profuse hemorrhage. Radiating pain under the scapula may indicate bleeding into the abdomen caused by phrenic nerve irritation. Hypovolemic shock is a major concern because systemic signs of shock may be rapid and extensive without external bleeding.

**DIAGNOSIS**

The combined use of transvaginal ultrasound examination (see Chapter 10) and determination of the beta subunit of human chorionic gonadotropin (β-hCG) are helpful in early detection of ectopic pregnancy. An abnormal pregnancy is suspected if hCG is present but at lower levels than expected. If a gestational sac cannot be visualized when hCG is present, a diagnosis of ectopic pregnancy may be made with great accuracy. Visualization of an intrauterine pregnancy, however, does not absolutely rule out an ectopic pregnancy. A woman may have an intrauterine pregnancy and concurrently have an ectopic pregnancy.

The use of sensitive pregnancy tests, maternal serum progesterone levels, and high-resolution transvaginal ultrasound has largely eliminated invasive tests for ectopic pregnancy. Laparoscopy (examination of the peritoneal cavity by means of a laparoscope) occasionally may be necessary to diagnose rupture of an ectopic pregnancy. A characteristic bluish swelling within the tube is the most common finding.

**THERAPEUTIC MANAGEMENT**

Management of tubal pregnancy depends on whether the tube is intact or ruptured. Medical management may be possible if the tube is unruptured. The goal of medical management is to preserve the tube and improve the chance of future fertility. The chemotherapeutic agent methotrexate (a folic acid antagonist) is used to inhibit cell division in the developing embryo.

Surgical management of a tubal pregnancy that is unruptured may involve a linear salpingostomy to salvage the tube (Figure 25-3). Linear salpingostomy also may be attempted if the tube is ruptured but damage to the tube is minimal. Salvaging the tube is particularly important to women concerned about future fertility.

When ectopic pregnancy results in rupture of the fallopian tube, the goal of therapeutic management is to control the bleeding and prevent hypovolemic shock. When the

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**Figure 25-3**  ■ Linear salpingostomy.
woman’s cardiovascular status is stable, removal of the tube (salpingectomy) with ligation of bleeding vessels may be required. With early diagnosis and medical management, salpingectomy has become uncommon in the treatment of ectopic pregnancy.

Rh immune globulin is given to appropriate Rh(D)-negative women.

**NURSING CONSIDERATIONS**

Nursing care focuses on prevention or early identification of hypovolemic shock, pain control, and psychological support for the woman who experiences ectopic pregnancy. Nurses monitor the woman for decreasing hematocrit levels and pain that would indicate a ruptured ectopic pregnancy. Nurses administer analgesics and evaluate their effectiveness so that pain can be controlled.

If methotrexate is used, the nurse must explain adverse side effects, such as nausea and vomiting, and the importance of communicating to the healthcare team any physical changes. Transient abdominal pain during methotrexate therapy occurs, probably because of expulsion of the products of conception from the tube (Heard & Buster, 2003). The woman must also be instructed to refrain from drinking alcohol, which decreases effectiveness, ingesting vitamins that contain folic acid, and having sexual intercourse until hCG is not detectable. If the treatment is successful, this hormone disappears from plasma within 2 to 4 weeks. Maintaining follow-up appointments is essential to identify whether the hCG titer becomes negative and remains negative. Continued presence of hCG in the serum requires follow-up to identify whether the ectopic pregnancy is still present (Cunningham et al., 2001).

The woman and her family will need psychological support to resolve intense emotions that may include anger, grief, guilt, and self-blame. The woman also may be anxious about her ability to become pregnant in the future. Because ectopic pregnancy may occur when a woman has an assisted reproductive technique, she may be more anxious about when she might become pregnant again and if similar risks exist for another pregnancy. The nurses should clarify the physician’s explanation and use therapeutic communication techniques that assist the woman to deal with her anxiety.

**Gestational Trophoblastic Disease**

*Hydatidiform Mole*

Hydatidiform mole is one form of gestational trophoblastic disease that occurs when the trophoblasts (peripheral cells that attach the fertilized ovum to the uterine wall) develop abnormally. As a result of the abnormal growth, the placenta, but not the fetal part of the pregnancy, develops. The condition is characterized by proliferation of villous edema of the chorionic villi. The fluid-filled villi form grapelike clusters of tissue that can rapidly grow large enough to fill the uterus to the size of an advanced pregnancy (Figure 25-4). The mole may be complete with no fetus present, or partial, in which fetal tissue or membranes are present.

**INCIDENCE AND ETIOLOGY**

In the United States and Europe, the incidence of hydatidiform mole is 1 in every 1500 to 2000 pregnancies. The rate is higher in Asian countries, reported as 1 in every 120 pregnancies. Age is a factor, with the frequency of molar pregnancies highest at both ends of reproductive life. Women who have had one molar pregnancy have four to five times the risk to have another in a subsequent pregnancy (Berman & Di Saia, & Tiwari, 2004; Li & Karlan, 2003). Persistent gestational trophoblastic disease may undergo malignant change (choriocarcinoma) and may metastasize to distant sites such as the lung, vagina, liver, and brain.

Complete mole is thought to occur when the ovum is fertilized by a sperm that duplicates its own chromosomes while the maternal chromosomes in the ovum are inactivated. In a partial mole, the maternal contribution is usually present but the paternal contribution is doubled, and therefore the karyotype is triploid (69,XXX or 69,XXY). If a fetus is identified with the partial mole, it is grossly abnormal because of the abnormal chromosomal makeup.

**CLINICAL MANIFESTATIONS**

Routine use of ultrasound allows earlier diagnosis of hydatidiform mole, usually before the more severe manifestations of the disorder develop. Possible signs and symptoms of molar pregnancy include the following:

- Elevated levels of hCG
- Characteristic ultrasonographic pattern that shows the vesicles and the absence of a fetal sac or fetal heart activity in a complete molar pregnancy
- A uterus that is larger than one would expect based on the duration of the pregnancy
- Vaginal bleeding, which varies from dark-brown spotting to profuse hemorrhage
- Excessive nausea and vomiting (HEG), which may be related to high levels of hCG from the proliferating trophoblasts
- Early development of preeclampsia, which is rarely diagnosed before 24 weeks in an otherwise normal pregnancy

**DIAGNOSIS**

Measurement of the hCG levels detects the abnormally high levels of the hormone before treatment. Following treatment, hCG levels are measured to determine if they fall and then disappear.

In addition to the characteristic pattern showing the vesicles, ultrasound examination allows a differential diagnosis to be made between two types of molar pregnancies: (1) a partial mole that includes some fetal tissue and membranes and (2) a complete mole that is composed only of enlarged villi but contains no fetal tissue or membranes.

**THERAPEUTIC MANAGEMENT**

Medical management includes two phases: (1) evacuation of the trophoblastic tissue of the mole and (2) continuous follow-up of the woman to detect malignant changes of any remaining trophoblastic tissue. At the same time the woman is treated for any other problems such as preeclampsia or HEG.

Before evacuation, chest radiography, computed tomography (CT), or magnetic resonance imaging (MRI) may be performed to detect metastatic disease. A complete blood count, laboratory assessment of coagulation status, and blood type and screen or cross-match are also necessary in case a transfusion is needed.

The mole usually is removed by vacuum aspiration followed by curettage. After tissue removal, IV oxytocin is given to contract the uterus. Avoiding uterine stimulation with oxytocin before evacuation is important. Uterine contractions can cause trophoblastic tissue to be pulled into large venous sinusoids in the uterus, resulting in embolization of the tissue and respiratory distress (Berman & Di Saia, & Tiwari, 2004). The tissue obtained is sent for laboratory analysis to be made between two types of molar pregnancies: (1) a partial mole that includes some fetal tissue and membranes and (2) a complete mole that is composed only of enlarged villi but contains no fetal tissue or membranes.

Follow-up is critical to detect changes suggestive of trophoblastic malignancy. Follow-up protocol involves evaluation of serum hCG levels every 1 to 2 weeks until undetectable. The test is then repeated every 1 to 2 months for a year. A persistent or rising hCG level suggests continued gestational trophoblastic disease. Pregnancy must be avoided during the 1-year follow-up because it would obscure the evidence of choriocarcinoma. Oral contraceptives are the preferred birth control method (Berman & Di Saia, & Tiwari, 2004).

**NURSING CONSIDERATIONS**

Bleeding and infection are the early possible complications after a molar pregnancy. The nurse should observe the vital signs for an elevated temperature and pulse and observe vaginal bleeding for excessive amount or foul odor.

Women who have had a hydatidiform mole experience emotions similar to those of women who have had any other type of pregnancy loss. In addition, they may be anxious about follow-up evaluations, the possibility of malignant change, and the need to delay pregnancy for at least a year.

**CHECK YOUR READING**

1. Why is ectopic pregnancy sometimes called a “disaster of reproduction”?
2. Why is the incidence of ectopic pregnancy increasing in the United States? How is ectopic pregnancy treated?
3. What is a hydatidiform mole, and why are two phases of treatment necessary?

**Application of the Nursing Process**

**Hemorrhagic Conditions of Early Pregnancy**

Regardless of the cause of early antepartum bleeding, nurses play a vital role in its management. Nurses are responsible for monitoring the condition of the pregnant woman and for collaborating with the physician to provide treatment.

**Assessment**

Confirmation of pregnancy and length of gestation are important initial data to obtain. Physical assessment focuses on determining the amount of bleeding and the description, location, and severity of pain. Estimate the amount of vaginal bleeding by examining linen and peripads. If necessary, make a more accurate estimation by weighing the linen and peripads (1 g weight equals 1 ml volume).

- When asking a woman how much blood she lost at home, ask her to compare the amount lost with a common measure such as a tablespoon or a cup. Ask also how long the bleeding episode lasted and what was done to control the bleeding.

Bleeding may be accompanied by pain. Uterine cramping usually accompanies spontaneous abortion; deep, severe pelvic pain is associated with ruptured ectopic pregnancy. Remember that in ruptured ectopic pregnancy, bleeding may be concealed and pain could be the only symptom.

The woman’s vital signs and urine output give a clue to her cardiovascular status. A rising pulse and respiratory rate and falling urine output are associated with hypovolemia. The blood pressure usually falls late in hypovolemic shock. Check laboratory values for hemoglobin and hematocrit and report abnormal values to the health care provider. Check laboratory values for coagulation factors to identify
CRITICAL THINKING EXERCISE 25-1

All women who have experienced prenatal bleeding and invasive procedures are at increased risk for infection.

Question
What common assumptions do nurses make about those who are at risk for developing infections?

TEACHING MEASURES TO PREVENT INFECTION

The risk for infection is greatest during the first 72 hours after spontaneous abortion or operative procedures. Personal hygiene should include daily showers and careful hand washing before and after changing perineal pads. Perineal pads, applied in a front-to-back direction, should be used instead of tampons until bleeding has subsided. The woman should consult with the health care provider about safe timing of resuming intercourse.

PROVIDING DIETARY INFORMATION

Nutrition and adequate fluid intake help maintain the body’s defense against infection, and the nurse must promote an adequate and culturally sensitive diet. The woman who has a hemorrhagic complication is also at risk for infection. She needs foods that are high in iron to increase hemoglobin and hematocrit values. These foods include liver, red meat, spinach, egg yolks, carrots, and raisins (Anderson, 2004; Shabert, 2004). Foods high in vitamin C include citrus fruits, broccoli, strawberries, cantaloupe, cabbage, and green peppers. Adequate fluid intake (2500 ml per day) promotes hydration after bleeding episodes and maintains digestive processes.

Iron supplementation also frequently is prescribed, and the woman may require information on how to lessen the gastrointestinal upset that many people experience when iron is administered. Less gastric upset is experienced when iron is taken with meals. Iron supplements having a slow release may also be better tolerated. A diet high in fiber and fluid helps reduce the commonly associated constipation.

TEACHING SIGNS OF INFECTION TO REPORT

Ensure that the woman has a thermometer and knows how to use it. Tell her to take her temperature every 8 hours for the first 3 days at home. Teach the woman to seek medical help if her temperature rises above 38° C (100.4° F) or as her physician instructs. She also should report other signs of infection, even if she does not have a fever, such as vaginal discharge with foul odor, pelvic tenderness, or persistent general malaise.

REINFORCING FOLLOW-UP CARE

A variety of follow-up procedures such as repeat ultrasonic examinations or serum hCG levels may be necessary for women with gestational trophoblastic disease such as hydatidiform mole. Immunologic or genetic testing and counseling may be advised for couples having recurrent abortions. All couples who have had a pregnancy loss should be seen and counseled.

At this time acknowledge their grief, which often manifests as anger. Many women have guilt feelings that must be recognized. They often need repeated reassurance that the loss was not a result of anything they did or anything they neglected.

Women who do not desire pregnancy right away will need contraception. Reliable contraception for at least 1 year

added risks for hemorrhage. Identify women who are Rh-negative so that they can receive Rh(D) immune globulin.

Because abortion or hydatidiform mole may be associated with infections, assess the woman for fever, elevated pulse, malaise, and prolonged or malodorous vaginal discharge. Determine the family’s knowledge of needed follow-up care and how to prevent complications such as infection.

Analysis

A variety of collaborative problems or nursing diagnoses should be considered in the woman who has a bleeding disorder of early pregnancy. Collaborative problems such as bleeding and potential for infection are present. Current diagnostic techniques often permit early diagnosis before hemorrhage occurs. A nursing diagnosis that would apply to these early pregnancy disorders is Deficient Knowledge: diagnostic and therapeutic procedures, signs and symptoms of additional complications, dietary measures to prevent infection or reduction in therapeutic drug levels, and importance of follow-up care.

Planning

Goals or expected outcomes for this nursing diagnosis are that the woman will:

- Verbalize understanding of diagnostic and therapeutic procedures
- Verbalize measures to prevent infection
- Verbalize signs of infection to report to the health care provider
- Maintain follow-up care

Interventions

PROVIDING INFORMATION ABOUT TESTS AND PROCEDURES

Women and their families experience less anxiety if they understand what is happening. Explain planned diagnostic procedures, such as transvaginal or transabdominal ultrasonography (see Chapter 10). Include the purpose of the tests, how long they will take, and whether the procedures cause discomfort. Briefly describe the reasons for blood tests such as hCG, hemoglobin, hematocrit, or coagulation factors. Explain that diagnostic and therapeutic measures sometimes must be performed quickly to prevent excessive blood loss. If surgical intervention is necessary, reinforce explanations of the anesthesia professional about planned anesthesia. Obtain needed consents before procedures.
CRITICAL THINKING EXERCISE 25-2

Alice Starkey, a 24-year-old primigravida, had an incomplete abortion at 12 weeks’ gestation. When she was admitted to the hospital, intravenous fluids were administered and blood was taken for blood typing and screen. A vacuum extraction with curettage was performed to remove retained placental tissue. When bleeding subsided, Alice was discharged to go home. Helen Claude, the nurse providing discharge instructions, comments to Alice, “These things happen for the best, and you are so lucky it happened early”; “You can have other children.”

Questions
1. What assumptions has Helen made? How might these affect Alice?
2. Is the comment that Alice can have other children comforting? Why or why not?
3. If the nurse’s response was not helpful, what responses from the nurse would be most helpful for Alice?

also will be essential for women who have had a molar pregnancy. Teach the woman how to use the prescribed contraceptive method correctly to enhance effectiveness (see Chapter 31).

Evaluation
Interventions are judged successful and the goals and expected outcomes are met if the woman does the following:
- Verbalizes understanding of diagnostic and therapeutic procedures
- Verbalize measures to prevent infection
- Verbalizes signs of infection that should be reported to a health care professional
- Helps develop and participate in a plan of follow-up care

HEMORRHAGIC CONDITIONS OF LATE PREGNANCY

After 20 weeks of pregnancy the two major causes of hemorrhage are the disorders of the placenta called placenta previa and abruptio placentae. Abruptio placentae may be further complicated by DIC, which was discussed earlier.

Placenta Previa

Placenta previa is an implantation of the placenta in the lower uterus. As a result the placenta is closer to the internal cervical os than the presenting part (usually the head) of the fetus. The three classifications of placenta previa (total, partial, and marginal) depend on how much of the internal cervical os is covered by the placenta (Figure 25-5). High-resolution ultrasound allows measurement of the distance between the internal cervical os and the lower border of the placenta:
- Marginal (sometimes called low-lying): Placenta is implanted in the lower uterus, but its lower border is more than 3 cm from the internal cervical os.
- Partial: Lower border of the placenta is within 3 cm of the internal cervical os but does not completely cover the os.
- Total: Placenta completely covers internal cervical os.

Margins (total, partial, and marginal) depend on how much of the internal cervical os is covered by the placenta (Figure 25-5). High-resolution ultrasound allows measurement of the distance between the internal cervical os and the lower border of the placenta:

In the United States the incidence of placenta previa averages 1 in 200 births. It is more common in women who have had previous placenta previa, cesarean birth, or pregnancy termination and in older women. The multipara is more likely to have placenta previa than the nullipara. African or Asian ethnicity also increases the risk. Cigarette smoking and cocaine use are personal habits that add to a woman’s risk for a previa.

CLINICAL MANIFESTATIONS

The classic sign of placenta previa is the sudden onset of painless uterine bleeding in the last half of pregnancy. However, many cases of placenta previa are diagnosed by ultrasound examination before any bleeding occurs. Bleeding results from tearing of the placental villi from the uterine wall, resulting in exposure of uterine vessels. Bleeding is painless because it does not occur in a closed cavity and does not cause pressure on adjacent tissue. It may be scanty or profuse, and it may cease spontaneously, only to recur later.

Bleeding may not occur until labor starts, when cervical changes disrupt placental attachment. The admitting nurse may be unsure whether the bleeding is just heavy “bloody show” or a sign of a placenta previa, particularly if the woman had no prenatal care.

If any doubt exists, the nurse never performs a vaginal examination or takes any action that would stimulate uterine activity. Digital examination of the cervical os when a placenta previa is present can cause additional placental separation or tear the placenta itself, causing severe hemorrhage and extreme risk to the fetus. Until the location and position of the placenta are verified by ultrasonography, no manual examinations should be performed, and administration of oxytocin should be postponed to prevent strong contractions that could result in sudden placental separation and rapid hemorrhage.

THERAPEUTIC MANAGEMENT

When the diagnosis of placenta previa is confirmed, medical interventions are based on the condition of the expectant mother and fetus. The woman is evaluated to determine the amount of hemorrhage, and electronic fetal monitoring is initiated to evaluate the fetus. Fetal gestational age is a third consideration.

Options for management include conservative management if the mother’s cardiovascular status is stable and the
fetus is immature and has a reassuring status by ultrasound examination and monitoring. Delaying birth may increase birth weight and maturity and administration of corticosteroids to the mother speeds maturation of the fetal lungs. Antepartal units are often designed to consider the woman’s needs for physical and occupational therapy and for diversion as well as care for her pregnancy complication. Conservative management may take place in the home or hospital.

**HOME CARE.** Criteria for home care include the following (Clark, 2004):
- No evidence of active bleeding is present.
- The woman is able to maintain bed rest at home.
- Home is a reasonable distance from the hospital.
- Emergency systems are available for immediate transport to the hospital 24 hours a day.

Nurses are often responsible for helping the woman and family understand the physician’s plan of care. Nurses help the woman and family develop a workable plan for home care that may include strict bed rest except for going to the bathroom, the presence of another adult to manage the home and be present if an emergency arises, and a procedure to follow if heavy bleeding begins. Teaching also includes emphasizing the importance of (1) assessing color and amount of vaginal discharge or bleeding, especially after each urination or bowel movement, (2) assessing fetal activity (kick counts) daily (see Chapter 10), (3) assessing uterine activity at prescribed intervals, and (4) refraining from sexual intercourse to prevent disruption of the placenta. Home care nurses may be responsible for making daily phone contact to assess the woman’s perception of uterine activity (cramping, regular or sporadic contractions), bleeding, fetal activity, and adherence to the prescribed treatment plan. In addition, they may make home visits for comprehensive maternal-fetal assessments with portable equipment, such as nonstress tests. The woman

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**Figure 25-5** The three classifications of placenta previa.
Abruptio Placentae

Separation of a normally implanted placenta before the fetus is born (called **abruptio placentae**, **placental abruption**, or **premature separation of the placenta**) occurs in cases of bleeding and formation of a hematoma (clot) on the maternal side of the placenta. As the clot expands, further separation occurs. Hemorrhage may be apparent (vaginal bleeding) or concealed. The severity of the complication depends on the amount of bleeding and the size of the hematoma. If bleeding continues, the hematoma expands and obliterates intervillous spaces. Fetal vessels are disrupted as placental separation occurs, resulting in fetal and maternal bleeding.

Abruptio placentae is a dangerous condition for both the pregnant woman and the fetus. The major dangers for the woman are hemorrhage and consequent hypovolemic shock and clotting abnormalities (see discussion of DIC, p. 626). The major dangers for the fetus are asphyxia, excessive blood loss, and prematurity.

**INCIDENCE AND ETIOLOGY**

Published incidence of abruptio placentae varies but is about 0.5% to 1%. Placental abruption extensive enough to cause the death of the fetus has declined to about 1 in 830 deliveries but accounts for 10% to 15% of perinatal deaths (Cunningham et al., 2001; Kay, 2003).

The cause is unknown; however, several factors that increase the risk have been identified. Maternal use of cocaine, which causes vasoconstriction in the endometrial arteries, is a leading cause of abruptio placentae. Other risk factors include maternal hypertension, maternal cigarette smoking, multigravida status, short umbilical cord, abdominal trauma, premature rupture of the membranes, and history of previous premature separation of the placenta. Maternal age is also associated with abruptio placentae, probably associated with a larger number of births for each mother (Clark, 2004; Cunningham et al., 2001).

**CLINICAL MANIFESTATIONS**

Although evidence of abruptio placentae may be quickly evident, it is not always a dramatic or acute event. Five classic signs and symptoms of abruptio placentae include:

- **Bleeding**, which may be evident vaginally or may be concealed behind the placenta
- **Uterine tenderness** that may be localized at the site of the abruption
- **Uterine irritability** with frequent low-intensity contractions and poor relaxation between contractions
- **Abdominal or low back pain** that may be described as aching or dull
- **High uterine resting tone** identified with use of an intravuterine pressure catheter

Additional signs include hypovolemic shock, fetal distress, and fetal death. Many women have a normal blood pressure, however, because the blood loss masks an undiagnosed hypertensive disorder (Clark, 2004).

Cases of abruptio placentae are divided into two main types: (1) those in which hemorrhage is concealed and (2) those in which hemorrhage is apparent. In either type the placental abruption may be complete or partial. In cases of concealed hemorrhage the bleeding occurs behind the placenta but the margins remain intact, causing formation of a hematoma. The hemorrhage is apparent when bleeding separates or dissects the membranes from the endometrium and blood flows out through the vagina. Figure 25-6 illustrates abruptio placentae with external and concealed bleeding. Apparent bleeding does not always correspond to the actual amount of blood lost, and signs...
of shock (tachycardia, hypotension, pale color, and cold, clammy skin) may be present when little or no external bleeding occurs.

Abdominal pain is also related to the type of separation. It may be sudden and severe when bleeding occurs into the myometrium (uterine muscle) or is intermittent and difficult to distinguish from labor contractions. The uterus may become exceedingly firm (boardlike) and tender, making palpation of the fetus difficult. Ultrasound examination is helpful to rule out placenta previa as the cause of bleeding, but it cannot be used to diagnose abruptio placentae reliably because the separation and bleeding may not be obvious on ultrasonography.

**THERAPEUTIC MANAGEMENT**

Any woman who exhibits signs of abruptio placentae should be hospitalized and evaluated at once. Evaluation focuses on the cardiovascular status of the expectant mother and the condition of the fetus. If the condition is mild and the fetus is immature and shows no signs of distress, conservative management may be initiated. This includes bed rest and may include administration of tocolytic medications to decrease uterine activity. Conservative management is rare, however, owing to the great risks of fetal death and maternal hemorrhage associated with abruptio placentae.

Immediate delivery of the fetus is necessary if signs of fetal compromise exist or if the expectant mother exhibits signs of excessive bleeding, either obvious or concealed. Intensive monitoring of both the woman and the fetus is essential because rapid deterioration of either can occur. Blood products for replacement should be available, and two large-bore IV lines should be started for replacement of fluid and blood.

Women who have experienced abdominal trauma are at increased risk for abruptio placentae. They may be monitored for 24 hours after significant trauma such as a motor vehicle accident, even if they are not having any signs of bleeding, because it may take this long for an abruptio placentae to develop. If they are not having contractions after the trauma and the fetal heart rate pattern is reassuring, monitoring for 4 to 6 hours may be sufficient (Clark, 2004).

**NURSING CONSIDERATIONS**

Abruptio placentae is frightening for a woman. She experiences severe pain and is aware of the danger to herself and to the fetus. She must be carefully assessed for signs of concealed hemorrhage.

If immediate cesarean delivery is necessary, the woman may feel powerless as the health care team hurriedly prepares her for surgery. If at all possible in the time available, nurses must explain anticipated procedures to the woman and her family to reduce their feelings of fear and anxiety.

Excessive bleeding and fetal hypoxia are always major concerns with abruptio placentae, and nurses are responsible for continuous monitoring of both the expectant mother and the fetus so that problems can be detected early before the condition of the woman or the fetus deteriorates.

**CRITICAL TO REMEMBER**

**Signs of Concealed Hemorrhage in Abruptio Placentae**

- Increase in fundal height
- Hard, boardlike abdomen
- High uterine baseline tone on electronic monitoring strip when an intrauterine pressure catheter is used
- Persistent abdominal pain
- Systemic signs of early hemorrhage (tachycardia [maternal and fetal], tachypnea, falling blood pressure, falling urine output, restlessness)
- Persistent late deceleration in fetal heart rate or decreasing baseline variability; absence of accelerations
- Slight or absent vaginal bleeding
Application of the Nursing Process

Hemorrhagic Conditions of Late Pregnancy

Assessment

For hemorrhagic conditions of late pregnancy, some nursing assessments should be performed immediately and others can be deferred until initial interventions have been taken to stabilize the cardiovascular status of the woman. Many nursing assessments are concurrent with medical assessments and include:

- Amount and nature of bleeding (time of onset, estimated blood loss before admission to hospital, and description of tissue or clots passed): Peripads and underpads should be saved as needed to accurately estimate blood loss.
- Pain (type [constant, intermittent, sharp, dull, severe]; onset [sudden, gradual]; and location [generalized over abdomen, localized in back]): Is uterine tenderness present with gentle palpation? Is the tenderness localized?
- Maternal vital signs: Are these within normal limits, or is hypotension, tachycardia, or both present? A normal blood pressure may be misleading in a woman with abruptio placentae because she may have been hypertensive before the blood loss caused her blood pressure to fall to normal or hypotensive levels.
- Condition of the fetus: An electronic fetal monitor determines fetal heart rate, presence of accelerations, and fetal response to uterine activity. The presence of late decelerations or poor variability is of particular concern.
- Uterine contractions: Application of an external monitor determines frequency and duration of contractions. An intrauterine pressure catheter can identify hypertonic contractions and an increased resting tone associated with abruptio placentae. Palpation can identify that the uterus does not relax fully between contractions. Thick abdominal fat reduces the ability to identify poor uterine relaxation by external means.
- Obstetric history (gravida, para, previous abortions, preterm infants, previous pregnancy outcomes). Does the history include previous abruptio placentae?
- Length of gestation (date of last menstrual period, fundal height, correlation of fundal height with estimated gestation): If bleeding occurs into the myometrium, the fundus enlarges as bleeding progresses. A piece of tape can mark the top of the fundus at a given time, and then the nurse can observe and report increasing fundal size, which indicates that bleeding into uterine muscles is occurring.
- Laboratory data (complete blood count and blood type and screen, coagulation studies): Laboratory data are obtained to prepare for transfusions if necessary and to determine whether signs of DIC are developing. Type and Rh factor identify possible need for Rh immune globulin (RhoGAM). Other tests may be done serially to identify whether the abruptio is stable or worsening. The Kleihauer-Betke (K-B) test identifies fetal blood cells in the maternal circulation and is usually done after a traumatic event such as a motor vehicle accident. Coagulation studies include fibrinogen, FSP, PT and PTT, and D-dimer.

Despite the emphasis on physical assessment, the emotional response of the mother and her partner must also be addressed. They will most likely be anxious, fearful, confused, and overwhelmed by the activity. They may have little knowledge of expected medical management and may not realize that the fetus will need to be delivered as quickly as possible and that a surgical procedure is necessary. They may fear for the life of the woman and the fetus.

Analysis

Nursing diagnoses vary, depending on the cause and severity of the bleeding. The most commonly used nursing diagnoses for antepartum bleeding appear in Nursing Care Plan 25-1. The most dangerous potential complication is hypovolemic shock, which jeopardizes the life of the mother as well as the fetus.

Planning

The nurse cannot independently manage hypovolemic shock but must confer with physicians for medical orders for treatment. Planning should reflect the nurse’s responsibility to:

- Monitor for signs of hypovolemic shock
- Consult with the physician if signs of hypovolemic shock are observed
- Perform actions to minimize the effects of hypovolemic shock

Interventions

MONITORING FOR SIGNS OF HYPOVOLEMIC SHOCK

Assess for any sign of developing hypovolemic shock. The body attempts to compensate for decreased blood volume and to maintain oxygenation of essential organs by increasing the rate and effort of the heart and lungs and by shunting blood from less essential organs, such as the skin and the extremities, to more essential organs, such as the brain and the kidneys. This compensatory mechanism results in the early signs and symptoms of hypovolemic shock:

- Fetal tachycardia (often the first sign of either maternal or fetal hypovolemia)
- Maternal tachycardia, diminished peripheral pulses
**Complications of Pregnancy**

**CHAPTER 25**

### ASSESSMENT:

Beth Dixon, a 28-year-old gravida 2, para 1, is admitted to the antepartum unit at 32 weeks’ gestation after an episode of vaginal bleeding resulting from total placenta previa. Vital signs are stable, and fetal heart rate is 140 to 150 beats per minute with no nonreassuring signs. Beth and her husband Bob appear anxious about the condition of the fetus and the plan of care. Beth is particularly worried about her 5-year-old son, who is at home with a neighbor.

### NURSING DIAGNOSIS:

Anxiety related to unknown effects of bleeding and lack of knowledge of predicted course of management

### CRITICAL THINKING:

Although this diagnosis is correct, what are priority nursing actions for Beth? Why?

**ANSWER:** To monitor the condition of the fetus and to observe Beth for vaginal bleeding or change in vital signs. These priorities are based on a hierarchy of needs; physiologic needs and the need for safety must be ensured before psychological needs are fully addressed.

### GOALS/EXPECTED OUTCOMES:

The couple will:

1. Verbalize expected routines and projected management by the end of the first day after admission.
2. Express less anxiety after the course of management is explained.

### INTERVENTION | RATIONALE

1. **Remain with the couple and acknowledge the emotions that they exhibit: “I know this is unexpected, and you must have many questions. Perhaps I can answer some of them.”**

2. **Determine the couple’s level of understanding of the situation and the projected management: “Tell me what you’ve been told to expect.”**

3. **Provide the couple with factual information about projected management.**
   
   a. Explain that Beth will need to remain in the hospital so that her condition and the condition of the fetus can be watched closely.
   b. Explain why a cesarean birth is necessary this time even though she delivered vaginally before.
   c. Provide information about hospital routines (meals, visiting hours) and any fetal surveillance techniques (such as frequent ultrasound exams and biophysical profiles).

4. **Explain corticosteroid therapy ordered to speed fetal lung maturation.**

5. **Ask Beth and her family if they would like to talk with a nurse from the special care nursery.**

6. **Allow Beth and her family to participate in the routine as much as possible. This may mean scheduling nursing care around times when Bob and their son can visit.**

### EVALUATION:

The interventions are considered successful if Beth and Bob demonstrate knowledge of the projected management and why it is necessary and verbalize reduced anxiety.

### ASSESSMENT:

Beth continues to have episodes of light vaginal bleeding, although none has been so heavy as to demand immediate delivery. Her doctor wants the fetus to mature at least 2 more weeks if possible. Beth cries frequently, telling the nurse, “I miss my son so much. He just started kindergarten and he is so shy. I feel useless and he really needs me now. It’s hard on Bob, too; he has to do everything.”

### NURSING DIAGNOSIS:

Situational Low Self-Esteem related to temporary inability to provide care for family

### GOALS OR EXPECTED OUTCOMES:

Beth will:

1. Identify positive aspects of self during hospitalization
2. Identify ways of providing comfort and affection for her son during the hospital stay

### INTERVENTION | RATIONALE

1. **Encourage Beth to express her concerns about the need for hospitalization: “What bothers you most about being away from home?”**

2. **重大关注可能不被识别或可能被误解，除非护士澄清它们。**
Normal or slightly decreased blood pressure
Increased respiratory rate
Cool, pale skin and mucous membranes

The compensatory mechanism fails if hypovolemic shock progresses and insufficient blood exists to perfuse the brain, heart, and kidneys. Later signs of hypovolemic shock include the following:
- Falling blood pressure
- Pallor; skin becomes cold and clammy
- Urine output less than 30 ml/hr
- Restlessness, agitation, decreased mentation

**MONITORING THE FETUS**

Use continuous electronic fetal monitoring so that signs of fetal compromise, such as decreasing baseline variability or late decelerations, can be seen (see Chapter 14). If non reassuring patterns are seen, contact the physician at once because the fetus often shows signs of compromise before maternal signs of hypovolemia are obvious. Give the physician a report on new laboratory data that suggest an increasing degree of placental abruption, such as rising K-B levels after abdominal trauma.

**PROMOTING TISSUE OXYGENATION**

To promote oxygenation of tissues:
- Place the woman in a lateral position, with the head of the bed flat to increase cardiac return and thus to increase circulation and oxygenation of the placenta and other vital organs.
- Limit maternal activity to decrease the tissue demand for oxygen.
- Provide simple explanations, reassurance, and emotional support to the woman to reduce anxiety, which increases the metabolic demand for oxygen.

**COLLABORATING WITH THE PHYSICIAN FOR FLUID REPLACEMENT**

To replace fluids:
- Insert IV lines according to hospital protocol; usually two lines that use large-gauge catheters (16- to 18-gauge) are recommended so that blood can be administered quickly if necessary.
- Administer fluids for replacement as directed by the physician to maintain a urinary output of at least 30 ml/hr.

**PREPARING THE WOMAN FOR SURGERY**

Quick preparation of the woman for cesarean delivery may be necessary. The nurse is responsible for the following:
- Validating that preoperative permits have been correctly signed.
levels of pregnancy-related hormones, such as estrogen and causes include possible allergy to fetal proteins. Elevated nancies, and in multifetal pregnancies. Other possible common among unmarried white women, during first preg-

Etiology

thiamine deficiency can cause encephalopathy. Etiology of vitamin K may cause coagulation disorders, and amounts of hydrochloric acid are lost in the vomitus. Defi-

imbalance (both sodium and potassium are lost from gastric fluids). Metabolic alkalosis may develop because large amounts of hydrochloric acid are lost in the vomitus. Defi-

nization of vitamin K may cause coagulation disorders, and thiamine deficiency can cause encephalopathy.

Evaluation

Although client-centered goals are not developed for collaborative problems, the nurse collects and compares data with established norms and judges whether the data are within normal limits. For hypovolemic shock, the maternal vital signs remain within normal limits and the fetal heart demonstrates no signs of compromise, such as abnormal rate, late decelerations, or decreasing baseline variability.

Therapeutic Management

The physician will exclude other causes for persistent nausea and vomiting, such as cholecystitis or peptic ulcer disease, before diagnosing hyperemesis. Laboratory studies include determining the hemoglobin and hematocrit, which may be elevated as a result of dehydration, which results in hemoconcentration. Electrolyte studies may reveal reduced sodium, potassium, and chloride. Elevated creatinine levels indicate renal dysfunction.

Treatment occurs primarily in the home, where the woman first attempts to control the nausea with methods that are used for morning sickness (see Chapter 7). In addition, some physicians prescribe vitamins, such as pyridoxine (vitamin B6), that may provide some relief. A daily vitamin and mineral supplement may be recommended.

Drug therapy may be required if the vomiting becomes severe. Drugs prescribed may include the following:

- Promethazine (Phenergan)
- Diphenhydramine (Benadryl)
- Histamine-receptor antagonists such as famotidine (Pepcid) or ranitidine (Zantac)
- Gastric acid inhibitors such as esomeprazole (Nexium) or omeprazole (Prilosec)
- Metoclopramide (Reglan)
- Ondansetron (Zofran)

The steroid methylprednisolone has recently been found to reduce the nausea and vomiting. Metoclopramide can be given with a subcutaneous infusion pump to provide continuous therapy at home. If drugs are required, a single drug is first prescribed in the lowest effective dose to minimize fetal effects. The benefit of the drug at controlling the adverse effects of the intractable vomiting is balanced against any fetal risk from the drugs (Magee, Mazzotta & Koren, 2002; Modigliani, 2000; Steinlauf, Magee, & Traube, 2004).

If simpler methods are unsuccessful and weight loss or electrolyte imbalance persists, IV fluid and electrolyte replacement or total parenteral nutrition may be necessary. In some women, IV fluid replacement improves the nausea and vomiting quickly. The woman usually can be managed at home with periodic home nursing visits if she must have total parenteral nutrition. Periodic brief hospitalizations may be needed until the hyperemesis problem lessens.

Nursing Considerations

Because management frequently occurs in the home, nurses are often responsible for assessing and intervening for the woman with HEG. Physical assessment begins with determining the intake and output. Intake includes IV fluids and

PROVIDING EMOTIONAL SUPPORT

Once the safety of the woman and the fetus is ensured, nursing interventions promote comfort and provide emotional support. Explain what is causing the discomfort, and reassure the woman that pain relief measures will be initiated as soon as possible without causing harm to the fetus. Although offering false reassurance about the condition of the fetus is unwise, remain with the woman and provide accurate and timely information. Find time to explain what is going on to the woman and her family. They can feel overwhelmed by all the activity.

HYPEREMESIS GRAVIDARUM

HEG is persistent, uncontrollable vomiting that begins before the twentieth week of pregnancy. HEG may continue throughout pregnancy, although its severity usually lessens. Unlike morning sickness, which is self-limited and causes no serious complications, HEG can have serious consequences. It can lead to loss of 5% or more of prepregnancy weight, dehydration, ketosis, acid-base imbalance, and electrolyte imbalance (both sodium and potassium are lost from gastric fluids). Metabolic alkalosis may develop because large amounts of hydrochloric acid are lost in the vomitus. Deficiency of vitamin K may cause coagulation disorders, and thiamine deficiency can cause encephalopathy.

Etiology

The cause of HEG is not known, but the condition is more common among unmarried white women, during first pregnancies, and in multifetal pregnancies. Other possible causes include possible allergy to fetal proteins. Elevated levels of pregnancy-related hormones, such as estrogen and hCG, are considered a possible cause, as is maternal thyroid dysfunction. More recently, an association with the organism that causes peptic ulcer disease, Helicobacter pylori, has been identified. Psychological factors may interact with the nausea and vomiting that occurs during early pregnancy to worsen it (Buckwalter & Simpson, 2002; Cunningham et al., 2001; Gilbert & Harmon, 2003; Scott & Abu-Hamda, 2004; Weyermann et al., 2003).

The cause of HEG is not known, but the condition is more common among unmarried white women, during first pregnancies, and in multifetal pregnancies. Other possible causes include possible allergy to fetal proteins. Elevated levels of pregnancy-related hormones, such as estrogen and
parenteral nutrition, as well as oral nutrition, which is allowed once vomiting is controlled. Output includes the amount and character of emesis and urinary output. As a rule of thumb, the normal urinary output is about 1 ml/kg/hr (1 ml/2.2 lb/hr). A record of bowel elimination also provides significant information about oral nutrition because a woman’s intake may have been so minimal that many days have passed since her last normal bowel movement.

Laboratory data may be evaluated to determine fluid and metabolic status. Elevated levels of hemoglobin and hematocrit may occur as a result of dehydration, which results in hemoconcentration. Concentrations of sodium, potassium, and chloride may be reduced, resulting in hypokalemia and alkalosis.

The woman weighs herself daily, first thing in the morning and in similar clothing each day. Her urine is tested for ketones. Weight loss and the presence of ketones in the urine suggest that fat stores and protein are being metabolized to meet energy needs.

Signs of dehydration include decreased fluid intake (less than 2000 ml/day), decreased urinary output, increased urine specific gravity (more than 1.025), dry skin or dry mucous membranes, and nonelastic skin turgor.

Nursing interventions focus on reducing nausea and vomiting, maintaining nutrition and fluid balance, and providing emotional support.

REDUCING NAUSEA AND VOMITING

When food is offered to the woman, portions should be small so that the amount does not appear overwhelming. Food should be attractively presented, and foods with strong odors should be eliminated from the diet because food smells often incite nausea. Low-fat foods and easily digested carbohydrates, such as fruit, breads, cereals, rice, and pasta, provide important nutrients and help prevent low blood sugar, which can cause nausea. Soups and other liquids should be taken between meals to avoid distending the stomach and triggering vomiting. Sitting upright after meals reduces gastric reflux.

MAINTAINING NUTRITION AND FLUID BALANCE

Women with nausea and vomiting should eat every 2 to 3 hours. Salting food helps replace chloride lost when hydrochloric acid is vomited. Eating potassium- and magnesium-rich foods and fluids should be encouraged when the woman can do so, because stores of these nutrients are likely to be depleted and magnesium deficiency can exacerbate nausea. Potassium is found in fruits, vegetables, and meat. Sources of magnesium include seeds, nuts, legumes, and green vegetables.

IV fluids and total parenteral nutrition are administered as directed by the physician. IV fluid containing potassium is often ordered until the low serum level returns to normal. Small oral feedings of clear liquids are started when nausea and vomiting subside. When oral fluids and adequate food intake are tolerated, parenteral nutrition is gradually discontinued. Continued inability to tolerate oral feedings or continued episodes of vomiting should be reported to the physician so that continued parenteral fluids and nutrition can be prescribed.

PROVIDING EMOTIONAL SUPPORT

The woman with HEG needs the opportunity to express how it feels to be pregnant and to live with constant nausea. The woman, and possibly her significant other, may have been surprised by the pregnancy and may not have accepted it. Helping the woman express reluctance to accept the pregnancy and identify her sources of support may reduce nausea, although its intensity may remain higher than in most women.

Often a curious lack of sympathy and support exists for these women, however. Nurses must use critical thinking to examine their own biases so that they can provide comfort and support. Case conferences or inservice educational programs may be necessary to overcome preset beliefs and to establish a level of care that meets the needs of the woman.

CHECK YOUR READING

11. How do “morning sickness” and HEG compare in terms of onset, duration, and effect on the client?
12. What are the nursing goals in therapeutic management of HEG?
13. Why is critical thinking particularly important in the care of the woman with HEG?

HYPERTENSIVE DISORDERS OF PREGNANCY

Terminology used to describe hypertension in pregnancy is not always uniform. Four categories of hypertensive disorders occurring during pregnancy were identified by a group working within the National Heart, Lung, & Blood Institute of the National Institutes of Health for the United States (2001). Hypertension of pregnancy and chronic hypertension that is present when not pregnant can coexist (Table 25-1).

- **Preeclampsia:** A systolic blood pressure of ≥ 140 mm Hg or diastolic blood pressure of ≥ 90 mm Hg occurring after 20 weeks of pregnancy that is accompanied by significant proteinuria (> 0.3 g in a 24-hour urine collection, which usually correlates with a random urine dipstick evaluation of ≥ 1+). Edema, although common in preeclampsia, is now considered to be nonspecific because it occurs in many pregnancies not complicated by hypertension.

- **Eclampsia:** Progression of preeclampsia to generalized seizures that cannot be attributed to other causes. Seizures may occur postpartum.

- **Gestational hypertension:** Blood pressure elevation after 20 weeks of pregnancy that is not accompanied by proteinuria. Gestational hypertension must be considered a working diagnosis because it may progress to preeclampsia. If gestational hypertension persists after birth, chronic hypertension is diagnosed.
Preeclampsia is a condition in which hypertension develops during the last half of pregnancy in a woman who previously had normal blood pressure. In addition to hypertension, renal involvement may cause proteinuria. Many women also experience generalized edema. The only known cure is delivery of the fetus. Maternal and fetal morbidity can be minimized if preeclampsia is detected early and managed carefully.

**INCIDENCE AND RISK FACTORS**

Preeclampsia is relatively common, affecting 8% of all pregnancies (American Academy of Pediatrics & American College of Obstetricians and Gynecologists, 2002). It is a major cause of perinatal death, and it often is associated with intrauterine fetal growth restriction (IUGR).

Although the cause of preeclampsia is not understood, several factors are known to increase a woman’s risk. Many risk factors may be interrelated such as overweight and prepregnancy diabetes.

Women with an increased risk for developing preeclampsia are those having their first baby. Women who are over 35, are African-American, or have a positive family history of pregnancy-induced hypertension, are more likely to be of low socioeconomic status and have eclampsia. Women who have eclampsia are more likely to be of low socioeconomic status.

Less well-known risk factors include both genetic and immunologic factors. The presence of the angiotensinogen gene T235 greatly increases the woman’s sensitivity to angiotensin, a powerful vasoconstrictor that could lead to hypertension. Also, a woman is more likely to have preeclampsia if her mother or sister also had the disorder. Antiphospholipid syndrome (APS) is also strongly associated with the development of preeclampsia. The syndrome results from the development of antiphospholipid antibodies (aPL). These antibodies are directed against phospholipids that are widely distributed in cell membranes. The clinical picture of APS includes thrombosis, recurrent fetal loss, IUGR, and a higher incidence of preeclampsia (Lockwood & Silver, 2004). Box 25-2 summarizes major risk factors for the development of preeclampsia.

The father’s contribution to the pregnancy also appears to play a role in development of preeclampsia. Women who had prior pregnancies without hypertension are more likely to have preeclampsia if the expectant father previously fathered a pregnancy in another woman who had the disorder.

**TABLE 25-1 Classifications of Hypertension in Pregnancy**

<table>
<thead>
<tr>
<th>Classification</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preeclampsia</td>
<td>Systolic blood pressure $\geq 140$ mm Hg or diastolic blood pressure $\geq 90$ mm Hg that develops after 20 weeks of pregnancy and is accompanied by proteinuria $&gt;0.3$ g in a 24-hr urine collection (random urine dipstick is usually $&gt;1+$. Progression of preeclampsia to generalized seizures that cannot be attributed to other causes. Development of new-onset proteinuria $&gt;0.3$ g in a 24-hr collection in a woman who has chronic hypertension. In a woman who had proteinuria before 20 weeks, preeclampsia should be suspected if the woman has a sudden increase in proteinuria from her baseline levels, a sudden increase in blood pressure when it had been previously well controlled, development of thrombocytopenia (platelets $&lt;100,000/\text{mm}^3$), or abnormal elevations of liver enzymes (AST or ALT).</td>
</tr>
</tbody>
</table>

| Eclampsia Gestational hypertension | Systolic blood pressure $\geq 140$ mm Hg or diastolic blood pressure $\geq 90$ mm Hg that was known to exist before pregnancy or develops before 20 weeks of gestation. Also diagnosed if the hypertension does not resolve during the postpartum period. |

| Chronic hypertension | Systolic blood pressure $\geq 140$ mm Hg or diastolic blood pressure $\geq 90$ mm Hg that is known to exist before pregnancy. |

| Preeclampsia superimposed on chronic hypertension | Development of new-onset proteinuria $>0.3$ g in a 24-hr collection in a woman who has chronic hypertension. |

**ALT:** Alanine aminotransferase (formerly SGPT); **AST:** aspartate aminotransferase (formerly SGOT).

**BOX 25-2 Risk Factors for Pregnancy-Related Hypertension**

- First pregnancy
- Age $\geq 35$ years
- Anemia
- Family history of pregnancy-induced hypertension
- Chronic hypertension or preexisting vascular disease
- Chronic renal disease
- Obesity
- Diabetes mellitus
- Antiphospholipid syndrome
- Multifetal pregnancy
- Angiotensin gene T235
- Mother or sister who had preeclampsia

PATHOPHYSIOLOGY

Preeclampsia is a result of generalized vasospasm. The underlying cause of the vasospasm remains a mystery, although some of the pathophysiologic processes are known. In normal pregnancy, vascular volume and cardiac output increase significantly. Despite these increases, blood pressure does not rise in normal pregnancy. This is probably because pregnant women develop resistance to the effects of vasoconstrictors, such as angiotensin II. Peripheral vascular resistance decreases because of the effects of certain vasodilators, such as prostacyclin (PGI₂), PGE, and endothelium-derived relaxing factor (EDRF).

In preeclampsia, however, peripheral vascular resistance increases because some women are sensitive to angiotensin II. They also may have a decrease in vasodilators. For instance, the ratio of thromboxane (TXA₂) to PGI₂ increases. TXA₂, produced by kidney and trophoblastic tissue, causes vasoconstriction and platelet aggregation (clumping). PGI₂, produced by placental tissue and endothelial cells, causes vasodilation and inhibits platelet aggregation.

Vasoconstriction also results in impeded blood flow and elevation of blood pressure. As a result, circulation to all body organs, including the kidneys, liver, brain, and placenta, is decreased. The following changes are most significant:

- Decreased renal perfusion reduces the glomerular filtration rate. Blood urea nitrogen, creatinine, and uric acid levels begin to rise.
- Reduced renal blood flow results in glomerular damage, allowing protein to leak across the glomerular membrane, which is normally impermeable to large protein molecules.
- Loss of protein reduces colloid osmotic pressure and allows fluid to shift to interstitial spaces. This may result in edema and a reduction in intravascular volume, which causes increased viscosity of the blood and a rise in hematocrit. In response to reduced intravascular volume, additional angiotensin II and aldosterone trigger the retention of both sodium and water. Generalized edema may occur.
- Decreased circulation to the liver impairs function and leads to hepatic edema and subcapsular hemorrhage, which can result in hemorrhagic necrosis. This is manifested by elevation of liver enzymes in maternal serum.
- Vasoconstriction of cerebral vessels leads to pressure-induced rupture of thin-walled capillaries, resulting in small cerebral hemorrhages. Symptoms of arterial vasospasm include headache and visual disturbances, such as blurred vision, “spots” before the eyes, and hypertensive deep tendon reflexes.
- Decreased colloid oncotic pressure can lead to pulmonary capillary leak that results in pulmonary edema. Dyspnea is the primary symptom.
- Decreased placental circulation results in infarctions that increase the risk for abruptio placentae and DIC.

In addition, the fetus is likely to experience intrauterine growth restriction and persistent hypoxemia and acidosis when maternal blood flow through the placenta is reduced. Figure 25-7 summarizes the pathologic processes of preeclampsia.

PREVENTIVE MEASURES

PRENATAL CARE. Proper prenatal care with attention to pattern of weight gain and monitoring of blood pressure and urinary protein may minimize maternal and fetal morbidity and mortality by allowing early detection of the problem.

Past attempts at prevention have included low-dose aspirin, calcium and magnesium supplements, and fish oil supplements. These measures have not proved to be beneficial for the general population, however.

More recent research assessed the benefits of antioxidant therapy with 1000 mg of vitamin C and 15 mg of vitamin E starting at 22 weeks. Although the results were promising, safety and effectiveness of antioxidant supplementation for the general population require further study (National Institutes of Health, 2001).

CLINICAL MANIFESTATIONS OF PREECLAMPSIA

CLASSIC SIGNS. The first indication of preeclampsia is usually hypertension. Blood pressure measurements vary with the woman’s position, so the blood pressure should be measured uniformly at each office visit. Blood pressure should be measured with the woman seated and her arm supported, and the cuff size should be appropriate for the size of her arm. The diastolic pressure should be recorded at Korotkoff phase V, disappearance of sound (National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy, 2000). Hospitalizing the woman for serial observations of her blood pressure may identify true elevations from those induced by anxiety.

Proteinuria can be identified by using a clean-catch specimen to prevent contamination of the specimen by vaginal secretions or blood. Women with a urinary tract infection often have erythrocytes and leukocytes in the urine, which would elevate urine protein in the absence of preeclampsia.

ADDITIONAL SIGNS. Careful assessment may reveal additional signs associated with preeclampsia. For instance, when the retina is examined, vascular constriction and narrowing of the small arteries are obvious in most women with preeclampsia. The vasoconstriction that can be seen in the retina is occurring throughout the body. Deep tendon reflexes may be very brisk (hyperreflexia) suggesting cerebral irritability secondary to decreased brain circulation and edema.

Laboratory studies may identify liver, renal, and hepatic dysfunction if preeclampsia is severe. Coagulation may be impaired, as evidenced by a fall in platelets, which are often in the high normal range in a woman without preeclampsia. See also the discussion of DIC, p. 626.

Although it is a nonspecific sign that may have many causes, generalized edema often occurs with preeclampsia,
and it may be severe. Edema may first manifest as a rapid weight gain caused by fluid retention. Edema may be present in the lower legs, which is common in pregnancy, and in the hands and face (Figure 25-8). Edema may be so massive that the woman’s appearance is distorted. Edema may not, however, be present in all women who develop preeclampsia, and it may be severe in women who do not have the disorder. Pulmonary edema is also more common in women with massive edema from any cause, including drug therapy such as that given to stop preterm labor.

![Figure 25-7](image-url) The pathologic processes of preeclampsia.

![Figure 25-8](image-url) Generalized edema is a possible sign identified with preeclampsia, although it may occur in normal pregnancy or in a pregnancy complicated by another disorder. A, Facial edema may be subtle. B, Pitting edema of the lower leg.
SYMPTOMS. Preeclampsia is dangerous for the woman and fetus for two reasons: (1) it can develop and worsen rapidly, and (2) the earliest symptoms are often not noticed by the woman. By the time she notices symptoms, the disease may have progressed to an advanced state with loss of valuable treatment time.

Certain symptoms, such as continuous headache, drowsiness, or mental confusion, indicate poor cerebral perfusion and may be precursors of seizures. Visual disturbances, such as blurred or double vision or spots before the eyes, indicate arterial spasms and edema in the retina. Numbness or tingling of the hands or feet occurs when nerves are compressed by retained fluid. Some symptoms, such as epigastric pain or “upset stomach,” are particularly ominous because they indicate distention of the hepatic capsule and often warn that a seizure is imminent. Decreased urinary output indicates poor perfusion of the kidneys and may preclude acute renal failure.

THERAPEUTIC MANAGEMENT OF MILD PREECLAMPSIA

The only cure for preeclampsia is delivery of the baby. However, the decision about delivery will be based on the severity of the hypertensive disorder and the degree of fetal maturity. If the fetus is less than 34 weeks of gestation, steroids to accelerate fetal lung maturity will be given and an attempt made to delay birth for 48 hours. However, if the maternal or fetal condition deteriorates, the infant will be delivered, regardless of fetal age or administration of steroids. Vaginal birth is preferred because of the multisystem impairments.

Preeclampsia is categorized as either mild or severe, depending on the presenting signs and symptoms (Table 25-2). However, an apparently mild condition can become severe in a very short time because the disease may progress rapidly.

HOME CARE. Management in the home may be possible for selected women if the condition is mild and they are not good candidates to have their labor induced, usually because of fetal immaturity. The woman must be in stable condition with a reassuring fetal status. She must be willing to adhere to a prescribed treatment plan that includes bed rest or reduced activity, home blood pressure monitoring, and follow-up visits to the physician every 3 to 4 days. The woman on home care should be taught how to check her blood pressure and the symptoms to report that suggest worsening preeclampsia, such as visual disturbance, severe headache, or epigastric pain. Symptoms that suggest a nonreassuring fetal status should also be taught, such as reduced fetal movement. If the woman is hospitalized with mild preeclampsia, the following assessments for home care are adapted for her inpatient care.

Activity Restrictions. The mother should rest frequently although full bed rest is not required for mild preeclampsia. A lateral position for at least 1 1/2 hr/day decreases pressure on the vena cava, thereby increasing cardiac return and circulatory volume and thus improving perfusion of the woman’s vital organs and the placenta.

Blood Pressure. If monitoring of blood pressure is prescribed, the family must be taught to use electronic blood pressure equipment, readily available in drug, grocery, and discount stores. Blood pressure should be checked in the same arm and in the same position two to four times each day. A large cuff should be used for a woman with a large upper arm.

Weight. The woman should weigh herself each morning, preferably on the same scale and in clothing of similar weight.

<table>
<thead>
<tr>
<th>TABLE 25-2 Mild Versus Severe Preeclampsia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Parameter Evaluated</strong></td>
</tr>
<tr>
<td>Systolic blood pressure</td>
</tr>
<tr>
<td>Diastolic blood pressure</td>
</tr>
<tr>
<td>Proteinuria (24-hr specimen is preferred to eliminate hour-to-hour variations)</td>
</tr>
<tr>
<td>Creatinine, serum (renal function)</td>
</tr>
<tr>
<td>Platelets</td>
</tr>
<tr>
<td>Liver enzymes (alanine aminotransferase [ALT] or aspartate aminotransferase [AST])</td>
</tr>
<tr>
<td>Urine output</td>
</tr>
<tr>
<td>Severe, unremitting headache not attributable to other cause; mental confusion (cerebral edema)</td>
</tr>
<tr>
<td>Persistent right upper quadrant or epigastric pain or pain penetrating to the back (distention of the liver capsule); nausea and vomiting</td>
</tr>
<tr>
<td>Visual disturbances (spots or “sparkles”; temporary blindness; photosphobia)</td>
</tr>
<tr>
<td>Pulmonary edema; heart failure; cyanosis</td>
</tr>
<tr>
<td>Fetal growth restriction</td>
</tr>
</tbody>
</table>

Urinalysis. A urine dipstick test for protein, using the first voided midstream specimen, should be performed daily. The physician may request that she test at other times also.

Fetal Assessment. Because vasoconstriction can reduce placental flow, the woman will have increased fetal assessments to observe for evidence of fetal compromise. Fetal compromise can be evidenced by reduced fetal movement noted by the mother (“kick counts”), a nonreactive nonstress test, reduced amniotic fluid on ultrasound examination, or a biophysical profile score of 6 or lower. See Chapter 10 for discussion of fetal surveillance methods.

Diet. The diet should have ample protein and calories. Sodium and fluid should not be limited (Castro, 2004). The woman should be taught symptoms that indicate worsening of the preeclampsia and to report these at once. Indications of disease progression or fetal deterioration necessitate admission to the hospital.

INPATIENT MANAGEMENT OF SEVERE PREECLAMPSIA

Preeclampsia is severe if the systolic blood pressure is \( \geq 160 \text{ mm Hg} \) or the diastolic blood pressure is \( \geq 110 \text{ mm Hg} \) or if evidence of multisystem involvement is present (see Table 25-2). Delivery is usually necessary, even if the gestation is less than 34 weeks, because of disease severity. A decreased volume of amniotic fluid is considered significant because it suggests reduced placental blood flow, even if the blood pressures are not high.

ANTEPARTUM MANAGEMENT. Goals of management are to improve placental blood flow and fetal oxygenation and to prevent seizures and other maternal complications such as stroke as the woman’s condition is stabilized before delivery.

BED REST. The woman is kept on bed rest in the lateral position, and her environment is kept quiet. External stimuli (lights, noise) that might precipitate a seizure should be reduced.

<table>
<thead>
<tr>
<th>Drug Guide</th>
<th>HYDRAZINE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Classification:</td>
<td>Antihypertensive.</td>
</tr>
<tr>
<td>Action:</td>
<td>Relaxes arterial smooth muscle to reduce blood pressure.</td>
</tr>
<tr>
<td>Indications:</td>
<td>Used in preeclampsia when blood pressure is elevated to a degree that might be associated with intracranial bleeding.</td>
</tr>
<tr>
<td>Dosage and Route:</td>
<td>Obstetric uses in pregnancy-induced hypertension: Intravenous doses: 5 to 10 mg may be administered as often as every 15 to 20 minutes if necessary. Duration of action is 3 to 8 hours (American College of Obstetrics and Gynecologists [ACOG], 2002; Roberts, 2004).</td>
</tr>
<tr>
<td>Absorption:</td>
<td>Widely distributed, crosses the placenta; enters breast milk in minimal concentrations.</td>
</tr>
<tr>
<td>Excretion:</td>
<td>Metabolized and excreted by the liver.</td>
</tr>
<tr>
<td>Contraindications and Precautions:</td>
<td>Contraindicated in coronary artery disease, cerebrovascular disease, and hypersensitivity to hydralazine. Used cautiously in pregnancy; pregnancy category C.</td>
</tr>
<tr>
<td>Adverse Reactions:</td>
<td>Headache, dizziness, drowsiness, hypotension that can interfere with uterine blood flow, epigastric pain, which may be confused with worsening preeclampsia.</td>
</tr>
<tr>
<td>Nursing Implications:</td>
<td>Obstetric clients are hospitalized before initiation of antihypertensive medications. Blood pressure and pulse must be monitored every 2 to 3 minutes for 30 minutes after initial dose and periodically throughout the course of therapy. Therapy is repeated only when diastolic pressure exceeds limits set by physician or facility protocol, usually ( \geq 105-110 \text{ mm Hg} ) (ACOG, 2002; Roberts, 2004).</td>
</tr>
</tbody>
</table>
to those of the expectant mother. As a result, the fetal monitor tracing may show decreased fetal heart rate variability. No cumulative effect occurs, however, because the fetal kidneys excrete magnesium effectively.

The therapeutic serum level for magnesium is 4 to 8 mg/dl. Adverse reactions to magnesium sulfate usually occur if the serum level becomes too high. The most important is CNS depression, including depression of the respiratory center. Magnesium is excreted solely by the kidneys, and the reduced urine output that often occurs in preeclampsia allows magnesium to accumulate to toxic levels in the woman. Assessment of serum levels, deep tendon reflexes (Procedure 25-1), and respiratory rate and oxygen saturation can identify CNS depression before it progresses to respiratory depression or cardiac dysfunction. Monitoring urine output, usually with an indwelling catheter, identifies oliguria that may allow magnesium to accumulate and reach excessive levels. Policies related to care of the woman with hypertension and magnesium administration, such as specific assessments and lab studies, provide an organized framework for medical and nursing care.

INTRAPARTUM MANAGEMENT. Most seizures occur during labor or the first 24 hours after birth. The fetus and the expectant mother must be monitored continuously to detect signs of decreased fetal oxygenation and imminent seizures. The woman should be kept in a lateral position to promote circulation through the placenta, and efforts should focus on controlling pain that may cause agitation and precipitate seizures.

Oxytocin to stimulate uterine contractions and magnesium sulfate to prevent seizures are often administered simultaneously during labor when a woman has preeclampsia. The woman will have two secondary infusions in addition to her primary infusion line, one for oxytocin and one for magnesium. Infusion pumps should be used to ensure that the medications and fluids are administered at the prescribed rate, and equipment and IV lines should be checked carefully for correct placement and function.

Narcotic analgesics or epidural analgesia may be administered to provide comfort and to reduce painful stimuli that could precipitate a seizure. However, some women with severe preeclampsia have coagulation abnormalities that may contraindicate use of epidural analgesia.

Continuous electronic fetal monitoring identifies changes in fetal heart rate patterns that suggest compromise (see Chapter 14). Late decelerations, associated with reduced placental perfusion, or decreased variability, associated with reduced placental perfusion or magnesium use, is more likely to occur, but any other nonreassuring pattern may occur as well. Interventions are tailored to the nonreassuring fetal heart pattern identified, such as maternal oxygen administration, stopping the oxytocin infusion, and increases in the IV fluid rates.

A pediatrician, neonatologist, or neonatal nurse practitioner must be available to care for the newborn at birth. A resuscitation team is called to the delivery if needed.

POSTPARTUM MANAGEMENT. After birth, careful assessment of the mother’s blood loss and signs of shock are essential because the hypovolemia caused by preeclampsia may be aggravated by blood loss during the delivery. Assessments for signs and symptoms of preeclampsia must be continued for at least 48 hours, and magnesium with its associated care usually is continued to prevent seizures for at least 24 hours.

### DRUG GUIDE

**MAGNESIUM SULFATE**

**Classification:** Miscellaneous anticonvulsant.

**Action:** Decreases acetylcholine released by motor nerve impulses, thereby blocking neuromuscular transmission. Depresses the central nervous system (CNS) to act as an anticonvulsant; also decreases frequency and intensity of uterine contractions. Produces flushing and sweating because of decreased peripheral blood pressure.

**Indications:** Prevention and control of seizures in severe preeclampsia. Prevention of uterine contractions in preterm labor.

**Dosage and Route:** A common intravenous (IV) administration protocol for preeclampsia includes a loading dose and a continuous infusion using a controlled infusion pump. The loading dose is 4 to 6 g of magnesium sulfate administered in 100 ml of IV fluid over 15 to 20 minutes. The continuous infusion to maintain control is 2 g/hr. Doses are individualized as needed. Deep intramuscular injection is acceptable but is painful. A primary IV infusion with no medication is maintained if the magnesium must be discontinued.

Magnesium sulfate may also be administered in a similar dose profile to stop preterm labor contractions.

**Absorption:** Immediate onset after IV administration.

**Excretion:** Excreted by the kidneys.

**Contraindications and Precautions:** Contraindicated in persons with myocardial damage, heart block, myasthenia gravis, or impaired renal function. Magnesium toxicity, possibly related to incomplete renal drug excretion, may be evidenced by thirst, mental confusion, or a decrease in reflexes.

**Adverse Reactions:** Result from magnesium overdose and include flushing, sweating, hypotension, depressed deep tendon reflexes, and CNS depression, including respiratory depression.

**Nursing Implications:** Monitor blood pressure closely during administration. Assess client for respiratory rate above 12 breaths per minute, presence of deep tendon reflexes, and urinary output greater than 30 ml/hr before administering magnesium. Place resuscitation equipment (suction, oxygen) in the room. Keep calcium gluconate, which acts as an antidote to magnesium, in the room, along with syringes and needles.
PROCEDURE 25-1 Assessing Deep Tendon Reflexes

PURPOSE: To identify exaggerated reflexes (hyperreflexia) or diminished reflexes (hyporeflexia)

You will need a reflex hammer to best assess both the brachial and the patellar reflexes.

Support the woman’s arm and instruct her to let it go limp while it is being held so that the arm is totally relaxed and slightly flexed as you assess the brachial reflex. If you have difficulty identifying the correct tendon to tap, have the woman flex and extend her arm until you can feel it moving beneath your thumb. Have her fully relax her arm after you identify the tendon.

Place your thumb over the woman’s tendon, as illustrated, to allow you to feel as well as see the tendon response when the tendon is tapped. Strike the thumb with the small end of the triangular reflex hammer. The normal response is slight flexion of the forearm.

The patellar, or “knee-jerk,” reflex can be assessed with the woman in two positions, sitting or lying. When the woman is sitting, allow her lower legs to dangle freely to flex the knee and stretch the tendons. If her patellar tendon is difficult to identify, have her flex and extend her lower legs slightly until you palpate the tendon. Strike the tendon directly with the reflex hammer just below the patella. The patellar reflex is less reliable if the woman has had epidural analgesia, and upper extremity reflexes should be assessed.

When the woman is supine, the weight of her leg must be supported to flex the knee and stretch the tendons. An accurate response requires that the limb be relaxed and the tendon partially stretched. Strike the partially stretched tendons just below the patella. Slight extension of the leg or a brief twitch of the quadriceps muscle of the thigh is the expected response.

For assessment of clonus, the woman’s lower leg should be supported, as illustrated, and the foot well dorsiflexed to stretch the tendon. Hold the flexion. If no clonus is present, no movement will be felt. When clonus (indicating hyperreflexia) is present, rapid rhythmic tapping motions of the foot are present.

DEEP TENDON REFLEX RATING SCALE*

<table>
<thead>
<tr>
<th>Rating</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Reflex absent</td>
</tr>
<tr>
<td>+1</td>
<td>Reflex present, hypoactive</td>
</tr>
<tr>
<td>+2</td>
<td>Normal reflex</td>
</tr>
<tr>
<td>+3</td>
<td>Brisker than average reflex</td>
</tr>
<tr>
<td>+4</td>
<td>Hyperactive reflex; clonus may also be present</td>
</tr>
</tbody>
</table>

*The rating scales of some facilities omit the plus signs.
Signs that the woman is recovering from preeclampsia include the following:
- Urinary output of 4 to 6 L/day, which causes a rapid reduction in edema and rapid weight loss
- Decreased protein in the urine
- Return of blood pressure to normal, usually within 2 weeks

**THERAPEUTIC MANAGEMENT OF ECLAMPSIA**

Eclampsia is a potentially preventable extension of severe preeclampsia marked by one or more generalized seizures, at times occurring before the woman goes to the hospital. Early identification of preeclampsia in a pregnant woman allows intervention before the condition reaches the seizure stage in most cases. Generalized seizures usually start with facial twitching, followed by rigidity of the body. Tonic-clonic movements then begin and last for about 1 minute. Breathing stops during a seizure but resumes with a long, noisy inhalation. The woman is temporarily in a coma and is unlikely to remember the seizure when she resumes consciousness. Transient fetal heart rate patterns may be non-reassuring, such as bradycardia, loss of variability, or late decelerations. Fetal tachycardia may occur as the fetus compensates for the period of maternal apnea during the seizures. Eclampsia may occur during pregnancy or in the intrapartum or postpartum period.

Magnesium sulfate is the drug of choice to control eclamptic seizures. Other anticonvulsants or sedatives are not routinely given.

The woman’s blood volume is often severely reduced in eclampsia, increasing the risk for poor placental perfusion. Fluid shifts from her intravascular space to the interstitial space, including the lungs, causing pulmonary edema and possibly heart failure as forward blood flow from the maternal heart is impeded. Renal blood flow is severely reduced, with oliguria (<30 ml/hr urine output) and possible renal failure. Cerebral hemorrhage may accompany eclampsia because of the high blood pressure and coagulation deficits. The woman’s lungs should be auscultated at regular intervals, usually hourly. A pulse oximeter provides continuous readings of oxygen saturation. Furosemide (Lasix) may be administered if pulmonary edema develops. Digitalis may be needed to strengthen heart function if circulatory failure results. Urine output should be assessed hourly. Urine output below 30 ml/hr may indicate renal failure.

The combination of hemolysis, elevated liver enzymes, and low platelets (HELLP syndrome) is associated with severe preeclampsia. DIC is an added complication of unexpected bleeding that may occur with coagulation abnormalities of severe preeclampsia or eclampsia. Lab studies are performed at frequent intervals to identify both falling and recovering coagulation values as well as identifying hemolysis and elevated liver enzymes.

The woman should be monitored for ruptured membranes, signs of labor, and abruptio placentae because eclampsia stimulates uterine irritability. While the woman is postictal (the unresponsive state after a seizure), she should be kept on her side to prevent aspiration and improve placental circulation. The side rails should be raised to prevent a fall and possible injury. After maternal and fetal conditions are stabilized, the fetus usually is delivered, either by induction of labor if the woman’s cervix is favorable or by cesarean birth if she is farther from term.

Aspiration may cause maternal morbidity after an eclamptic seizure. After initial stabilization the nurse should anticipate orders for chest radiography and arterial blood gas determination to identify aspiration.

Magnesium may be given intravenously to control the seizures. A sedative such as phenobarbital or diazepam is used only if magnesium fails to bring the seizures under control. Sedatives should not be given if birth is expected within 2 hours because of their depressant effects on the fetus.

Pulmonary edema, circulatory or renal failure, and intracranial hemorrhage are additional complications that may occur with severe preeclampsia or eclampsia. The woman’s lungs should be auscultated frequently, and furosemide (Lasix) may be administered if pulmonary edema develops. Digitalis may be needed to strengthen heart function if circulatory failure results. Urine output should be assessed hourly. Urine output below 30 ml/hr may indicate renal failure.

**CHECK YOUR READING**

14. What are the effects of vasospasm on the fetus?
15. What are the signs and symptoms of preeclampsia?
16. What is the effect of vasospasm on the brain?
17. What are the effects of magnesium sulfate, including the primary adverse effect?
18. What are the major complications of eclampsia?

**Application of the Nursing Process**

**Preeclampsia**

**Assessment**

Nursing assessment is one of the most important components of successful management of preeclampsia. Careful assessment is the only way to determine whether the condi-
tion is responding to medical management or whether the disease is worsening. A one-to-one nurse-patient ratio is needed for the woman with severe pregnancy-induced hypertension.

Weigh the woman on admission and daily. Check vital signs and auscultate the chest at least every 4 hours for moist breath sounds that indicate pulmonary edema. Assess the location and severity of edema at least every 4 hours. Table 25-3 provides a guideline to describe edema. Insert an indwelling catheter to measure hourly urine output. Check the urine for protein every 4 hours. Apply an external electronic fetal monitor to identify changes in fetal heart rate, variability, or nonreassuring patterns. Consider maternal medications and their relationship to the fetal monitoring pattern.

Check reflexes such as brachial, radial, and patellar reflexes for hyperreflexia, which indicates cerebral irritability. Determine if clonus is present with hyperactive reflexes by dorsiflexing the woman’s foot sharply and then releasing it while her knee is held in a flexed position. Clonus (rapidly alternating muscle contraction and relaxation) may occur when reflexes are hyperactive. If clonus is present, it should be reported to the physician. Procedure 25-1 illustrates how to assess and rate deep tendon reflexes.

Question the woman carefully about symptoms she may be experiencing, such as headache, visual disturbances, epigastric pain, nausea or vomiting, or a sudden increase in edema.

Detailed questions are usually needed to identify important symptoms. Ask targeted questions, such as “Do you have a headache? Describe it for me.” “Do you have any pain in the abdomen? Show me where it is and describe it.” “Do you see spots before your eyes? Flashes of light?” “Do you have double vision?” “Is your vision blurred?” “Does the light bother you?” “I see you have removed your rings. Did you do that because your hands were swollen? When did that happen?”

**ASSESSMENTS FOR MAGNESIUM TOXICITY**

Obstetric units have protocols that address routine assessments when magnesium is being administered and their frequency. Reflexes may be slightly hypotonic but should not be absent at therapeutic levels of magnesium. Absent reflexes suggest CNS depression that precedes respiratory depression if magnesium levels are too high. Determining the respiratory rate and oxygen saturations by pulse oximetry identifies the adequacy of maternal respirations. Checking urine output identifies oliguria (<30 ml/hr) that may result in magnesium toxicity as the drug accumulates. Assess the woman’s level of consciousness (alert, drowsy [expected], confused, oriented). Table 25-4 summarizes nursing assessments and their implications.

**PSYCHOSOCIAL ASSESSMENT**

The development of preeclampsia places added stress on the childbearing family. The woman may be on reduced activity at home if the condition is mild and her gestation is early. She may be hospitalized, complicating child care. This creates anxiety about the condition of the fetus and that of the expectant mother. Many families do not understand the seriousness of the disease. After all, the woman often feels well after its onset, especially if her preeclampsia does not advance rapidly. The possibility that a preterm birth may be

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**TABLE 25-3** Assessment of Edema

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Grade</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minimal edema of lower extremities</td>
<td>1</td>
</tr>
<tr>
<td>Marked edema of lower extremities</td>
<td>2</td>
</tr>
<tr>
<td>Edema of lower extremities, face, hands,</td>
<td>3</td>
</tr>
<tr>
<td>and sacral area</td>
<td></td>
</tr>
<tr>
<td>Generalized massive edema that includes asci-</td>
<td>4</td>
</tr>
<tr>
<td>ties (accumulation of fluid in peritoneal</td>
<td></td>
</tr>
<tr>
<td>cavity</td>
<td></td>
</tr>
</tbody>
</table>

**TABLE 25-4** Nursing Assessments for Preeclampsia and Magnesium Toxicity

<table>
<thead>
<tr>
<th>Assessment</th>
<th>Implications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daily weight</td>
<td>Provides estimate of fluid retention. To determine worsening condition, response to treatment, or both.</td>
</tr>
<tr>
<td>Blood pressure</td>
<td>Drug therapy (magnesium sulfate) causes respiratory depression, and drug should be withheld and the physician notified if respiratory rate is &lt;12/min or as specified by hospital policy. Pulse oximeter readings 95% or greater.</td>
</tr>
<tr>
<td>Respiratory rate, pulse oximeter</td>
<td>Hyperreflexia indicates increased cerebral irritability and edema; hyporeflexia is associated with magnesium excess.</td>
</tr>
<tr>
<td>Breath sounds</td>
<td>For estimation of interstitial fluid.</td>
</tr>
<tr>
<td>Deep tendon reflexes</td>
<td>Output of at least 30 ml/hr indicates adequate perfusion of the kidneys (25 ml/hr is used by some authorities). Magnesium levels may become toxic if urinary output is inadequate.</td>
</tr>
<tr>
<td>Edema</td>
<td>Normal protein in a random dipstick urine sample is negative or trace. Higher protein levels suggest greater leaking of protein secondary to glomerular damage with worsening preeclampsia. A 24-hour urine sample is most accurate for quantitative urine protein level.</td>
</tr>
<tr>
<td>Urinary output</td>
<td>Drowsiness or dulled sensorium indicates therapeutic effects of magnesium; no responsive behavior or muscle weakness is associated with magnesium excess.</td>
</tr>
<tr>
<td>Urine protein</td>
<td>These symptoms indicate increasing severity of the condition caused by cerebral edema, vasospasm of cerebral vessels, and liver edema. Eclampsia may develop quickly.</td>
</tr>
<tr>
<td>Level of consciousness</td>
<td>Rate should be between 110 and 160 beats per minute in a term fetus. Decreasing baseline variability may be caused by therapeutic magnesium level or by inadequate placental perfusion.</td>
</tr>
<tr>
<td>Headache, epigastric pain, visual</td>
<td>Elevated serum creatinine, elevated liver enzymes, or decreased platelets (thrombocytopenia) are significant signs of increasing severity of disease. Serum magnesium levels should be in the therapeutic range designated by the physician.</td>
</tr>
<tr>
<td>problems</td>
<td></td>
</tr>
<tr>
<td>Fetal heart rate and baseline</td>
<td></td>
</tr>
<tr>
<td>variability</td>
<td></td>
</tr>
<tr>
<td>Laboratory data</td>
<td></td>
</tr>
</tbody>
</table>
necessary to reduce harm to mother and infant adds to the family’s concerns about the outcome.

Explore how the family will function while the expectant mother is hospitalized. Determine how the woman is adapting to the “sick role” and the necessity of being dependent on others instead of functioning in her primary role. Ask how much support is available and who is willing to participate. Determine if referrals to manage loss of income are needed. Finally, determine the priority concerns of the family.

Analysis

Analysis of the data collected can lead to both nursing diagnoses (see Nursing Care Plan 25-2) and collaborative problems for potential complications. Both physician-prescribed and nurse-prescribed interventions are used to minimize the com-

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**Nursing Care Plan 25-2**  
**Preeclampsia**

**Assessment:** Julie Frost, a 16-year-old primigravida, is seen in the prenatal clinic at 30 weeks of gestation. Her blood pressure is 136/90, and there is some edema of the lower legs and trace proteinuria. She is given instructions about home care for pregnancy-induced hypertension. The regimen includes bed rest; frequent monitoring of blood pressure, weight, and urine; and doing fetal “kick counts.” Julie is told she must return to the clinic in a week. She states that she feels fine and doesn’t want to miss school. She says that she doesn’t see the reason for bed rest.

**Nursing Diagnosis:** Impaired Adjustment related to lack of knowledge of health status and the need for a change in lifestyle

**Goals/Expected Outcomes:** Julie will:
1. Verbalize the benefits of the recommended regimen by the end of the first prenatal appointment.
2. Comply with the recommended care for the next week.

**Intervention**

<table>
<thead>
<tr>
<th>Intervention</th>
<th>Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Allow Julie to verbalize her feelings about the recommended regimen: “What concerns you most about missing school?” Acknowledge her feelings as important: “It must be difficult to think of falling behind in your schoolwork. It isn’t any fun to miss all the after-school activities or time with your friends.”</td>
<td>1. When feelings are identified and acknowledged as important, anxiety decreases and teaching and learning can begin.</td>
</tr>
<tr>
<td>2. Identify family support that is likely to improve compliance with the recommended regimen of bed rest and home care.</td>
<td>2. Compliance with the regimen is impossible without family assistance, which includes assistance with activities of daily living and necessary assessments.</td>
</tr>
<tr>
<td>3. Describe in general terms the pathophysiologic processes that affect Julie and her fetus.</td>
<td>3. Expectant mothers are usually motivated to comply with a therapeutic regimen that will benefit the fetus.</td>
</tr>
<tr>
<td>4. Explain that Julie may feel well even when the condition worsens and that she must be observed for signs and symptoms at home and at the clinic.</td>
<td>4. The expectant mother does not notice hypertension and proteinuria. Although edema is not always present in hypertensive complications during pregnancy, clients may not be aware that it may also be associated with other disorders.</td>
</tr>
<tr>
<td>5. Instruct Julie to call the clinic if she notices headache, double vision, or spots before her eyes.</td>
<td>5. These signs indicate rapid progression of the disease and the prompt necessity for additional management.</td>
</tr>
<tr>
<td>6. Collaborate with Julie to arrange for contact with her boyfriend or selected friends and to arrange for ongoing home-bound classes.</td>
<td>6. Such an agreement will allow a schedule to provide peer support but allow for prolonged periods of quiet. Home-bound classes alleviate the concern that she is falling behind with schoolwork.</td>
</tr>
</tbody>
</table>

**Evaluation:** Despite following the recommended regimen of bed rest with the help of her mother and sister and keeping prenatal appointments, Julie’s condition worsens. She develops a rise in blood pressure and a rapid gain in weight, indicating generalized edema, at 32 weeks.

**Assessment:** Julie is admitted to the hospital at 32 weeks of gestation with a blood pressure of 160/110, heart rate of 92, and respiratory rate of 22 per minute. There is 2+ proteinuria and marked edema of the hands and face as well as her lower extremities. Fetal heart rate is 136 with average variability. An intravenous infusion of magnesium sulfate is started, seizure precautions are initiated, and environmental stimuli are reduced. Julie is agitated and verbalizes concern that the procedures are going to hurt her or the fetus. She frequently asks, “How sick am I?” “Is the baby going to be okay?” Her hands are perspiring, and they shake when she reaches for a tissue.

**Nursing Diagnosis:** Anxiety related to hospitalization and concern about her health and the health of the fetus
plication. Potential complications for the woman with preeclampsia are eclamptic seizures and magnesium toxicity.

Planning

Client-centered goals are inappropriate for the potential complications of eclamptic seizures and magnesium toxicity because the nurse cannot independently manage these conditions but must confer with physicians and use established protocols for treatment. For seizures, planning should reflect the nurse’s responsibility to do the following:

- Perform actions that minimize the risk of seizures and prevent injury if seizures do occur
- Monitor for signs of impending seizures
- Consult with the physician if signs of impending seizures are observed
- Support the family of the woman with eclampsia

GOALS/EXPECTED OUTCOMES: Julie will:

1. Verbalize her concerns and describe the benefits of the treatment while her family is present
2. Manifest less anxiety (agitation, physiologic signs such as tremors, tachycardia, and perspiration)

INTERVENTION

1. Initiate measures to reduce anxiety:
   a. Provide positive reassurance that a solution for anxiety can be found: “I can see you are really worried, and I will try to answer all your questions.”
   b. Allow Julie to cry, get angry, or express any feeling that is present.
   c. Encourage a discussion of feelings: “Tell me more about how you feel.”
   d. Reflect observations: “I see you wringing your hands; do you want to talk about it?”
   e. Convey empathy and positive regard; use nonverbal behavior, including touch, when appropriate.

2. Provide information about hospital routines and procedures when Julie’s anxiety has diminished enough for learning to take place:
   a. Be very specific about procedures, such as fetal monitoring, assessment of deep tendon reflexes, taking of vital signs, and care specific for magnesium sulfate therapy. Explain the reasons for these procedures, who will perform them, and how long they will be continued after birth.
   b. Focus on Julie’s present concerns; she is not able to be future oriented at this time.
   c. Speak slowly and calmly, give very short directions, and do not ask Julie to make decisions: “Turn on your side.” “Breathe slowly.”
   d. Allow a friend or family member to remain with Julie, and instruct the person about the need for a low-stimulus environment.

RATIONALE

1. Anxiety is an ominous feeling of tension resulting from a physical or emotional threat to the self. It is a global, often unnamed sense of doom, a feeling of helplessness, isolation, and insecurity. Anxiety needs to be ventilated and then addressed by conveying that the person is not alone and will be protected.

2. Knowledge of procedures to be performed and the purpose of these procedures provides a sense of control that reduces anxiety. Perception is somewhat narrowed when anxiety is high; therefore brief instructions are easier for the anxious person to understand than long explanations.

EVALUATION: Julie discusses her feelings with the nurse and with her sister. She feels in control of anxiety, as manifested by fewer signs of agitation and fewer physiologic signs (tachycardia, tachypnea) and by the ability to use relaxation techniques.

CRITICAL THINKING:

1. What two potential complications cause the greatest concern for nurses who care for Julie?
2. Why do nurses not develop goals for these problems?
3. What are the nurses’ responsibilities for these complications?

ANSWERS

1. The most common complications that cause the greatest concern are magnesium toxicity and generalized seizures.
2. These are collaborative problems that require collaboration with physicians for management. The nurse does not independently manage magnesium toxicity or seizures.
3. The nurse must monitor for signs of these conditions, administer prescribed medications, observe and report Julie’s response to the medications, and collaborate with the physicians to lessen the chance that these complications will occur.
The following steps are necessary in cases of magnesium toxicity:
- Monitor for signs of magnesium toxicity.
- Consult with the physician if signs of magnesium toxicity are observed.
- Perform actions that reduce the possibility of magnesium toxicity.

**Interventions**

**INTERVENTIONS FOR SEIZURES**

**MONITORING FOR SIGNS OF IMPENDING SEIZURES.** Signs of impending seizures include the following:
- Hyperreflexia, possibly accompanied by clonus
- Increasing signs of cerebral irritability (headache, visual disturbances)
- Epigastric or right upper quadrant pain, nausea, or vomiting

None of these signs is a predictor of imminent seizure. Nurses must be alert for subtle changes and be prepared for seizures in all women with preeclampsia.

**INITIATING PREVENTIVE MEASURES.** In the presence of cerebral irritability, generalized seizures may be precipitated by excessive visual or auditory stimuli. Nurses should reduce external stimuli by doing the following:
- Admitting the woman to a private room in the quietest section of the unit and keeping the door to the room closed. Intense nursing observation is needed regardless of the specific room location that is available.
- Padding the door to reduce noise when the door must be opened and closed.
- Keeping lights low and noise to a minimum. This may include blocking incoming telephone calls and turning the noises of the electronic monitors (fetal monitor, pulse oximeter, IV pump) as low as possible.
- Grouping nursing assessments and care to allow the woman periods of undisturbed quiet.
- Moving carefully and calmly around the room and avoiding bumping into the bed or startling the woman.
- Collaborating with the woman and her family to restrict visitors.

**PREVENTING SEIZURE-RELATED INJURY.** Hard side rails should be padded and the bed kept in the lowest position with the wheels locked to prevent trauma during a seizure.

Oxygen and suction equipment should be assembled and ready to use to suction secretions and to provide oxygen if it is not already being administered. Check equipment and connections at the beginning of each shift because sufficient time for setup will not exist if seizures occur.

A preeclampsia tray or box should be in the room. Typical contents include a medium plastic airway, an Ambu bag with mask, an ophthalmoscope, a tourniquet, a reflex hammer, and syringes and needles. Medications that should be on hand include magnesium sulfate, sodium bicarbonate, heparin, epinephrine, phenytoin, and calcium gluconate.

**PROTECTING THE WOMAN AND FETUS DURING A SEIZURE.** Nurses must protect the woman and the fetus during a seizure. The nurse’s primary responsibilities are the following:
- Remain with the woman and press the emergency bell for assistance.
- If she is not on her side already, attempt to turn the woman onto her side when the tonic phase begins. A side-lying position permits greater circulation through the placenta and may prevent aspiration.
- Note the time and sequence of the seizure. Eclampsia is marked by a tonic-clonic seizure that may be preceded by facial twitching that lasts for a few seconds. A tonic contraction of the entire body is followed by the clonic phase, which may last about a minute.
- Insert an airway after the seizure, and suction the woman’s mouth and nose to prevent aspiration. Administer oxygen by mask at 8 to 10 L/min to increase oxygenation of the placenta and all maternal body organs.
- Notify the physician that a seizure has occurred. This is an obstetric emergency that is associated with cerebral hemorrhage, abruptio placenta, severe fetal hypoxia, and death.
- Administer medications and prepare for additional medical interventions as directed by the physician.

**PROVIDING INFORMATION AND SUPPORT FOR THE FAMILY.** Explain to the family what has happened without minimizing the seriousness of the situation. A generalized seizure is frightening for anyone who witnesses it, and the family often is reassured when the nurse explains that the seizure lasts for only a few minutes and that the woman will be unconscious, then drowsy for some time afterward. Acknowledge that the seizure indicates worsening of the condition and that it will be necessary for the physician to determine future management, which may include delivery of the infant as soon as possible. Vaginal birth is preferred if the maternal and fetal conditions permit because of abnormalities in the coagulation and other body systems.

**INTERVENTIONS FOR MAGNESIUM TOXICITY**

**MONITORING FOR SIGNS OF MAGNESIUM TOXICITY.** Magnesium excess depresses the entire CNS, including the brainstem, which controls respirations and cardiac function, and the cerebrum, which controls memory, mental processes, and speech. Carbon dioxide accumulates if the respiratory rate is reduced, leading to respiratory acidosis and further CNS depression, which could culminate in respiratory arrest.

Signs of magnesium toxicity include the following:
- Respiratory rate of less than 12 breaths per minute (hospitals may specify a rate of less than 14 breaths per minute)
- Maternal pulse oximeter reading lower than 95%
Absence of deep tendon reflexes
- Sweating, flushing
- Altered sensorium (confused, lethargic, slurred speech, drowsy, disoriented)
- Hypotension
- Serum magnesium above the therapeutic range of 4 to 8 mg/dl

**RESPONDING TO SIGNS OF MAGNESIUM TOXICITY.** Discontinue magnesium if the respiratory rate is below 12 breaths per minute, a low pulse oximeter level (<95%) persists, or deep tendon reflexes are absent. Additional magnesium will make the condition worse. Notify the physician of the woman’s condition for additional orders. If the urinary output falls below 30 ml/hr, the physician is notified so that the drug’s administration can be adjusted to maintain a therapeutic range.

Calcium opposes the effects of magnesium at the neuromuscular junction, and it should be readily available whenever magnesium is administered. Magnesium toxicity can be reversed by IV administration of 1 g (10 ml of 10% solution) calcium gluconate at 1 ml/min.

**Evaluation**

Collect and compare data with established norms and then judge whether the data are within normal limits. For seizures, interventions are judged to be successful if:
- Reflexes remain within normal limits (+1 to +3)
- The woman is free of visual disturbances, headache, and epigastric or right upper quadrant pain
- The woman remains free of seizures or free of preventable injury if a seizure occurs

For magnesium toxicity, determine whether respiratory rates remain at least 12 breaths per minute, deep tendon reflexes are present and not hyperactive, and maternal plasma levels of magnesium do not exceed the therapeutic range of 4 to 8 mg/dl.

**CHECK YOUR READING**

19. What nursing assessments should be made for the woman with preeclampsia? Why?
20. What measures may be initiated to prevent or manage seizures?
21. How can injury during seizure be prevented?
22. What are the signs of magnesium toxicity? How should it be managed?

**HEMOLYSIS, ELEVATED LIVER ENZYMES, AND LOW PLATELETS (HELLP) SYNDROME**

The acronym HELLP (hemolysis, elevated liver enzymes, low platelets) refers to a life-threatening occurrence that complicates about 10% of pregnancies. Half of the women affected with HELLP also have severe preeclampsia, although hypertension may be absent. As in preeclampsia, HELLP syndrome may occur during the postpartum period (Abramovici, Mattar, & Sibai, 2000; Moldenhauer & Sibai, 2003).

Hemolysis is believed to occur as a result of the fragmentation and distortion of erythrocytes during passage through small damaged blood vessels. Liver enzyme levels increase when hepatic blood flow is obstructed by fibrin deposits. Hyperbilirubinemia and jaundice may occur as a result of liver impairment. Low platelet levels are caused by vascular damage resulting from vasospasm; platelets aggregate at sites of damage, resulting in thrombocytopenia, which increases the risk for bleeding, usually in the liver.

The prominent symptom of the HELLP syndrome is pain in the right upper quadrant, the lower right chest, or the midepigastric area. There may also be tenderness because of liver distention. Additional signs and symptoms include nausea, vomiting, and severe edema. It is important to avoid traumatizing the liver by abdominal palpation and to use care in transporting the woman. A sudden increase in intraabdominal pressure, including a seizure, could lead to rupture of a subcapsular hematoma, resulting in internal bleeding and hypovolemic shock. Hepatic rupture can lead to fetal and maternal mortality (August, 2004; Moldenhauer & Sibai, 2003; Riely & Fallon, 2004).

Women with the HELLP syndrome should be managed in a setting with intensive care facilities available. Treatment includes magnesium sulfate to control seizures and hydralazine to control the blood pressure. Fluid replacement is managed to avoid worsening the woman’s reduced intravascular volume without giving her too much, which could cause pulmonary edema or ascites. Cervical ripening with labor induction usually is done if the gestation is at least 34 weeks. Delivery may be delayed if the gestation is less than 34 weeks and the woman’s condition is stable to give steroids a chance to stimulate fetal lung maturation (Landon, 2004). After delivery, most women begin recovering within 72 hours.

**CHRONIC HYPERTENSION**

A diagnosis of chronic hypertension is made whenever evidence suggests that hypertension preceded the pregnancy or when a woman is hypertensive before 20 weeks’ gestation. Chronic hypertension is seen most often in older women, in those who are obese, and in those with diabetes. Heredity, including race, plays a role in the development of chronic hypertension, which is more common in African-Americans at any age than in people of other races (National Center for Health Statistics, 2004). Late childbearing and rising obesity rates will no doubt fuel an increase in hypertension. Chronic hypertension is usually essential, or primary. However, it may be secondary to another problem, such as renal disease or an autoimmune disorder (Cunningham et al., 2001; Moldenhauer & Sibai, 2003).

Because of the natural fall in blood pressure during early pregnancy, the woman’s blood pressure may appear normal when she enters prenatal care. If she already is taking an an-
tihypertensive drug, she usually continues this drug unless her blood pressure becomes low because of the vasodilators of pregnancy. If she is not on an antihypertensive drug, she may be placed on one if her diastolic pressure is consistently above 90 mm Hg.

The most common maternal hazard is the development of preeclampsia, which occurs in 20% of pregnant women with chronic hypertension. New-onset proteinuria or a significant rise in preexisting proteinuria identifies the development of superimposed preeclampsia. The rise in blood pressure with preeclampsia is likely to be greater in these women (Cunningham et al., 2001; National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy, 2000; Roberts, 2004).

A diettian should be consulted about the appropriate diet and weight gain, because many of these women are obese and they frequently have diabetes. Adequate intake of protein helps counteract the protein lost in urine. A reduced salt intake may be advised, unlike recommendations for the woman with preeclampsia alone. More frequent prenatal visits will be needed. Regular fetal surveillance by biophysical profile and kick counts (see Chapter 10) is the usual method for identification of poor growth patterns or signs that are nonreassuring, such as a falling amount of amniotic fluid.

Antihypertensive medications must be chosen carefully because they may reduce placental blood flow. Antihypertensive medication should be initiated if the diastolic pressure remains higher than 100 mm Hg in early pregnancy (Roberts, 2004). Methyldopa (Aldomet) is the drug of choice because of its record of safety and effectiveness in pregnancy. β-Blockers and calcium channel blockers may also be used if methyldopa is not effective, but their record of safety in pregnancy is less well established. Angiotensin-converting enzyme (ACE) inhibitors are not recommended in pregnancy but may be used in the postpartum period. Hydralazine is a vasodilator reserved for hypertensive crisis. Diuretics are avoided if possible because they may shrink the blood volume, which may already be reduced if preeclampsia exists with the chronic hypertension. If regular diuretics are needed, thiazides are considered safe for pregnancy.

**CHECK YOUR READING**

23. What is the unabbreviated form of the term HELLP? What are the prominent signs and symptoms of this syndrome? Why should the liver not be palpated?

**INCOMPATIBILITY BETWEEN MATERNAL AND FETAL BLOOD**

**Rh Incompatibility**

Rhesus (Rh) factor incompatibility during pregnancy is possible only when two specific circumstances coexist: (1) the expectant mother is Rh-negative, and (2) the fetus is Rh-positive. For such a circumstance to occur, the father of the fetus must be Rh-positive. Rh incompatibility is a problem that affects the fetus; it causes no harm to the expectant mother.

Rh-negative blood is an autosomal recessive trait, and a person must inherit the same gene from both parents to be Rh-negative. Approximately 15% of the white population in the United States is Rh-negative. The incidence is lower in the African-American and Asian populations.

**PATHOPHYSIOLOGY**

People who are Rh-positive have the Rh antigen on their red blood cells, whereas people who are Rh-negative do not have the antigen. When blood from a person who is Rh-positive enters the bloodstream of a person who is Rh-negative, the body reacts as it would to any foreign substance: it develops antibodies to destroy the invading antigen. To destroy the Rh antigen, which exists as part of the red blood cell, the entire red blood cell must be destroyed. Exposure of the Rh-negative male or female to Rh-positive blood may occur unrelated to a pregnancy, such as emergency blood transfusion Rh-negative blood. Destruction of Rh-positive cells occurs in the Rh-negative person after they have become sensitized to the Rh-positive antigens.

Theoretically, fetal and maternal blood do not mix during pregnancy. In reality, small placental accidents occur that allow a drop or two of fetal blood to enter the maternal circulation and initiate the production of antibodies to destroy the Rh-positive blood. Sensitization also can occur during a spontaneous or elective abortion or during antepartal procedures such as amniocentesis and chorionic villus sampling (Figure 25-9). A rapid immune response against Rh-positive blood occurs with an extensive fetal-maternal hemorrhage in complications like placenta previa or abruptio placentae (p. 634) or with an uncomplicated birth.

Most exposure of maternal blood to fetal blood occurs during the third stage of labor, when active exchange of fetal and maternal blood may occur from damaged placental vessels. In this case the woman’s first child is not usually affected because antibodies are formed after the birth of the infant. Subsequent Rh-positive fetuses may be affected, however, unless the mother receives Rh(D) immune globulin (RhoGAM) to prevent antibody formation after the birth of each Rh-positive infant.

**FETAL AND NEONATAL IMPLICATIONS**

If antibodies to the Rh factor are present in the expectant mother’s blood, they cross the placenta and destroy fetal erythrocytes. The fetus becomes deficient in red blood cells, which are needed to transport oxygen to fetal tissue. As fetal red blood cells are destroyed, fetal bilirubin levels increase (icterus gravis), which can lead to neurologic disease (kernicterus, leading to bilirubin encephalopathy). This hemolytic process results in rapid production of erythroblasts (immature red blood cells), which cannot carry oxygen. The entire syndrome is termed erythroblastosis fetalis. The fetus may become so anemic that generalized fetal edema (hydrops fetalis) results and can end in fetal congestive heart failure.

Management of the infant born with erythroblastosis fetalis is discussed in Chapter 30.
Women should have a blood test to determine blood type and Rh factor at the initial prenatal visit. Rh-negative women should have an indirect Coombs’ test to determine whether they are sensitized (have developed antibodies) as a result of previous exposure to Rh-positive blood. If the indirect Coombs’ test is negative, it is repeated at 28 weeks of gestation to identify if they have developed subsequent sensitization.

Rho(D) immune globulin (such as RhoGAM) is administered to the unsensitized Rh-negative woman at 28 weeks of gestation to prevent sensitization, which may occur from small leaks of fetal blood across the placenta. Rho(D) immune globulin is a commercial preparation of passive antibodies against Rh factor. It effectively prevents the formation of active antibodies against Rh-positive erythrocytes if a small amount of fetal Rh-positive blood enters the circulation of the Rh-negative mother during the remainder of the pregnancy. Rho(D) immune globulin is repeated after birth if the woman delivers an Rh-positive infant.

If the indirect Coombs’ test result is positive, indicating maternal sensitization and the presence of antibodies, it is repeated at frequent intervals throughout the pregnancy to determine whether the antibody titer is rising. An increase in titer indicates that the process is continuing and that the fetus will be in jeopardy.

Amniocentesis may be performed to determine the Rh factor of the fetus and to evaluate change in the optical density (ΔOD) of amniotic fluid. If the fluid optical density remains low, it may indicate that the fetus is Rh-negative or that the fetus is Rh-positive but in no jeopardy. DNA analysis allows determination of the Rh factor of the fetus from amniotic fluid cells with high accuracy, reducing uncertainty when other testing results fall in the borderline zone. For example, if the optical density is slightly elevated but DNA testing shows the fetus to be Rh-negative, amniocentesis can eliminate the need to test further for Rh-related problems.

Ultrasound examination also is used to evaluate the condition of the fetus. Generalized fetal edema, ascites, enlarged heart, or hydramnios indicates serious fetal compromise. A cordocentesis may be performed to evaluate the
fetal hematocrit, and an intrauterine transfusion may follow if the preterm fetus is anemic (Figure 25-10).

**POSTPARTUM MANAGEMENT**

If the mother is Rh-negative, umbilical cord blood is taken at delivery to determine blood type, Rh factor, and antibody titer (direct Coombs’ test) of the newborn. Rh-negative, unsensitized mothers who give birth to Rh-positive infants are given an intramuscular injection of Rho(D) immune globulin (RhoGAM) within 72 hours after delivery. If RhoGAM is given to the mother in the first 72 hours after delivery of an Rh-positive infant, Rh antigens present in her blood are destroyed before she forms antibodies to the Rh factor. If the infant is Rh-negative, Rh antibody formation does not occur and RhoGAM is not necessary.

Families often are concerned about the fetus, and nurses must be sensitive to cues that indicate that the family is anxious and must be able to offer honest reassurance. This is especially important if the expectant mother is sensitized and fetal testing is necessary throughout pregnancy (Box 25-3).

**BOX 25-3 Nursing Diagnoses for the Woman with a Complication of Pregnancy**

<table>
<thead>
<tr>
<th>Diagnosis</th>
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<tbody>
<tr>
<td>Anxiety*</td>
</tr>
<tr>
<td>Deficient Diversional Activity</td>
</tr>
<tr>
<td>Fear</td>
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<tr>
<td>Impaired Adjustment*</td>
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<tr>
<td>Deficient Knowledge*</td>
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<tr>
<td>Risk for Dysfunctional Family Processes</td>
</tr>
<tr>
<td>Risk for Infection</td>
</tr>
<tr>
<td>Situational Low Self-Esteem*</td>
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</tbody>
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*R：Nursing diagnoses explored in this chapter.

During labor the nurse must carefully label the tube of cord blood obtained for analysis of the newborn’s blood type and Rh factor. During the postpartum period, nurses are responsible for follow-up to determine whether RhoGAM is necessary and for administering the injection within the prescribed time.

**ABO Incompatibility**

ABO incompatibility occurs when the mother is blood type O and the fetus is blood type A, B, or AB. Types A, B, and AB blood contain a protein component (antigen) that is not present in type O blood.

People with type O blood develop anti-A or anti-B antibodies naturally as a result of exposure to antigens in the foods that they eat or to infection by gram-negative bacteria. As a result, some women with blood type O have developed high serum anti-A and anti-B antibody titers before pregnancy. The antibodies may be either IgG or IgM. When the woman becomes pregnant, the IgG antibodies cross the placental barrier and cause hemolysis of fetal red blood cells. Although the first fetus can be affected, ABO incompatibility is less severe than Rh incompatibility because the primary antibodies of the ABO system are IgM, which do not readily cross the placenta.

No specific prenatal care is needed; however, the nurse must be aware of the possibility of ABO incompatibility. During the delivery, cord blood is taken to determine the blood type of the newborn and the antibody titer (direct Coombs’ test). The newborn is carefully screened for jaundice, which indicates hyperbilirubinemia. See Chapter 30 for medical and nursing management of hyperbilirubinemia in newborns.
CHAPTER 25

Complications of Pregnancy

**Rh(D) Immune Globulin (RhoGAM, Hyprhod, Gamulin RH)**

**Classification:** Concentrated immunoglobulins directed toward the red blood cell antigen Rh(D).

**Action:** Prevents production of anti-Rh(D) antibodies in Rh-negative women who have been exposed to Rh-positive blood by suppressing the immune reaction of the Rh-negative woman to the antigen in Rh-positive blood; prevents antibody response and thereby prevents hemolytic disease of the newborn in future Rh-positive pregnancies. Used for both males and females who are Rh-negative but exposed to Rh-positive blood or for immune thrombocytopenic purpura (ITP).

**Indications (Pregnancy Related):** Administered to Rh-negative women who have been exposed to Rh-positive blood by

1. Delivering an Rh-positive infant
2. Aborting an Rh-positive fetus
3. Undergoing chorionic villus sampling, amniocentesis, or intraabdominal trauma while carrying an Rh-positive fetus
4. Receiving inadvertent transfusion of Rh-positive blood

**Dosage and Route:** One standard dose (300 mcg) administered intramuscularly:

1. At 28 weeks of pregnancy and within 72 hours of delivery
2. Within 72 hours after termination of a pregnancy of 13 weeks or more of gestation
   - One microdose (50 mcg) within 72 hours after the termination of a pregnancy of less than 13 weeks of gestation.

**About Rh Incompatibility**

- **What does it mean to be Rh-negative?**
  
  Those who are Rh-negative lack a substance that is present in the red blood cells of those who are Rh-positive.

- **How can the expectant mother be Rh-negative and the fetus be Rh-positive?**
  
  The fetus can inherit the Rh-positive factor from the father.

- **What does sensitization mean?**
  
  Sensitization means that the Rh-negative person has been exposed to Rh-positive blood and has developed antibodies against the Rh factor.

- **Do the antibodies harm the woman?**
  
  No, because she does not have the Rh factor.

- **Do Rh-positive men always father Rh-positive children?**
  
  No. Rh-positive men who have an Rh-positive gene and an Rh-negative gene can father Rh-negative children.

- **Why is Rh(D) immune globulin (RhoGAM) necessary during pregnancy and after childbirth?**
  
  RhoGAM prevents the development of Rh antibodies in the mother, which might be harmful to subsequent fetuses who are Rh-positive. Administering Rh(D) immune globulin during pregnancy to the mother who delivers an Rh-positive infant is not harmful to the baby.

- **Why will the next fetus be jeopardized if RhoGAM is not administered?**
  
  If RhoGAM is not administered when a baby is Rh-positive, the expectant mother may develop antibodies that cross the placental barrier and affect the next Rh-positive fetus.

**CHECK YOUR READING**

25. Why do unsensitized Rh-negative expectant mothers receive Rh(D) immune globulin during pregnancy and after an abortion, amniocentesis, and childbirth?

26. What are the effects of maternal Rh sensitization?

27. Why is the first fetus sometimes affected if ABO incompatibility occurs? Why are the effects of ABO incompatibility milder than those of Rh-sensitization?

**SUMMARY CONCEPTS**

- Spontaneous abortion is one of the leading causes of pregnancy loss. Treatment is aimed at preventing complications, such as hypovolemic shock and infection, and providing emotional support for grieving.

- The incidence of ectopic pregnancy is increasing in the United States as a result of pelvic inflammation associated with sexually transmissible diseases. The goals of therapeutic management are to prevent severe hemorrhage and to preserve the fallopian tube so that future fertility is retained.
Management of gestational trophoblastic disease (hydatidiform mole) involves two phases: (1) evacuation of the molar pregnancy, and (2) continued follow-up for 1 year to detect malignant changes in the remaining trophoblastic tissue.

Disorders of the placenta (placenta previa and abruptio placenta) are responsible for hemorrhagic conditions of the last half of pregnancy. Either condition may result in maternal hemorrhage and fetal or maternal death.

Disseminated intravascular coagulation is a life-threatening complication of missed abortion, abruptio placenta, and severe hypertension, in which procoagulation and anticoagulation factors are simultaneously activated. DIC may occur with problems unrelated to pregnancy.

The cause of hyperemesis gravidarum remains unclear, but the goals of management are to prevent dehydration, malnutrition, excess weight loss, and electrolyte imbalance. Emotional support is an important responsibility of nurses, in addition to physical care.

Classifications of hypertension during pregnancy include preeclampsia, eclampsia, gestational hypertension, chronic (preexisting or persistent) hypertension, and preeclampsia superimposed over existing chronic hypertension. The underlying process is generalized vasospasm, which decreases circulation to all organs of the body, including the placenta. Major maternal organs affected include the liver, kidneys, and brain.

Treatment of preeclampsia includes reduced activity, reduction of environmental stimuli, and administration of medications to prevent generalized seizures.

Magnesium sulfate, used to prevent preeclampsia from progressing to eclamptic seizures, may have adverse effects. The most serious of these is central nervous system depression, which includes depression of the respiratory center. Adverse effects such as respiratory depression or absent deep tendon reflexes are more likely to occur if the blood level of magnesium rises over the therapeutic range.

Nurses monitor the woman with preeclampsia to determine the effectiveness of medical therapy and to identify signs that the condition is worsening, such as increasing hyperreflexia. Nurses also control external stimuli and initiate measures to protect her in case of eclamptic seizures.

Women who have chronic hypertension are at increased risk for preeclampsia and should be monitored for worsening hypertension, proteinuria, or generalized edema. Antihypertensive medication should be continued or initiated if diastolic blood pressure is consistently higher than 100 mm Hg.

Rh incompatibility can occur if an Rh-negative woman conceives a child who is Rh-positive. As a result of exposure to the Rh-positive antigen, maternal antibodies may develop that cause hemolysis of fetal Rh-positive red blood cells in subsequent pregnancies.

Administration of Rh(D) immune globulin (RhoGAM) prevents production of anti-Rh antibodies, thereby preventing destruction of Rh-positive red blood cells in subsequent pregnancies.

ABO incompatibility usually occurs when the mother has type O blood and naturally occurring anti-A and anti-B antibodies, which cause hemolysis if the fetus’s blood is not type O. ABO incompatibility may result in hyperbilirubinemia of the infant, but it usually presents no serious threat to the health of the child.

Nurses often assume that patients know how to use a thermometer and that they know the signs of infection. Many nurses assume that patients realize the connection between blood loss and the tendency to develop infection. As a result, nurses may not emphasize the need for a diet that is high in nutrients that increase hemoglobin and hematocrit (iron and vitamin C). Nurses also may assume incorrectly that women know foods that contain these nutrients.