METHODICAL POINTING
for the independent work of students for preparation to practical lesson

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Obstetrical bleeding in II half of pregnancy, in labor and during postpartum term.

Obstetric hemorrhage (OH) can be a very serious complication of pregnancy. According to the World Health Organization, OH is a cause of death of 125-150 thousands women every year. Each 4th the incidence of maternity mortality is OH. Rate of OH - 3-11% (4% - of vaginal deliveries; 6-8% - of cesarean section).

The acceptable blood loss is 250-400 ml, accounting for 0,3-0,5% of the body weight, that usually tolerates without complications. Blood loss of more than 0,5% of the body weight is considered to be pathological. Considering that pregnant, parturient and puerperal women is lowered tolerance to blood loss and symptoms of shock may develop in its volume of 750-1000 ml, blood loss up to 1-1,5% of the body weight is regarded as a moderate, but more than 1,8% of body weight as a massive.

Classification of causes of the obstetrical hemorrhages

1. Causes of the hemorrhages in the first trimester of pregnancy:
   - spontaneous abortion;
   - hydatidiform mole;
   - ectopic pregnancy (including cervical pregnancy).

2. Causes of the hemorrhages in the second trimester of pregnancy:
   - placental presentation;
   - premature abruption of normally situated placenta;
   - uterine rapture (see section IV).

3. Causes of the hemorrhages during labor:
   Stage I:
   - premature abruption of normally situated placenta;
   - placenta previa;
   - uterine rupture;
   - cervical laceration;
Stage II:
- premature abruption of normally situated placenta;
- uterine rapture;

Stage III:
- pathological placentation;
- placental retention, entrapment;
- laceration of the soft tissues of the parturient canal.

4. Postpartum hemorrhage:
- hypotonic hemorrhage;
- retention of the placental fragments;
- laceration of the soft tissues of the parturient canal;
- uterine rupture;
- amniotic fluid embolism;
- coagulopathy.

5. Hemorrhages, not related to pregnancy:
- cervical polyp;
- cervical cancer.

6. Secondary (late) postpartum hemorrhage
- retained placental fragments;
- purulo-septic complications after the delivery;
- untimely diagnosis of the vaginal hematoma;
- dehiscence of the uterine scars and uterine injuries (after cesarean section or uterine rapture).
- causes not related to pregnancy and labor (perineal caviar lesion rupture, cervical carcinoma, etc.).
I. ANTEPARTUM HEMORRHAGE
(in the first trimester of pregnancy)

1. Spontaneous abortion – let see section “Prematurity”.

2. Hydatidiform mole

**Definition.** Hydatidiform mole is characterized by the hyperplasia of both trophoblast layers, rapid increase and swelling of chorion villi, resembling the bunch of grapes (hydro tropic transformation of chorion villi), the presence of vesicles with a volume up to 15 mm, filled with fluid, consisting of albumin and mucin. Hydatidiform mole develops due to abnormal fertilization and refers to gestational trophoblastic disease. The complete hydatidiform mole develops into trophoblastic malignant tumors in about 20% of cases. Trophoblastic disease mainly occurs in women of the late reproductive age.

**Classification** of trophoblastic disease:

1. Hydatidiform mole:
   - complete (traditional);
   - incomplete (partial);
   - invasive (destructive).

2. Choriocarcinoma (gestational trophoblastic tumor, malignant gestational trophoblastic disease):
   - non-metastatic (the process is limited to the uterus);
   - metastatic (the process extends the uterus):
     - low risk (favorable prognosis)
     - high risk (unfavorable prognosis).

**Etiology.** There are two basic theories of the onset of trophoblastic disease. According to one of them, the disease is caused by the pathology of chorial epithelium of the fertilized eggs. The second theory explains the development of the disease by the pathological changes in the body of the mother, lowering of host defense, hormonal disorders. 46XX chromosome complement is distinguished, where both X- chromosomes have the parental origin. The reason for this may be the fertilization of the egg simultaneously by two sperm or doubling of the sperm chromosomes in the absence/inadequacy of the maternal X- chromosome.
Complete and partial hydatidiform moles are distinguished. In partial hydatidiform mole the fetus or its elements, and partially the villi, is found in the uterus. This is caused by chromosomal abnormalities, mainly by triploidy or trisomy. Such pathology is rarely transformed into malignant tumors of the trophoblas (fig 26).

![Scheme of hydatidiform mole](image)

Fig. 26 Scheme of hydatidiform mole

**Destructive form of** hydatidiform mole, when due to high proteolytic activity the decidual membrane, uterine wall, vessels is destroyed, resembling the tumor which is growing. Sometimes such destructions reach the serous layer and lead to intraabdominal hemorrhages.

**Clinical manifestations** are the occurrence of symptoms of threatened miscarriage at the early or late terms of pregnancy; sometimes vaginal bleeding with vesicles’ content occurs. Gynecological examination reveals larger size of uterus than expected at to-date term of gestation, as well as *lutein cysts of the ovaries* (pathognomonic symptom). Sometimes the patients are admitted to the hospital with the signs of intraabdominal hemorrhage (destructive hydatidiform mole).

**Diagnosis:**

1. Bimanual gynecological examination.
3. Ultrasonography of the pelvis.

Discharge of cystically altered swollen chorionic villi from the uters is the absolute sign of hydatidiform mole. Parts of the fetus, its heartbeat and movements are not detected on palpation. Ultrasonography shows the absence of the fetus in the
uterus, instead, homogenous fine-grained mass is detected. Elevated blood hCG over 100 000 IU/l (50—100 times higher the norm) in the enlarged uterus and bleeding is another sign of hydatidiform mole.

**Differential diagnosis** is made with regard to ectopic pregnancy, abnormal uterine hemorrhage, stillbirth, spontaneous abortion, multiple pregnancy, polyhydramnios, and myoma.

**Indications for hospitalization:**
1. Suspicion of hydatidiform mole.
3. Disparity in uterine size and the term of pregnancy.
4. Blood stained vaginal discharge during 1 month after evacuation of the hydatidiform mole.
5. Sustained high level of blood hCG.
   - complete blood count,
   - urinalysis,
   - kidney and liver function biochemical tests,
   - blood group and Rh factor determination,
   - chest X-ray.

**The main method of treatment of** hydatidiform mole is careful evacuation of the uterus (mainly vacuum excochleation). Intravenous infusion of uterotonic is made during and after manipulations. In cases of the significant treat of uterine perforation during the curettage the complete evacuation of the uterus is not advocated, but repeated operation is made following the 2-3 days. In the destructive hydatidiform mole there is a high risk for uterine perforation. In this case laparotomy and hysterectomy are made. Chemotherapy is provided to women who experience uterine subinvolution after the curettage, sustained bloody vaginal discharge and elevated level of hCG. Vinblastin, methotrexat, mercaptopurin, rubomycin, dactinomycin medications are prescribed. Once the course of treatment finished, women should undergo regular medical check-up to prevent the presumed development of choriocarcinoma.

**Prophylactic medical examination service**
1. Close follow-up checkup over 1 year:
   - Monitoring of the level of hCG once a 14 days (until return to normal).
   - Ultrasound of the pelvis in dynamics.
   - Contraception (barrier methods) over 1 year.

2. In case of the occurrence of the positive reaction the patient is hospitalized for thorough examination, clarification of diagnosis and treatment. Oncologist’s medical advice is recommended. It is advisable to avoid pregnancy for 3 years.

3. Cervical ectopic pregnancy

   Cervical ectopic pregnancy is one of the rare and severe types of ectopic pregnancy, when the fertilized ovum has been implanted in the endocervical canal (see fig. 27).

   ![Fig. 27 Types of ectopic pregnancy](image)

Diagnosis

1. History, including gynecological one. Attention is paid to number of abortions and course of the follow up period, previous vaginal inflammatory diseases, including the cervix.
2. Vaginal speculum examination. Visualization of the cyanotic ballooned-out cervix.

3. Careful bimanual gynecological examination reveals the typical hour-glass configuration of the uterus with cervix.

4. Ultrasound of the pelvis.

**Ultrasound signs of cervical pregnancy:**

- absence of the gestational sac in the uterine cavity;
- hyperechogenicity of the endometrium (decidual tissue);
- heterogeneity of the myometrium;
- hour-glass configuration of the uterus;
- dilated cervical canal;
- gestational sac is in the cervical canal;
- placental tissue is in the cervical canal;
- internal os is closed.

**Differential diagnosis.** Cervical pregnancy should be distinguished from the spontaneous abortion, myoma, cervical carcinoma, origination of pedunculated submucous myoma, choriocarcinoma, placenta previa and low-lying placenta. Ultrasound examination provides with definite diagnosis.

**Treatment.**

- In case of the confirmed cervical pregnancy it is strongly recommended NOT to do curettage which can lead to profuse bleeding.
- The method of treatment of cervical pregnancy is **surgical (hysterectomy).**
- Blood group and Rh-factor should be determined, venous catheter is set; the informed agreement on hysterectomy is obtained from the patient.
- Fresh frozen plasma of the same group, packed RBC, blood substitutes are to be stocked in advance.

I. ANTEPARTUM HEMORRHAGE

*(in the second trimester of pregnancy)*

**ABNORMAL PLACENTATION**

1. Placenta previa
**Definition.** When the placenta is implanted partially or completely over the lower uterine segment (over and adjacent to the internal os) it is called *placenta previa.* The incidence of placenta previa, the most common type of abnormal placentation, is 0.5%. Approximately 20% of all cases of antepartum hemorrhage are due to placenta previa. Seventy percent of patients with placenta previa present with painless vaginal bleeding in the third trimester, 20% have contractions associated with bleeding, and 10% have the diagnosis made incidentally on the basis of ultrasonography or at term.

**Predisposing factors.** Factors that have been associated with a higher incidence of placenta previa include:

- multiparity;
- increased maternal age;
- prior placenta previa;
- multiple gestation;
- cesarean delivery.

Patients with a prior placenta previa have a 4-8% risk of having placenta previa in a subsequent pregnancy.

**Classification**

Placenta previa is classified according to the relationship of the placenta to the internal cervical os.

1. Complete (placenta praevia centralis)
2. Incomplete (partial, placenta praevia parcialis):
   - (marginal placental presentation)
   - (lateral placental presentation)

Complete placenta previa implies that the placenta totally covers the cervical os. A complete placenta previa may be central, anterior, or posterior, depending on where the center of the placenta is located relative to the os. Partial placenta previa implies that the placenta partially covers the internal cervical os. A marginal placenta previa is one in which the edge of the placenta extends to the margin of the internal cervical os. Placenta praevia lateralis, when 2/3 of the internal os has been covered with placenta (fig. 28).
**Diagnosis.**

**Clinical signs of placenta previa:**

- upper positioning of the fetal presentation,
- pathological fetal positioning,
- bleeding in the absence of pain or contact bleeding,
- during the ultrasonography after 26 weeks of gestation – the edge of the placenta is less than 20 mm from the internal os or 20 mm overlies it.

**Currently, the detection of placenta previa by the manual examination of the area of the internal os during labor is to be avoided. The definite diagnosis should be made by ultrasonography (see fig.29)**
The classic presentation of placenta previa is painless vaginal bleeding in a previously normal pregnancy. The mean gestational age at onset of bleeding is 30 weeks, with one-third presenting before 30 weeks. Placenta previa is almost exclusively diagnosed on the basis of ultrasonography. Between 4% and 6% of patients have some degree of placenta previa on ultrasonic examination before 20 weeks’ gestation. With the development of the lower uterine segment, a relative upward placental migration occurs, with 90% of these resolving by the third trimester. Complete placenta previa is the least likely to resolve, with only 10% of cases resolving by the third trimester.

When placenta previa is diagnosed in the second trimester, a repeat sonogram is indicated at 30 to 32 weeks for follow-up evaluation. Transabdominal ultrasonography has an accuracy of 95% for placenta previa detection. If the placenta is implanted posteriorly and the fetal vertex is low, the lower margin of the placenta may be obscured and the diagnosis of placenta previa missed. Transvaginal ultrasonography can accurately diagnose placenta previa in virtually 100% of cases.

Management
Management decisions depend on the gestational age of the fetus and the extent of the vaginal bleeding.

With a preterm pregnancy, the goal is to attempt to obtain fetal maturation without compromising the mother’s health.

If bleeding is excessive, delivery must be accomplished by cesarean, regardless of gestational age. When the bleeding episode is not profuse or repetitive, the patient is managed expectantly in the hospital on bed rest.

With expectant management, 70% of patients will have recurrent vaginal bleeding before completion of 36 weeks’ gestation and will require delivery. If the patient reaches 36 weeks, fetal lung maturity should be determined by amniocentesis and the patient delivered by cesarean if the fetal lungs are mature. Elective delivery is preferable, as spontaneous labor places the mother at greater risk for hemorrhage and the fetus at risk for hypovolemia and anemia. The favorable prognosis of successful vaginal delivery can be made if at 35-36 weeks of pregnancy the edge of placenta is more than 20 mm from the internal os. Any overlying of the internal os by placenta after 35-36 weeks of pregnancy (0 to 20 mm) is the indication to cesarean section.

2. Low-lying placenta

A patient with a low-lying placenta (placental margin within 7 cm of the endocervical os) may present in the same way as a patient with placenta previa. It may be difficult to distinguish a low-lying placenta from a marginal placenta previa, but a transvaginal ultrasound is typically diagnostic. Vaginal delivery is not contraindicated, because during labor the fetal head compresses the edge of the placenta, decreasing the risk of bleeding. The same level of monitoring should be maintained for maternal hemodynamic stability and fetal well-being.

Maternal-fetal risks. Maternal mortality from placenta previa has dropped from 30% to less than 1% over the past 60 years. This has primarily been due to the liberal use of cesarean delivery and careful expectant management. The rare maternal death is generally associated with complications of cesarean delivery or uncontrolled hemorrhage from the placental site. The lower uterine segment does not contract well, especially after a lower uterine incision. DIC may also result if a massive hemorrhage or an associated abruption occurs.
The risk of antepartum or intrapartum hemorrhage, or both, is a constant threat to the patient with placenta previa. Bleeding may be exacerbated by an associated placenta accreta or uterine atony. Placenta previa predisposes the patient to preterm delivery, which poses the greatest risk to the fetus. As a result of advances in obstetric and neonatal care, the perinatal mortality rate (PMR) for patients with placenta previa has declined over the past decade. The incidence of malpresentation with placenta previa is 30%, presumably due to the mass effect of the placenta and distortion of the lower uterine segment.

**ABRUPTIO PLACENTAE**

*(Premature abruption of normally situated placenta)*

**Definition.** Premature abruption of normally situated placenta (abruptio placentae) is the separation of the normally situated placenta during pregnancy or in the I-II stages of labor, leading to bleeding. Abruptio placentae or premature separation of the normally implanted placenta, complicates 0.5-2% of all pregnancies (1 in 120 births). Abruption severe enough to result in fetal death occurs in 1 in 500 deliveries.

**Etiology and pathophysiology.** The most common of these risk factors is maternal hypertension, either chronic or as a result of preeclampsia. The risk of recurrent abruption is 10% after one abruption and 25% after two. The etiology may be the opposite of that for placenta accreta. Because abruption is associated with maternal hypertension or preeclampsia, there may be a failure of adequate placental implantation. Its inciting cause is unknown, but placental separation may be due to an inherent weakness or anomaly in the spiral arterioles. Placental separation is initiated by hemorrhage into the decidua basalis with formation of a decidual hematoma. The resulting separation of the decidua from the basal plate predisposes to further separation and bleeding, as well as to compression and destruction of placental tissue. Blood may either dissect upward toward the fundus, resulting in a concealed hemorrhage, or extend downward toward the cervix, resulting in an external or revealed hemorrhage.

**Classification**
(according to the degree of separation)

1. Complete (separation of the entire placenta)
2. Partial:
   - marginal;
   - central.

The clinical course of the premature abruption of normally situated placenta depends on degree of separation and severity of the concomitant pathology.

Sometimes it is not possible to diagnose the partial separation of placenta on the small area during the labor. It is diagnosed after delivery when placenta is examined and a clot is found adhering to the maternal surface.

**The major symptoms of the premature abruption of normally situated placenta are bleeding and pain.** The bleeding can be **external** (vaginal) and **internal** (formation of the retroplacental clot). Internal bleeding, when the clot is in the middle of the placenta and external bleeding, when separation begins at the periphery and combined (fig 30).

![Fig. 30 Types of abruptio placentae](image)

**A**-Partial separation (concealed hemorrhage)  **B**- Partial separation (apparent hemorrhage)  **C**- Partial separation (concealed hemorrhage)

**The internal bleeding is the most life-threatening:**

- maternal vital signs are worsening,
- blood pressure is decreasing,
• tachycardia,
• weakness,
• pallor skin and mucous membranes.

**Another main symptom is pain:**
• located in the site of placenta implantation,
• occurs due to formation of retroplacental clot,
• arching pain.

**Diagnosis.** The vaginal examination shows:
• asymmetry of uterus,
• tension of uterus,
• uterine hypertonus,
• tenderness on palpation,
• fetal parts are difficult to detect.

Vaginal examination shows permanently tensed fetal sac, blood stained amniotic fluid. Placenta is delivered right after the birth of the baby. Separation of 1/3 of the surface area of the placenta leads to fetal distress, and separation of over 1/3 of the surface area results in fetal death, especially in case of morphological or functional placental impairments. Ultrasonography is critical in the diagnosis of the premature abruption of normally situated placenta and placenta previa.

**Management.** Management of the patient with an abruption includes careful maternal hemodynamic and fetal monitoring, serial evaluation of the hematocrit and coagulation profile, and delivery. Intensive monitoring of both the mother and the fetus is essential because rapid deterioration of the condition of either one can occur. Blood products for replacement should always be available. Red blood cells should be given liberally if indicated. In the setting of placental abruption, the use of tocolytics or uterine relaxants is not advisable. Uterine tone must be maintained to control bleeding following delivery, or at least to control the bleeding sufficiently to allow a safe hysterectomy to be performed, if necessary.

**Management of the placental abruption in the II stage of labor:**
- prompt amniotomy, if the fetal sac is intact;
- cephalic presentation calls for vacuum extraction or forceps-assisted vaginal
delivery;
- pelvic presentation calls for the prompt cesarean section;
- a transverse lie of the second twin requires an internal version with extraction of the fetus;
- manual detachment and removal of placenta;
- manual revision of the uterine cavity, removal of blood clots;
- uterotonics (10 U IV drop Oxytocin in 500 ml normal saline 60 drops per min.; 0,2 mg Ergometrin, 800 mcg Misoprostol per rectum, 100 mcg Carbetocin IV, if available);
- thorough dynamic observation of the uterus in the postpartum period.

During the cesarean section due to premature abruption of normally situated placenta the revision of the uterine wall (especially the external surface) is mandatory to exclude the **uterineplacental apoplexy** (Couvelaire uterus) (fig.31).

Maternal-fetal risks.

Abruption places the fetus at significant risk of hypoxia and, ultimately, death. The perinatal mortality rate due to placental abruption is 35%. Each second live-born infant has significant neurologic impairment.
Placental abruption is the most common cause of disseminated intravascular coagulation (DIC) in pregnancy. This results from release into the maternal circulation of thromboplastin from the disrupted placenta and subplacental decidua, causing a consumptive coagulopathy. Clinically significant DIC complicates 20% of cases and is most commonly seen when the abruption is massive or fetal death has occurred. Hypovolemic shock and acute renal failure as a result of massive hemorrhage may be seen with a severe abruption if hypovolemia is left uncorrected. Sheehan syndrome (amenorrhea as a result of maternal postpartum pituitary necrosis) may be a delayed complication resulting from coagulation within the portal system of the pituitary stalk. Assessment of pituitary function should be considered in the postpartum follow-up of women after a serious abruption with a coagulation disorder.

II. INTRAPARTUM HEMORRHEGE
   (in the third stage of labor)

1. Placenta accreta
   
   **Definition.** Placenta accreta implies an abnormal attachment of the placenta through the uterine myometrium as a result of defective decidual formation. This abnormal myometrial attachment of the placental villi is usually superficial (accreta), but the villi may invade more deeply into the myometrium (increta) or extend through to the uterine serosa (percreta). Two-thirds of patients with this complication require hysterectomy when an attempt to remove the placenta leads to severe hemorrhage intrapartum. Patients with a history of uterine surgery are at greatest risk of developing an accreta. In fact, those with prior cesarean delivery have a 10-50% risk of abnormal implantation. If ultrasonic imaging shows accreta prior to delivery, elective hysterectomy may be performed to prevent hemorrhage. The etiology of placenta accreta is complex, but recent evidence suggests the aggressive invasion of the placenta into the spiral arteries is a process unique to primates that has been conserved through evolution. It ensures that invasion of the uterine arteries is complete to maximize fetal access to the maternal circulation for maximal nutrition (fig. 32).
Causes:
- abnormal implantation of the placenta (pl.accreta, increta, percreta);
- abnormal release of placenta: incarcerated placenta.

The amount of blood loss depends on the kind of abnormal implantation of the placenta:
- complete or partial;
- pl. adhaerens or pl.accreta;
- pl.increta or pl.percreta;
- detached placenta retention in the uterine cavity.

Clinical signs:
- bleeding that begins after the childbirth, or before the release of placenta.
- absence of signs of detachment of the placenta within 30 minutes in the active management of the III stage of labor and expectant management of the III stage of labor.

In the occurrence of bleeding in the active management of the III stage of labor and absence of signs of detachment of the placenta within 15 minutes the prompt removal of the placenta is performed.

Management:
1. Check up for the signs of detachment of the placenta.

2. In occurrence of signs of detachment of the placenta controlled cord traction is applied to deliver the placenta.

3. When the placenta fails to deliver the manual detachment and removal of placenta is made by the controlled cord traction under intravenous anesthesia;

4. In the absence of bleeding, unsuccessful attempt of the manual detachment of the placenta and the diagnosis of the placenta accreta do not try to tear off the parts of placenta. This can lead to a massive uncontrolled hemorrhage caused by the uterine injury!

5. Prompt laparotomy with hysterectomy is performed in case of unsuccessful attempt of the manual detachment of the placenta and the diagnosis of the placenta accreta/increta/percreta.

2. Fetal bleeding

Rupture of a fetal umbilical vessel complicates 0.1-0.8% of pregnancies. This often results when the cord insertion is velamentous, implying that the vessels of the cord insert between the amnion and chorion, away from the placenta. The incidence of velamentous cord insertion varies from 1% in singleton pregnancies to 10% in twins and 50% in triplets. If the unprotected vessels pass over the cervical os, this is termed a vasa previa. The incidence of vasa previa is 1 in 5000 pregnancies. Velamentously inserted vessels need not pass over the os to rupture.

The diagnosis of fetal bleeding is made by performing an Apt-test. After obtaining blood from the vagina and putting it into a red-topped test tube, tap water or distilled water is added. The water will lyse blood cells and release hemoglobin into the solution. Adding 1 mL of KOH results in a brown discoloration when the hemoglobin is maternal. If the blood is fetal in origin, the color of the fluid will remain red because the fetal hemoglobin will not be denatured by the KOH. Rupture of a fetal vessel necessitates immediate abdominal delivery.

III. POSTPARTUM HEMORRHAGE
Postpartum hemorrhage is defined as blood loss of more than 500 ml or any bleeding, accompanied by the impairment of the hemodynamic state of the puerpera following the 6 weeks after the childbirth.

Postpartum hemorrhage, the leading cause of maternal mortality, is defined as blood loss in excess of 500 mL at the time of vaginal delivery or blood loss in excess of 1000 mL following cesarean delivery. The excessive blood loss usually occurs in the immediate postpartum period, but it can occur slowly over the first 24 hours. This is usually due to subinvolution of the uterus and disruption of the placental site “scab” several weeks postpartum or to the retention of placental fragments that separate several days after delivery.

**Classification:**

- **Primary (early) postpartum hemorrhages** occurred in the early postpartum period or **within the 24 hours after the childbirth**. About 70% of the postpartum hemorrhages are caused by the uterine atony. It occurs when the uterus fails to contract adequately after the childbirth.

- **Secondary (late) postpartum hemorrhages** occur following the first 24 hours postpartum or up to **6 weeks following the childbirth**. Most of the late hemorrhages are associated with retention of the placental fragments, infection or both.

**Causes of obstetric hemmorhages in III stage of labor and postlabor period**

1. Violation of placenta detachment and removal (T-tissue)
2. Impaired motor function of the uterus (T-tension)
3. Trauma of the maternal passages (T-trauma)
4. Impaired blood cloting (T-trombin)

**Evidence-based methods of the prevention of the postpartum hemorrhage.**

Active management of the third stage of labor reduces the amount of blood loss and risk of the postpartum hemorrhage. Preventive routine administration of the uterotonics in the third stage of labor reduces the risk of the development of postpartum hemorrhage by 60% in all puerperal women (10 IU oxytocin
intramuscular). In case of cesarean delivery intravenous 10 IU oxytocin is infused slowly.

**Uterine atony**

The majority of postpartum hemorrhages cases (75-80%) are due to **uterine atony**.

**The factors predisposing to postpartum uterine atony**

- **Fatigue of uterus**: rapid labor, prolonged labor, stimulation of labor by uterotonics
- **Dilatation of uterus**: polyhydroamnion, multiple pregnancy, macrosomia of fetus, dilatation of uterus by lost bleeding, mioma of uterus
- **Anesthesia or analgesia of uterus**
- **Others**: uterus hypotony when horizontal labor, amnionit (sepsis), amnion embolia, using magnio-sulfat in labor, vitamin D deficiency, fetal genetic factors.

Most of the blood loss due to uterine atony occurs from the myometrial spiral arterioles and decidual veins that previously supplied and drained the intervillous spaces of the placenta. As the contractions of the partially empty uterus cause placental separation, bleeding occurs and continues until the uterine musculature contracts around the blood vessels and acts as a physiologic-anatomic ligature. Failure of the uterus to contract after placental separation (uterine atony) leads to excessive placental site bleeding.

During pregnancy, uterine relaxation is facilitated by progesterone and parathyroid hormone-related peptide (PTHrP). The latter plays an important role in maintaining uterine relaxation during pregnancy; however, as soon as the uterus is emptied (delivery of the fetus and placenta), the gene controlling this hormone is turned off and the uterus is allowed to contract more completely. If there is a failure of complete expulsion of the placenta or poor uterine contractility leading to excessive bleeding, the uterus will fill with blood. The distention is thought to reactivate the expression of PTHrP and cause uterine relaxation, thereby leading to excessive hemorrhage.
Management of patients at risk for postpartum hemorrhage

Because the major cause of postpartum hemorrhages is uterine atony, the initial focus should be on prevention of uterine atony by considering the following steps:

1. All women in early labor who have risk factors for PPH should be identified and their hemoglobin checked. For medium-risk women, their blood should be typed and screened for irregular antibodies such as Rh and Kell. For high-risk women, 2 units of blood should be typed and crossmatched.

2. As soon as the fetus has been delivered, an infusion of oxytocin 10-40 U/L should be started and maintained during the first 6 hours postpartum.

3. The vagina and perineum should be inspected to rule out any lacerations that could cause excessive bleeding.

4. The placenta should be carefully assessed at delivery to make certain there are no missing cotyledons (lobules of placenta).

5. The uterus should be evaluated by abdominal palpation during the first 1 to 2 hours before transfer to the postpartum unit. The nurses on the postpartum unit should frequently assess the status of uterine contractility, instructing the patient on how to assess uterine firmness and reporting any excessive bleeding. For high-risk patients, continuation of the oxytocin infusion during the early postpartum hours should be considered.

Coagulation disorders

Peripartum coagulation disorders are high-risk factors for PPH, but fortunately they are quite rare. Patients with thrombotic thrombocytopenia have a rare syndrome of unknown etiology characterized by thrombocytopenic purpura, microangiopathic hemolytic anemia, transient and fluctuating neurologic signs, renal dysfunction, and a febrile course. In pregnancy, the disease is usually fatal.

An amniotic fluid embolus is also rare and is associated with an 80% mortality rate. This syndrome is characterized by a fulminating consumption coagulopathy, intense bronchospasm, and vasomotor collapse. It is triggered by an intravascular infusion of a significant quantity of amniotic fluid during a tumultuous or rapid labor in the presence of ruptured membranes. During the process of placental abruption, a
small amount of amniotic fluid may leak into the vascular system, and the thromboplastin in the amniotic fluid may trigger a consumption coagulopathy.

Patients with idiopathic thrombocytopenic purpura have platelets with abnormal function or a shortened lifespan. This causes thrombocytopenia and a tendency to bleed. Circulating antiplatelet antibodies of the immunoglobulin G type may occasionally cross the placenta and result in fetal and neonatal thrombocytopenia as well.

Willebrand disease is an inherited coagulopathy characterized by a prolonged bleeding time due to factor VIII deficiency. During pregnancy, these patients are likely to have a decreased bleeding diathesis because pregnancy elevates factor VIII levels. In the postpartum period, they are susceptible to delayed bleeding as factor VIII levels fall.

**Management.**

When PPH is associated with coagulopathy, the specific defect should be corrected by the infusion of blood products. Patients with thrombocytopenia require platelet concentrate infusions; those with von Willebrand disease require factor VIII concentrate or cryoprecipitate.

A packed red cell infusion is given to a patient who has bled sufficiently to compromise the delivery of oxygen to the tissues. Therefore, institution of blood transfusion is best judged by symptoms of oxygen deprivation rather than by some empirical hemoglobin level. No important physiologic impairment has been noted at hemoglobin levels as low as 6 to 8 g/dL (hematocrit of 18-24%). In general, a 1-U transfusion of packed red blood cells will increase the hemoglobin level by 1 g/dL (and the hematocrit by 3-4%).

Massive blood replacement (when total blood volume is replaced in a 24-hour period) may be associated with thrombocytopenia, prolonged PT, and hypofibrinogenemia. Thrombocytopenia is the most common abnormality, so platelet transfusion following determination of a low platelet count is not an uncommon scenario. Fresh frozen plasma may be transfused for prolonged PT or hypofibrinogenemia.
In the setting of active bleeding greater than 1000 mL, the hemorrhage care protocol should be activated. Maternal mortality and morbidity have been reduced when a protocol of packed red blood cells, fresh frozen plasma, and platelets, given in a ratio of 6:4:1, is implemented. Treatment should not be delayed while awaiting laboratory results or blood product crossmatching.

**Evidence-based methods of the prevention of the postpartum hemorrhage.**

Active management of the third stage of labor reduces the amount of blood loss and risk of the postpartum hemorrhage.

Preventive routine administration of the uteritontics in the third stage of labor reduces the risk of the development of postpartum hemorrhage by 60% in all puerperal women (10 IU oxytocin intramuscular).

In case of cesarean delivery intravenous 10 IU oxytocin is infused slowly.

**ABC—diagnosis**

Assessment of the state of the airways (A) and respiration (B) (resuscitation in case of asphyxia), artificial support of blood circulation (C):

- cardiac massage: the frequency of compression: 100 per1 min, compression to ventilation ratio = 30:2,
- catheterization of 2 peripheral veins with catheters of adequate diameters, venostomy or central vein catheterization, if necessary,
- urinary bladder catheterization,
- warm the puerpera up.
- prompt infusion therapy,

**Temporary blood arrest:**

- abdominal aortic compression.

**Clinical and laboratory examination:**

- blood group and rh-factor,
- complete blood count,
- blood clotting time,
- coagulogram,
- bedside test,
- blood sampling for compatibility.
**Determination of the cause of hemorrhage:**

- assessment of the uterine tone:

**In case of uterine tone disorder:**

- infusion of uterotonics (oxytocin 10 U/500ml normal saline solution with the rate of 60 drops/min);
- fundal massage;
- in continuing bleeding: manual examination of the uterine walls under intravenous anesthesia;
- use of uterotonic medications: ergometrine, prostaglandins, carbetocin;
- in continuing bleeding: bimanual uterine compression or aortic compression;
- if bleeding continues, balloon tamponade is performed in the operational theatre and tranexamic acid is prescribed (1g, repeat after 30 minutes).
- if the blood loss is greater than 1,5% and more of the body weight, laparotomy is performed (use of organ-preservation techniques, i.e., ligation of the great vessels (stepwise partial devascularization of the uterus), uterine compression sutures, bilateral laceration of the internal iliac (hypogastric) arteries (by the medical clinician or vascular surgeon only). In case of ineffectiveness, hysterectomy without adnexa is advocated.

**If the uterus is firm exclude the trauma:**

- manual examination of the uterine walls to exclude the uterine rapture. Prompt laparotomy is performed in case of uterine rapture.
- thorough examination to exclude traumas (the perineum, vagina, cervix). Suture of the tears in the operational theatre with a good light and available assistant. Presence of traumas is the indication to prescribe tranexamic acid (1g, repeat after 30 minutes). Absence of traumas is indicative of blood clotting disorder.
  - assessment of blood clotting by the bedside test, blood clotting time, coagulogram (if possible).
management

1. Constant control of the puerpera’s state, indices of hemodynamics and blood clotting.

2. Prescription of broad-spectrum antibiotics.

3. 24 hour observation in the critical care unit following the stabilization of the woman’s state (BP not less than 100 mm Hg, 90/min heart rate).

Hemorrhages caused by dehiscence of the uterine scars and uterine injuries are required prompt laparotomy.

**External abdominal aortic compression**

- the point for compression through the abdominal wall is right above the umbilicus and slightly on the left;
- another hand palpates the pulse on the femoral artery to assess the effectiveness of the compression;
- if the pulse is recorded then the pressure with a closed fist is insufficient and vise versa (fig. 33).

![Fig. 33 External abdominal aortic compression](image)

**Bimanual uterine compression.**

Introduce one hand into vagina and clench your fist with the back of your hand positioned posteriorly and your knuckles in the anterior fornix. Place your other hand on the abdomen behind the uterus and squeeze the uterus firmly between both hands, as shown in fig. 34.
Uterine balloon tamponade.

Special balloon is inserted into the uterine cavity (beyond the external os). Inflate the balloon with sterile saline (300-500 ml) using the syringe to ensure counter-pressure to stop bleeding. Continue oxytocin infusion for 24 hours. Thereafter slowly deflate the balloon by letting down the saline over 2 hours, then remove the balloon. Fully inflated balloon ensures the tamponade effect. The effectiveness of the balloon tamponade accounts of 77.5 – 88.8%, i.e., in most cases this method allows preventing the follow up surgical treatment (fig. 35).
Methods of surgical hemostasis

The following methods of surgical hemostasis of massive obstetric hemorrhage are distinguished:

I. Bilateral ligation of the uterine vessels.
II. Bilateral ligation of the ovarian vessels.
III. Compressive uterine suture.
IV. Bilateral ligation of the internal iliac (hypogastric) arteries.
V. Radical surgeries (subtotal or total hysterectomy).

B-Lynch compressive suture

Uterine compression suture has been widely used to manage the postpartum hemorrhage for the last decade. Two of the most common techniques are the B-Lynch suture and multiple square suture, described by Cho et al. The effectiveness of the compressive suturing is 70-90% (fig.36).

Indications for hysterectomy

1. Premature abruption of normally situated placenta with intermuscular hemorrhages, uterineplacental apoplexy and development of the Couvelaire uterus.
2. Uterine atony is not sensitive to uterotonic
3. Failed balloon tamponade, compressive suture, ligation of the greater uterine vessels (uterine, ovarian).
4. Uterine rapture with uterine vessels detachment and massive hemorrhage. Subtotal hysterectomy is performed in placenta accreta only without heavy blood loss and DIC-syndrome.

**Ligation of the internal iliac (hypogastric) arteries**

**Indications:**
- Following the hysterectomy in continuing bleeding.
- In the comprehensive therapy of coagulopathic hemorrhage.
- Diffuse hemorrhage from the site without definite detection of the source of bleeding and vascular bed.
- Uterine rapture with detachment of uterine artery.
- Deep cervical lacerations and vaginal fornix with technical difficulties of their suturing.

**Main stages of the internal iliac arteries ligation:**
1. Laparotomy.
2. Longitudinal incision of the posterior leaf of the parietal peritoneum.
3. Dissection of the internal iliac artery.
4. Placing of ligation under the artery.
5. Ligation of the hypogastric artery.

**TESTS**

1. All of the following is associated with massive placental abruption EXCEPT:
   - **A. painless vaginal bleeding**
   - B. uterine rigidity
   - C. uterine pain
   - D. maternal cardiovascular collapse
   - E. absent fetal heart sound

2. Vasa previa diagnosed in early labor is best treated with:
   - **A. cesarean section**
   - B. voorhees bag
   - C. forceps delivery
3. Which form of therapy is often most effective for patients with "Couvelaire uterus"?

A. Intravenous ampicillin, total abdominal hysterectomy
B. bedrest
C. cervical cerclage
D. total abdominal hysterectomy
E. cesarean section

4. A 32-year-old G2P1 at 28 weeks gestation presents to labor and delivery with the complaint of vaginal bleeding. Her vital signs are: blood pressure 115/67 mm Hg, pulse 87 beats per minute, temperature 37.0°C, respiratory rate 18 breaths per minute. She denies any contraction and states that the baby is moving normally. On ultrasound the placenta is anteriorly located and completely covers the internal cervical os. Which of the following would most increase her risk for hysterectomy?

A. placenta accrete
B. desire for sterilization
C. development of disseminated intravascular coagulopathy (DIC)
D. prior vaginal delivery
E. smoking

5. After delivery and revision of placenta there was found the defect of placental lobule. General condition of woman is normal, uterus is firm, there is moderate bloody discharge. Speculum inspection of birth canal shows absence of lacerations and raptures. What action is necessary?

A. manual exploration of the uterine cavity
B. external massage of uterus
C. introduction of uterine contracting agents
D. urine drainage, cold on the lower abdomen
E. introduction of hemostatic medications

6.10 minutes after delivery a woman discharged placenta with a tissue defect 5x6 cm large. Discharges from the genital tracts were profuse and bloody. Uterus tonus was low, fundus of uterus was located below the navel. Examination of genital tracts revealed that the uterine cervix, vaginal walls, perineum were intact. There was uterine bleeding with following blood coagulation. Your actions to stop the bleeding:

A. to make manual examination of uterine cavity
B. to apply hemostatic forceps upon the uterine cervix
C. to introduce an ether-soaked tampon into the posterior fornix
D. to put an ice pack on the lower abdomen
E. to administer uterotonics

7. A 26 year old woman had the second labour within the last 2 years with oxytocin application. The child's weight is 4080 g. After the placent birth there were massive bleeding, signs of hemorrhagic shock. Despite the injection of contractive agents, good contraction of the uterus and absence of any cervical and vaginal disorders, the bleeding proceeds. Choose the most probable cause of bleeding:

A. atony of the uterus
B. injury of cervix of the uterus
C. hysterorrhexis
D. delay of the part of placenta
E. hypotonia of the uterus

8. A 34 y.o. woman in her 29-th week of pregnancy, that is her 4-th labor to come, was admitted to the obstetric department with complaints of sudden and painful bloody discharges from vagina that appeared 2 hours ago. The discharges are profuse and contain grumes. Cardiac function of the fetus is rhythmic, 150 strokes in the minute, uterus tone is normal. The most probable provisional diagnosis will be:

A. placental presentation
B. detachment of normally located placenta
C. vasa previa  
D. bloody discharges  
E. disseminated intravascular coagulation syndrome

9. A pregnant woman (35 weeks), aged 25, was admitted to the hospital because of bloody discharges. In her medical history, there were two artificial abortions. In a period of 28-32 weeks, there was noted the onset of hemorrhage and USD showed a placental presentation. The uterus is in normotonus, the fetus position is transversal (Ist position). The heartbeats is clear, rhythmical, 140 bpm. What is the further tactics of the pregnant woman care?

A. **to perform a delivery by means of Cesarean section**  
B. to perform the hemotransfusion and to prolong the pregnancy  
C. to introduct the drugs to increase the blood coagulation  
D. to continue observation stimulate the delivery by intravenous introduction of oxytocin  
E. to keep the intensity of hemorrhage under observation and after the bleeding is controlled to prolong the pregnancy

10. A 39-year-old woman, gravida 2, para 1, at 36 and 4/7th weeks of gestation with a history of prior cesarean section in the setting of placental abruption presents with abdominal pain and vaginal bleeding. She admits to using cocaine. Her vital signs are significant for T 5 99.9, HR 5 120, BP 5 170/100. Fetal heart rate baseline is in the 160s with minimal variability and repetitive late decelerations. Her bloodwork is significant for a hemoglobin of 7.5, platelets of 110,000, and a fibrinogen level of 250 mg/dL. All of the following are risk factors for this patient’s condition except:

A. **prior cesarean section**  
B. advanced maternal age  
C. cocaine  
D. hypertension  
E. prior placental abruption
SITUATIONAL TASKS

A 33-year-old woman, gravida 7, para 3214, presents at 28 weeks with complaints of vaginal bleeding. She denies abdominal or back pain. She has had no prenatal care. She reports recent intercourse. On presentation, she has light vaginal bleeding and fetal heart tones are reassuring. The most useful next step in her evaluation would be:

2. A 20-year-old woman, gravida 1, para 0, at 33 weeks of gestation arrives to labor and delivery reporting profuse vaginal bleeding and abdominal pain. Her vitals are as follows: T 5 96.8, BP 5 78/40, P 5 138, R 5 28. Her abdomen is firm and tender to touch. Fetal heart tones are in the 160s with minimal variability and late decelerations. Tocimeter demonstrates contractions every 1 to 2 minutes. Ultrasound demonstrates a cephalic fetus, placenta is fundal and free of the os without a retroplacental clot. Cervical examination is 3/90/ 2 1. Which is the most appropriate management plan?

3. A 34-year-old woman, gravida 5, para 4004, at 30 and 27th weeks of gestation presents to labor and delivery reporting vaginal bleeding. She reports vague back pain. Her blood pressure is 110/78 and her pulse is 106. She has slow, continuous bleeding from her vagina. Her cervix appears long and closed on speculum examination. Fetal monitoring reveals one uterine contraction every 30 minutes, and the fetal heart rate is reassuring. Transabdominal ultrasound demonstrates a complete placenta previa. The most appropriate next step would be:

4. The woman who has delivered twins has early postnatal hypotonic uterine bleeding reached 1,5\% of her bodyweight. The bleeding is going on. Conservative methods to arrest the bleeding have been found ineffective. The conditions of patient are pale skin, acrocyanosis, oliguria. The woman is confused. The pulse is 130 bpm, BP– 75/50 mm Hg. What is the further treatment?
5. A 20 y.o. pregnant woman with 36 weeks of gestation was admitted to the obstetrical hospital with complains of pain in the lower abdomen and bloody vaginal discharge. The general condition of the patient is good. Her blood pressure is 120/80 mm Hg. The heart rate of the fetus is 140 bpm, rhythmic. Vaginal examination: the cervix of the uterus is formed and closed. The discharge from vagina is bloody up to 200 ml per day. The head of the fetus is located high above the minor pelvis entry. A soft formation was defined through the anterior fornix of the vagina. What is the probable diagnosis?