***Manual of Obstetrical and Gynecological ward practice***

***For 5th class***

***First Edition***

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***Note: The medical conditions included in this manual are the must admitted to the ward***

***Part –A-***

***Obstetrics***

***Terminology***

In medicine, gravidity refers to the number of times a woman has been pregnant.

*1. Parity:* is the no. of live birth at any age or stillbirth after 24 weeks of gestation.

*2. Nullipara:* a woman who has never delivered a fetus or fetuses beyond 20 weeks of gestation.

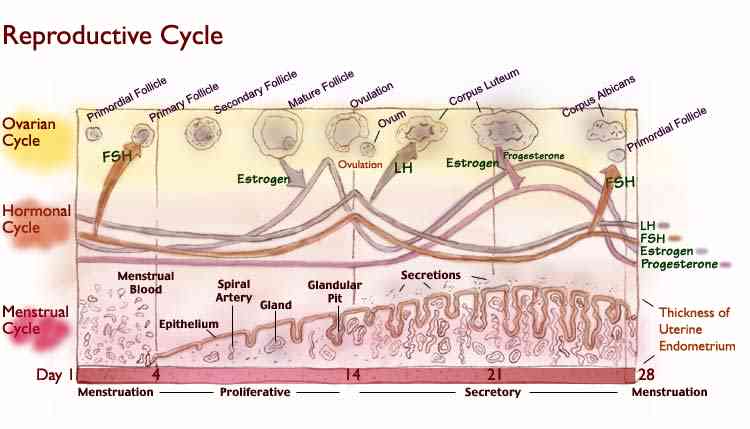
*3. Gravida:* a is the total no. of pregnancy regardless of how they ended( abortion, normal pregnancy).

*4. Nullgravida:* a nulligravida or gravida 0 is a woman who has never been pregnant.

*5. Primigravida:* a primigravida or gravida 1 is a woman who is pregnant for the first time or has been pregnant one time.

*6. elderly primigravida:* an elderly primigravida is a woman in her first

pregnancy, who is at least 35 years old.

Example : a woman who has 2 complete abortion and 1 normal pregnancy may be termed as G3P1A2.

***Obstetrical Abbreviations***

-Expected Date of Delivery **(EDD)**

- First Missed Period **(FMP)**

- Last Menstrual Period **(LMP)**

- Fetal Movement **( FM)**

- Poly Cystic Ovary Syndrome **(PCOS)**

- Neural Tube Defect **(NTD)**

- Fetal Life **( FL)**

- Pregnancy Test **(PT)**

- Caesarean Section **(C/S)**

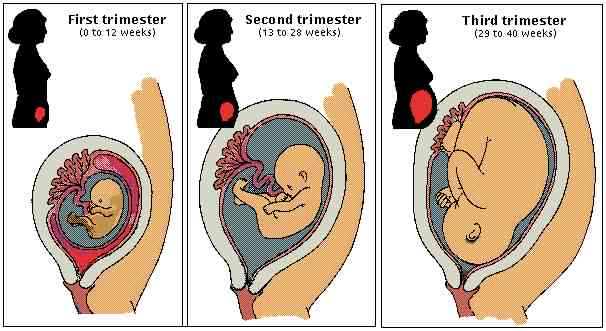
- Normal Vaginal Delivery **(NVD)**

- Premature Uterine Contractions **(PUC)**

- Respiratory Distress Syndrome **(RDS)**

***Trimesters of Pregnancy***

The human gestation period is 36-42 weeks, and is divided into three stages called trimesters. Each trimester is three months. The stages of development are the pre-embryonic, embryonic and fetal. The pre-embryonic stage is when the fertilized ovum consolidates, and it lasts for 17 days postconception. The major organ systems are formed during the embryonic stage (18-56 days), with maturation, development and growth continuing during the fetal stage (18-38 weeks).



***A.First trimester of pregnancy***

* First trimester pregnancy is the early stage of pregnancy from conception to 12 weeks gestation, or about 14 weeks from the first day of the last normal menstrual period (LNMP).
* During this stage pregnant may experience the dreaded morning sickness and sore and enlarged breasts. A woman may notice no period or a light period; blue lines under the skin over her breasts and abdomen; waistline expansion; breasts that grow larger protruding nipples.
* food aversions and cravings; heartburn and indigestion; fatigue; tender breasts; complexion problems; a need to urinate often ; constipation; headaches, dizziness, or faintness. The most dramatic changes and development occur during the first trimester.

1. ***Second trimester of pregnancy***

* This second stage of pregnancy lasts until the end of the seventh month and is many times the easiest stage of pregnancy as most women will art to regain some of their energy.
* Women may notice the abdomen begins to swell. By the end of the second trimester, the uterus is near the rib cage; the skin on the abdomen and breasts stretches.
* movements made by the fetus. Known as quickening, this often occurs sometime around weeks 6 to 20
* a dark line forming from the navel down to the middle of the abdomen; brown, uneven marks on the face or other changes in skin pigment; darkening of the area around the nipples.

In the fetus, growth continues quickly from now until birth. Organs such as the heart and kidneys develop further, eyebrows and finger nails form, the skin is wrinkled and covered with fine hair, periods of activity and quiet occur as the fetus moves, kicks, sleeps, and wakes.

The second trimester is the most physically enjoyable for most women. Morning sickness usually abates by this time and the extreme fatigue and breast tenderness usually subsides. These changes can be attributed to a decrease in levels of human chorionic gonadotropin (hCG) hormone and an adjustment to the levels of estrogen and progesterone hormones.

1. ***Third trimester of pregnancy***

The third trimester of pregnancy generally spans weeks 28 through 40, though healthy babies may be born a bit sooner or later. The fetus is continuing to grow in weight and size and the body systems finish maturing. The mother may feel more uncomfortable now as she continues to gain weight and begins to have false labor contractions (called Braxton-Hicks contractions).

***Normal pregnancy and Prenatal Care***

***A. Pregnancy Signs and Symptoms***

***1. Nausea and vomiting :***

Recurrent nausea and vomiting during the 1st trimester occurs in about one-half of pregnancies.

The etiology of this problem is not clear but hormonal and emotional factors have been investigated. Symptoms can be mild or so severe that the patient becomes dehydrated and risks electrolyte imbalance and caloric malnutrition , this condition is known as **Hyperemesis gravidarum** that required hospitalization .

Management include **non pharmacological** measures as avoidance of fatty or spicy foods, eating small, more frequent meals, drinking ginger teas, inhaling peppermint oil vapors, wearing motion sickness.

**Pharmacological therapy** in severe cases include administration of pyridoxine, a variety of antihistamins, promethazine, metoclopramide ,more recently intravenous droperidol and diphenhydramine.

***2. Heartburn:***

Heartburn is a reflux esophagitis caused by both mechanical factors (enlarging uterus displacing the stomach above the esophageal sphincter) and hormonal factors (by progesterone).

Management include **non pharmacological** therapy avoidance of acidicand spicy foods, decreasing the amount of food and liquid at each meal, limiting food and liquid intake before bedtime, sleeping a semi-Fowlers position or propped up on pillows.

**Phrmacological therapy** include use of liquid forms of antacids H2-receptor inhibitors.

***3. Constipation:***

Progesterone-induced relaxation of the intestinal smooth muscle .peristalsis and increase bowel transit time is the causative factor of constipation. Dietary management include increased fluids and liberal intake of foods. Iron salts may exacerbate the problem. Enemas, laxative , and strong cathartics should be avoided.

**4. *Varicosities and Hemorrhoids:***

Varicosities most often occur in the lower extremities but may be seen in the vulva as well. Contributing factors include genetic predisposition, advanced maternal age, increased parity, and prolong standing. Treatment includes avoidance of garments that constrict at the knee and upper leg, support stocking, and increased periods of rest with the legs elevated.

Hemorrhoids, Varicosities of the rectal veins, are due to mechanical compression by the enlarging uterus, as well as from constipation and straining at stool. Treatment includes OTC topical preparations, cool sitz baths , and stool softeners.

***4. Ptyalism:***

Ptyalism is the increased production of saliva, probably induced by the consumption of starch. It is cured by reducing carbohydrate intake.

***6. Leg Cramps:***

Almost half of all pregnant women suffer from recurrent painful spasms of the muscles of the lower extremities, especially the calves. Leg cramps are more frequent at night and usually occur during the 3rd trimester. Treatment include massage and placing the affected muscle(s) on stretch relieves the cramps when occur.

**7. *Backache:***

Most pregnant women experience lower backache as pregnancy progresses. These are usually alleviated by minimizing the amount of time spent standing, by increasing rest, by wearing a specially designed support belt over the lower abdomen, and by taking an analgesic such as paracetamol.

***8. Headache:***

Muscle tension headaches may occur intermittently. Headaches during the 2nd and 3rd trimesters are not an expected symptom of pregnancy.

***9. Urinary Frequency:***

Urinary Frequency occur mostly during the 1st trimester, as the enlarging uterus compresses the bladder , and again during the last weeks, as the fetal head descends into the pelvis.

***10. Leukorrhea:***

An increase in the amount of vaginal discharge is physiologic and suspected during pregnancy. Douching has no place in the treatment

***11.* *Syncope:***Venous pooling in the lower extremities increases as the pregnancy regresses. This can lead to dizziness or lightheadedness, especially after standing upright abruptly or for long periods of time. Other causes include dehydration, hypoglycemia, and the taunting of blood flow to the stomach after eating a large meal.

***Laboratory Evaluation***

***1. Maternal Screening Tests:***

***A. . Amniocentesis*** (also referred to as ***amniotic fluid test or AFT):*** is a medical procedure used in prenatal diagnosis of genetic abnormalities and fetal infections, in which a small amount of amniotic fluid, which contains fetal tissues, is extracted from the amnion or amniotic sac surrounding a developing fetus, and the fetal DNA is examined for genetic abnormalities usually no earlier than the 14th week of pregnancy.

***B***. ***Chorionic villus sampling (CVS:)*** is a form of prenatal diagnosis to determine chromosomal or genetic disorders in the fetus. It entails getting a sample of the chorionic villus (placental tissue) and testing it. The advantage of CVS is that it can be carried out 10-13 weeks after the last period, earlier than amniocentesis (which is carried out at 15-18 weeks).

***Indications***: Possible reasons for having a CVS can include:

• Mother's age of 35 years or greater

• Increased nuchal translucency or other abnormal ultrasound findings

• Family history of a chromosomal abnormality or other genetic disorder

• Parents are known carriers for a genetic disorder

***C. Alfa-fetoprotein (AFP or MSAFP) test:*** is a maternal blood test done in the second trimester that checks for a protein normally secreted by the fetal liver. The levels of this protein alert the obstetrician to the possibility of a chromosomal abnormality, such as Down syndrome or the presence of twins. Abnormal AFP levels may also indicate developmental problems in the fetus, such as neural tube defects like spina bifida or defects in the abdominal wall of the fetus.

***2. Screening for Gestational Diabetes:***

The 1-hour, 50-g oral glucose screen is used to detect glucose intolerance in pregnancy. Routine screening is performed on all patients between 24 and 28 weeks gestation. The significance of GDM lies not in an increased risk of fetal loss but in the risk of excessive fetal growth with its attendant birth-related morbidities.

***3. Researching for Rh Antibodies:***

All Rh-ve women who are unsensitized at the beginning of pregnancy should be retested at approximately 26-28 weeks gestation. If the antibody screen remains -ve , the mother should receive Rho(D) immune globulin .

***4. Screening for Bacterial Vaginosis***

Bacterial vaginosis(BV) is a condition in which the normal flora of the vagina( speciaaly lactobacilli) are reduced in number and replaced by overgrowth of anaerobic organisms. Some studies have linked BV with an increased incidence of preterm labor, endometriosis and premature rupture of the membranes.

**5. *Testing for Group B Streptococci( GBS):***

GBS are part of the normal vaginal flora and implicated in preterm labor, as well as in amnionitis, endometriosis, and wound infection in the mother. Vertical transmission during labor or delivery may result in generalized sepsis in the newborn and related long-term morbidity or neonatal death. Cultures obtained at 35-37 weeks gestation from the lower third of the vagina and perianal area. Culture+ve women are treated during labor with antibiotic prophylaxis to prevent fetal-neonatal GBS infection.

***Nutrition in pregnancy***

1. ***Folic acid:*** Folate is needed to make DNA and RNA (the building blocks of cells), is a B vitamin that occurs naturally in food, folate-deficient women who become pregnant are at greater risk of giving birth to low-weight, premature infants with neural tube defects, which can result in the malfunction of the spine (spina bifida), skull and brain. The Recommended Dietary Allowance (RDA) for folate equivalents for pregnant women is 600 micrograms.
2. ***Iron:*** iron is essential for the manufacture of red blood cells that carry oxygen around the body. During pregnancy iron is needed in larger amounts because the mother's blood volume increases and the baby's blood is also developing. Lack of iron can cause anemia, which means the red blood cells are not able to carry enough oxygen around the body leaving you tired and less able to fight off infections. Anemia during pregnancy can persist after the birth of the baby and can also affect the baby's iron stores. The dose is 200 mg tid.
3. ***Calcium:*** Total serum calcium decreases gradually thought pregnancy. Substantial increases in absorptive efficiency and positive balance begin in the 1st trimester. This must represent maternal accumulation of calcium, since the fetal calcium content is negligible at this time. It is possible that calcium added to maternal bone during early pregnancy is transferred to the fetus in later gestation. Calcium supplementation is not necessary in women with a diet that includes adequate dairy foods. Absent this, Calcium supplementation may be used on as may be used on as-needed basisto meet the recommended dietary allowance (RDA) of 1200 mg May during pregnancy.
4. ***Zinc:*** Zinc is a trace mineral. A zinc deficiency may be teratogenic in humans. Zinc levels in amniotic fluid correlate with antimicrobial activity, suggesting that zinc plays a role in protecting against intrauterine infection. Low dietary intake of zinc has been associated with IUGR. The RDA during pregnancy is increased from 15 to 20 mg /day.
5. ***Vitamin D:*** Most vitamin D is synthesized from a precursor in the skin after exposure to UV light from the sun and relatively few foods are good sources of the vitamin. In human pregnancy, high maternal intake of vitamin D was implicated as the cause of a syndrome that included mental and physical retardation and hypercalcemia. 400-500IU vitamin D supplementation have been reported to be safe and adequate.
6. ***Vitamin A:*** Vitamin A appears to be important for fetal growth and poor maternal vitamin A status was associated with preterm birth, intrauterine growth retardation ( IUGR), and decreased birth weight. Vitamin A may be important for lung growth. However an excess leads to teratogenicity mainly in the 1st trimester as CNS abnormalities(hydrocephalus or micricephaly), CVS abnormalities , facial abnormalities and altered growth.

***Abortion***

An abortion is the spontaneous or induced loss of an early pregnancy. The period of pregnancy prior to fetal viability outside of the uterus is considered early pregnancy. Most consider early pregnancy to end at 20-24 weeks' gestation. The term miscarriage is used often in the lay language and refers to spontaneous abortion.

***Maternal causes of spontaneous miscarriage***

1. *Genetic:* (>30% in people aged 40 year). Couples with recurrent miscarriages have a 2-3% incidence of a parental chromosomal anomaly (i.e, balanced translocation).
2. *Structural abnormalities of the reproductive tract* :
3. • Congenital uterine defects (particularly uterine septum)
   * Fibroids
   * Cervical incompetence
4. *Iatrogenic causes* (ie, Asherman syndrome)
5. *Acute maternal factors* :
   * Corpus luteum deficiency
   * Active infection (eg, rubella virus, cytomegalovirus, *Listeria* infection, toxoplasmosis)
6. *Chronic maternal health factors* :
   * Polycystic ovary syndrome
   * Poorly controlled diabetes mellitus (A successful pregnancy requires much tighter control.)
   * Renal disease
   * Systemic lupus erythematosus (SLE)
   * Untreated thyroid disease
   * Severe hypertension
   * Antiphospholipid syndrome
7. *Exogenous factors*:
   * Tobacco
   * Alcohol

• Cocaine

• Caffeine (high doses)

*1. Threatened Abortion*

Threatened abortion is a clinical condition occur in women who are at less than 20 weeks' gestation with a viable pregnancy and have vaginal spotting or bleeding, a closed cervical os, and, possibly, mild uterine cramping. The rate of miscarriage increases with maternal age.

Threatened abortions may continue on as healthy term pregnancies or complete, or incomplete, spontaneous, inevitable may progress to abortions. Vaginal spotting or frank bleeding is very common and is experienced in approximately 25% of clinically apparent pregnancies at less than 20 weeks' gestational age. The bleeding and pain that accompany threatened abortion are not usually intense. Threatened abortion rarely manifests with severe vaginal bleeding. Often, the bleeding is temporary and self-limited and possibly due to trophoblastic implantation within the decidualized endometrium. On examination, blood or brownish discharge may exist in the vagina. The cervix is not tender, and the cervical os is closed. No fetal tissue or membranes have passed. The ultrasound shows a continuing intrauterine pregnancy. If an ultrasound was not performed previously, it is required at this time to rule out an ectopic pregnancy, which could present similarly. If the uterine cavity is empty on ultrasound, obtaining a human chorionic gonadotropin (hCG) level is necessary to determine if the discriminatory zone has been passed.

*Treatment*

1. *Medical Care*: No effective therapy is available for a threatened intrauterine abortion.

1. Bed rest.

2. In general, most do not administer progesterone or sedatives. In most instances of threatened abortions that ultimately result in complete abortion, the embryo is already dead; thus, the administration of progesterone drugs is ineffective and only prolongs the natural course of abortion. However, progesterone (vaginal administration) may be indicated in unique circumstances, including viable pregnancies achieved with advanced reproductive technology or patients with a history of an inadequate luteal phase. progesterone administration may reduce the severity of symptoms such as pain from cramping and uterine contractions.

3. Treat any vaginal infections.

*B. Surgical Care*

*2. Incomplete abortion:* an incomplete abortion is the partial expulsion of the products of conception before the 20th week of gestation. Incomplete abortion in a pregnancy is associated with vaginal bleeding, dilatation of the cervical canal, and passage of products of conception. Usually, the cramps are intense, and the vaginal bleeding is heavy. Patients describe passage of tissue, or the examiner observes evidence of tissue passage within the vagina. The ultrasound confirms that some of the products of conception are still present in the uterus.

*3. Complete Abortion :*Complete abortion is a completed miscarriage. Typically, a history of vaginal bleeding, abdominal pain, and passage of tissue exists. After the tissue passes, the patient notes that the pain subsides and the vaginal bleeding significantly diminishes. The examination reveals some blood in the vaginal vault; a closed cervical os; and no tenderness of the cervix, uterus, adnexa, or abdomen. The ultrasound demonstrates an empty uterus.

*4. Missed Abortion:*a missed abortion is a nonviable intrauterine pregnancy that has been retained within the uterus without spontaneous abortion. Typically, no symptoms exist besides amenorrhea, and the patient finds out that the pregnancy stopped earlier when a fetal heartbeat is not observed or heard at the appropriate time. An ultrasound usually confirms the diagnosis. No vaginal bleeding, abdominal pain, passage of tissue, or cervical changes are present.

*5. Habitual Abortion:* recurrent miscarriage or recurrent pregnancy loss (RPL) is the occurrence of repeated (three or more consecutive) pregnancies that end in miscarriage of the fetus, usually before 20 weeks of gestation.

***Teratology and Drugs in Pregnancy***

A *teratogen:* is an agent that interferes with the normal growth and development of the fetus, and is used to describe drugs or chemicals that cause major or gross birth defects.

The food and Drug Administration (FDA) lists five categories of labeling

for drug use in pregnancy.

*Category A :* no fetal risk shown in controlled human studies.

*Category B :* no human data available and animal studies shown no fetal risk or animal studies show a risk but human studies do not show fetal risk.

*Category C :* no controlled studies on fetal risk available for humans or animals or fetal risk shown in controlled animal studies but no human.

data available (benefit of drug use must clearly justify potential fetal risk in this category).

*Category D :* studies show fetal risk in humans ( use of drug may be acceptable even with risks such as in life threatening illness or where safer drugs are ineffective).

*Category X :* risk to fetus clearly outweighs any benefits from these drugs.

***Gestational Disorders***

***A. Gestational Diabetes (GD)***

GD is the most common type of diabetes complicating pregnancy, and most patients are obese. The women with GD has a normal oral glucose tolerance test(OGTT) when she is not pregnant, so her disease usually is mild. pregnancy is associated with increased tissue resistance to insulin, resulting in increased levels of blood insulin as well as glucose and trigclyerides. These changes are due to placental lactogen and elevated circulating esterogens and progesterone.

***Risk factors for GDM***

1. Maternal age greater than 30 years.

2. Previous macrosomic, malformed, or stillborn infant.

3. GDM in a previous pregnancy.

4. Family history or diabetes.

5. Maternal obesity.

6. Persistent glucosuria.

7. Chronic use of certain drug such as β-agonists or corticosteroids.

*Maternal Problems*

1. Hypoglycemia: occurs during the first half of pregnancy due to increased insulin sensitivity.

2. Hyperglycemia: occurs during the second half of pregnancy.

3. UTI.

4. Hypertension: the abnormal blood vessels of pregnant women with DM can lead to the development of hypertension in the later weeks of gestation since the abnormal endothelium cannot produce enough prostacyclin to antagonize the elevated angiotensin II vasopressor levels.

5. Hydromnios: excess amounts of aminotic fluid can occur with DM especially if glucose is poorly controlled since maternal hyperglycemia produces fetal hyperglycemia and fetal glucosuria.

6. Retinopathy.

*Infant Problems*

1. Spontaneous abortion.

2. Congenital abnormalities: CVS and CNS most affected systems

3. Respiratory distress: since hyperglycemia interferes with ability of cortisol to accelerate surfactant production.

4. Hypoglycemia: since the fetus exposed to high glucose levels coming across the placenta from a hyperglycemic mother reacts by producing large amounts of insulin in an attempt to reduce glucose.

5. Macrosomia: more than 4 kg.

6. Hypocalcemia.

7. Hyperbilirubinemia: results from a higher hematocrit developed in utero especially if oxygen availability is decreased.

8. Perinatal mortality: since acute deprivation caused by glucose binding to Hb or sudden shifts in water and electrolytes with glucose movements have been suspected.

***Management***

1. Diet control.

2. Insulin therapy: the beginning dose is higher in late pregnancy therefore starting dose is 0.5 u/kg in the first half of gestation and then 0.7 u/kg in the second half, and increase the dose twofold-threefold during the 20-30 week period at a total dose of 15-20 u/day.

***B. Hypertensive Disorder of Pregnancy***

**I. Gestational hypertension** is defined as a persistent systolic blood pressure level of 140 mm Hg or greater or a diastolic blood pressure level of 90 mm Hg or greater that occurs on two occasions 4 hours apart after 20 weeks of gestation in a woman with previously normal blood pressure.

**II. Preeclampsia: see below**

**III. Eclampsia: see below**

**IV. Chronic hypertension: see below**

**Chronic hypertension**

Patients with a persistent elevation of blood pressure to at least 140/90 mm Hg on two occasions before 20 weeks’ gestation, and patients with hypertension that persists for more than 6 weeks postpartum.

Complications related to chronic hypertension include superimposed preeclampsia, fetal growth restriction, pre-term birth, and placental abruption. The risk of developing one of these complications correlates with the degree of maternal blood pressure elevation; the higher the blood pressure, the greater the risk of one of these complications.

A number of antihypertensives have been shown to be safe and effective during pregnancy in controlling maternal blood pressure. Treatment of elevated blood pressure with antihypertensives reduces the risk of maternal morbidities related to hypertension but does not reduce the risk of fetal complications such as intrauterine growth restriction, preeclampsia, and placental abruption.

***Antihypertensive Therapy in Chronic Hypertension***

Several choices for initial antihypertensive therapy during pregnancy are available.

**1-Methyldopa** has been studied extensively and is recommended by many as the first-line antihypertensive agent in pregnancy. It is a centrally acting alpha-adrenergic agonist that appears to inhibit vasoconstricting impulses from the medullary vasoregulatory center. The total daily dosage of 500 mg to 2 g is administered in 2–4 divided doses

Sedation and postural hypotension are the most common side effects. A positive direct Coombs’ test may be seen, usually after 6–12 months of therapy. Hemolytic anemia may occur in these patients and is an indication to stop the medication. Fever, liver function abnormalities, granulocytopenia, and thrombocytopenia are rare side effects.

**2-Labetalol** is an alpha1-adrenergic blocker and a nonselective beta-adrenergic blocker. The betablockade/ alpha-blockade ratio is 7:1. A large body of clinical evidence suggests that use of labetalol is safe during pregnancy. One randomized study showed no advantages of labetalol over methyldopa. The usual starting dose is 100 mg twice per day (BID), and the dose can be increased weekly to a maximum of 2400 mg daily. Titration increments should not exceed 200 mg BID.

**3-Nifedipine** is a calcium channel blocker that has been used during pregnancy for tocolysis and treatment of hypertension. Several reports suggest that nifedipine use is safe during pregnancy;

When nifedipine is used for treatment of chronic hypertension during pregnancy, the long acting formulation (Procardia XL, Adalat CC) may improve patient compliance. The principal benefit of this agent is once-daily dosing. The usual starting dose is 30 mg daily. If necessary, the dose may be increased to 60–90 mg daily. The neuro-muscular-blocking action of magnesium may be potentiated by simultaneous calcium channel blockade; therefore, nifedipine should be used with caution in patients receiving magnesium sulfate. The sublingual route of administration is associated with unpredictable blood levels and should be avoided.

**Other antihypertensive medications** used in pregnancy include atenolol, metoprolol, prazosin, minoxidil, hydralazine, thiazide diuretics, and clonidine. Published experience with these agents is limited, and they should not supplant methyldopa, labetalol, or nifedipine as first-line agents in pregnancy.

* Fetal bradycardia, growth retardation, and neonatal hypoglycemia have been reported in patients treated with blockers.

**Note:**Use of angiotensin-converting enzyme inhibitors (enalapril, captopril) during pregnancy is associated with fetal hypocalvaria, renal defects, anuria, and fetal and neonatal death. These agents are contraindicated in pregnancy.

With few exceptions, diuretics (furosemide, hydrochlorothiazide) should be avoided during pregnancy.

**PREECLAMPSIA**

• **Preeclampsia**: a syndrome of gestational hypertension plus end-organ manifestations includingnproteinuria with proteinuria defined as urinary excretion of 0.3 g protein or more in a 24-hour urine specimen or a protein/creatinine ratio ≥0.3 mg/dL. In the absence of proteinuria, new-onset hypertension with thrombocytopenia (less than 100,000 platelets/mL) or renal insufficiency (serum creatinine concentration greater than 1.1 mg/dL) or impaired liver functions (transaminases twice the upper limits of normal concentration) constitute diagnostic criteria of preeclampsia. There are only two types of preeclampsia: mild and severe.

***Etiology***

Preeclampsia is a disorder of unknown etiology that is peculiar to human pregnancy. Many theories regarding its etiology have been suggested, including

• Abnormal placentation

• Immunologic phenomena

• Coagulation abnormalities

• Abnormal cardiovascular adaptation

• Dietary factors

• Genetic factors

• Angiogenesis factors

• Vascular endothelial damage

• Abnormal prostaglandin metabolism

***Risk Factors***

• Nulliparity

• Multiple gestation

• Previous pregnancy with preeclampsia

• Family history of preeclampsia or eclampsia

• Preexisting hypertension or renal disease

• Pregestational diabetes

• Use of donor oocytes

• Nonimmune hydrops fetalis

• Molar pregnancy

• Obesity

**Management**

Once the diagnosis of preeclampsia has been made, definitive therapy in the form of delivery is the desired goal because it is the only cure for the disease. The ultimate goals of the therapy must always be first the safety of the mother and then the delivery of a mature newborn that will not require intensive and prolonged neonatal care.

**MILD PREECLAMPSIA**

**Evaluation**

• At the time of diagnosis, all patients with preeclampsia should be evaluated regardin maternal/fetal condition

***Maternal Evaluation***

**History**

Markers for possible severe preeclampsia:

• Persistent occipital or frontal headaches

• Visual disturbances

• Right upper quadrant abdominal or epigastric pain

**Physical Evaluation**

• Blood pressure assessment at diagnosis, then twice weekly by the health care provider.

• Urine protein assessment at diagnosis. If significant proteinuria is identified, subsequent proteinuria evaluation is not necessary as the amount or change in the amount of proteinuria will not influence the need for delivery.

• Weight daily.

**Laboratory Evaluation**

• Hematocrit and platelet count once per week

• Liver function tests once per week

• Twenty-four–hour urine collection at diagnosis for total protein excretion and creatinine

clearance or a protein/creatinine ratio to confirm the diagnosis

***Fetal Evaluation***

• Daily fetal movement assessment (kick counts)

• Nonstress test (NST) twice weekly

• Biophysical profile if nonreactive NST

• Amniotic fluid volume assessment weekly

• Ultrasound evaluation of fetal growth every 3 weeks

**Management**

• Women with mild disease who achieve a gestational age of 37 weeks should undergo delivery.

Therapy for patients with mild disease can be conducted by either outpatient management or hospitalization.

* Outpatient management is acceptable for patients who are compliant, who can have frequent office visits including laboratory assessments, and who can perform some form of adequate blood pressure monitoring at home.
* Hospitalization should be required for noncompliant patients and those who show unsatisfactory progress as outpatients.

• If outpatient management is used, the regimen described below is recommended for mild preeclampsia.

* A patient is considered a candidate for induction of labor if she has reached 37 weeks’ gestation.
* She is also a candidate for induction if her blood pressure continues to rise despite conservative management.

**PATIENT EDUCATION**

• Close communication between the patient and physician is obligatory for successful outpatient management of mild gestational hypertension and preeclampsia. Patients are instructed to contact their managing physician for one for more of the following symptoms specific to preeclampsia:

• Blood pressure above a chosen target level

• A severe, long-lasting headache

• Epigastric or right upper quadrant abdominal pain

• Visual disturbances

• Nausea and vomiting

• The patient should also be instructed to notify her physician for the following complications of pregnancy, regardless of preeclampsia:

• Vaginal bleeding

• Leakage of fluid from the vagina

• Regular preterm uterine contractions

• Decreased fetal movement

**SEVERE PREECLAMPSIA**

• The clinical course of severe preeclampsia is usually characterized by progressive deterioration in both maternal and fetal status.

• Most of the fetal or neonatal complications are related to intrauterine fetal growth retardation, placenta abruption, or prematurity.

**Diagnosis**

1• Blood pressure ≥160 mm Hg systolic or ≥110 mm Hg diastolic on two occasions atleast 4 hours apart with the patient on bed rest.

2• Cerebral or visual disturbances.

3• Severe and persistent epigastric or right upper quadrant abdominal pain.

4• Pulmonary edema or cyanosis.

**Management**

• All patients with severe preeclampsia should be admitted to the labor and delivery area for close observation of maternal and fetal condition and provided steroids for lung maturity if less than 34 weeks’ gestation during initial evaluation and with the decision for delivery.

• All patients should receive intravenous magnesium sulfate to prevent convulsions.

• Control of maternal blood pressure within a safe range

• Initiating delivery

• Management of patients with severe disease remote from term (less than 34 weeks 0 days) is controversial.

**ECLAMPSIA**

***Definition***

• Eclampsia is the development of convulsions or coma unrelated to other cerebral conditions during pregnancy or in the postpartum period in patients with signs and symptoms of preeclampsia.

**Treatment**

***Management***

1• Support of Cardiorespiratory Functions

2• Control of Convulsions and Prevention of Recurrent Convulsions

3• Magnesium Sulfate Therapy

* Parenteral magnesium sulfate is the drug of choice for convulsions resulting from eclampsia.Its major advantages include relative maternal and fetal safety when properly used. The mother is awake and alert most of the time, and laryngeal reflexes are intact, which helps protect against aspiration problems.
* There are several regimens of magnesium sulfate used to prevent convulsions. The most commonly used is an intravenous loading dose of 6 g of magnesium sulfate (MgSO4·7H2O) prepared as 6 g diluted in 150 mL D5W or lactated Ringer solution is administered via infusion pump over 20 to 30 minutes.
* If the patient develops recurrent convulsions after the initial infusion of magnesium sulfate, a further dose of 2 g can be infused over 5 to 10 minutes.On completion of the magnesium sulfate loading infusion, a maintenance infusion of 2 to 3 g/h is used.
* The infusion rate of magnesium sulfate should be adjusted on the basis of physical examination and maternal urine output. Serial serum magnesium levels need not be followed except in situations with increased serum creatinine or decreased urine output.
* Patients receiving magnesium sulfate therapy must be monitored for evidence of drug toxicity.Magnesium is excreted by the kidneys, and renal dysfunction may cause toxic accumulation.
* Magnesium toxicity can be avoided by:
  + Confirming adequate renal function with hourly urinary output assessment
  + Serial evaluation for the presence of patellar deep tendon reflexes
  + Closely observing respiratory rate
  + Monitoring serial serum magnesium levels when toxicity is suspected
* If magnesium toxicity is suspected, the following steps should be taken:

1-The magnesium sulfate infusion should be discontinued.

2-Supplemental oxygen should be administered.

3-A serum magnesium level should be assessed.

4-If magnesium toxicity is recognized, 10 mL of 10% calcium gluconate is administered (1 g total) intravenously. This medication must be given slowly (i.e., 2 to 5 mL/min) to avoid hypotension, bradycardia, and vomiting.

-Calcium competitively inhibits magnesium at the neuromuscular junction, but its effect is only transient because the serum concentration is unchanged. Symptoms of magnesium toxicity can recur following calcium gluconate administration if the magnesium level remains elevated.

• At delivery, neonatal side effects of maternal administration of magnesium sulfate include

* + Hypotension
  + Hypotonia
  + Respiratory depression
  + Lethargy
  + Decreased suck reflex

***Control of Severe Hypertension***

• The objective of treating severe hypertension is to prevent maternal cerebrovascular accidents and congestive heart failure without compromising cerebral perfusion or jeopardizing

uteroplacental blood flow, which is already reduced in eclampsia.

**• Labetalol**

* Parenteral labetalol has a rapid onset of action and produces a smooth reduction in blood pressure with rare overshoot hypotension.
* Labetalol is contraindicated in patients with a greater than first-degree heart block.
* Labetalol is administered in intermittent intravenous boluses of 20 to 80 mg.

**• Hydralazine**

* Hydralazine is a direct arteriolar vasodilator.
* Intravenous hydralazine has an onset of action of 10 to 20 minutes, with a peak effect in 60 minutes, and a duration of action of 4 to 6 hours.
* Hydralazine is administered in intermittent bolus injections with an initial dose of 5 mg. Blood pressure should be recorded every 5 minutes.
* If an adequate reduction in blood pressure is not achieved 20 to 30 minutes after the initial dose, then a repeat dose of 5 mg or a dose increased to 10 mg in increments of every 20 to 30 minutes should be given for a maximum of 25 mg/h.

**• Nifedipine**

* Nifedipine is a calcium channel antagonist.
* Nifedipine improves renal function with a beneficial effect on urine output when treating preeclampsia in the postpartum period.
* Nifedipine is administered 10 to 20 mg orally every 4 hours.
* Profound reductions in blood pressure with nifedipine can be partially reversed by the slow intravenous administration of calcium gluconate.

**• Sodium nitroprusside**

* Sodium nitroprusside relaxes arteriolar and venous smooth muscle by interfering

with both influx and the intercellular activation of calcium.

* Onset of action is immediate, and duration of action is very short (1 to 10 minutes).
* Because preeclamptic patients have a propensity for depleted intravascular volume, they are especially sensitive to its effects. The initial infusion dose should therefore be 0.2 μg/kg/min, rather than 0.5 μg/kg/min as is standard in nonpregnant patients.
* Cyanide and thiocyanate are products of metabolism of this drug with potential deleterious effects for the fetus.

**Complications of Eclampsia** Maternal complications with eclampsia convulsions including

1• Abruptio placentae

2• Pulmonary edema

3• Acute renal failure

4• Aspiration pneumonia

**5-**HELLP SYNDROME Hemolysis, abnormal liver function tests, and thrombocytopenia have long been recognized as complications of preeclampsia and eclampsia.

• *H* for hemolysis

• *EL* for elevated liver enzymes

• *LP* for low platelet count

• The incidence of severe preeclampsia or eclampsia complicated by HELLP syndrome has been reported to range from 2% to 12%

**Criteria for the Diagnosis of HELLP Syndrome**

Hemolysis:Abnormal peripheral smear

Total bilirubin >1.2 mg/dL

Lactic dehydrogenase >600 U/L

Elevated liver functions:Serum aspirate aminotransferase >70 U/L

Low platelets Platelet count <100,000/mm3

Patients with HELLP syndrome may present with a variety of signs and symptoms, including

• Epigastric or right upper-quadrant abdominal pain

• Nausea or vomiting

• Nonspecific viral syndrome–like symptoms

• History of malaise for the past few days before presentation

***Management of HELLP Syndrome***

• Maternal condition is assessed and stabilized.

• If disseminated intravascular coagulopathy (DIC) present, coagulopathy is corrected.

• Antiseizure prophylaxis is given with magnesium sulfate.

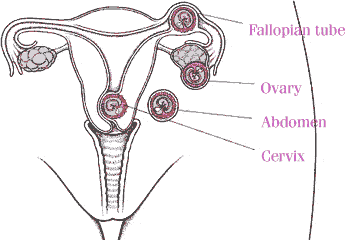
• Treatment of severe hypertension is begun.

• Computed tomography or ultrasound of the abdomen is done if a subcapsular hematoma of the liver is suspected.

• Fetal well-being is evaluated.

***C. Gestational Trophoblastic Disease (Hydatidiform Mole & Choriocarcinoma)***

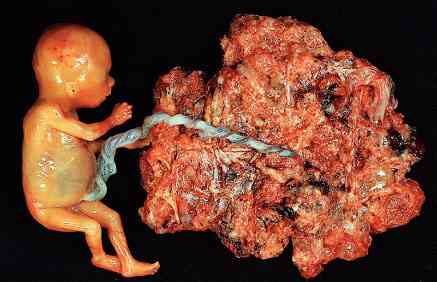
Gestational trophoblastic disease is a spectrum of dis­orders that includes hydatidiform mole, invasive mole, and choriocarcinoma. Partial moles generally show ev­idence of an embryo or gestational sac; are polyploid, slower-growing, and less symptomatic; and often present clinically as a missed abortion. Partial moles tend to follow a benign course, while complete moles have a greater tendency to become choriocarcinomas.



***Symptoms and diagnosis***

Excessive nausea and vomiting occur in over one-third of patients with hydatidiform mole. Uterine bleeding, beginning at 6-8 weeks, is observed in virtually all in­stances. In about one-fifth of cases, the uterus is larger than would be expected in a normal pregnancy of the same duration. Bilaterally enlarged cystic ovaries are sometimes palpable. They are the result of ovarian hyperstimulation due to excess of hCG. Preeclampsia-eclampsia, frequently of the fulmi­nating type, may develop during the second trimester of pregnancy, but this is unusual.

Choriocarcinoma may be manifested by continued or recurrent uterine bleeding after evacuation of a mole or following delivery, abortion, or ectopic preg­nancy. The presence of an ulcerative vaginal tumor, pelvic mass, or evidence of distant metastatic tumor may be the presenting observation. The diagnosis is established by pathologic examination of curettings or by biopsy.



***Laboratory findings***

A serum hCG (3-subunit value above 40,000 mU/mL ) or a urinary hCG value in excess of 100,000 units/24 h increases the likelihood of hydatidiform mole.

*Imaging*

Ultrasound has virtually replaced all other means of preoperative diagnosis of hydatidiform mole. A preoperative chest film is indicated to rule out pulmonary metastases of trophoblast.

*Treatment*

*A .Specific surgical measures*

The uterus should be emptied as soon as the diagnosis of hydatidiform mole is established, preferably by suc­tion. Ovarian cysts should not be resected nor ovaries removed; spontaneous regression of theca lutein cysts will occur with elimination of the mole.

If malignant tissue is discovered at surgery or during the follow-up examination, chemotherapy is indicated.

Thyrotoxicosis indistinguishable clinically from that of thyroid origin may occur. While hCG usually has minimal TSH-like activity, the very high hCG lev­els associated with moles result in the release of T3 and T4 and cause hyperthyroidism. Patients thyrotoxic on this basis should be stabilized with (3-blockers prior to induction of anesthesia for their surgical evacuation. Surgical removal of the mole promptly corrects the thyroid overactivity.

*B .Follow up measures*

Effective contraception (preferably birth control pills) should be prescribed. Weekly quantitative hCG level measurements are initially required. Following successful surgical evacuation, moles show a progres­sive decline in hCG. After two negative weekly tests (< 5 mU/mL), the interval may be increased to monthly for 6 months and then to every 2 months for a total of 1 year. If levels plateau or begin to rise, the patient should be evaluated by repeat chest film and dilatation and curettage (D&C) before the initiation of chemotherapy.

***D. Seizure disorders***

Epileptic women contemplating pregnancy who have not had a seizure for 5 years should consider a prepregnancy trial of withdrawal from treatment. Those with recurrent epilepsy should use a single drug with blood level monitoring. valproate is contraindicated during pregnancy; phenytoin and carbamazepine may be teratogenic in the first trimester and should not be used unless absolutely necessary. Phenobarbital is considered the drug of choice. Serum levels should be measured in each trimester and dosage adjustments made to keep serum levels in the low normal therapeutic range. Pregnant women taking phenobarbital and phenytoin should receive vitamin supplements, in­cluding folic acid and vitamin D, throughout pregnancy. Vitamin K, 10-20 mg/d, is administered during the last month to help prevent bleeding problems in the new­born, who is at risk of bleeding tendencies due to de­creased levels of clotting factors. Such infants should re­ceive an injection of vitamin K- 1 mg subcutaneously immediately after delivery, and should have clotting stud­ies 2—4 hours later. Breast-feeding is not contraindicated for infants of mothers taking anti-seizure medications.

***E.Thyroid Disease***

Thyrotoxicosisduring pregnancy may result in fetal anomalies, late abortion, or preterm labor and fetal hyperthyroidism with goiter. Thyroid storm in late pregnancy or labor is a life-threatening emergency.

Radioactive isotope therapy must never be given during pregnancy. The antithyroid drug of choice is propylthiouracil, which acts to prevent further thyroxine formation by blocking iodination of tyrosine. There is a 2- to 3-week delay before the pretreatment hormone level begins to fall. The initial dose of pro­pylthiouracil is 100—150 mg three times a day; the dose is lowered as the euthyroid state is approached. It is desirable to keep free T4 in the high normal range during pregnancy. A maintenance dose of 100 mg/d minimizes the chance of fetal hypothyroidism and goiter.

Maternal hypothyroidism even subclinical hy­pothyroidism manifested only by elevated levels of TSH—may adversely affect subsequent neuropsychological development of the child. Mothers with known or suspected hypothyroidism should have the TSH level measured at the first prenatal visit. Replacement therapy with levothyroxine should be adjusted to maintain levels of TSH in the normal range.

***F.Urinary Tract Infection*  -**

The urinary tract is especially vulnerable to infections during pregnancy because the altered secretions of steroid sex hormones and the pressure exerted by the gravid uterus upon the ureters and bladder cause hypotonia and congestion and predispose to urinary stasis. Labor and delivery and urinary retention post-partum also may initiate or aggravate infection. *Eschrichia coli* is the offending organism in over two-thirds of cases.

From *2%* to 8% of pregnant women have asymp­tomatic bacteriuria, which some believe to be associ­ated with an increased risk of prematurity. It is esti­mated that pyelonephritis will develop in 20-40% of these women if untreated.

A first-trimester urine culture is indicated in women with a history of recurrent or recent episodes of urinary tract infection. If the culture is positive, treatment should be initiated as a therapeutic measure. Nitrofurantoin (100 mg twice daily), ampicillin (500 mg four times daily), and cephalexin (500 mg four times daily) are acceptable medications for 3—7 days. Acute pyelonephritis re­quires hospitalization for intravenous administration of antibiotics until the patient is a febrile; this is followed by a full course of oral antibiotics.

***G. Anemia***

Anemia in pregnancy is defined as a Hb below 10 g/dl or hematocrit below 30%.

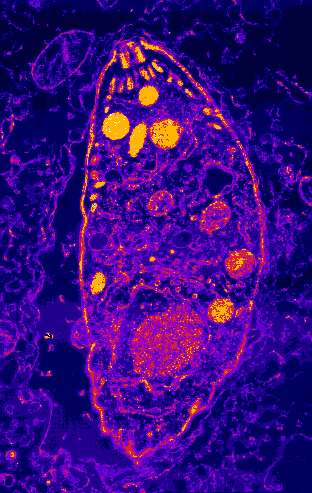
Plasma volume increases 50% during pregnancy, while red cell volume increases 25% causing lower HB and hematocrit values, which are maximally changed around the 24th-28th weeks. Anemia is very common in pregnancy causing fatigue, anorexia, dyspnea and edema.

1. Iron deficiency anemia: many women enter pregnancy with reduced iron stores resulting from heavy menstrual periods, previous pregnancies, breast-feeding, or poor nutrition. It is difficult to meet the increased requirement for iron through diet and anemia often develops unless iron supplements are given. RBCs may not become hypochromic and microcytic until the hematocrit has fallen significantly. Treatment consists of a diet containing iron-rich foods and 60 mg of elemental iron 3 times daily with meals. Iron is best absorbed if taken with a source of vitamin C.
2. Folic acid deficiency anemia: folic acid deficiency is the main cause of macrocytic anemia in pregnancy, since vitamin B12 deficiency anemia is rare in the childbearing years. The daily requirements of folic acid doubles from 0.4mg -0.8 mg in pregnancy.

Twin pregnancies, infections, malabsorption, and use of anticonvulsants such as phenytoin can precipitate folic acid deficiency. The anemia may first be seen in the puerperium owing to the increased need for folate during lactation. Good sources of folate in food are leafy green vegetables, orange juices, peanuts and beans. Cooking and storage of food destroy folic acid.

***Toxoplasmosis***

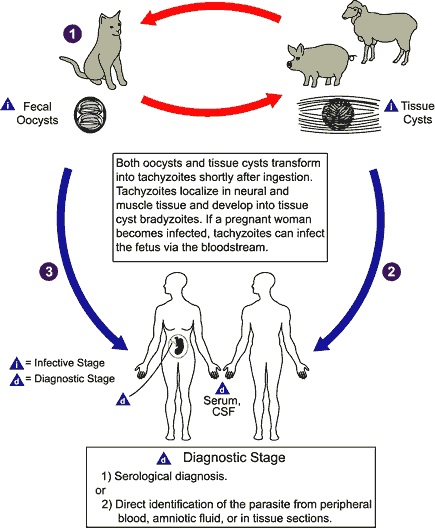
Toxoplasmosis is an infection caused by the protozoal parasite *Toxoplasma* *gondii.*

**

***Transmission Routes***

1. Cats: ingestion of oocysts shed by cats which are the definitive hosts for *T.gondii* and the cats become infected by eating infected wild rodents and birds.

2.Meat: ingestion of infected meat containing tissue cysts is an important cause of toxoplasmosis.



1. In Utero Transmission: Newborn with congenital toxoplasmosis become infected in utero by transplacental passage of the parasite when the mother has acute infection. Incidence and severity vary with the trimester of gestation during which the mother acquired the infection in which 10-20% (1st trimester),30-54%(2nd trimester), 60-65%(3rd trimester) may occur.

Congenital infection occurring in the 1st trimester is the most severe. 89-100% of infections in the 3rd trimester are asymptomatic and risk to the fetus is not correlated with symptoms in the mother.

*Physical findings and clinical presentation*

1. Acquired (immunocomptent host): 80-90% of the cases are asymptomatic but adenopathy(usually cervical),fever, myalgias, malaise, sore throat, maculopapular rash, hepatosplenomegaly may occur.
2. Congenital: results from acute infection acquired by the mother within 6-8 weeks before conception or during gestation. Usually the mother is asymptomatic with no signs of disease but the following conditions may occur as chorioretinitis, blindness, epilepsy, psychomotor or mental retardation, intracranial calcifications, hydrocephalus, encephalitis, anemia, thrombocytopenia, hepatosplenomegaly, lymphadenopathy, jaundice, rash, and pneumonitis.



*Diagnosis*

1. *Acute infection, immunocomptent host:* CBC, *Toxoplasma* serology (IgG,Ig) in serial blood specimens 3 week apart, and lymph node biopsy if diagnosis uncertain.
2. *Toxoplasmosis in pregnancy:*
3. Initial maternal screening with IgM and IgG.
4. Acute maternal infection not excluded or documented: fetal blood sampling ( for culture, Ig, IgA, IgE.
5. *Congenital toxoplasmosis:*
6. Placental histology.
7. Specific IgM or IgA in infants blood.

*Lab. Studies: by antibody studies including:*

1. *IgM:* appears 5 days into infection, peaks at 2 week and falls to low level or disappears within 2 weeks. It may persist at low levels for 1 year or more.
2. *IgG :*appears 1-2 week after infection, peaks at 6-8 weeks and gradually declines over months to years.

*Treatment*

1. *Acute infection in pregnancy*: treatment should be started immediately and risk of fetal infection reduced by 60% with treatment. In 1st trimester treatment is done with spiramycin 3g orally in 3-4 divided doses per day or sulfadiazine 4 g orally in 4 divided doses. In the 2nd and 3rd trimesters, sulfadiazine as above plus pyrmethamine 25 mg orally /day plus lucovorin 5-15 mg orally /day or spiramycin as in the same dose mentioned above.

*B. Congenital infection:* sulfadiazine 50mg/kg orally bid plus pyrimethamine 2 mg/kg orally for 2 days then 1mg/kg orally 3 times weekly plus lecoverin 5-20 mg orally 3 times weekly for 12 months as minimum duration of treatment.

*Prevention*

Prevention is most important in seronagative pregnant women and include ccoking meat to 66 C◦ and cooking eggs, no drinking of unpasteurized milk, washing hands thoroughly after handling raw meat, washing kitchen surfaces that come in contact with raw meat, washing fruits and vegetables and avoiding contact with materials potentially contaminated with cat feces.

***Erythrocyte Immunization (Rh Disease)***

Erythroblastosis fetalis (i.e., hemolytic disease of the newborn) is caused by an incompatibility between fetal and maternal blood. The Rh-negative mother becomes immunized by exposure to Rh-positive fetal erythrocytes during pregnancy or delivery, and antibodies formed by the mother pass through the placenta to the fetal circulation, where they react with the Rh-positive fetal erythrocytes, causing a hemolytic anemia.

*Factors influencing Rh immunization*

1 . Aminocentesis

2. Threatened abortion, placenta previa, placental abruption

3. Abdominal truma

4. External version

5. Fetal death

6. Sinusoidal fetal heart tracing

7. Multiple pregnancy

8. Cesarean section

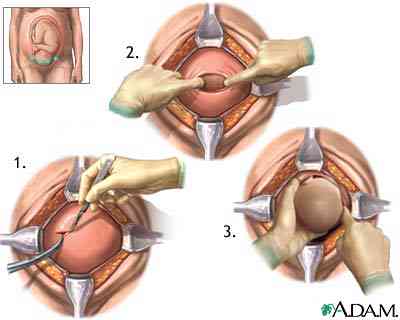
9. Anemic infant

*Treatment*

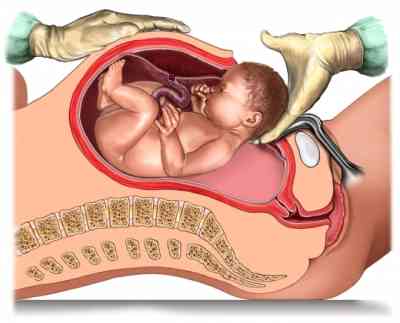
Passive immunization against hemolytic disease of the newborn is achieved with Rho(D) immune globuline, a purified concentrate of antibodies against Rho(D) antigen. The Rho(D) immune globuline (one vial of 300 mcg IM) is given to the mother within 72 hours after delivery( or spontaneous or induced abortion or ectopic pregnancy). the antibodies in the immune globuline destroy fetal Rh-positive cells so that the mother will not produce anti- Rho(D). during her next Rh-positive gestation, erythroblastosis will be prevented. An additional safety measure is the administration of immune globulin at the 28th week of pregnancy. the passive antibody titer that results is too low to significantly affect an Rh-positive fetus. The maternal clearance of the globulin is slow enough that protection will continue for 12 weeks.

***Cesarean section***

Cesarean section is a term used to describe the delivery of a vaiable fetus through an incision in the abdominal wall (laproscopy) and the uterus.The majority of cesarean sections are performed for fetal indications, afew are soly for maternal reasons, and some benefit both fetus and mother.



*Common indications for cesarean delivery*

1. precious (high risk) Fetus
2. prolonged labour or a failure to progress (dystocia)
3. apparent fetal distress
4. apparent maternal distress
5. complications (pre-eclampsia, active herpes)
6. catastrophes such as cord prolapse or uterine rupture
7. multiple births
8. abnormal presentation (breech or transverse positions)
9. failed induction of labour
10. placental problems (placenta praevia, placental abruption or placenta accreta)
11. umbilical cord abnormalities (vasa previa, multi-lobate including bi-lobate and succenturiate-lobed placentas, velamentous insertion)
12. contracted pelvis
13. Sexually transmitted infections such as genital herpes (which can be passed on to the baby if the baby is born vaginally, but can usually be treated in with medication and do not require a c-section)
14. previous caesarean section prior problems with the healing of the
15. perineum (from previous childbirth or Crohn's Disease).
    1. 

***Complications of C-section***

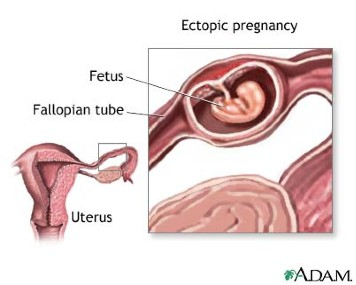
Most of the serious complications associated with cesarean section are not due to the operation itself. Instead, the complications arise from the indication for the cesarean section. For example, a woman whose placenta separates prematurely (placental abruption) may require an emergency cesarean section. Under these circumstances, complications arise primarily from the placental abruption itself.Fortunately, serious complications are rare. However, the following minor complications can occur in women having cesarean sections

1. Infection
2. Bleeding
3. Atony
4. Lacerations
5. Placenta Accreta
6. Blood Clots

**Ectopic Pregnancy**

**Definition**

An ectopic pregnancy is one in which the fertilized ovum implants at any site other than the endometrial cavity. The fallopian tube is the most common site, accounting for more than 95% of ectopic pregnancies, but other implantation sites include the cervix, abdominal cavity, and ovary.



**Incidence**

Currently, ectopic locations are diagnosed in approximately 2% of clinically recognized pregnancies. The prevalence of ectopic among women presenting with first-trimester bleeding and/or abdominal pain can be up to 18%. Ectopic pregnancy is the most common cause of nonpuerperal maternal mortality and is the leading cause of first-trimester maternal death.

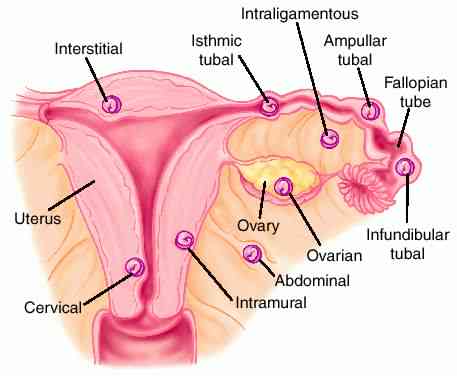
**Etiology**

A number of risk factors for ectopic pregnancy have been identified including the following conditions:

* Salpingitis. Approximately 50% of ectopic pregnancies can be attributed to a history of salpingitis. Chlamydial salpingitis may pose a greater risk than gonorrheal infection.
* Prior ectopic pregnancy
* Peritubal adhesions following postabortal infections, appendicitis, or endometriosis.
* Tubal surgery, including tubal ligation, and tubal reconstruction for fertility.
* Intrauterine device. Intrauterine devices (IUDs) are highly effective at preventing intrauterine pregnancy. Thus, any pregnancy in an IUD user is more likely to be tubal.
* Progestin-only contraceptives. Users of progestin-only oral contraceptives as well as injectable progestins are at increased risk of ectopic pregnancy if pregnancy occurs, possibly because of altered tubal motility.
* History of infertility.
* Increased maternal age.

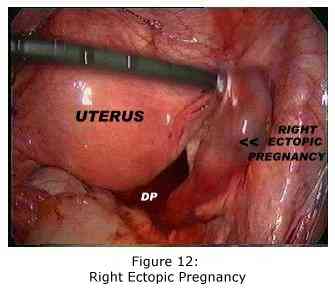
*Clinical Findings*

1. *Symptoms and signs:* they may be acute or chronic.
2. *Acute**(40%):* Severe lower quadrant pain occurs in almost every case. It is sudden in onset, lancinating, intermittent, and does not radiate. Backache is present during attacks. Shock occurs in about 10%, often after pelvic examination. At least two-thirds of patients give a history of abnormal menstruation; many have been infertile.
3. *Chronic (60%):* Blood leaks from the tubal am­pulla over a period of days, and considerable blood may accumulate in the peritoneum. Slight but persis­tent vaginal spotting is reported, and a pelvic mass can be palpated. Abdominal distention and mild paralytic ileus are often present.



1. *Laboratory findings*
2. *hCG:* if normal intrauterine pregnancy(IUP), 85% have doubling time of 2 days. If abnormal gestation, will show < 66% increase of QhCG within 2 days. However, 13% of ectopic pregnancies have a normal doubling time.
3. *Progesterone:* decreased production in ectopic pregnancy, < 5 ng/ml strongly predictive of abnormal pregnancy. If > 25 ng/ml strongly predictive of normal pregnancy.
4. *Drooping Hct associated with tubal rupture.*
5. Leukocytosis.
6. *Imaging*

*In U/S* presence of IUP rules out ectopic pregnancy*.* If QhCG>6000mIU/ml, should see IUP on abdominal scan, and QhCG >1500mIU/ml for transvaginal scan. Findings on U/S in ectopic pregnancy include empty uterus, adnexal mass, Cul-ed-sac fluid, fetal sac in tube and fetal cardiac activity in adnexa.



*D. Special examination*

With the advent of high-resolution transvaginal ultra­sound used in evaluation of possible ectopic pregnancy. Laparoscopy is the surgical procedure of choice both to confirm an ectopic preg­nancy and in most cases to permit pelviscopic removal of the ectopic pregnancy without the need for explor­atory laparotomy.

**TREATMENT**

Ectopic pregnancies can present as life-threatening emergencies. The patient presenting in shock with an acute abdomen should be stabilized and taken to surgery immediately. Fluid resuscitation must be carried out immediately. Laboratory tests needed are minimal. Blood should be drawn for hematocrit and crossmatched for four units of red cells. A β-hCG level should be obtained, but it is not necessary to wait for the results. The patient should be taken to surgery as quickly as possible.

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**• Surgical management**

• Surgical options are operative laparoscopy or laparotomy. The first choice for surgical management is operative laparoscopy with either salpingostomy or salpingectomy. Laparotomy is reserved for specific indications.

• Laparoscopic salpingostomy is the procedure of choice in most circumstances. Salpingectomy is selected if future fertility is not desired (e.g., ectopic pregnancy after tubal ligation) or if rupture has destroyed the tube

• Laparotomy should be performed if laparoscopy is unsatisfactory because of extensive adhesions, if the patient becomes unstable, or if there are medical limitations to laparoscopy. After conservative surgery (when the tube is not removed), weekly β-hCG levels should be obtained until they are less than negative (values vary by laboratory). If there is concern about the completeness of removal of the pregnancy, postoperative prophylactic methotrexate using the single-dose regimen significantly decreases the rate of persistence. Early detection of trophoblastic persistence is facilitated by persistently elevated or rising β-hCG levels.

• **Medical management**

• Medical management has the advantage of avoiding surgery with its attendant risks. Patients who are clinically stable with a small, unruptured ectopic pregnancy may be offered medical management with systemic methotrexate, a folic acid antagonist that preferentially inhibits rapidly replicating cells such as trophoblast. In properly selected patients, methotrexate is 75% to 85% effective in resolving ectopic pregnancy, with the remaining women requiring surgery.

• Criteria for medical management include hemodynamic stability, gestational sac less than 3.5 cm in diameter, β-hCG at diagnosis less than 5000 IU, no ultrasound fetal cardiac activity, minimal hemoperitoneum, no underlying liver or renal disease, no blood dyscrasia, not breast-feeding, and ability to have regular follow-up.

• Pretreatment complete blood count and platelet count, β-hCG level, and liver and renal function tests should be obtained.

• Methotrexate is given in either single-dose or multidose regimens (Table 7-4). The two regimens have been widely studied for treatment of ectopic pregnancy and both are acceptable. The multidose regimen has a lower failure rate; however, the risk of complications including diarrhea, abnormal liver function, and stomatitis is greater with the multidose regimen.The single-dose regimen is slightly less successful, requiring a second dose in up to 20% of women; however, there is a lower incidence of side effects. The failure rate is approximately 15% with the single-dose regimen when the initial β-hCG level is greater than 5000 IU but drops to approximately 4% with lower initial β-hCG levels. The failure rate of either regimen increases when a live embryo, a high initial β-hCG level, or a large adnexal mass is present.

Follow-up after methotrexate includes measurement of β-hCG on days 4 and 7 after the single dose. The day 4 level is usually increased over baseline due to lysis of trophoblast. The day 7 level should be at least 15% less than the day 4 level or the dose may be repeated. Repeat dosing may also be required if β-hCG levels increase or plateau. Levels of β-hCG should be followed weekly until reaching a nonpregnant level (threshold will vary by laboratory). Surgical intervention is rarely required but may be needed to manage severe pain, hemorrhage, or treatment failure.

**Long-Term Prognosis**

• Women who have had one ectopic pregnancy are at significant risk for future infertility and for recurrent ectopic pregnancies.

• Women who have had an ectopic pregnancy should be educated about the symptoms associated with ectopic pregnancy and should be counseled to seek care immediately upon diagnosis of a subsequent pregnancy, regardless of symptoms. β-hCG levels should be monitored, and an early ultrasound should be performed.



***Preterm Labor***

*Risk factors for preterm labor*

1. Twin pregnancy

2. Uterine abnormalities

3. vaginal fibronectin

4. Age and race: increased age will increase the incidence of preterm labor. Black people have a short gestational period and their infants are of lower weight per week of age

5. Prior preterm labor

6.UTI

7. bacterial vaginosis

8. vaginal pH >4.5

*Signs of preterm labor*

* uterine contractions and cramps
* vaginal discharge
* bleeding
* backache
* leaking amniotic fluid

*Treatment of preterm labor*

*1. Bed rest*

*2. Hydration:* 500 ml of balanced electrolyte solution, such as Ringers lactate IV over 30 min.peroid. Hydration is continued at a rate of at least 125 ml/hour.

*3. Tocolytics*

*A. Magnesium sulfate*: as high conc. have been shown to decrease uterine activity. The dose is 6 g IV as bolus dose in 250 ml of sol. over 30 min. period; the infusion is then maintained at 2-4 g /hr.

*B*. *β- Mimetic drugs*: like ritodrine as it causes uterine relaxation is administered IV and slowly titrated upward until a response is achieved. The dose is 100 µ /min. IV, with increases of 50 µ/min.every 10 min. to a max. of 350ug/min.

*4.Glucocorticosteroid*: these drugs are administered for the reduction of respiratory distress syndrome in preterm infants. The mechanism by which these drugs decrease lung disease is enzyme induction in type II pneumocytes of increased production of surfactant, which in turn reduces alveolar surface tension. All women between 24 and 34 weeks of pregnancy at risk for preterm delivery are candidates for antenatal corticosteroid therapy. Treatment should consist of either tow doses of 12 mg of betamethasone IM 24 hours apart or 4 doses of 6 mg of dexamethasone IM 12 hours apart. Some benefit begins at 24 hours, with a max. benefit at 48 hours after imitation of therapy and lasting for 7 days. Treatment is given weekly until fetal maturity.

5. *Group B Streptococcus Treatment:* Premature infants are very susceptible to early GBS infections. So the use of pencillin is recommended.

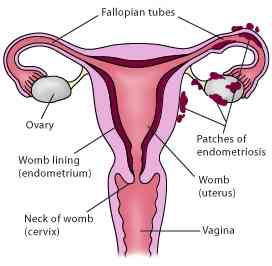
*6.Calicum Channel Blockers:* these drugs have been used for preterm labor as nifedipine, nicardipine and verapamil as they inhibit contractions. Dosage of nifedipine is 10-20 mg every 4-6 hours orally sublingually in the first hour, followed by 60-160 mg/day of slow-release nifedipine.

***Part –B-***

***Gynecology***

***Endometriosis***

Endometriosis is the growth of cells similar to those that form the inside of the uterus (endometrial cells), but in a location outside of the uterus. Endometrial cells are the same cells that are shed each month during menstruation. The cells of endometriosis attach themselves to tissue outside the uterus and are called endometriosis implants. These implants are most commonly found on the ovaries, the Fallopian tubes, outer surfaces of the uterus or intestines, and on the surface lining of the pelvic cavity. They can also be found in the vagina, cervix, and bladder, although less commonly than other locations in the pelvis. Rarely, endometriosis implants can occur outside the pelvis, on the liver, in old surgery scars, and even in or around the lung or brain. Endometrial implants, while they can cause problems, are benign (not cancerous),most cases of endometriosis are diagnosed in women aged around 25-35 years.



*Causes of Endometriosis*

Several different hypotheses have been put forward as to what causes endometriosis. Unfortunately, none of these theories have ever been entirely proven, nor do they fully explain all the mechanisms associated with the development of the disease. Thus, the cause of endometriosis remains unknown.

1. Metaplasia

2. Retrograde menstruation

3. Genetic predisposition

4. Lymphatic or vascular distribution

5. Immune system dysfunctions

6. Environmental influences

*Symptoms of endometriosis*

**•** Very painful menstrual cramps

**•** Pain with periods that getsworse over time

**•** Chronic pain in the lower back and pelvis

**•** Pain during or after sex

**•** Intestinal pain

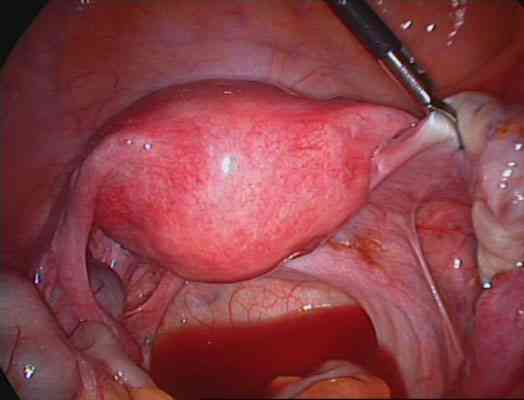
**•** Painful bowel movements or painful urination during menstrual periods

**•** Heavy and/or long menstrual periods

**•** Spotting or bleeding between periods

**•** Infertility (not being able to get pregnant)

**•** Fatigue



*Diagnoses of endometriosis*

These include ultrasound, MRI scans, and gynaecological examinations. None of these can definitively confirm endometriosis (though they can be suggestive of the disease), nor can they definitively dismiss the presence of endometriotic lesions/cysts.

*Treatment*

There is no cure for endometriosis, but there are many treatments for the pain and infertility that it causes. The treatment chosen will depend on symptoms, age, and plans for getting pregnant.

1. *Pain Medication:* for some women with mild symptoms over-the-counter medicines can be used for pain. These include: ibuprofen or naproxen.
2. *Hormone Treatment*: when pain medicine is not enough, hormone medicines are used to treat endometriosis. Only women who do not wish to become pregnant can use these drugs. Hormone treatment is best for women with small growths who don't have bad pain. Medical treatment suppresses endometriosis, rather than removing it and is effective only for short term management of symptoms, the active endometriosis returning gradually over 12-24 months after stopping the drugs. Medical treatment suppresses endometriosis, rather than removing it and is effective only for short term management of symptoms, the active endometriosis returning gradually over 12-24 months after stopping the drugs.

The aim of drug therapy is to break the cycle of stimulation and bleeding. By stopping the ovary's usual hormonal cycle and reducing estrogen levels, the endometrial deposits shrink down and become inactive. The endometriosis is still there, and will gradually become reactivated when the normal menstrual cycle starts again.Ovarian endometriomas of greater than 3cm diameter are unlikely to respond to medical treatment, and similarly if there is a significant amount of adhesions - these will respond best to [laparoscopic](http://www.endo-resolved.com/laparoscopy.html) surgery.

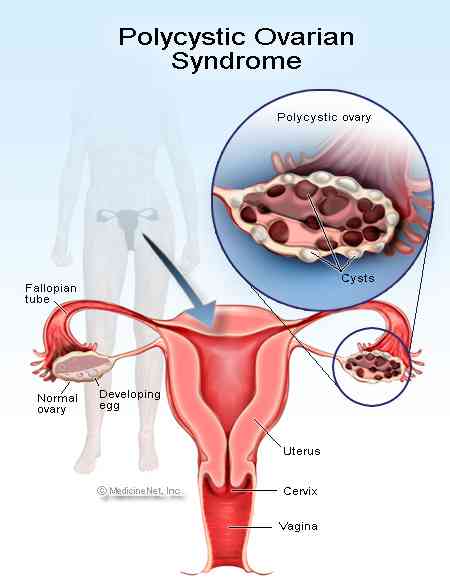
This aim of drug treatment is to alter the chemical and hormone levels in the body which in turn will affect the natural bodily processes. This will also affect the behaviour of the Endometrial growths. These including:

1. *Birth control pills*: block the effects of natural hormones on growths. So, they prevent the monthly build-up and breakdown of growths. This can make endometriosis less painful. Birth pills also can make a woman's periods lighter and less uncomfortable. Most birth control pills contain two hormones, estrogen and progestin which can be used for 6-12 months. Breakthrough bleeding can be treated with conjugated estrogen 1.25 mg daily for 1 week or esteradiol 2mg daily for 1 week.
2. *Progestins:*or progesterone medicines work much like birth control pills and can be taken by women who can't take estrogen oppose the estrogen effects on the endometrial growths which causes them to ‘shrink’. Progesterone also prevents ovulation which lowers the estrogen levels.When a woman stops taking progestins, she can get pregnant again. But, the symptoms of endometriosis return too. Medroxyprogesterone acetate 100 mg every 2 weeks for 4 doses and then 100 mg every 4 weeks. Add oral estrogen or esteradiol valearate 30 mg IM for breakthrough bleeding for 6-9 months.
3. *Gonadotropin releasing hormone agonists or GnRH agonists:* slow the growth of endometriosis and relieve symptoms. They work by greatly reducing the amount of estrogen in a woman's body, which stops the monthly cycle. Leurprolide 3.75 mg IM/month for 6 months is a GnRH agonist often used to treat endometriosis. GnSH agenists should not be mused alone for more than six months. This is because they can lead to osteoporosis. But if a woman takes estrogen along with GnRH agonists, she can use them for a longer time. When a woman stops taking this medicine, monthly periods and the ability to get pregnant return. But, usually the problems of endometriosis also return. Nafarelin nasal spray 0.2-0.4 mg twice daily can also be used.
4. *Danazol*: is a weak male hormone. Nowadays, it is rarely recommend for endometriosis. Danazol lowers the levels of estrogen and progesterone in a woman's body. This stops a woman's period or makes it come less often. Danazol also gives pain relief , but it often causes side effects like oily skin, weight gain, tiredness, smaller breasts, and hot flashes. Danazol does not prevent pregnancy and can harm a baby growing in the uterus. It is used for 4-6 months in the lower doses to suppress menstruation at a dose of 200-400 mg daily.

1. *Surgery*: Surgery is usually the best choice for women with endometriosis who have a severe amount of growths, a great deal of pain, or fertility problems.
2. *Laparoscopy****:*** can be used to diagnose and treat endometriosis. During this surgery, doctors remove growths and scar tissue or destroy them with intense heat. The goal is to treat the endometriosis without harming the healthy tissue around it. Women recover from laparoscopy much faster than from major abdominal surgery.
3. *Laparotomy or major abdominal surgery*::is a last resort treatment for severe endometriosis. In this surgery, a much bigger cut in the abdomen than with laparoscopy will be done. This allows to reach and remove growths of endometriosis in the pelvis or abdomen. Recovery from this surgery can take up to two months.
4. *Hysterectomy:* should only be considered by women who do not want to become pregnant in the future. During this surgery, the uterus will be removed as well as the ovaries and fallopian tubes at the same time. This is done when the endometriosis has severely damaged them.

***Poly Cystic Ovary Syndrome***

Polycystic ovary syndrome (PCOS), previously known as Stein-Leventhal syndrome, is a disorder in which numerous benign cysts form on the ovaries under a thick, white covering. It is most common in women under 30 years old.Elevated serum LH concentrations and an increased serum LH:FSH ratio result either from an increased GnRH hypothalamic secretion or less likely from a primary pituitary abnormality. This results in dysregulation of androgen secretion and increased intraovarian androgen, the effect of which in the ovary is follicular atresia, maturation arrest, polycystic ovaries, and anovulation. Hyperinsulinemia is a contributing factor to ovaian hyperandrgenism, independent of LH excess. A role for insulin growth factor (IGF) receptors has been postulated for the association of PCOS and DM. Imbalance of these hormones prevents the ovaries from releasing an egg each month. It also results in an increased production of the male hormone testosterone by the ovaries.



*Symptoms of PCOS*

* . Amenorrhea (no menstrual period), infrequent menses, and/or oligomenorrhea (irregular bleeding).
* Oligo or anovulation (infrequent or absent ovulation).
* Hyperandrogenism.
* Infertility
* Cystic ovaries
* Enlarged ovaries.
* Obesity or weight gain.
* Insulin resistance, hyperinsulinemia, and diabetes.
* Dyslipidemia (lipid abnormalities).
* Hypertension.
* Hirsutism.
* Alopecia
* Acne/Oily Skin/Seborrhea
* Acanthosis nigricans (dark patches of skin, tan to dark brown/black).



*Diagnoses*

1. *Biochemical analysis:* Fasting comprehensive biochemical and lipid panel,2-hour GTT with insulin levels (also called IGTT), LH:FSH ratio, serum total testosterone level, Serum Hormone Binding Globulin (SHBG) level, serum androstenedione level, serum prolactin level and serum TSH, T4,T3 level.
2. *Imaging studies:* pelvic U/S (or CT scan) reveals the presence of 2-fold-5-fold ovarian enlargement with a thickened tunica albuginea, thecal hyperplasia, and 20 or more subcapsullar follicles from 1-15 mm in diameter.

*Treatment*

1. *Metabolic derangements*: diet and exercise in patients with PCOS who are obese , endocrine-metabolic parameters markedly improve after 4-12 weeks of dietary restriction. Their SHBG levels rise and free testosterone levels fall by 2-fold. Serum insulin and IGF-1 levels also decrease. Weight loss in patients with PCOS who are obese is associated with a reduction of hirsutism and a return of ovulatory cycles in 30% of women. A moderate amount of daily exercise increases of levels of IGF-1 binding protein and decreases IGF-1 levels by 20%. Modest weight loss of 2-5% of total body weight can help restore ovulatory menstrual periods in obese patients with PCOS. A daily 500-1000 calorie deficit with 150 minutes of exercise per week can cause ovulation.

***Investigational Therapies***

New evidence suggests that using medications which lower insulin levels in the blood may be effective in restoring menstruation and reducing some of the health risks associated with PCOS. Lowering insulin levels also helps to reduce the production of testosterone, thus diminishing many of the symptoms associated with excess testosterone: hair growth on the body, alopecia (scalp hair loss), acne, and, possibly, cardiovascular risk. Metformin improves insulin resistance and decrease hyperinsulinemia in patients with PCOS. The usual starting dose is 500 mg given orally twice a day. A decrease in body fat will lower the conversion of androgens to esterone thereby help restore ovulation.

Pioglitazone (Actos®) and Rosiglitazone (Avandia®) are insulin-sensitizing agents that improve glucose tolerance and insulin resistance.

1. *Anovulation:* metformin can reduce hyperinsulinemia and hyperandrogenemia in PCOS. Metformin combined with clomiphene resulted in ovulation in 76% of patients  
   compared with 42% in patients who received clomiphene alone. Metformin also has a small but beneficial effect on metabolic syndrome at a dose of 500 mg 3 times daily for 3-6 months. Management of unfertilized patients with PCOS include the usage of clomiphene. Other, more aggressive, treatments for [infertility](http://www.medicinenet.com/script/main/art.asp?articlekey=40638) (including injection of gonadotropin hormones and assisted reproductive technologies) may also be required in women who desire pregnancy and do not become pregnant on clomiphene therapy.
2. *Hirsutism*
3. *Hair removal:* short-term nonpharmacologic treatments of hirsutism include shaving and use of chemical depilatories and/or bleaching cream.. Weight reduction decreases androgen production in women who are obese; therefore, losing weight can slow hair growth.
4. *Oral contraceptives*: women who do not wish to become pregnant can be effectively treated for hirsutism with oral contraceptives. Oral contraceptives slow hair growth in 60-100% of women with hyperandrogenemia. Therapy can be started with a preparation that has a low dose of estrogen and a nonandrogenic progestin. Preparations that have norgestrel and levonorgestrel should be avoided because of their androgenic activity.

*C. Spironolactone:* antiandrogens, such as spironolactone, are effective for hirsutism. Spironolactone 50-100 mg twice daily is an effective primary therapy for hirsutism. Because of its potential teratogenic effects, spironolactone should be prescribed with an oral contraceptive. Adverse effects of spironolactone include GI discomfort, and irregular  
menstrual bleeding (which can be managed by adding an oral contraceptive).

*D. Flutamide:* 250 mg daily or finasteride 5 mg daily.

*E. Eflornithine:* Eflornitliine (Vaniqa®) is a topical cream that can be used to slow the hair growth. Eflornithine works by inhibiting ornithine decarboxylase, which is essential for the rapidly dividing cells of hair follicles.

*4*. *Menstrual irregularity :*this is treated with an oral contraceptive, which not only  
inhibits ovarian androgen production but also increases SHBG production.

*5.Surgical Care:* surgical management is aimed mainly at restoring ovulation.

***Placental problems***

**Role of placenta**

The placenta is an organ that develops in the uterus during pregnancy. This structure provides oxygen and nutrients to the growing baby and removes waste products from the baby's blood. The placenta attaches to the wall of the uterus, and the baby's umbilical cord arises from it. In most pregnancies, the placenta attaches at the top or side of the uterus.

**Factors affect placental health**

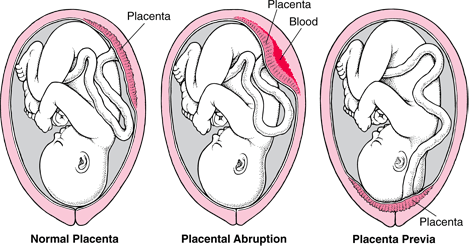
Various factors can affect the health of the placenta during pregnancy, some modifiable and some not. For example:

* **Maternal age.** Certain placental problems are more common in older women, especially after age 40.
* **Premature rupture of the membranes.** During pregnancy, baby is surrounded and cushioned by a fluid-filled membrane called the amniotic sac. If the sac leaks or breaks before labor begins, the risk of certain placental problems increases.
* **High blood pressure.** High blood pressure can affect the placenta.
* **Twin or other multiple pregnancy.** pregnant with more than one baby, might be at increased risk of certain placental problems.
* **Blood-clotting disorders.** Any condition that either impairs blood's ability to clot or increases its likelihood of clotting increases the risk of certain placental problems.
* **Previous uterine surgery.** If woman had a previous surgery on her uterus, such as a C-section or surgery to remove fibroids, she at increased risk of certain placental problems.
* **Previous placental problems.** A placental problem during a previous pregnancy might be at increased risk of experiencing it again.
* **Substance abuse.** Certain placental problems are more common in women who smoke or use illegal drugs, such as cocaine, during pregnancy.
* **Abdominal trauma.** Trauma to the abdomen — such as from a fall or other type of blow to the abdomen — increases the risk of certain placental problems.

**The most common placental problems**

During pregnancy, the most common placental problems include placental abruption, placenta previa and placenta accreta. These conditions can cause potentially heavy vaginal bleeding. After delivery, retained placenta is also sometimes a concern.

* **Placental abruption (abruptio placentae).** If the placenta peels away from the inner wall of the uterus before delivery — either partially or completely — it's known as placental abruption. Placental abruption can cause varying degrees of vaginal bleeding and pain or cramping. It might also deprive the baby of oxygen and nutrients. In some cases, early delivery is needed.
* **Placenta previa.** This condition occurs when the placenta partially or totally covers the cervix — the outlet for the uterus. Placenta previa is more common early in pregnancy and might resolve as the uterus grows. Placenta previa can cause severe vaginal bleeding before or during delivery. A C-section delivery usually is required if the placenta previa is present at the time of delivery.
* **Placenta accreta.** This condition occurs when the blood vessels of the placenta grow too deeply into the uterine wall. Placenta accreta can cause vaginal bleeding during the third trimester of pregnancy and severe blood loss after delivery. Treatment might require a C-section delivery followed by surgical removal of the uterus (abdominal hysterectomy). More-aggressive forms of this problem can also occur if the placenta invades the muscles of the uterus (placenta increta) or if the placenta grows through the uterine wall (placenta percreta).
* **Retained placenta.** If the placenta isn't delivered within 30 to 60 minutes after childbirth, it's known as retained placenta. Retained placenta might occur because the placenta becomes trapped behind a partially closed cervix or because the placenta is still attached to the uterine wall — either loosely (adherent placenta) or deeply (placenta accreta). Left untreated, a retained placenta can cause severe infection or life-threatening blood loss in the mother.



***Female infertility***

 According to the [World Health Organization](https://en.wikipedia.org/wiki/World_Health_Organization) (WHO), infertility can be described as the inability to become pregnant, maintain a pregnancy, or carry a pregnancy to live birth.[[3]](https://en.wikipedia.org/wiki/Female_infertility#cite_note-WHO.2C_2013-3) A clinical definition of infertility by the [WHO](https://en.wikipedia.org/wiki/WHO) is “a disease of the reproductive system defined by the failure to achieve a clinical pregnancy after 12 months or more of regular unprotected sexual intercourse.” [[4]](https://en.wikipedia.org/wiki/Female_infertility#cite_note-Zegers-Hochschild-4)Infertility can further be broken down into primary and secondary infertility. [Primary infertility](https://en.wikipedia.org/wiki/Primary_infertility) refers to the inability to give birth either because of not being able to become pregnant, or carry a child to live birth, which may include miscarriage or a stillborn child. [[5]](https://en.wikipedia.org/wiki/Female_infertility#cite_note-WHO_terminology.2C_2013-5)[[6]](https://en.wikipedia.org/wiki/Female_infertility#cite_note-Rutstein_2004-6) [Secondary infertility](https://en.wikipedia.org/wiki/Secondary_infertility) refers to the inability to conceive or give birth when there was a previous pregnancy or live birth.[[6]](https://en.wikipedia.org/wiki/Female_infertility#cite_note-Rutstein_2004-6)[[5]](https://en.wikipedia.org/wiki/Female_infertility#cite_note-WHO_terminology.2C_2013-5)

### Factors Affect Fertility:

#### Hypothalamic-pituitary factors

* [Hypothalamic dysfunction](https://en.wikipedia.org/wiki/Hypothalamic_dysfunction)
* [Hyperprolactinemia](https://en.wikipedia.org/wiki/Hyperprolactinemia)

#### Ovarian factors

* [Chemotherapy](https://en.wikipedia.org/wiki/Chemotherapy) with certain agents have a high risk of toxicity on the ovaries.
* [Polycystic ovary syndrome](https://en.wikipedia.org/wiki/Polycystic_ovary_syndrome)
* [Anovulation](https://en.wikipedia.org/wiki/Anovulation). Female infertility caused by anovulation is called "anovulatory infertility", as opposed to "ovulatory infertility" in which ovulation is present.
* [Premature menopause](https://en.wikipedia.org/wiki/Premature_menopause)
* [Menopause](https://en.wikipedia.org/wiki/Menopause)
* Luteal dysfunction
* Gonadal dysgenesis ([Turner syndrome](https://en.wikipedia.org/wiki/Turner_syndrome))
* [Ovarian cancer](https://en.wikipedia.org/wiki/Ovarian_cancer)

#### Tubal (ectopic)/peritoneal factors

* [Endometriosis](https://en.wikipedia.org/wiki/Endometriosis)
* Pelvic [adhesions](https://en.wikipedia.org/wiki/Adhesion_(medicine))
* [Pelvic inflammatory disease](https://en.wikipedia.org/wiki/Pelvic_inflammatory_disease) (PID, usually due to [chlamydia](https://en.wikipedia.org/wiki/Chlamydia_infection))
* [Tubal occlusion](https://en.wikipedia.org/wiki/Tubal_occlusion)
* Tubal dysfunction
* Previous [ectopic pregnancy](https://en.wikipedia.org/wiki/Ectopic_pregnancy)

#### Uterine factors

* [Uterine malformations](https://en.wikipedia.org/wiki/Uterine_malformation)
* [Uterine fibroids](https://en.wikipedia.org/wiki/Uterine_fibroids)
* [Asherman's Syndrome](https://en.wikipedia.org/wiki/Asherman%27s_Syndrome)
* [Implantation failure](https://en.wikipedia.org/wiki/Implantation_failure) without any known primary cause. It results in negative pregnancy test despite having performed e.g. [embryo transfer](https://en.wikipedia.org/wiki/Embryo_transfer).

Previously, a [bicornuate uterus](https://en.wikipedia.org/wiki/Bicornuate_uterus" \o "Bicornuate uterus) was thought to be associated with infertility, but recent studies have not confirmed such an association.

#### Cervical factors

* [Cervical stenosis](https://en.wikipedia.org/wiki/Stenosis_of_uterine_cervix)
* Antisperm antibodies
* Non-receptive cervical [mucus](https://en.wikipedia.org/wiki/Mucus)

#### Vaginal factors

* [Vaginismus](https://en.wikipedia.org/wiki/Vaginismus)
* Vaginal obstruction

***Appendix***

***Some drugs used in obstetrics and gynecology***

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
| ***Dose and duration of therapy*** | ***Dosage form(s)*** | ***Mechanism of action*** | ***Indications*** | ***Trdae name®*** | | ***Drug name*** | |
| *1 trab. Daily for 5 days starting within about 5th day of the cycle* | *50mg/tab.* | *Antiesterogenic action (partial esterogen agonist)* | *Anovulatory infertility* | *Clomide* | | *Clomifene* | |
| *Infertility: 20 mg/day on days 2,3,4,5 of the cycle; 20/day in BC* | *10, 20 mg/ tab.* | *Antiesterogenic action (partial estradiol inhibitor)* | *Anovulatory infertility, breast cancer(BC)* | *Nolvadex* | | *Tamoxifen* | |
| *1 tab./day* | *2.5 mg/ tab* | *Aromatase inhibitor (an enzyme required for esterogen synthesis)* | *BC in postmenopausal women* | *Femara* | | *Letrozole* | |
| *By IM use according to patient´s response* | *1500,5000 mg/amp.* | *Increase FSH, LH secretion cause induction of ovulation* | *Infertility in women with hypopitutarism* | *Pregnyl* | | *Human Chorionic Gonadotophin(hCG)* | |
| *By IM use according to patient´s response* | *FSH 75 units, LH 75 units/vial* | *Stimulant of  FSH , LH to stimulate the ovaries to produce eggs* | *Women infertility with hypopitutarism* | *Pergonal* | | *Human Menopausal Gonadotrophins(hMG) contain FSH, LH* | |
| *By IM, SC use according to patient´s response* | *FSH 75 units units/vial* | *Stimulant of  FSH to stimulate the ovaries to produce egg* | *Women infertility with hypopitutarism* | *Gonal –F* | | *FSH* | |
| *1-2 times daily at bed time and increased gradually according to patient´s response* | *2.5 mg/tab.* | *Stimulate dopamine receptors and reduce prolactine release* | *Treatment of hypogonadism , galactorrhea, infertility* | *Parlodel* | | *Bromocriptin* | |
| *1 mg/week as asingle dose or 2 divided doses in different days* | *0.5mg/tab.* | *Same as bromocriptine* | *Same as bromocriptine* | | *Dostinex* | *Cabergolin* | |
| *By S.C. inj. 3.6 mg/28 day(max. 6 months)* | *3.6 mg/syringe* | *Gondorelin analogue stimulate the production of estrogen which then suppressed by body´s feedback mechanism* | *Endomertiosis and BC* | | *Zoladex* | *Goseleine* | |
| *By S.C. inj. 3.75 mg as a single dose repeat monthly (max. 6 months)* | *3.75 mg /1 ml syringe* | *Gondorelin analogue stimulate the production of estrogen which then suppressed by body´s feedback mechanism* | *Endomertiosis and BC* | | *Lupron depot* | *Leuprorelin* | |
| *1-2 tab tid ; deep IM inj. Within 5 days of cycle and last for 12 weeks.* | *2.5, 5 ,10 mg/tab.; 150 mg/ml inj.* | *A progersterone analgoue* | *Contraception, endometriosis* | | *Provera, Depoprovera* | *Medroxyprogesterone* | |
| *2-3 tab./day in divided doses* | *5 mg/tab.* | *A progersterone analgoue* | *HRT, Contraception, endometriosis* | | *Primolute- N* | *Northisterone* | |
| *1 tab. /day* | *6.25, 1.25 mg/tab.* | *A mixture of esterogen hormones substitute the loss of esterogen and alleviates the menopausal symptoms* | *Menopausal symptoms, osteoporsis prophylaxis, HRT* | | *Premarin* | *Conjugated esterogen* | |
| *blue tab./day from day 5 of the cycle then 1 tab. day in sequence* | *12 blue estradiol 2 mg; 10 whilte estradiol 2mg and northisteron 1mg; 6 red estradiol 1 mg* | *esterogen hormones substitute the loss of esterogen and alleviates the menopausal symptoms, progestine( reduce the esterogen- induced risk of endometriosis),selective inhibition of pituitary function results in ovulation inhibition* | *Menopausal symptoms, osteoporsis prophylaxis, HRT* | | *Trisequens* | *Estradiol + progesterone* |
| *2-3 tab./day* | *10 mg/tab.* | *A progesterone analogue* | *Recurrent miscarriage, endometriosis* | | *Duphastone* | *Dydrogesteron* |
| *Cyclogest :200-400mg vaginally or rectally; Gestone deep IM inj. 5-10 mg/day for 5-10 days; Crinone: 1 application vaginally* | *Cyclogest: 200,400 mg pessaries; Gestone: 50 mg/ml amp.; Crinone 90 mg/aaplication* | *Its action on the womb lining causes it to thicken in preparation for the fertilized egg to implant; it supports the placenta and prevents the uterus from spontaneously aborting the fetus* | *Cyclogest : premenstrual syndrome and natal depression; Gestone : recurrent miscarriage due to inadequate luteal phase; Crinone : infertility due to inadequate luteal phase* | | *Cyclogest pessaries; Gemstone inj.; Crinone vaginal gel* | *Progesterone* |
| *By IM inj. According to patient´s condition* | *0.5 mg/tab.; 0.5 mg/ml amp.* | *Ergot alkaloid act as blood vessels constrictor to decrease mother blood loss* | *Induction of utrine contactions and minimize postpartum haemorrhage* | | *Methergin* | *Methylergometrine* |
| *2-3 times /day according to the case* | *500 mg/tab.; 100 mg/ml amp.* | *Antifibrinolytic agent stops bleeding* | *Reduce haemorrhage and stop vaginal bleeding* | | *Cyklokapron* | *Tranxemic acid* |
| *By slow IV inj. According to patient´s condition* | *5 units/ml amp., 10 units/ml amp.* | *A hormone release from the posterior lob of pituitary to induce utrine contractions* | *Induce labour* | | *Pitocin* | *Oxytocin* |
| *By mouth or vaginal route according to the case* | *0.2 mg/tab.* | *PG cause vaginal bleeding* | *Induce medical abortion* | | *Cytotec* | *Misopristol* |
| *1 tab.twice daily* | *10,20 mg/tab.* | *Vasodilator, increase blood flow* | *Uterine relaxant* | | *Duvadilan* | *Isoxsopurine* |
| *Endometriosis 200-800 mg in divided doses; BC 300 mg in divided doses* | *100,200 mg/ cap* | *A synthetic androgen hormone, inhibit pituitary gonadotropins( ES and PG)* | *Endometriosis therapy; benign fibrocystic breast disease* | | *Danol* | *Danazol* |

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* ***Polycystic Ovarian Syndrome: Treatment & Medication. Author: Mukhtar I Khan, MD, Assistant Professor of Medicine, Division of Endocrinology, Diabetes & Metabolism, SUNY Upstate Medical University, Syracuse, NY.*** [***Contributor Information and Disclosures***](javascript:showcontent('active','authordisclosures');)***. Updated: Sep 4, 2009.***
* ***Polycyctic ovary syndrome,www.diagnose-me.com.***
* ***www.uchospitals.edu › ... ›*** [***Polycystic Ovary Syndrome***](http://www.google.com/url?q=http://www.uchospitals.edu/specialties/pcos/index.html&sa=X&ei=OtAxTJuKHeiIOJ2TiP8B&ved=0CCEQ6QUoAQ&usg=AFQjCNHK-inisXBP5C2UfJIsQcV6h_a4kQ)
* ***www.medicinenet.com › ... ›*** [***polycystic ovary index***](http://www.google.com/url?q=http://www.medicinenet.com/script/main/forum.asp%3Farticlekey%3D453&sa=X&ei=OtAxTJuKHeiIOJ2TiP8B&ved=0CCsQ6QUoAQ&usg=AFQjCNGAo2Gl4yTN4rELwqONmemlBAocoQ)
* ***Endometriosis.*** [***www.medicinenet.com***](http://www.medicinenet.com)***.***
* ***Endometriosis.*** [***www.medic8.com/healthguide/articles/endometriosis***](http://www.medic8.com/healthguide/articles/endometriosis)
* [***www.healthopedia.com***](http://www.healthopedia.com)
* ***www.endo-resolved.com/treatment.html***
* ***Early Pregnancy Loss.Author: Elizabeth E Puscheck, MD, Professor, Department of Obstetrics and Gynecology, Wayne State University School of Medicine; In Vitro Fertilization Director, Gynecologic Ultrasound Director, Clinical Endocrine Laboratory Consultant, Department of Obstetrics and Gynecology, University Women's Care.***[***Contributor Information and Disclosures***](javascript:showcontent('active','authordisclosures');)***.Updated: Feb 17, 2010.Obstetrics: Normal and Problem Pregnancies, 5th ed.Copyright ©*** [***2007 Churchill Livingstone, An Imprint of Elsevier***](http://www.mdconsult.com/about/book/churchill.html)
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* ***www.healthline.com/.../complications-cesarean-section***