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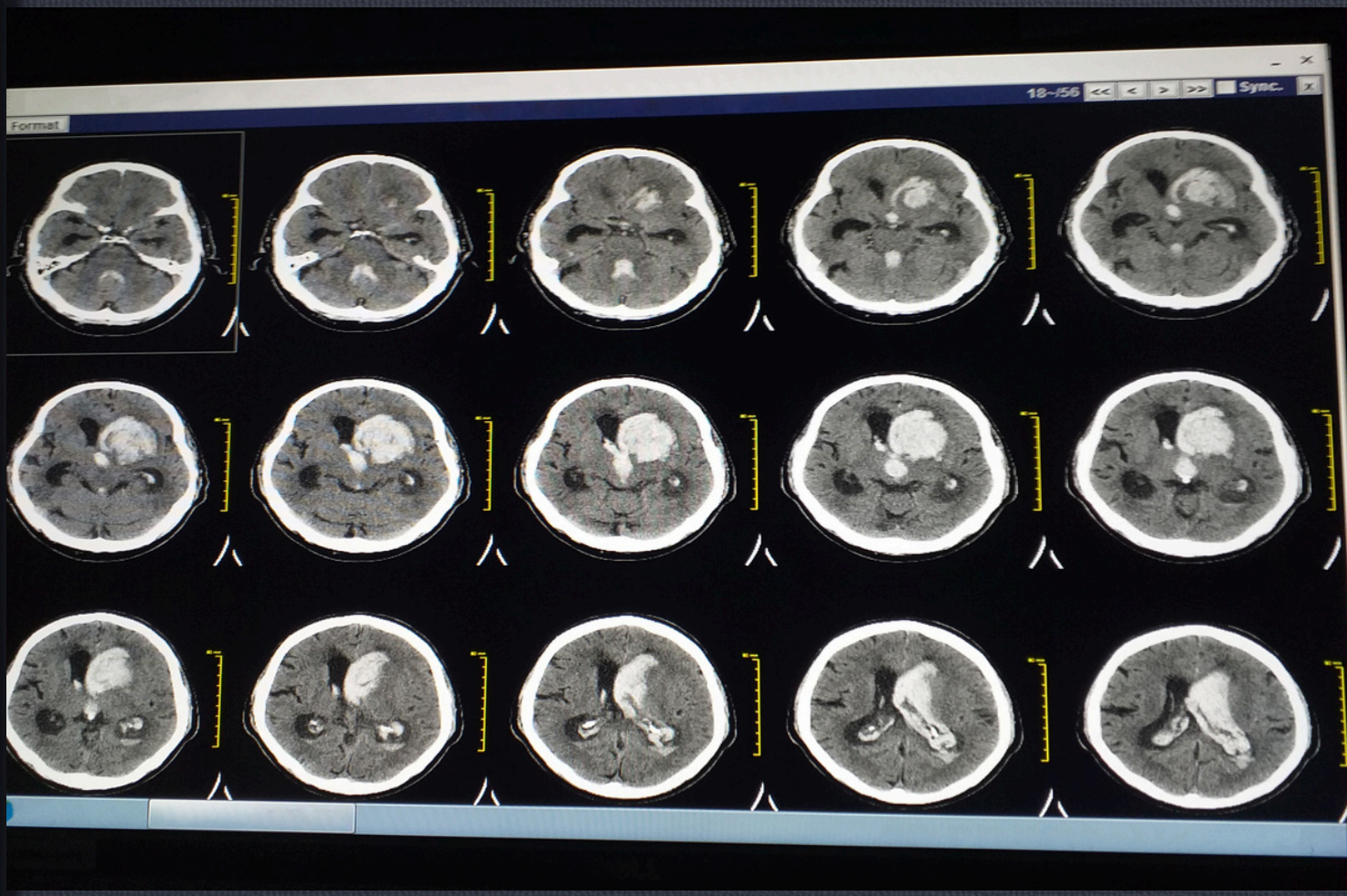
OBSTETRIC HEMORRHAGE: PATHOGENESIS, RISK FACTORS AND TREATMENT ALGORITHMS



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MONOGRAPH

Compilied by

Kh.SH.Shavkatov

SAMARQAND – 2026

This monograph is devoted to one of the most urgent problems of modern obstetrics — obstetric hemorrhage. Hemorrhagic complications during pregnancy, childbirth, and the postpartum period remain a leading cause of maternal morbidity and mortality worldwide. The book presents contemporary views on the pathogenesis of obstetric hemorrhage, identifies major risk factors, and analyzes mechanisms of hemostatic disorders associated with pregnancy and delivery. Special attention is paid to early diagnosis, prediction of hemorrhagic complications, and evidence-based clinical decision-making. The monograph systematizes modern approaches to prevention and management, including pharmacological therapy, blood-saving technologies, surgical methods, and intensive care principles. Practical treatment and management algorithms are proposed based on international recommendations and national clinical guidelines.

The publication is intended for obstetricians-gynecologists, anesthesiologists, residents, master's students, and healthcare professionals involved in perinatal care.

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Obstetric hemorrhage: pathogenesis, risk factors and treatment algorithms

monograph /compiled by Kh. Sh. Shavkatov - 2026 –108 p

LIST OF ABBREVIATIONS

1. **PPH** — Postpartum Hemorrhage
2. **APH** — Antepartum Hemorrhage
3. **CS** — Cesarean Section
4. **VBAC** — Vaginal Birth After Cesarean
5. **DIC** — Disseminated Intravascular Coagulation
6. **Hb** — Hemoglobin
7. **Hct** — Hematocrit
8. **BP** — Blood Pressure
9. **HR** — Heart Rate
10. **ICU** — Intensive Care Unit
11. **FFP** — Fresh Frozen Plasma
12. **PRBC** — Packed Red Blood Cells
13. **TXA** — Tranexamic Acid
14. **WHO** — World Health Organization
15. **FIGO** — International Federation of Gynecology and Obstetrics

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INTRODUCTION

The key indicators of population health are closely linked to social, economic, and healthcare-related factors. Among these indicators, maternal mortality occupies a particularly important place, as it reflects not only the quality of obstetric care but also the overall level of women's health, accessibility of medical services, and the effectiveness of healthcare systems. When maternal mortality is analyzed separately, it becomes evident that it is strongly associated with living conditions, the health status of pregnant women, and the timely provision of qualified medical care during pregnancy, childbirth, and the postpartum period. One of the most critical and widely studied problems in modern obstetrics is obstetric hemorrhage. Despite significant advances in diagnostic technologies, surgical techniques, and intensive care, bleeding during pregnancy, childbirth, and the postpartum period remains one of the leading causes of maternal morbidity and mortality worldwide. The clinical and social importance of this problem is determined by the fact that obstetric hemorrhage often develops rapidly, is unpredictable, and may lead to life-threatening conditions within a very short period of time. According to international data, obstetric hemorrhage accounts for approximately 25.8% of maternal deaths as a direct cause and contributes indirectly to up to 78% of cases by aggravating pre-existing conditions or triggering severe complications. The American College of Obstetricians and Gynecologists reports that globally, one woman dies from hemorrhage approximately every four minutes. In the Republic of Uzbekistan, obstetric hemorrhage has historically remained one of the leading causes of maternal mortality, accounting for an average of 26% of all maternal deaths. Despite ongoing reforms in maternal healthcare, improvements in prenatal care, and the introduction of modern clinical protocols, postpartum hemorrhage continues to occupy a dominant position among the causes of maternal mortality. This indicates that obstetric bleeding remains not only a medical but also an organizational and educational challenge that requires continuous attention and improvement.

Maternal death resulting from obstetric hemorrhage is most commonly associated with hemorrhagic shock and its severe complications, including acute circulatory failure, disseminated intravascular coagulation, multiple organ dysfunction, and irreversible metabolic disturbances. In this context, the management of blood loss

during the earliest stages of bleeding is of paramount importance. Early recognition of hemorrhage, prompt control of bleeding, and adequate replacement of blood loss are the key determinants that significantly influence patient outcomes and survival rates. In obstetric practice, the issue of developing effective strategies and tactics for the management of acute blood loss remains especially urgent, as the success of hemorrhage control largely depends on how quickly and accurately the medical team responds to the situation.

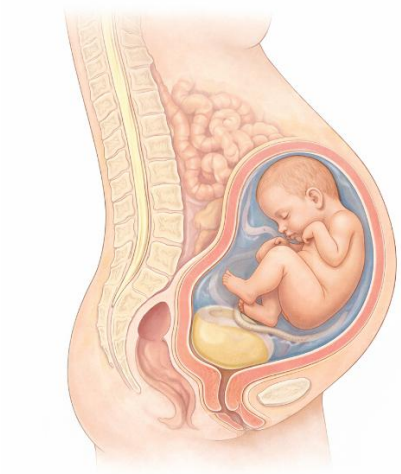
Practical experience from obstetric and gynecological healthcare facilities shows that the greatest difficulties for physicians arise in clinical situations requiring urgent medical intervention. These emergencies often develop unexpectedly and demand immediate decision-making under conditions of time pressure and limited resources. In such situations, the primary objective is to save the patient's life; therefore, delayed actions, incorrect tactical decisions, or inappropriate selection of treatment methods may result in serious complications or even fatal outcomes. Conversely, timely, well-planned, and rational emergency medical care, implemented using accessible and effective methods, can not only prevent maternal death but also preserve a woman's reproductive potential, which is particularly important for patients of reproductive age.

An essential aspect of effective management of obstetric hemorrhage is the constant preparedness of the practicing physician. This preparedness includes not only technical skills and clinical knowledge but also psychological readiness to act decisively in emergency situations. Adequate theoretical training, practical experience, and a clear understanding of emergency diagnostic criteria, prevention strategies, and treatment algorithms for obstetric bleeding play a crucial role in determining clinical outcomes. Continuous professional education, simulation-based training, adherence to standardized clinical guidelines, and coordinated interdisciplinary teamwork significantly enhance the effectiveness of emergency obstetric care.

This work emphasizes the clinical and social significance of obstetric hemorrhage as a leading cause of maternal mortality and highlights the importance of early diagnosis, timely intervention, and well-organized emergency care. All comments, suggestions, and recommendations aimed at improving the structure, clarity, and practical value of this material are gratefully acknowledged by the authors, as they contribute to the continuous development and refinement of obstetric care practices

I. TOPOGRAPHIC ANATOMY OF THE PREGNANT UTERUS

If an abdominal incision is performed (either transverse or longitudinal), upon opening the abdominal cavity, the pregnant uterus can be observed as an ovoid-shaped organ. It is located predominantly in the anterior part of the abdominal cavity and is usually positioned slightly to the right of the midline.



The right lateral aspect of the uterus extends deeply toward the inferior margin of the liver, while its left portion lies closer to the anterior abdominal wall and is directed toward the left hypochondrium along the greater curvature of the stomach. The right border of the anterior uterine wall is directly covered by the posterior surface of the lateral abdominal wall; it overlies the cecum and displaces it upward and posteriorly.

Thus, in late pregnancy the cecum is no longer located in the right inguinal region as in non-pregnant women; instead, it is displaced upward to the right, above the level of the umbilicus, and is often found near the inferior margin of the liver. The left border of the anterior uterine wall is covered by intestinal loops that are displaced to the left at the level of the iliac fossa; these loops come into close contact with the prominent fold of the sigmoid colon projecting anteriorly. In conditions of inadequate anesthesia, meteorism, or peritonitis, the loops of the small intestine and the sigmoid colon (S-romanum) become distended with gas, protrude markedly anteriorly, and may cover the anterior surface of the uterus up to the linea alba of the abdominal wall.

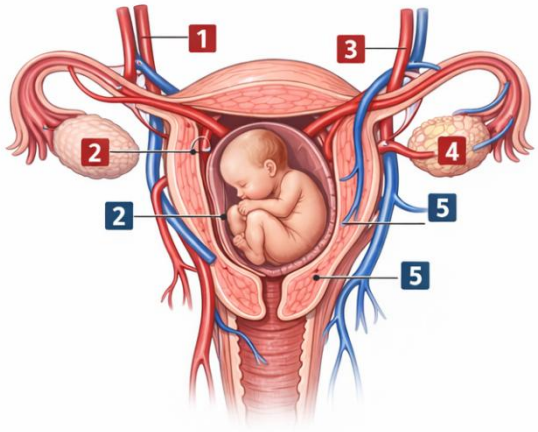
When the woman is placed in a position with the lower extremities elevated, it is possible to completely free the anterior uterine wall, allowing the intestines and mesentery to shift upward above the umbilical region. In late pregnancy, the lower uterine segment more or less enters the lesser pelvis, where it is laterally bordered by the lateral pelvic walls and anteriorly by the iliac portion of the abdominal wall. The urinary bladder, located between the anterior wall of the lower uterine segment and the posterior surface of the abdominal wall, assumes an arcuate or bean-shaped configuration. As a rule, the bladder is displaced to the left or right of the fetal head, depending on its position.

An analysis of the position of the uterus within the abdominal cavity shows that it is inclined to the right and slightly rotated around its longitudinal axis from left to right. As a result, the right adnexa and the right round ligament are usually not visible. In this situation, the left round ligament lies anteriorly and somewhat obliquely,

extending toward the left iliac region. A few centimeters superior and posterior to the left round ligament, the left fallopian tube and its mesosalpinx are stretched, partially covering the left ovary.

When the surgeon performs an incision in the lower uterine segment, the left uterine artery (a. uterina) is placed in a more vulnerable position, particularly at the site of its bifurcation into ascending and descending branches as it crosses the transverse plane, which significantly increases the technical difficulty of the procedure (Figure 1).

Blood Supply of the Uterus in a Pregnant Woman

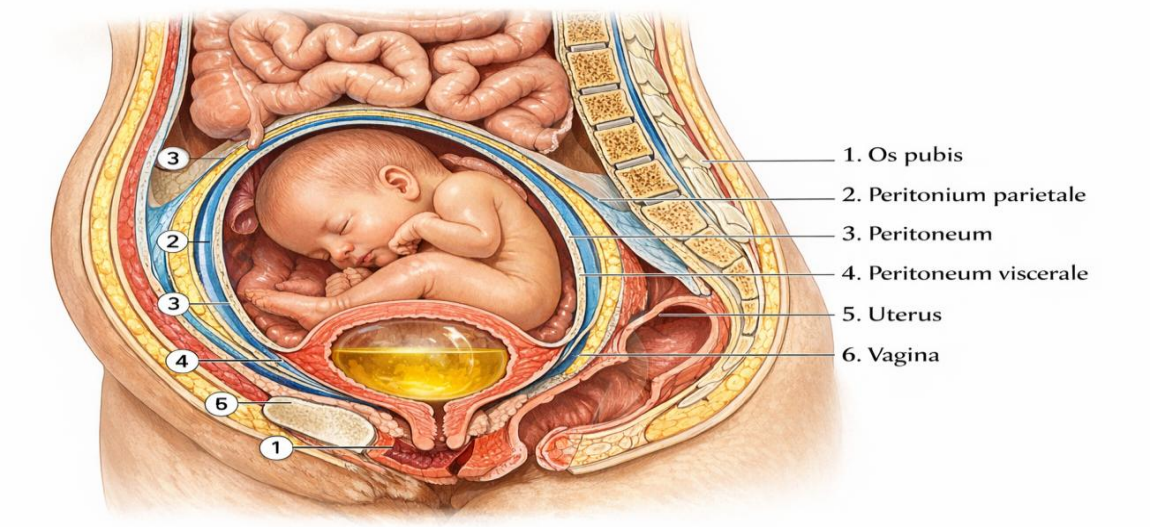


Number	Description
1	Descending Branch of Uterine Artery
2	Uterine Vein
3	Ascending Branch of Uterine Artery
4	Ovarian Artery
5	Ovarian Vein

Most often, this error occurs due to insufficient knowledge of the anatomical features of the course of the uterine vascular bundles during pregnancy. It is well known that the lower uterine segment increases predominantly in its transverse dimension, especially in the period immediately preceding labor. Therefore, in late pregnancy, the bifurcation of the left uterine artery occurs not at a distance of approximately 1 cm, as in non-pregnant women, but much closer to the lateral border of the uterus.

Thus, uneven stretching of the lower uterine segment significantly increases the risk of injury to the uterine artery. The main trunk of the left uterine artery is located approximately 6 cm above the pubic symphysis, whereas on the right side it lies at a distance of about 10 cm. Due to the physiological rotation of the uterus from left to right, the right uterine artery is positioned deeper and more posteriorly; it often appears tortuous and may even form loops. In contrast, the left uterine artery becomes elongated, is displaced closer to the anterior abdominal wall, lies in close proximity to the uterine wall, and bends sharply upward.

Such an atypical position of the left uterine artery increases the likelihood of its injury during a cesarean section performed through the lower uterine segment, particularly when delivering a large or macrosomic fetal head and when the uterine incision is inadvertently extended toward the left side



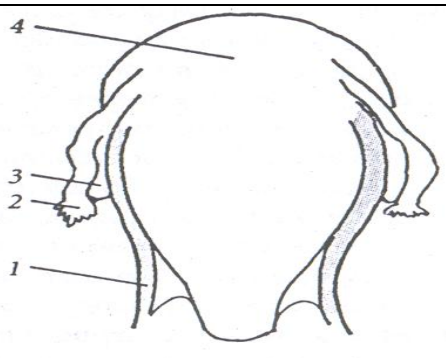
From the above, it becomes clear that the anterior lower wall of the uterus is located extraperitoneally over a considerable distance and is only partially covered by the urinary bladder. Between the bladder and the uterus, there is a loose layer of connective tissue reaching up to 20 mm in thickness. This anatomical feature makes it possible, during a cesarean section performed via an abdominal approach, to quickly and safely separate the posterior wall of the urinary bladder from the anterior uterine wall (Figure 2). During pregnancy, the weight of the uterus reaches approximately 1000 g, and its length increases up to 250 mm. By the end of the first half of pregnancy, the uterine walls reach their maximum thickness (30–40 mm). During labor, the uterus represents a hollow muscular organ that undergoes periodic contractions and relaxations. Under the influence of contractions, the uterine walls become denser and the uterus acquires an ovoid shape. In cases of strong labor contractions, the uterus tends to assume a pronounced ovoid configuration, with the fundus slightly elongated anteriorly and approaching the midline.

The lower uterine segment is located between the uterine body and the cervix. The upper border of the lower segment is considered to be the area where the peritoneum begins to separate slightly from the uterine wall. Determining the lower boundary of this segment is difficult, as the junction between the lower uterine segment and the cervix does not have clearly defined anatomical limits. In addition, beneath the vesicouterine fold, the urinary bladder is adjacent to the lower uterine segment, which is not covered by peritoneum.

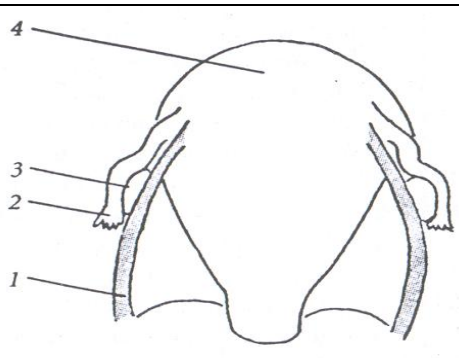
In cases of a clinically narrow pelvis, when the fetal head is unable to progress along the birth canal, the lower uterine segment rises even higher. In such situations, the upper margin of the lower segment may reach the level of the umbilicus, the contraction ring becomes oblique, and the risk of uterine rupture increases. It should be remembered that surgical access to the lower uterine segment is easiest only after labor has begun and the lower segment has ascended from the pelvic cavity into the abdominal cavity. Lowering the patient’s head during surgery facilitates better access to the lower uterine segment. This technique may be particularly useful when the fetal head is impacted at the pelvic inlet.

Simultaneously with the thickening of the uterine musculature, significant changes occur in the uterine vascular system. Both arteries and, especially, veins

dilate during pregnancy, forming wide vascular loops along the uterus, particularly in its lateral portions and around the uterine tubes. In the second half of pregnancy, the uterine walls become thinner, reaching 20–30 mm. At the time of delivery, the thickness of the uterine wall is not uniform in different regions: it is greatest near the lower part of the uterine body (20–30 mm), considerably thinner in the lower uterine segment (approximately 5 mm), and becomes even thinner—clinically and anatomically—near the pelvic cavity (about 2 mm), especially in cases of pathological labor. During cesarean section, it is important to know the site of placental attachment to the uterine wall. To determine placental localization on the uterine wall, the signs described by Palm may be used (Figure 3).



The placenta is attached to the posterior wall of the uterus (according to Palm). The round ligament of the uterus (ligamentum teres uteri).



The placenta is attached to the anterior wall of the uterus (according to Palm): the round ligament of the uterus (ligamentum teres uteri).

figure. 3. 1 - Lig. teres uteris; 2 - Tuba uterina; 3 - Ovarium; 4 - Uterus

According to the observations of the author (Palm), when the placenta is located on the posterior wall of the uterus, the round ligaments become thickened, reaching approximately the size of the little finger. The ligaments are attached to the uterine fundus at an acute angle and are positioned at a short distance from each other, giving them the appearance of being thick and short.

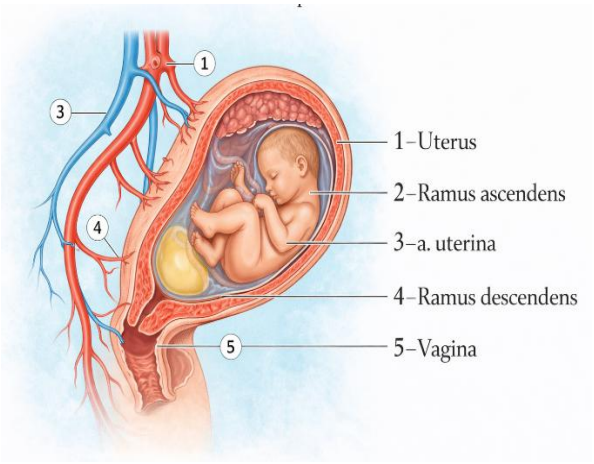
When the placenta is attached to the anterior wall of the uterus, the round ligaments are elongated and extend from the uterus at a right angle, more posteriorly; they are also located at a considerable distance from each other.

In cases where it is necessary to control bleeding from the uterine tube or ovary, the asymmetric position of the suspensory ligament of the ovary (*ligamentum suspensorium ovarii*) should be taken into account. On the right side, this ligament extends from the pelvic wall between the cecum and the lateral border of the *psoas* muscle. The left *ligamentum suspensorium ovarii* is located slightly lower than the right one. To identify this ligament, the uterus should be sharply retracted to the right, while the sigmoid colon should be displaced to the left.

Each of these ligaments contains a vascular bundle consisting of the ovarian artery and markedly dilated veins. Thick veins draining blood from the uterine tubes and ovaries join the veins extending from the uterine fundus and the round ligaments, forming the pampiniform plexus. Laterally from the adnexa, a plexus consisting of three veins is present; these veins unite into a single common vein that drains into

the inferior vena cava on the right side and most often into the renal vein on the left side.

After originating from the internal iliac artery, the uterine artery courses medially and anteriorly over the fascia of the *levator ani* muscle, enters the base of the broad ligament, and gives off vesical branches (*rami vesicales*) to the urinary bladder.



The artery passes through the periuterine tissues anterior to the ureter, giving off a thin branch to it, then approaches the uterine isthmus and, at a distance of 15–20 mm in non-pregnant women (or very close to the origin of the branch in pregnant women), divides into two branches: a descending branch (*ramus descendens*) and an ascending branch (*ramus ascendens*).

The descending branch of the uterine artery is thinner than the ascending branch and runs obliquely from superior to inferior and from lateral to medial. At a lower level, additional branches extend from the main trunk of the *ramus descendens*, coursing downward at an acute angle toward the cervix, where they anastomose with similar branches from the opposite side.

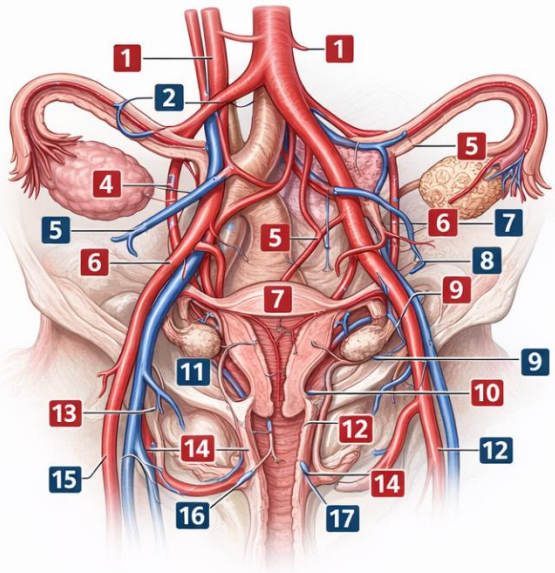
Based on these anatomical features, it becomes clear why arcuate incisions with the apex directed upward are required in the region of the uterine body, and why blood loss during cesarean section is significantly greater than during many other abdominal operations. As the artery approaches the uterine fundus, the obliquely oriented branches of the *ramus ascendens* change their direction to a more horizontal course. Therefore, transverse incisions in the corresponding areas of this organ are recommended.

The main trunk of the *ramus ascendens* of the uterine artery, upon reaching the origin of the ovarian ligament, divides into two branches. The first branch (*ramus ovaricus*) changes its course from vertical to horizontal, reaches the superior pole of the ovary, and supplies it through the mesovarium. It gives off additional branches to the mesosalpinx and subsequently anastomoses near the lateral border with the ovarian artery, which has a smaller diameter.

The second branch of the *ramus ascendens a. uterina* arches upward and supplies the uterine fundus. From one of these branches arises the tubal branch of the uterine artery (*ramus tubarius a. uterina*), which runs from the inferior margin of the uterus through the isthmic portion of the tube to the ampullary part, where it anastomoses with the tubal branch of the ovarian artery (*ramus tubarius a. ovarica*). The branches of both arteries extend through the mesosalpinx to the tubal wall, branching up to the 6th–7th order and forming numerous interconnected vessels that create a dense plexus. These characteristics of uterine blood supply likely account for the massive blood loss observed in disrupted ectopic pregnancy.

The third (thinnest) branch of the ascending uterine artery passes to the round ligament of the uterus, where it anastomoses with a branch of the inferior epigastric artery (*a. epigastrica inferior*).

Arteries of the Pelvic Organs



Number	Description
1	Abdominal aorta
2	Ureter
3	Inferior mesenteric artery
4	Median sacral vein
5	Common iliac artery
6	Internal iliac artery
7	External iliac artery
8	Obturator artery
10	Umbilical artery
11	Vaginal artery
12	Inferior rectal artery
15	Superior vesical artery
16	Urinary bladder
17	Vagina
20	Descending branch of uterine artery
23	Rectum

II. FEATURES OF VITAL ORGAN RESPONSES IN ACUTE BLOOD LOSS IN PREGNANT AND LABORING WOMEN

During a normally developing pregnancy, a number of important changes occur in a woman's body; understanding and taking them into account is necessary for diagnosing possible complications and providing adequate treatment. A pregnant woman's total body weight increases by an average of 12 kg. Moreover, 75% of this increase consists of fetal body weight, placenta, uterus, and amniotic fluid, as well as circulating blood volume (UQO), which begins to increase in the first trimester. In the second trimester, UQO growth reaches its maximum, and by the end of the third month it is on average 40–50% higher than baseline, reaching 74 ml/kg of body weight, which is almost 1.5 liters more than in a non-pregnant woman. The volumes of circulating plasma and red blood cells increase in different proportions. The intravascular plasma volume rises from 2.5 L to 3.8 L by the 40th week of pregnancy, while the circulating erythrocyte volume increases from 1.4 L to 1.65 L during this period. As a result, when hemoglobin concentration and hematocrit decrease, the phenomenon of physiological hemodilution, or dilutional anemia, appears. Blood dilution observed during pregnancy plays an important role in preventing thrombosis.

Hemodynamic changes in the cardiovascular system of a pregnant woman contribute to increased uterine blood flow and ensure fetal vital activity. By the end of the

first trimester, cardiac output already increases from 4.5 L/min to 6.8 L/min due to increased stroke volume and heart rate. As a result, uterine blood flow rises from 50 ml/min at the beginning of pregnancy to 500 ml/min at the end, before delivery. At the same time, total peripheral and pulmonary resistance decrease, so central venous pressure (MVO) remains normal.

Lung function also changes significantly. Pregnant women are characterized by a tendency to hyperventilation. By the end of pregnancy, during labor, minute ventilation increases by an average of 50%, reaching 10 L/min due to increased tidal volume and respiratory rate. The increase in minute ventilation is accompanied by increased oxygen delivery and consumption and a decrease in arteriovenous oxygen difference. One-third of the increased oxygen consumption is used to compensate for increased cardiac work, almost half by the kidneys, and the remainder by the uterus and placenta. During labor, physiological hyperventilation is accompanied by hypocapnia (during contractions pCO2 decreases to 20 mm Hg), which is the most important condition for normal transplacental diffusion of CO2 from the fetus to the mother. During pregnancy, renal blood flow increases. As a result, slight enlargement of kidney size, dilation of calyces, and an increase in glomerular filtration from 100 to 150 ml/min/m² are observed. At the same time, sodium reabsorption increases significantly, resulting in an increase in total body water.

Special attention should be paid to changes in the coagulation system during pregnancy and childbirth. In uncomplicated pregnancy, the concentration of plasma coagulation factors increases. The level of factor I (fibrinogen) increases most significantly—from an average of 2.5 to 3.8 g/L—as do factors VII, VIII, IX, and X. A decrease in coagulation inhibitors, in particular protein S, is noted in pregnant women. By the 12th week of pregnancy, its amount decreases by 40–50% from the initial level. The level of prothrombin, activated partial thromboplastin time (APTT), bleeding time, and antithrombin III level remain normal.

In the placenta, plasminogen activator inhibitor (PAI-2), which regulates fibrinolytic activity, is formed and released into maternal plasma; together with another plasminogen activator inhibitor (PAI-1), it blocks tissue plasminogen activator, preventing plasmin formation.

Thus, a number of changes occur in a pregnant woman’s body. On the one hand, processes preventing thrombus formation before childbirth (hemodilution) are present; on the other hand, processes ensure rapid thrombosis of the placental bed after delivery (the listed changes in the hemostatic system). They are activated when the uterus contracts or fails to contract postpartum, when a zone of damaged placental vessels appears. Acute blood loss is one of the most common injuries to the body throughout evolution and can lead to significant disturbances of vital activity; therefore, medical intervention is always necessary.

The definition of acute massive blood loss requiring transfusion intervention is associated with the availability of necessary reserves, because these reserves are precisely what give the physician the right to perform the very risky operation of transfusing blood components. Acute blood loss is considered massive and requiring transfusion support if, within 1–2 hours, estimated blood loss is at least 30% of the

initial volume. In this case, collapse of peripheral vessels ("empty vessels" sign), persistent hypotension, and decreased hourly diuresis are noted.

The body responds to massive blood loss with several standard reactions aimed, on the one hand, at creating favorable conditions to stop bleeding and, on the other hand, at ensuring oxygen transport to organs and systems that make survival possible. Oxygen transport is provided in the "order of importance" (heart–lungs–brain–liver–kidneys). The secretory glands of the gastrointestinal tract are functionally suppressed (therefore one of the most important signs of bleeding, including internal bleeding, is dry mouth), intestinal motility almost stops, urination decreases, and the vessels of skin and muscles are in a dormant state (peripheral vasoconstriction).

At the same time, lymph and interstitial fluid enter the bloodstream, and blood dilution increases—hemodilution. The blood dilution process is not rapid. Therefore, in the first hours of acute blood loss, its severity cannot be assessed by hemoglobin concentration, which decreases when blood is diluted by lymph: despite major blood loss and marked skin pallor, hemoglobin level may remain close to the initial value for several hours. From the splenic depot, skin, and muscle capillaries, red blood cells are released into the circulation. A kind of blood autotransfusion and maximal centralization of circulation occur. The described physiological reactions ensure replenishment of circulating blood volume and, to some extent, circulating red blood cell volume, maintaining oxygen transport and its delivery to tissues.

In this, body immobilization plays a very important role. The area of working muscle capillaries is approximately 20:1 compared with resting muscles. Therefore, when discussing the required volume of blood replacement, one should consider the sharply reduced demand of an immobilized body for circulation and oxygen delivery. Along with the listed compensatory reactions, increased oxygen release to tissues and its utilization are observed. Thus, for physiological protection from blood loss, the maternal body responds with hemodilution, which improves blood fluidity, mobilization from the red blood cell depot, and after delivery a sharp decrease in blood volume demand (muscle relaxation) and oxygen demand due to increased respiratory rate, cardiac output, and left ventricular work.

It is known that approximately 70% of total UQO is in veins, 15% in arteries, 12% in capillaries, and 3% in heart chambers. In women, circulating blood volume is not identical and depends on constitution: for normosthenics—6.5% of body weight, for asthenics—6%, for hypersthenics—5.5%, for athletic muscular women—7%; therefore absolute UQO values may differ, which should be considered in clinical practice.

A specific feature of obstetric hemorrhage is its massive and sudden onset. As a rule, when obstetric bleeding occurs during pregnancy, the fetus suffers. This necessitates urgent delivery and does not allow waiting for stable stabilization of hemodynamic parameters and full infusion-transfusion therapy. In some cases, blood loss is accompanied by severe pain. Specific pathophysiological changes in pregnant women, especially in hypertensive disorders or complicated labor, lead to rapid activation of compensation and defense mechanisms. In addition, obstetric hemorrhage is characterized by acute volumetric deficit of circulating blood, cardiac dysfunction, and anemic and circulatory forms of hypoxia. The main causes of hemodynamic

disorders in obstetric bleeding are deficiency of circulating blood volume and mismatch between this volume and intravascular capacity. Disturbances of water-electrolyte balance, acid-base balance, hormonal ratios, and enzymatic processes occur. It should be remembered that massive obstetric bleeding is almost always accompanied by impaired blood coagulation properties (DVS syndrome). Even with relatively small blood loss (15–20% of UQO), biphasic changes in the hemostatic system are often pronounced: a short hypercoagulation phase is replaced by a hypocoagulation phase. These body reactions are aimed at preserving viability and guide the physician in providing appropriate care.

CLASSIFICATION OF OBSTETRIC HEMORRHAGES

Bleeding during pregnancy occurs in 2.5–4% of pregnant women. This article does not discuss bleeding in the first and second trimesters, since it is interpreted as incipient abortion.

The causes listed below are referred to as **late obstetric hemorrhage**:

1. **Bleeding in the second half of pregnancy**, including:
   A) placenta previa
   B) premature placental abruption
   C) rupture of umbilical vessels with varicose changes
   D) cervical polyps
   E) cervical erosion
   F) blood diseases (Werlhof disease, APS, Schönlein–Henoch, etc.).
2. **Bleeding during labor**:
   A) premature placental abruption
   B) rupture of placental lateral sinuses
   C) rupture of umbilical vessels
   D) uterine rupture
3. **Bleeding in the postpartum period** occurs in 2.5–8% of cases; in 20–45% of cases, these hemorrhages cause maternal death (V.I. Kulakov, 2009):
   A) retained placenta
   B) pathological placental attachment (placenta adherens, true accreta)
   C) incomplete placental separation
   D) uterine hypotonia and atony
   E) birth canal trauma
   F) hereditary and acquired hemostatic disorders
4. **Postpartum hemorrhage**:
   A) retained placental tissue in the uterine cavity
   B) uterine hypotonia and atony resulting from mismanagement of labor, multiple pregnancy, macrosomia, and prolonged labor
   C) uterine rupture
   D) tears of soft tissues of the birth canal
   E) hereditary and acquired hemostatic disorders

Causes of bleeding in the second half of pregnancy (according to Beckmann, 2004):

- **Vulva** – injury of varicose veins

- **Vagina** – trauma or injury
- **Cervix** – polyps, cervicitis, cancer, erosion, ectropion
- **Uterine cavity** – placenta previa, premature placental abruption, vasa previa

Classification of obstetric hemorrhage according to ICD-11:

- **O44** – Placenta previa:
  - **O44.0** – Placenta previa specified without bleeding;
  - **O44.1** – Placenta previa with bleeding.
- **O45** – Premature placental separation (placental abruption):
  - **O45.8** – Other premature placental separations;
  - **O45.9** – Premature placental separation, unspecified.
- **O46** – Antepartum hemorrhage, not elsewhere classified:
  - **O46.0** – Antepartum hemorrhage with coagulation defect;
  - **O46.8** – Other antepartum hemorrhage;
  - **O46.9** – Antepartum hemorrhage, unspecified.
- **O67** – Labor and delivery complicated by intrapartum hemorrhage, not elsewhere classified:
  - **O67.0** – Intrapartum hemorrhage with coagulation defect;
  - **O67.8** – Other intrapartum hemorrhage;
  - **O67.9** – Intrapartum hemorrhage, unspecified.
- **O72** – Postpartum hemorrhage:
  - **O72.0** – Hemorrhage in the third stage of labor;
  - **O72.1** – Other immediate postpartum hemorrhage;
  - **O72.2** – Delayed or secondary postpartum hemorrhage.

III. CLINICAL CHARACTERISTICS OF OBSTETRIC HEMORRHAGE

According to the World Health Organization, 585,000 women die every year from complications of pregnancy and childbirth; according to recent data, mortality has increased by 85,000. Maternal mortality is high in Asian countries, particularly India, Bangladesh, and Pakistan, as well as in Afghanistan and African countries (150,000 annually). The causes of maternal mortality differ across countries. In Uzbekistan, obstetric hemorrhage has held the leading position for many years, followed by hypertensive conditions, amniotic fluid embolism, and purulent-septic complications. Among all causes of obstetric hemorrhage, hypo- and atonic bleeding occurs in 60–70% of cases and is mainly observed during prolonged labor, when the woman in labor is exhausted. At present, this rate is 3–8% of all deliveries.

In the Republic of Uzbekistan, hypo- and atonic hemorrhage accounts for 25% of all causes of obstetric bleeding, while in Samarkand region it accounts for 35%.

An important feature of obstetric hemorrhage is that it occurs in pregnant women, women in labor, and postpartum women, often suddenly, and within a short period a woman may lose a large amount of blood. As a result, compensatory capacity decreases and hemodynamic and hemostatic disturbances develop, especially in women with a pathological course of pregnancy and childbirth.

According to the authors of *Am Fam* (2007), the causes of hemorrhage are: 70% due to uterine atony, 20% due to trauma (hematomas, ruptures, etc.), 10% due to tissue

causes (retained placenta, placenta accreta), and a total of 1% due to coagulopathic bleeding.

Today, the frequency of obstetric hemorrhage in our Republic is 25.8%. In our country, the causes of this complication during pregnancy and childbirth include acute premature placental abruption and placenta previa, pathological placental attachment to the uterine wall, birth canal injury, hypoatonic hemorrhage, and uterine rupture. If adequate care is not provided, these hemorrhages may lead to hemorrhagic shock, coagulation disorders, disseminated intravascular coagulation syndrome, and subsequently to multiple organ and multisystem failure. Bleeding during labor and in the early postpartum period (within 2 hours after birth) accounts for 50% of all hemorrhage cases. In 34.7% of cases, secondary uterine inertia leads to pathological hemorrhage. Excessive and discoordinated uterine contractions also result in atony.

The second group of causes of hypo- and atonic hemorrhage is overdistension of the uterine wall, as a result of which the uterus loses its contractile ability. This is observed in multiple pregnancy (twins), macrosomia, and polyhydramnios. Placental pathology—placenta previa or low-lying placenta, and premature abruption of a normally located placenta—also causes hemorrhage due to poor contraction of the placental bed.

Bleeding is observed in women with anatomical uterine defects, uterine fibromatosis, adhesions, tumors, a history of multiple abortions, and infantilism; functional uterine defects develop due to decreased levels of biologically active substances in the uterus.

Disturbance of cerebral circulation in the mother leads to circulatory disorders in all organs, which in turn causes hypo- and atonic hemorrhage.

Severe forms of hypertensive disorders, extragenital diseases, and intrauterine fetal death lead to coagulation system disorders, disseminated intravascular coagulation syndrome, and bleeding. Hypotonic hemorrhage occurs in waves. Bleeding usually begins in the postpartum and early postpartum periods.

There are two clinical variants of early postpartum hemorrhage:

- In the **first variant**, bleeding is abundant and massive from the very beginning. The uterus is relaxed, in a state of atony, and does not respond to uterotonic agents, external massage, or manual examination of the uterine walls. Hypovolemia develops rapidly, and hemorrhagic shock and disseminated intravascular coagulation syndrome develop.
- In the **second variant**, there is alternation of recurrent blood loss with temporary restoration of uterine muscle tone and temporary cessation of bleeding in response to conservative therapy. Blood is lost in portions of 150–250 ml. Small-volume blood loss allows temporary adaptation of the postpartum woman to developing hypovolemia. Blood pressure remains within normal limits; the skin and visible mucosa become pale, and tachycardia increases. This period of temporary compensation is often overlooked. Without adequate treatment in the early initial period of uterine hypotension, the severity of disturbances increases and blood loss volume rises. At a certain stage, bleeding intensifies, often accompanied by worsening of the general condition—signs

of shock rapidly increase and DVS syndrome develops. If the condition is not severe and timely care is provided, bleeding can be stopped within 15–30 minutes.

Regarding determination of blood loss volume, obstetricians have certain advantages. In most cases, hemorrhage develops in their presence—collect it and measure it. A separate issue is determining blood loss in hemorrhage that occurs outside the hospital. When assessing blood loss, interviewing witnesses (emergency physicians, relatives) may not always be accurate. In such situations, use of the **"shock index"** is helpful. Patients with hypertension are an exception.

In most cases, when calculating blood loss volume, one should initially rely on a known percentage of the woman's body weight and, depending on blood loss volume, determine the main ITT program. Each ITT component should have its own indications. In obstetric hemorrhage, one of the first and primary tasks of ITT is restoration of blood volume.

ERRORS AND DEFICIENCIES IN PROVIDING CARE FOR OBSTETRIC HEMORRHAGE

Early recognition of obstetric hemorrhage is a very important factor in treatment. Prevention, early detection, and rapid response are key to minimizing the consequences of obstetric hemorrhage. Delayed diagnosis of postpartum hemorrhage was identified in 68 (71%) cases, of which 48 (84%) occurred after cesarean section and 20 after vaginal delivery. Medical records lacked information on uterine tone and the amount of vaginal discharge at specified time intervals. According to the national standard for obstetric hemorrhage, routine postpartum monitoring should include assessment of uterine tone and vaginal discharge; monitoring of hemodynamic parameters every 15 minutes during the first hour after birth and at the end of the 2nd, 3rd, and 4th hours; then every 4 hours during the first 24 hours. Inadequate postpartum monitoring leads to delayed recognition of massive hemorrhage and, accordingly, delayed measures to stop bleeding. In all deceased cases, there was no monitoring chart documenting heart rate, A/D, blood loss, respiratory rate, oxygen saturation, diuresis, administered drugs, as well as infusion volume during observation.

It is known that blood loss volume can be determined by three methods:

- weighing blood clots and multiplying the result by two;
- weighing blood-soaked pads and used materials.

However, in practice, blood loss is often assessed visually; in such cases, errors of 200–300 ml, and sometimes up to 1000 ml, are made, which negatively affects the choice of obstetric tactics.

This clinical assessment still remains the primary method in obstetric practice for determining blood loss volume and selecting therapy. It should be taken into account that changes in pulse and A/D, however significant, appear in late stages of hemorrhage and indicate the beginning of failure of compensatory mechanisms. It should also be considered that in the postoperative period fluid infusions affect hemodynamic parameters and may prevent a significant drop in A/D and marked tachycardia. Sudden blood loss may not present with decreased hematocrit or hemoglobin in the first 4 hours or longer. During normal vaginal delivery, blood loss is usually

assessed visually and is often incorrect. During labor, the doctor estimates blood loss by eye, but the true amount is often much higher than the visual estimate. Underestimation of blood loss and inadequate monitoring of hemodynamic parameters and hemoglobin lead to delays in adequate infusion therapy and blood transfusion, resulting in subsequent complications of posthemorrhagic anemia. All lost blood should be collected and measured in calibrated containers or trays with volume markings. Each institution should also know the weight of each pad, sheet, dressing material, and gauze of different calibers used during surgery. In blood loss analysis, all collected blood in graduated containers and trays should be evaluated. Hemodynamic assessment should include not only A/D, pulse, and diuresis, but also respiratory rate and oxygen saturation measurement, which may indicate oxygen deficiency in the body. During monitoring, hemoglobin and hematocrit should be assessed regularly.

Delayed surgical hemostasis. Delay in necessary obstetric care. Incorrect assessment of blood loss, passivity and negligence of physicians, and refusal of the patient's relatives to consent to manipulations lead to insufficient obstetric care. As a result, first-stage measures are ineffective. Analysis of medical records and questionnaire data identified the following causes of delayed surgical hemostasis:

- insufficient assessment or underestimation of blood loss volume;
- staff lacking sufficient skills to perform surgical hemostasis;
- absence of a protocol for uterine balloon tamponade;
- insufficient use of organ-preserving technologies.

According to national standards, surgical hemostasis should be initiated when blood loss reaches 1500 ml; nevertheless, in 50% of cases, hemostasis is performed late. This is often explained by the fact that laparotomy hysterectomy is associated with hemorrhage and the decision to remove the uterus in first delivery is made late. Delays in surgical hemostasis also occur after cesarean section.

It should be emphasized that if first-stage measures are ineffective, immediate transition from conservative methods to surgical methods, i.e., surgical hemostasis, is required. In all cases, the interval between diagnosis of hemorrhage and initiation of surgical control should be minimized. After manual uterine examination, if incomplete uterine rupture is present, laparotomy should be started immediately. If uterine atony is identified after manual examination and blood loss continues despite bimanual compression and repeated uterotonics, global practice recommends balloon tamponade, with reported effectiveness of 88–98%. If bleeding continues despite uterine balloon tamponade, laparotomy should be performed. If bleeding persists, surgical hemostasis should not be delayed, as it is the only way to save the woman. International practice recommends hemostatic measures according to the principle: **"Earlier rather than later!"**

Despite clinical guideline recommendations, organ-preserving technologies are insufficiently used in practice. The reason is lack of adequate experience and knowledge among surgeons regarding these techniques. Currently, many methods of compression sutures exist—B-Lynch, Hyman, Cho, Pereyra, Kurtser, and others. Vessel ligation and compression sutures require consistent, step-by-step adherence to organ-preserving principles.

Ineffectiveness of organ-preserving technologies is an immediate indication for hysterectomy. The decision to remove the uterus should be made by an experienced physician, and the operation should be performed by a surgeon experienced in hysterectomy. Hysterectomy should not be postponed as a last resort if less radical options are unlikely to work or surgeon experience is limited. Subtotal hysterectomy is the method of choice in most postpartum hemorrhage cases requiring hysterectomy if there is no cervical or lower-segment injury; the risk of cervical stump neoplasia developing years later is not significant in life-threatening hemorrhage. Hysterectomy should be performed regardless of hemodynamic parameters and hemorrhagic shock status when the bleeding source cannot otherwise be excluded and stabilization is unlikely.

Surgical treatment should be started under any conditions—hemorrhagic shock, DVS syndrome, etc.—and no condition should prevent surgical control of bleeding. In severe hemorrhagic shock and technical difficulties with surgical hemostasis, the principle of **"damage control surgery"** should be used:

1. **Stage 1** – after laparotomy, bleeding is stopped by any method: compression, clamping, ligatures, tamponade, and even aortic cross-compression.
2. **Stage 2** – the anesthesiologist-resuscitator stabilizes vital body functions, which occurs faster and more effectively than under ongoing bleeding conditions.
3. **Stage 3** – after shock is resolved, the obstetrician-gynecologist provides definitive hemostasis required for this case in a fundamentally different clinical situation.

It is known that the woman's initial status during bleeding is critically important: hypovolemia, hypoxic-ischemic disturbances in vital organs, and coagulopathy in the coagulation system.

To prevent these complications, ITT, particularly transfusion, is used and should be timely and rational. However, in practice, physicians focus more on stopping bleeding, while the above measures are delayed or insufficiently implemented. In the first 5 minutes, only in 25% of cases were two veins catheterized and crystalloid infusion started. In the first 15 minutes, among women with major blood loss, only 20% received 1000 ml crystalloids. The second task after stopping bleeding is restoration of circulating blood volume, as well as stabilization of hemodynamics, adequate oxygen delivery, and increase of hemoglobin concentration. UQO restoration is provided by crystalloids and colloids. In major severe blood loss over 1500–2000 ml, a massive transfusion protocol should be followed. Administration of blood components (plasma, red blood cells) should begin as early as possible (within the first 2 hours), because with such blood loss, >2000 ml crystalloids or colloids already lead to hemodilutional coagulopathy and increase blood loss volume, multiorgan failure, and death. This may be due to shortage of blood products or even simple solutions, and may result only from negligence of obstetric care organizers or practitioners. According to national standards, circulating blood volume should be calculated by the woman's weight, blood loss severity should be determined according to blood loss volume, and blood loss should be replaced according to tables. In maternity institutions, it is recommended to visibly display infusion-program tables by blood

loss level, train staff to use these tables, and organize blood reserves in institutions with more than 2500 births per year.

Inadequate antenatal care. When reviewing medical records of women with massive blood loss, almost all women had antenatal risk factors: preeclampsia, uterine scar, large fetus, multiple births, placenta previa, placenta accreta (invasion), severe anemia, thrombocytopathies, antenatal fetal death, septic pneumonia, and acute fatty liver. Despite these risk factors, in 90% of cases they were not hospitalized in time.

Deficiencies in laboratory services and diagnostic equipment.

Most often, as a rule, these deficiencies are found in regional maternity hospitals. What is necessary in obstetric practice is not at the proper level: diagnostics of the coagulation system, thromboelastography, coagulogram. This leads to untimely diagnosis of coagulopathic disorders, which in turn causes incorrect differentiation of phases. According to WHO, in addition to the above, there are the following errors and deficiencies that can lead to maternal mortality (WHO Chronicle, 2017). These are:

- **Low level of women's medical knowledge.**
  No one except the woman herself can be responsible for her health. Since a doctor sees a woman once a month, at most once a week, only the woman herself and her close relatives can observe her around the clock. However, knowledge of simple warning signs such as YMOK, premature rupture of amniotic fluid, and antenatal fetal death indicates the need for immediate consultation with a doctor.
- **Insufficient qualification of doctors.**
  This especially applies to primary care doctors, who must quickly identify life-threatening pathologies and hospitalize patients in specialized medical institutions (WHO, Geneva 2002; Rooney S., 2012).
- **Poor infrastructure of medical care.**
  This concerns transporting a woman from peripheral areas to a specialized medical institution. It includes lack of transport, poor roads, and weak communication (WHO/FHE/2004).
- **Negligent attitude of medical staff.**
  Sometimes, even if the woman arrives on time, passivity of medical staff, delay of necessary manipulations, untimely availability of blood products or other medications, and delayed prescriptions can lead to maternal death. Every specialized obstetric hospital should have blood reserves, everything necessary for emergency care, fully equipped additional operating rooms, as well as highly qualified specialists ready to receive patients (Campbell O.M., 2000; WHO/FHE/2004). Elimination of all these deficiencies can lead to a reduction in maternal mortality.

IV. PLACENTA PRAEVIA (PLACENTA PREVIA)

Placenta previa (POK) is an abnormal attachment of the placenta to the uterus. If it is located in the lower uterine segment above the internal os, it partially or completely covers it and lies in the fetal passage. The term “previa” consists of two parts, “prae” and “via,” meaning “on the way.” Normally, when the placenta is attached in the uterine body, its lower edge is located 7 cm or more away from the internal os.

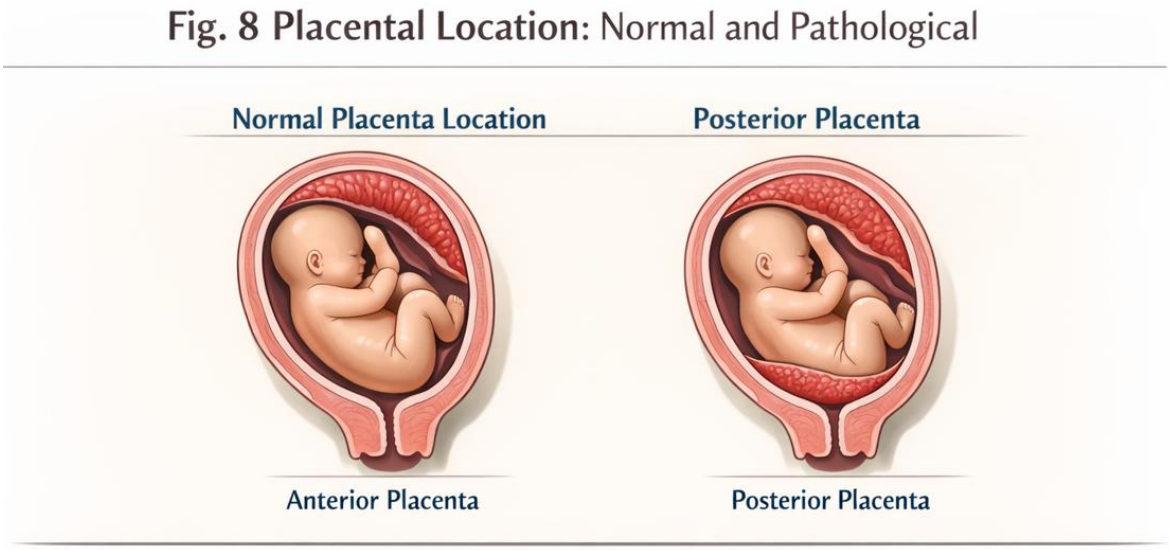
Problems associated with placentation anomalies:

- Increase in the number of cesarean sections;
- In these patients, delivery is accompanied by bleeding;
- Need to manage blood loss during surgery;
- Performance of organ-preserving surgical measures;
- Timely diagnosis of placenta invasion (accreta spectrum).

Frequency.
According to literature analysis, placenta previa occurs in 0.2–0.6% of cases. In fact, the frequency of this pathology is much higher, confirmed by ultrasound data from early pregnancy. A large proportion of miscarriages in the first half of pregnancy occur as a result of low placentation. According to WHO, the main cause of maternal death in placenta previa is hemorrhagic shock. Maternal morbidity is 22–25%, and preterm birth is 20–25%. Perinatal mortality varies from 17% to 25% and is mainly associated with prematurity, fetal immaturity, and antenatal fetal distress. Fetal death depends on the volume of blood loss. With placenta previa, preterm birth is observed in 47% of cases. Fetal anomalies are detected 2.5 times more often than in the general population. Fetal growth restriction is also identified.

Significant variation in statistical data is due to the following reasons:

1. In maternity institutions where high-risk pregnant women are hospitalized, placenta previa is observed more often than in institutions where births occur without complications;
2. There is no unity of opinion regarding the degree of cervical dilation (2–4–6 cm) at which the degree of placenta previa is finally determined;
3. Differences in applied research methods.
   Currently, the most informative methods are ultrasound and MRI.



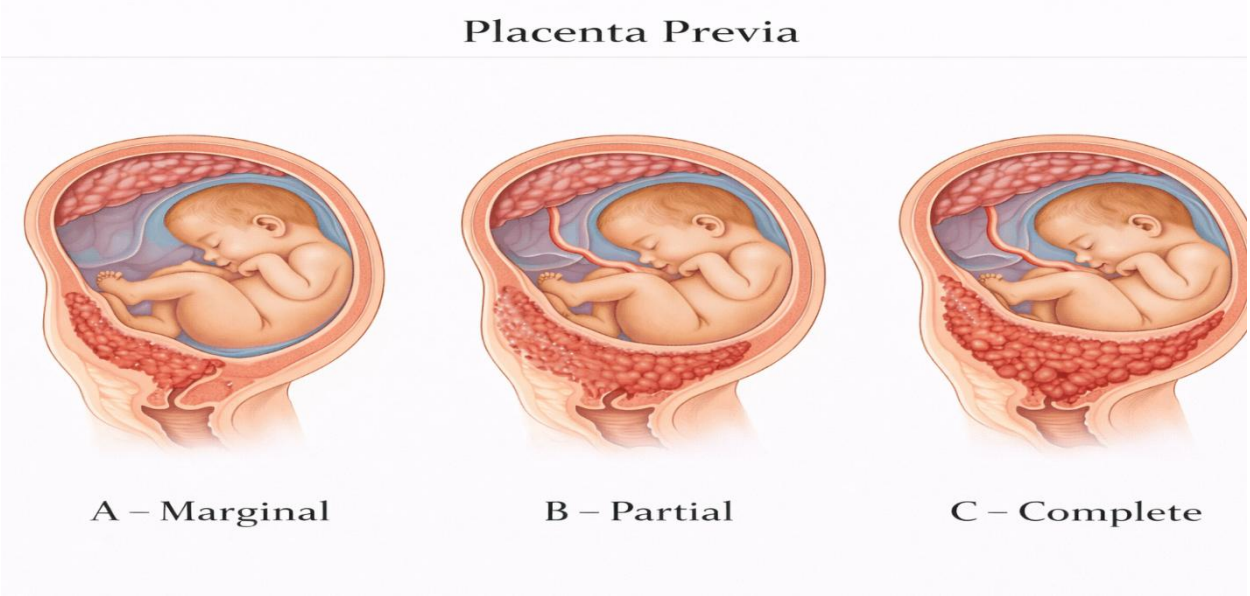
Classification of placenta previa

There are different classifications of placenta previa. Some authors use the following:

1. **Central placenta previa (placenta previa centralis)** — the internal os is covered by placental lobules; fetal membranes are not detected on vaginal examination.
2. **Lateral placenta previa (placenta previa lateralis)** — parts of one or two placental lobules are identified at the internal os; during vaginal examination, fetal membranes are detected near the placental lobules.
3. **Marginal placenta previa (placenta previa marginalis)** — the lower edge of the placenta is located at the margins of the internal os; only fetal membranes are present within the os.

In addition, there is a simpler classification, commonly used abroad (Williams Obstetrics, 2001):

1. **Complete placenta previa (total placenta previa)** — the internal os is completely covered by the placenta.
2. **Partial placenta previa (partial placenta previa)** — the internal os is partially covered by the placenta.
3. **Marginal placenta previa (marginal placenta previa)** — the placental edge is at the margin of the internal os.
4. **Low-lying placenta** — the placenta is located in the lower uterine segment, but its edge does not reach the internal os.



Etiology and pathogenesis

The specific cause of placenta previa is unknown. During pregnancy, the localization of implantation of the fertilized ovum is influenced by many factors. Traditionally, they can be divided into 2 groups:

1) Fetal factor

If the fertilized ovum is initially located low or if its proteolytic properties are reduced, it may fail to implant in the uterine fundus in time. In this case, implantation occurs after the ovum descends into the lower uterine segments. A similar mechanism is possible with artificial insemination, IVF, and embryo transfer.

2) Uterine factor

The presence of dystrophic and atrophic changes in the endometrium contributes to placenta previa. In this situation, the zygote cannot implant in the altered mucosa of the uterine fundus/body and descends lower.

Main causes of dystrophic and atrophic endometrial changes:

- inflammatory uterine processes (chronic endometritis);
- history of multiple births and frequent abortions;
- postpartum and post-abortion septic diseases;
- uterine fibroids;
- deformation of the uterine cavity (uterine scars after cesarean section and other surgeries);
- congenital uterine anomalies;
- conservative myomectomy, repair of uterine rupture, etc.

When cesarean section is performed in placenta previa, the rate of hysterectomy increases further. If 25% of hysterectomies in placenta previa cases were performed in women with a history of two previous cesarean sections, then after the first cesarean section for placenta previa, hysterectomy was performed in only 6%.

At 18 weeks of pregnancy, 25% of pregnant women had low placental location; by the end of pregnancy, this persisted in 7% of women. During ultrasound examination at 18–20 weeks, low placental location was found in 12% of cases. If the placenta

did not cover the internal os, placenta previa was not diagnosed. If the placenta was located over the internal os, placenta previa persisted in 40% of cases.
During transvaginal ultrasound at 18–23 weeks, placenta previa was diagnosed in 1.5% of women examined. Of these, only 20% had the placental edge located less than 15 mm from the internal os at delivery. When located 25 mm or more from the os, placenta previa persisted in 40% of cases.

Table 1. Risk factors for placenta previa development

No.	Factors	Relative risk
1	History of previous placenta previa	2.6
2	Previous cesarean sections	2.5
3	History of 1 cesarean section	2.2
4	History of 2 cesarean sections	4.1
5	History of 3 cesarean sections	22.4
6	Previous pregnancy termination	—
7	Multiparity	—
8	Advanced maternal age (>40 years)	—
9	Multiple pregnancy	—
10	History of endometrial insufficiency: uterine scar; endometritis; manual removal of placenta; curettage; submucosal myoma	—

In the first half of pregnancy, placental growth has been found to predominate over uterine growth. By the end of the second trimester and the beginning of the third trimester, on the contrary, due to formation of the lower uterine segment, the placenta is displaced upward. It may move by 3 to 9 cm; that is, placenta previa diagnosed in early pregnancy does not always persist until term.
Due to insufficient development of the decidua in the lower uterine segment, dense placental attachment (placenta adhaerens), and sometimes true invasion (placenta increta or percreta), may occur. With placenta previa, pathological placental attachment occurs in 7% of cases. Some researchers, when comparing maternal risk factors for placenta previa and placental abruption, concluded that abruption is more associated with conditions arising during pregnancy, whereas previa is more associated with conditions present before pregnancy.

Clinical presentation

Placenta previa has 2 phases:

1. **“Silent phase”** — no obvious symptoms;
2. **“Manifest phase”** — external bleeding appears, indicating placental separation in placenta previa.

Before bleeding begins, the clinical picture of placenta previa is difficult to determine. In the third trimester and late pregnancy, there may be a high station of

the presenting part, unstable fetal position, a high percentage of oblique and transverse lies, breech presentation, and frequent signs of threatened pregnancy, etc. Before the use of ultrasound, placenta previa was usually diagnosed only when external bleeding occurred, which is the main clinical sign. This bleeding has characteristic features: the blood is usually bright red, with no pain syndrome ("painless bleeding"). Bleeding may occur without any apparent cause—at night during sleep, at rest, etc. Another characteristic feature is recurrence of bleeding. A second important feature is progressive anemia in the pregnant woman, because with repeated bleeding, even if small, adequate blood regeneration does not occur. Under such conditions, even relatively small bleeding during labor can produce a clinical picture of decompensated blood loss and become life-threatening.

With placenta previa, uterine bleeding most often occurs at 30–35 weeks of pregnancy. It occurs very rarely in early pregnancy (9–12 weeks) and rarely in the first stage of labor. The appearance of bleeding is associated with premature placental separation. It is known that uterine contractions occur not only during labor but also during normal pregnancy (Braxton-Hicks contractions), although they are often not felt by the pregnant woman. In this case, distraction (stretching) of uterine muscle fibers occurs, especially in the isthmus region during formation of the lower uterine segment. The placenta has no ability to stretch. As a result, displacement occurs along two surface planes—the lower uterine segment and part of the placenta—whereby the maternal placental surface separates from the uterine wall, and bleeding occurs from placental bed vessels. This bleeding may stop only after contractions cease, vascular thrombosis develops, and placental separation stabilizes. When uterine contractions resume, bleeding also recurs.

Another explanation of the bleeding mechanism during pregnancy is possible. At the end of the second trimester and beginning of the third trimester, when the lower segment is forming, its length increases and lags behind placental growth, resulting in placental "migration," which may lead to separation and bleeding.

In the third trimester, painless bleeding—regardless of severity—should be considered placenta previa until correct diagnosis is established.

One factor in bleeding onset with placenta previa at the beginning of labor is tension of the membranes at the lower pole of the ovum, which holds the placental edge so it does not follow contraction of the lower uterine segment; as a result, their connection is disrupted and bleeding occurs. After membrane rupture, relations change, because the placenta no longer separates further with lower-segment contraction. During labor, bleeding may stop after rupture of membranes and mechanical compression of the placental edge by the fetal head descending into the pelvis.

With complete placenta previa, spontaneous cessation of bleeding is not possible, because during cervical dilatation the membranes do not open in the usual way, and placental separation continues.

Diagnosis

When diagnosing placenta previa, features of obstetric and gynecologic history should be considered, including inflammatory diseases of the uterus and adnexa,

post-abortion and postpartum diseases, infertility, uterine anomalies, previous abortions, uterine surgery (conservative myomectomy, cesarean section), and ovarian dysfunction. Pregnancy often proceeds with threatened miscarriage in the first and second trimesters.

In the third trimester, placenta previa can be suspected if the fetus has an abnormal lie (transverse, oblique), breech presentation, or if the presenting part is high above the pelvic inlet. On palpation, the presenting part (head) is less clearly felt, as if through spongy tissue (uterine wall and placenta), and it may be mistaken for the pelvic end. If the placenta is located on the posterior wall of the lower segment, the presenting part often protrudes toward the pubis; displacement backward toward the promontory may cause a feeling of resistance and lead to a decrease in fetal heart rate.

A Doppler device may be used to successfully determine placental location; in addition to fetal heart tones, it allows detection of placental vessel sounds and localization of the placenta. Fetal heart activity is usually not affected by placenta previa.

The main sign of placenta previa is frequent recurrent painless bleeding from the genital tract in the second half of pregnancy. Bright red bleeding may be heavy or slight.

When a pregnant woman presents with bleeding, the physician should limit the examination to general and external obstetric assessment. The most informative diagnostic method for placenta previa is ultrasound. The method accuracy is 98%. Vaginal examination is not performed, because bleeding may intensify.

According to national guidelines on antepartum hemorrhage, the following definitions are used:

- **Spotting** — appearance of spots, streaks, or blood stains on underwear or sanitary protection.
- **Minor bleeding** — blood loss <50 ml.
- **Major bleeding** — blood loss 50–1000 ml, without clinical signs of shock.
- **Massive bleeding** — blood loss >1000 ml and/or clinical signs of shock.
- **Recurrent antepartum bleeding** — term used when bleeding episodes occur multiple times.

When a pregnant woman with bleeding is admitted to hospital, to уточнить diagnosis, speculum cervical examination and bimanual vaginal examination are performed **with the operating room prepared** (for cesarean section). On speculum examination, the bleeding source can be identified and several possible causes excluded: cervical canal polyp, cervical erosion and cancer, vaginal varicosities with ruptured node.

Bleeding from the cervical canal can also occur with abruption of a normally located placenta, rupture of marginal placental sinus, rupture of umbilical vessels, uterine rupture, etc.

Treatment

Choice of treatment method for placenta previa depends on several factors:

- timing of bleeding onset (during pregnancy or labor);
- severity and volume of blood loss;

- general condition of the pregnant/laboring woman;
- condition of the birth canal (degree of cervical dilatation);
- type of placenta previa;
- gestational age;
- fetal condition and position;
- hemostatic status.

Management of pregnancy and labor

If ultrasound in early pregnancy reveals placenta previa but there is no clinical manifestation (bleeding), what should be done? Bleeding may occur at any time, and not only the pregnant woman but also her relatives should be informed. In the woman's best interest, hospitalization in a qualified center for evaluation (repeat ultrasound, Rh test, hemostasiogram) is recommended.

If the pregnant woman's condition is satisfactory, there is no bleeding, and she insists on discharge, this is permitted only after a written request from the woman and informed acknowledgment by close relatives regarding possible complications. The woman must receive clear instructions on which hospital to attend in case of bleeding. Bed rest at home is required, with control ultrasound every 2 weeks. It is recommended to prolong pregnancy to 36–37 weeks (fetal viability), then choose an adequate delivery method based on the obstetric situation if previa persists.

In women with placenta previa, the decisive factor is bleeding severity. Thus, if bleeding threatens the pregnant woman's life, regardless of fetal status (nonviable or dead fetus), the only correct method is delivery by cesarean section. In all other cases, many factors should be considered, first of all gestational age, type of previa, fetal condition, and readiness of the birth canal.

If a pregnant woman is hospitalized with slight genital bleeding and her condition is stable, strict bed rest, full examination, and treatment aimed at prolonging pregnancy (to 36–37 weeks) and assessing fetal condition are recommended.

If bleeding continues, an intravenous catheter should be inserted, blood pressure and pulse measured, blood group and Rh status determined, CBC performed, hemostasiogram done, and IV fluid therapy started. The hospital should have emergency reserve blood of group 0 (I), Rh-negative.

Evaluation first includes external obstetric examination. Attention is paid to uterine tone, tenderness, distension, nature of the presenting part and its entry into the pelvis, nature and amount of bleeding, etc.

To assess fetal condition, fetal heart rate, character of heart activity, fetal movements, Doppler assessment, etc. are performed.

The most informative test is ultrasound, which allows determination of placental location, thickness, volume, maturity, and detection of separation areas (often difficult because blood exits and does not accumulate at separation sites), fetal heartbeat, estimated fetal weight (hypotrophy), umbilical cord insertion, etc.

If placenta previa is confirmed, the pregnant woman should remain hospitalized, where symptomatic treatment is required to reduce uterine excitability, strengthen vascular wall stability, etc. Bleeding of varying severity may lead to anemia. This condition must be treated, since labor is approaching and is always associated with

some blood loss. In preterm fetus, due to risk of neonatal respiratory distress syndrome, glucocorticoid administration is indicated.
In partial or marginal placenta previa, and in absence of aggravating factors (bloody discharge, oblique/transverse fetal lie, multiple pregnancy, uterine scar, etc.), expectant management may be followed until spontaneous labor begins. If labor pains occur, and with head engagement bleeding appears, amniotomy is indicated. Before amniotomy, it is necessary to ensure cervical opening, whether the fetal head is engaged, whether placental tissue is present and how much it covers the internal os, and whether membranes are accessible for opening. After amniotomy, if the head descends to pelvic inlet, bleeding may stop, provided contractions are regular and adequate.
In this group, to stop bleeding, not only membrane rupture but also IV oxytocin should be started (5 IU in 500 ml isotonic sodium chloride) to increase uterine contractility. If after amniotomy the fetal head does not press the placenta firmly against the lower uterine segment and bleeding continues, cesarean section is indicated.
Indications for cesarean section include complete, partial, or marginal placenta previa with significant blood loss. According to literature, surgical delivery rate in placenta previa is 70–82%. The rationale is that immediate delivery of fetus and placenta allows uterine contraction and bleeding control. In addition, surgery helps avoid cervical injury; complete or partial placenta previa can cause serious complications.
With central placenta previa, the only correct mode of delivery is planned or emergency cesarean section. Planned surgery is recommended at 37 weeks. This allows delivery of a viable fetus, preparation for surgery, provision of necessary blood and infusion media, preparation for reinfusion during surgery, adequate anesthesia planning, and avoidance of antenatal blood loss.

MANAGEMENT OF PATIENTS WITH PLACENTA PREVIA AND PLACENTA INVASION, AND STAGES OF LABOR MANAGEMENT:

If placenta previa (with signs of accreta) is diagnosed, after the 2nd ultrasound all patients should be referred for consultation;

1. Perform 3 screenings; refer for hospitalization at 35 weeks;
2. Patients living in remote areas should be placed in hospital from 34 weeks onward.

According to researchers, introducing screening at 6–8 weeks of pregnancy plays an important role in identifying chorionic ingrowth formation in women with a previous cesarean section.

“Placenta accreta” criteria:

- thinning or absence of the uterine muscular wall in the placental area;
- absence of a clear border between the placenta and myometrium;
- presence of “Swiss cheese” and “Moth-eaten” placental lacunae;

- a distinct vascular network (vascularization) at the placenta accreta site.

PRE-DELIVERY HOSPITALIZATION STAGE:

1. If anemia is present (hemoglobin less than 100 g/L), treat with anti-anemia drugs for parenteral use;
2. Ultrasound findings to determine the level of the placental edge and signs of bladder wall involvement;
3. Carry out prophylaxis of neonatal RDS (taking gestational age into account);
4. Hematologist's conclusion.

Conservative hemostasis methods and blood-saving technologies

Use of uterotonics:
Intravenous oxytocin administration
Use of carbetocin
Use of misoprostol
Antifibrinolytics (tranexamic acid)
Intraoperative normovolemic hemodilution
Device-assisted reinfusion of autoerythrocytes
Blood components (FFP, platelets, cryoprecipitate)
Coagulation factors

Carbetocin is an effective uterotonic agent, and its use significantly reduces blood loss during abdominal delivery compared to oxytocin in patients with a uterine scar, large fetus, or multiple pregnancy. When using this drug for prevention of bleeding, additional methods to stop bleeding are required less often. Complex clinical cases combining placenta previa and placenta accreta do not serve as a direct indication for carbetocin use if bleeding is caused by a number of reasons and tone disturbance is not leading. Its use in organ-preserving operations makes it possible to ensure reliable hemostasis and reduce the volume of blood loss. Carbetocin demonstrates greater efficacy than continuous oxytocin infusion and has a similar safety profile. When used after placental delivery, a single intravenous administration of 100 mcg carbetocin is more effective and reliable than a standard continuous oxytocin infusion in maintaining adequate uterine tone and preventing excessive blood loss during cesarean section. This helps prevent excessive bleeding that is aggravated by low uterine tone. Carbetocin acts quickly and causes normal uterine contractions within 2–3 minutes after administration. Carbetocin is well absorbed and has a longer duration of action than oxytocin (approximately 5 hours) (oxytocin lasts 1 hour 30 minutes).

Step-by-step surgical hemostasis method

Controlled balloon tamponade;
Ligation of uterine vessels;
Uterine compression sutures;
Compression sutures + controlled balloon tamponade (uterine sandwich);
Ligation of the internal iliac arteries;
Hysterectomy;

Electrosurgery (argon plasma coagulation);
Local hemostatic sutures;

Pregnancy management tactics in the first half

If placenta previa is detected by ultrasound in early pregnancy and there is no bleeding, the pregnant woman may be observed in the clinic. If bloody discharge occurs, inpatient treatment is indicated. Therapy is aimed at reducing uterine excitability and strengthening the vascular wall:

¬ etamsylate 2 ml IM 3–4 times daily until bleeding stops;
¬ Magne B6, 2 tablets, 2 times daily until uterine tone normalizes and bleeding stops;
¬ magnesium sulfate 25% solution, 5 ml IM 1–2 times daily until uterine tone normalizes;
¬ dicynone 2–4 ml IM 1–2 times daily until bleeding stops;
¬ papaverine suppositories every night until uterine tone normalizes;
¬ no-shpa 1 tablet or 2 ml IM 1–2 times daily until uterine tone normalizes.

In placenta previa with severe uterine bleeding, cesarean section is an absolute indication regardless of gestational age. Conservative treatment is indicated at the end of the second trimester and the beginning of the third trimester (25–35 weeks) if bleeding is minimal. In such cases, strict bed rest must be ensured. Hemostatic agents (dicynone, vikasol) and restorative agents (40% glucose, vitamins) are prescribed.

If a pregnant woman presents to an antenatal clinic in the 2nd or 3rd trimester with complaints of bloody discharge from the genital tract, the doctor must urgently transport her in a supine position to the maternity hospital. In the maternity hospital, in the emergency room, blood group, Rh factor, and hemoglobin are determined again. An anesthesiologist is invited, the operating team is alerted, and only after that is an internal examination performed to determine the cause of genital tract bleeding.

Tactics of pregnancy and delivery in the second half of pregnancy

If a pregnant woman presents at the end of the second or third trimester with complaints of bloody genital discharge, the doctor must immediately send her to the maternity hospital, personally accompanying her in a supine position.

For complete, incomplete, and lateral placenta previa without bleeding, planned delivery by cesarean section is indicated at 37 weeks or more.

With heavy bleeding, regardless of the degree of placenta previa and regardless of gestational age, operative delivery by cesarean section is indicated. If the placenta is localized on the posterior uterine wall, a transverse incision in the lower uterine segment should be used. In partial (marginal) placenta previa, one may wait for spontaneous labor onset, then perform amniotomy.

During cesarean section for placenta previa, some authors also recommend citrate-free plasma for hemostatic purposes, and in severe blood loss, red blood cells as well. We consider these recommendations absolutely correct and fully justified.

In addition, the following are used:

- aminocaproic acid 100 ml IV once daily for 3–4 days;
- aminomethylbenzoic acid 50–100 mg or 100 ml IM once daily for 4–6 days;
- 10% calcium gluconate solution, 5–10 ml IV 2–3 times daily until bleeding stops;
- etamsylate IV 250 mg 3 times daily until bleeding stops;
- tranexamic acid IV 1000 mcg.

To prevent bleeding in the postoperative period, uterotonic drugs are indicated:

- methylergometrine 0.02% solution, 2 ml in 10 ml 5% glucose solution or 0.9% sodium chloride solution IV;
- oxytocin IV, 15–20 units in 500 ml 0.9% sodium chloride solution, infused once daily for 1–2 days.

Because the risk of purulent-inflammatory diseases is high, after fetal extraction and umbilical cord clamping, IV drip administration of 2 g ceftriaxone or cefepime is indicated, followed by antibacterial therapy in the postoperative period:

- cefazolin IV 1 g 3 times daily or claforan IM 1 g 3 times daily for 3–5 days;
- gentamicin 80 mg IM 2 times daily for 3–5 days;
- according to indications, doxylan 100 mg, 1 tablet, 2 times daily for 5 days;
- Metrogyl 100 ml IV drip 2 times daily for 3 days.

Treatment is considered effective if bleeding can be stopped and the life and health of the mother and her child can be preserved. Complications, side effects, and physician errors are considered unjustified.

Treatment of placenta previa

Bleeding from placenta previa requires emergency care. All actions of medical personnel (in clinic, hospital, by physicians) must be rapid and precise.

With delayed initiation of treatment or transport of a pregnant woman, or with inadequate hemostatic and blood replacement therapy, there is a high risk of developing hemorrhagic shock and disseminated intravascular coagulation (DIC syndrome) due to possible massive bleeding. This increases risk to the life of both mother and fetus. In practice, the most common error is delayed initiation of adequate hemostatic, surgical, and blood replacement therapy.

At 18–24 weeks of pregnancy, in addition to the above drugs, the following tocolytics may be prescribed:

- hexoprenaline IV 0.005 mg in 500 ml of 0.9% sodium chloride solution or 500 ml of 5–10% glucose/dextrose until uterine contractions are suppressed, then 0.5 mg orally 4–6 times daily for 2–3 weeks;
- fenoterol IV (15–20 drops per minute), 0.5 mg in 500 ml of 0.9% sodium chloride solution or 500 ml of 5–10% glucose/dextrose until uterine contractions are suppressed, then orally 5 mg 4–6 times daily for 3–4 weeks.

For prevention and treatment of anemia in pregnant women, the following are prescribed:

- ferretab containing iron fumarate and folic acid as active substances. Taken orally, 1 capsule daily for 4 weeks.

- Vitamins B1 100 mg, B6 200 mg, and B12 200 mcg: 1 tablet 3 times daily. In early pregnancy, for prevention of respiratory distress syndrome, large doses of glucocorticoid steroids should be used, specifically dexamethasone 8 mg IM every 12 hours, total dose 24 mg.

Intraoperative stage

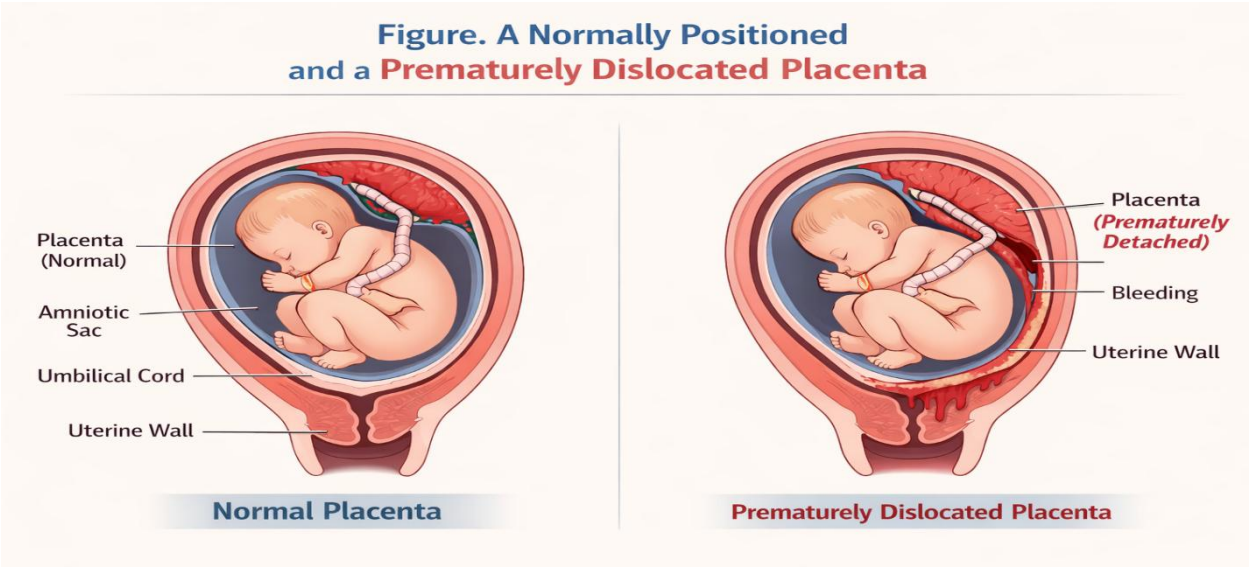
If bladder wall injury is suspected, cystoscopy and ureteral catheterization are performed. Perform normovolemic hemodilution.
Anesthesia options – spinal anesthesia 16%, intubation anesthesia 84%.
Removal of the uterus with fetus, determining the method of uterine incision according to the size of the "uterine hernia."
Corporal – 30%, lower – 43%, lower segment – 27%.
Fetal extraction.
Application of clamps or ligatures to the edges of the uterine incision.

Consequences of placenta previa

The prognosis depends on pregnancy duration and timely medical care. During cesarean section in pregnant women with placenta previa, bleeding increases due to poor contraction of the lower segment. Because the lower segment area is insufficiently developed, placenta previa is often associated with firm attachment and sometimes invasion. In such cases, the uterus should be removed without delay.

V. PREMATURE ABRUPTION OF A NORMALLY LOCATED PLCENTA

Premature abruption of a normally located placenta *(separatio placentae normaliter insertae spontanea, NORMAL JOYLASHGAN YO`LDOSH MUDDATIDAN OLDIN AJRALISHI)* is the separation of the placenta before fetal birth, during pregnancy or labor (usually in the first stage). This pathology is dangerous for the woman's health and sometimes life, and is very dangerous for the fetus.



During pregnancy and labor, due to its soft and elastic structure, the placenta easily adapts to changes in intrauterine pressure and to the pressure of the uterine wall muscles with which it is closely connected. Pressure of the uterine muscles on the placenta is compensated by intrauterine pressure, which prevents its separation. By balancing two forces acting in opposite directions, the connection between the placenta and uterine wall remains intact. In addition, preservation of placental attachment is supported by the considerable elasticity of placental tissue and by reduced contractility of the uterine wall in the placental zone during labor ("progesterone block"). Any disturbance in the connection between the placenta and uterine wall during pregnancy and labor is accompanied by bleeding.

Frequency. Premature separation of a normally located placenta ranges from 0.8% to 2.5%, although in practice it is more common. Physicians often do not pay sufficient attention to pain and bleeding in the second trimester, when early placental abruption may occur. Maternal mortality ranges from 2% to 15% and is due to consequences of this pathology—disseminated intravascular coagulation syndrome, followed by multiple organ and multisystem failure (acute renal failure in the anuric stage, cerebral edema, pulmonary edema, etc.). In premature placental abruption, perinatal mortality is 25–45%, due to hypoxia and fetal immaturity.

Etiology and pathogenesis

The main cause of premature abruption of a normally located placenta cannot always be identified.

What are the risk factors for placental abruption?

A number of clinical and epidemiological studies have identified risk factors. The most predictive is prior abruption in a previous pregnancy. A large observational study in Norway reported a recurrence rate of 4.4% (adjusted OR 7.8, 95% CI 6.5–9.2). Abruption recurs in 19–25% of women whose previous two pregnancies were complicated by abruption. Other risk factors include: preeclampsia, fetal growth restriction, advanced maternal age, multiple pregnancy, low body mass index, pregnancy after assisted reproductive technology, premature rupture of membranes, abdominal trauma (both accidental and related to domestic violence during pregnancy), smoking, and drug abuse (cocaine and amphetamines).

First-trimester bleeding increases the risk of abruption later in pregnancy. A retrospective study in Denmark showed that threatened pregnancy increased the risk of placental abruption from 1.0% to 1.4%. A systematic review showed that first-trimester bleeding is associated with increased risk of placental abruption; when retroplacental hematoma is detected by ultrasound in the first trimester, the risk of subsequent abruption increases.

Etiological factors include hypertension in pregnancy, gestosis (especially severe forms), pyelonephritis, allergic conditions, blood disorders (thrombocytopenia), and hypertension associated with very young or advanced maternal age in primigravidas. Placental abruption may also occur in multiparous women, with uterine developmental anomalies and tumors, polyhydramnios, autoimmune conditions (lupus antigen), etc.

One of the main causes of premature separation of a normally located placenta is preeclampsia, especially severe forms (12.6–90%).
Maternal hypertension plays a major role in placental abruption, where reduced intravascular blood flow and, in many cases, chronic vascular disease are present; under such conditions, abruption manifests in severe form. Reduced platelet activity also contributes to abruption. External trauma, a short umbilical cord, uterine anomalies, or tumors may also contribute.
Autoimmune conditions associated with hemostatic disorders and development of destructive placental changes (antiphospholipid syndrome, presence of lupus antigen, etc.) deserve special attention; in this case, the antigen-antibody reaction plays an important role.
The risk of abruption is high in subsequent pregnancies. According to some authors, recurrent placental abruption is observed in 1 out of 6–8 women in subsequent pregnancies. The timing of placental abruption is difficult to predict. Cases with normal non-stress and normal contractile tests have been described up to 4 hours before onset of abruption and rapid fetal death.
Placental abruption should not be viewed only as an acute pathology. It is the final stage of severe, often clinically unrecognized pathological conditions.
Premature placental separation is the result of vasculopathy, increased capillary permeability and fragility, and microcirculatory disorders. Similar vascular changes occur in gestosis, pyelonephritis, hypertension, etc.
Early placental abruption is more often observed in multiparous women, which is associated with changes in the uterine mucosa.
Many believe placental abruption occurs as a result of gross placental bed changes in severe gestosis, nephritis, hypertension, and heart defects.
We emphasize that the leading pathogenetic factor in premature placental separation is disseminated intravascular coagulation. Most consider that coagulopathy during early placental abruption develops through a thrombohemorrhagic mechanism, in which two phases should be distinguished: first—an early phase of sharp increase in intravascular coagulation due to entry of thromboplastic substances into the bloodstream, leading to defibrination; second—a late phase characterized by increased anticoagulant blood activity. In this phase, lysis of the formed fibrin occurs. In defibrination during early placental abruption, mobilization of fibrin in the retroplacental hematoma area and major blood loss also play a known role.
Placental abruption begins with hemorrhage into the decidua basalis. The decidua then ruptures, leaving a thin layer over the myometrium. Subsequently, a hematoma forms in decidual tissue, leading to placental separation adjacent to this area, compression, and destruction. In the early stage, clinical signs may be absent. After delivery, when examining the maternal placental surface, changes may be identified as impressions several centimeters in diameter covered with a dark blood clot.
In some cases, decidual arteries rupture, leading to formation of a retroplacental hematoma, which disrupts vessel integrity, separates the placenta, and increases bleeding. The abruption area grows rapidly and may reach the placental edge. As the uterus is stretched by expansion of the hematoma, its contractile ability decreases, and ruptured vessels at the placental attachment site are not compressed.

Accumulated blood may separate membranes from the uterine wall and flow outward, or may collect in the uterus as a retroplacental hematoma. In some cases, blood penetrates into the myometrial thickness and reaches the serosal membrane; less often, it enters between leaves of the broad ligament, into the ovaries, and even into the free abdominal cavity. This pathological condition is called **utero-placental apoplexy**, first described by **A. Couvelaire in 1911**.

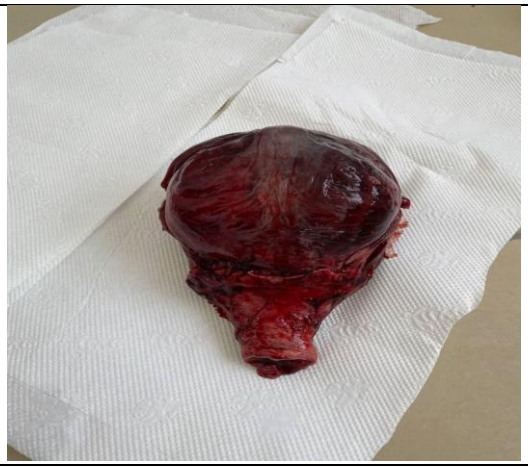


Figure 15. Uterine apoplexy (Couvelaire uterus) in premature separation of a normally located placenta (massive hemorrhages into the uterine wall and beneath the peritoneum).

In this pathological condition, uterine contractility is often impaired, and severe bleeding is observed in the postpartum period as a manifestation of disseminated intravascular coagulation (DIC syndrome).
In placental abruption, if placental integrity is preserved (20%) or damaged (30%), fetal blood may enter the maternal bloodstream (fetomaternal hemorrhage), which can be detected by examining the maternal blood smear (Kleihauer cells).
Despite numerous experimental and clinical studies, the pathogenesis of placental abruption cannot be considered fully elucidated. Most authors dealing with this problem reasonably believe that the most important factors in the pathogenesis are hemodynamic and microcirculatory disturbances in the utero-placental circulation, resulting in impairment of the most important functions of the entire fetoplacental system.
In conclusion, it should be emphasized that premature detachment of a normally located placenta cannot be considered, as it is often interpreted by obstetricians, an acute, sudden coagulopathic catastrophe. Placental abruption is the result of the influence of long-standing background and newly emerging pathogenetic factors.

Risk factors:
¬ Arterial hypertension associated with pregnancy
¬ History of multiple deliveries
¬ Short umbilical cord
¬ Combination of arterial hypertension and history of 3 (or more) deliveries
¬ History of placental abruption
¬ Premature rupture of membranes
¬ Smoking (especially more than one pack per day)

¬ Alcohol abuse and drug use
¬ Maternal age over 35 years
¬ Polyhydramnios and multiple pregnancy
Classification of Premature Detachment of a Normally Located Placenta (PDNLP)

In classification, it is considered appropriate to base it on the presence or absence of external bleeding caused by the type of abruption (E.A. Chernukha, 2003).
In this case, the following forms are distinguished:

1. External or visible bleeding, in which blood flows from the vagina;
2. Internal or concealed bleeding, in which blood is located between the placenta and the uterine wall (retroplacental hematoma);
3. Combined or mixed bleeding, in which both visible and concealed bleeding are present.

Most obstetricians distinguish two types of bleeding — visible and concealed.
According to the severity of the clinical presentation, mild and severe forms of premature placental abruption are distinguished.
There are partial (progressive and non-progressive) and complete detachments of a normally located placenta. Non-progressive abruption is referred to by foreign authors as chronic when the retroplacental hematoma does not increase in size.

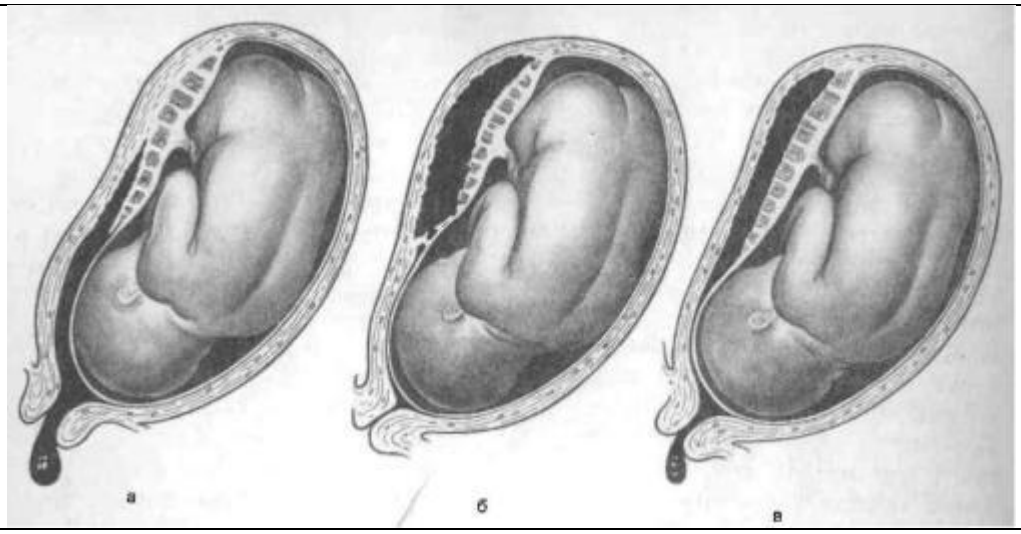


Figure 16. Variants of separation of a normally located placenta:
a) partial placental separation with external bleeding;
b) complete placental separation (retroplacental hematoma, internal bleeding);
c) complete placental separation with both internal and external bleeding.

Clinical presentation The main signs of abruption are **bleeding** and **pain**. Their severity varies widely: sometimes they are barely noticeable or may even be absent. In other cases, these symptoms are so pronounced that even one of them is sufficient

to make the diagnosis and conclude that there is a threat to the life of both mother and fetus.
Mild abruption often does not manifest clinically and is detected only after delivery when examining the maternal placental surface (small blood clots are found).
With internal bleeding (retroplacental hematoma), diagnosis is much more difficult than with external bleeding. In such cases, the main symptoms are pain and fetal hypoxia. In combined bleeding, the severity of internal bleeding cannot be judged by the intensity of external bleeding. According to experts, external bleeding may prevent further placental separation. If bleeding begins internally, it may remain concealed, or it may dissect membranes away from the uterine wall and then appear externally. Rarely, a Couvelaire uterus forms. Very rarely, with widespread subserous hematomas, peritoneal integrity is disrupted and blood flows into the abdominal cavity.
Retroplacental hematomas may be single or multiple. The degree of bleeding depends on placental separation site (more pronounced with central separation), area of separation, vessel involvement, and coagulation status.
If external bleeding appears soon after separation, the blood is usually bright red. If some time passes between separation and blood appearance, the blood is darker.
In premature separation of a normally located placenta, external bleeding—unlike bleeding in placenta previa—does not increase during contractions; on the contrary, it stops**.**

Another major symptom is abdominal pain. During placental separation, pain occurs due to uterine wall stretching, blood infiltration of the uterine wall, and peritoneal irritation. Pain is especially pronounced in internal bleeding. However, there is no strict parallel between the degree of bleeding and pain intensity. Sometimes pain is so severe that it can only be compared with pain preceding uterine rupture or tubal rupture in ectopic pregnancy. Pain may radiate to the symphysis and thigh and is often prolonged. On objective examination, sharp tenderness is noted on abdominal palpation. Uterine contours are preserved, but uterine shape and size often change. Due to pain, fetal palpation is often difficult. Fetal motor activity may be increased or reduced; fetal heart rate is often rapid (tachycardia) and may frequently be inaudible. With slight blood loss, visible mucosa and skin are usually normal or mildly pale; pulse is rapid but remains well filled. With moderate blood loss, visible mucosa and skin become pale, skin is cool to touch, sweat appears on the forehead, and the whole body becomes clammy. Body temperature falls below normal. Pulse becomes rapid, weak, and tense. Blood pressure decreases. Breathing becomes rapid.
With massive blood loss, severe abdominal pain is often present and signs of shock are expressed to varying degrees. In such cases, skin and visible mucosa are markedly pale, pulse is rapid, and blood pressure is low. Dizziness, severe weakness, dyspnea, and depression occur.

If bleeding is not stopped in time and anemia/shock are not corrected, pulse becomes thready, yawning appears, sticky sweat covers the body, and complaints of visual disturbance, thirst, and vomiting occur. Later, the patient becomes apathetic,

indifferent to surroundings, gradually loses consciousness, then coma and death may follow.

In shock development during placental abruption, not only blood loss volume but also the amount of thromboplastin entering maternal circulation from the placenta plays an important role, causing intravascular coagulation and acute cor pulmonale. Experimental data showed that rapid IV administration of a large thromboplastin dose causes severe shock.

Signs of anemia and consequences do not always develop in parallel with blood loss. Sometimes a patient tolerates large blood loss relatively well; in others, even moderate bleeding causes a severe reaction.

Current classification

I. By severity:

- Mild placental abruption — separation of a small area
- Severe degree — separation of 1/3, 2/3, or complete placental separation

II. By bleeding pattern:

- Separation with external bleeding
- Separation with internal bleeding
- Separation with combined bleeding (both external and internal)

III. By area/progression:

- Partial separation — progressive or non-progressive
- Complete placental separation

In mild form, pain is absent, uterus fully relaxes between contractions, and fetal heart activity is not compromised. The only sign may be slight dark-red genital discharge. Diagnosis is usually made after placental examination, when small crater-like depressions filled with dark clots are found on the placental surface.

Severe form (acute placental insufficiency) occurs when 1/4–2/3 of placental surface or more than 2/3 is separated. Initial symptoms may develop gradually or suddenly, with persistent abdominal pain and subsequent dark genital bleeding. Sometimes severe shock signs are present. Uterine tone is increased diffusely or locally; complete relaxation between contractions is absent. The fetus suffers from hypoxia, and intrauterine death may occur. Due to marked uterine tone, fetal heart sounds are difficult to auscultate. Onset is usually sudden (acute abdominal pain, marked weakness, dizziness, often syncope). The patient is restless, moaning. Skin and visible mucosa are pale; face covered with cold sweat; respiration and pulse are rapid; pulse weak and tense. Blood pressure is reduced. Abdomen is markedly enlarged; uterus tense, with “local swelling”; fetal small parts and heart sounds are not detected. External genital bleeding may be absent or moderate.

In severe placental abruption, condition severity is determined not only by volume/rate of blood loss, but also by massive entry of active thromboplastins into maternal circulation.

In severe cases, renal failure may develop. Beyond major blood loss, this is primarily due to reduced renal perfusion from decreased cardiac output, intrarenal vasospasm, and hypovolemia. Renal failure may present as cortical glomerular necrosis. Cases requiring hemodialysis have been described. Renal dysfunction is often caused by delayed/inadequate treatment of hypovolemia. To prevent renal

dysfunction, timely administration of blood components, blood substitutes, and colloid/crystalloid solutions is required.

Diagnosis

During pregnancy, small abruption of a normally located placenta with mild uterine pain and without external bleeding can often only be suspected clinically unless special investigations (ultrasound, MRI) are used. Final diagnosis may be made after delivery when depressions and clots are identified on the maternal placental surface. During examination of pregnant women, premature separation of a normally located placenta must be differentiated from placenta previa manifestations, rupture of marginal placental sinus, rupture of umbilical vessels, uterine rupture, and others (Table 2).

Differential Diagnosis of Bleeding in Late Pregnancy

Clinical Signs	Placenta Previa	Premature Placental Abruption (Abruptio Placentae)	Vasa Previa
Volume of blood loss	Variable	Variable	Usually small
Duration of bleeding	Often stops within 1–2 hours	Usually prolonged	Stops within a short time
Abdominal pain	Absent	May be severe	Absent
Fetal heart rate	Normal	Tachycardia → bradycardia → possible antenatal fetal death	Tachycardia → bradycardia → possible antenatal fetal death
Coagulation disorders	Sometimes	Rare, but may occur; possible development of severe disseminated intravascular coagulation (DIC)	Not observed
Anamnesis features	Frequent abortions, postpartum/post-abortal infections, hypomenstrual syndrome, infertility	Abdominal trauma, maternal hypertension, multiple pregnancy, polyhydramnios, preeclampsia	None

With a clear clinical picture of placental abruption, diagnosis is established based on history (sudden onset, acute uterine pain, gestosis, hypertension, pyelonephritis, abdominal trauma, etc.) and objective findings. The uterus is tense, firm, asymmetric, and sharply tender in a specific area. Small fetal parts are not palpable. On auscultation, fetal tachycardia or bradycardia is noted; sometimes fetal heart sounds are absent. Usually, at the onset, there is no vaginal bloody discharge.

During pregnancy, vaginal examination shows the cervix preserved, external os closed, and presenting part high. For diagnosis, the type of placental separation is important. With marginal separation, external bleeding appears and is usually not accompanied by pain. With central separation and hematoma formation, external

bleeding may be absent even with severe pain. This is a very dangerous form, leading to rapid fetal death and severe hypovolemic disorders in the mother.
The classic clinical picture of placental abruption is observed in only 10% of women. In one-third of pregnant women, pain syndrome is absent, although it is an important diagnostic sign. The leading clinical signs of this pathology are bleeding and signs of fetal hypoxia. With placental abruption, increased or decreased fetal motor activity is often observed.

A clear clinical picture is characterized by changes in the hemostatic system. While physiological pregnancy is associated with hypercoagulation, in early placental abruption isocoagulation or hypocoagulation is detected due to consumption of clotting factors. At this stage, platelet count decreases, fibrinogen concentration falls, antithrombin III declines further, and fibrinogen degradation products increase.
With complete premature placental separation and fetal death, varying degrees of hypocoagulation are observed, and antithrombin III concentration may be three times higher than in uncomplicated pregnancy. Increased fibrinogen degradation products indicate consumption coagulopathy causing circulatory disorders and hemorrhagic shock.
Even in mild placental abruption, thromboplastic substances of tissue and cellular origin enter maternal circulation, resulting in hyperbilirubinemia and intravascular coagulation. The scale of this process depends on the size of placental separation and its duration.
The most important indicator of hemostatic system function in placental abruption is the degree of intravascular conversion of fibrinogen to fibrin. This can be most accurately assessed by tracking antithrombin III (heparin cofactor, natural anticoagulant) dynamics and measuring fibrinogen degradation products in serum. Decreased antithrombin III in women with placental abruption indicates reduced total plasma anticoagulant activity due to intensive antithrombin III consumption during activation of clotting factors (XII, XI, IX, VII, V).
It has been shown that concentrations of soluble fibrin-monomer complexes and fibrinogen degradation products are directly related to severity of placental separation. Thus, with complete separation, fibrinogen degradation product concentration is five times higher than in uncomplicated pregnancy. Severity of coagulopathy also depends on gestational age at onset of abruption.
In placental abruption, the main mechanism of coagulation defects is almost certainly initiation of intravascular coagulation and, to a lesser extent, retroplacental coagulation. Although significant fibrin is present in the uterus in severe abruption and hypofibrinogenemia, it is insufficient to account for blood levels. It has been shown that fibrinogen breakdown in peripheral blood serum is higher than in serum from blood located within the uterine cavity.
When abruption occurs in the first stage of labor, the amniotic sac is usually tense; sometimes moderate bloody uterine discharge with clots appears. After membrane rupture, amniotic fluid is markedly blood-stained. Detection of placental tissue on vaginal examination indicates placenta previa. In placental abruption, signs of

anemia appear early: pallor of mucous membranes and skin, tachycardia, and later decreased blood pressure.
Ultrasound is an important aid in diagnosing premature separation of a normally located placenta, especially concealed bleeding. Longitudinal and transverse scanning allow detection of separation site, retroplacental hematoma size, and structure. With marginal separation and external bleeding, ultrasound may fail to detect pathology.
Clinical diagnosis is confirmed by ultrasound in only 25% of cases; therefore, negative ultrasound findings do not exclude life-threatening placental abruption. It is more often observed when the placenta extends to one lateral uterine wall or is located on the anterior wall. When localized in the fundal region, separation is less common.
If the clinical picture is not clearly expressed, diagnosis is made by excluding other diseases.

Clinical recommendations

Therapy of premature separation of a normally located placenta should be aimed at treatment of causative factors, reduction of uterine tone, correction of hemostasis, and management of anemia and shock. Choice of treatment method depends on volume and severity of blood loss, general condition of pregnant/laboring woman, fetal condition, timing of bleeding (pregnancy vs labor), bleeding type (concealed/external), birth canal status (degree of cervical dilatation), and hemostatic status. The main criteria in choosing treatment are severity of bleeding and condition of mother and fetus. An important factor is timing of separation—during pregnancy or labor. In cases occurring during pregnancy, considering maternal and fetal condition, when there are no conditions for rapid vaginal delivery, conservative management may be attempted or delivery may be performed by cesarean section. Pregnant women with suspected early placental abruption should be hospitalized in a maternity unit, preferably where anesthesiology-resuscitation services, operating surgeons, laboratory services, organized blood service, and operative capacity are available.
The main task is timely control of uterine bleeding.

Management tactics at 34–36 weeks in Premature separation of a normally located placenta

During pregnancy (up to 34–36 weeks), in mild placental abruption**,** if the condition of the pregnant woman and fetus is not significantly worsened and there is no significant external or internal bleeding, conservative management may be used:

- bed rest
- ultrasound monitoring
- cardiotocography
- coagulation monitoring
- mild antispasmodics
- multivitamins

- iron therapy, etc.

Examples of therapy used:

- **Magne B6**: 2 tablets twice daily for 1 month
- **12.5% sodium etamsylate solution**: 4 ml IV or IM, then 2 ml 4 times daily for 7–10 days
- **Drotaverine 2% solution**: 4 ml IM, then after 4 hours 2 ml IM, 4–6 times daily for 7–10 days
- **Dicynone**: 4 ml IV, then 2 ml IM 3–4 times daily
- multivitamins, iron and zinc preparations (zinc sulfate solution, 1 tablespoon 4–6 times daily)

ß-mimetics are contraindicated in placental abruption, because they relax the uterus, reduce pain, may promote further separation, cause vasodilation, and negatively affect maternal cardiac function.

If antiphospholipid syndrome, lupus, thrombocytopenia, or other blood disorders are present, hemostatic parameters must be closely monitored and appropriate treatment provided.

Evaluation for signs of early placental abruption includes:

- blood tests
- urine tests
- biochemical blood tests
- blood electrolytes
- hemostasiogram
- ultrasound
- CTG
- Doppler assessment, etc.

The prognosis of premature placental separation is difficult. Progression from mild to severe form is always possible. Special caution is needed with recurrent small bleeding episodes indicating progression. In such cases, even if maternal status is satisfactory, the issue of abdominal delivery should be raised.

If the clinical picture during pregnancy is severe (bleeding, fetal hypoxia, severe pain), urgent cesarean delivery is recommended. If uterine apoplexy (Couvelaire uterus) is found during cesarean section, hysterectomy without adnexa is performed; if adnexa are also blood-infiltrated, hysterectomy with adnexa may be required due to risk of postoperative bleeding from uterine hypotonia and coagulopathy in DVS syndrome.

In such cases, it may be preferable to perform surgery with ligation of the internal iliac arteries, which can significantly reduce pelvic blood flow within 1.5–2 hours.

Management tactics in normal placental abruption

If the woman is in the first stage of labor, and there is no major bleeding, and maternal/fetal conditions are satisfactory, amniotomy and conservative labor management under continuous monitoring are possible.

Labor stimulation is not recommended**;** oxytocin is not advised for labor augmentation in this context. Increased uterine contractile force may promote thromboplastin entry into maternal circulation, trigger/worsen coagulopathy, and may contribute to amniotic fluid embolism. If, during monitoring, bleeding

increases, uterine tension rises, fetal distress signs appear (decelerations, etc.), and rapid vaginal birth is not feasible, cesarean section is recommended.
In the second stage of labor**,** the key sign of placental abruption is acute fetal hypoxia. If conditions are present (full cervical dilation, fetal head in pelvis), urgent delivery is performed with obstetric forceps.
In severe abruption, the placenta is often delivered immediately after fetal birth, with large amounts of blood and clots. In partial separation, manual placental removal and inspection of placental integrity with uterine cavity/wall control are performed.
After vaginal birth, prophylactic uterotonics are indicated in the postpartum and early postpartum periods. Postpartum and early postpartum hemorrhage occurs in 5.0–13.1% of cases.
With clear clinical deterioration of the mother (shock) and fetus, the only correct delivery method is cesarean section**.**
In the early postoperative period, uterotonics and hemostasiogram monitoring are indicated to prevent bleeding.
To restore blood loss, transfusion is used (packed RBCs), fresh frozen plasma, blood substitutes, colloids, etc. In bleeding, blood and crystalloids are administered to maintain hematocrit around 30% or slightly higher, and urine output at least 30 ml/h (preferably 60 ml/h). Furosemide should not be used for oliguria**.** If intensive infusion does not correct oliguria, central venous pressure should be assessed. If early pulmonary congestion cannot be detected directly, monitor for dyspnea, cough, and crackles. Furosemide is indicated when pulmonary congestion is present.
In newborns, trauma, prematurity, hypoxia, and sepsis predispose to intravascular coagulopathy.
After vaginal birth, bleeding often results from coagulation disorders and impaired uterine contractility. To stop bleeding, IV uterotonics (oxytocin, prostaglandins) are recommended. To exclude traumatic causes, manual uterine examination and inspection of cervix and birth canal are necessary.
For coagulopathic bleeding, effective therapy includes IV fresh frozen plasma, fresh donor blood, tranexamic acid, fibrinogen, ethoxylated starch, and cryoprecipitate. In thrombocytopenia, platelet transfusion is indicated to increase platelets to 50,000. After delivery, coagulation defects may recover within 24 hours; platelets usually normalize within 2–4 days.
Maternal mortality in premature separation of a normally located placenta is 1.6–15.6%. Main causes of death are shock and bleeding.
Perinatal mortality is 20.0–35.0%, due to intrauterine hypoxia, fetal immaturity, and depending on abruption extent and bleeding severity. Many newborns have neurological injury.
In postpartum and early postpartum periods after vaginal birth, uterotonics are used prophylactically (oxytocin 40 IU, methylergometrine 2 ml IV).
Currently, plasma substitutes are widely used—aminoselmin, infuzol, refortan, stabizol, etc. Usual daily IV dose is at least 1000–1200 ml. In Premature separation of a normally located placenta transfusion therapy and resuscitation

in pregnant, laboring, and postpartum women should be coordinated with a general practitioner and especially an anesthesiologist-resuscitator.

Postpartum and postoperative therapy

If postpartum or intraoperative bleeding continues, the following blood products may be used:

- cryoprecipitate 300–400 ml IV
- aprotinin IV drip 50,000–100,000 units up to 5 times/day
- hydroxyethyl starch derivatives
- non-citrated plasma 150–200 ml
- fibrinogen IV infusion 1–2 times/day
- platelet mass
- intensive therapy continued until bleeding stops

Surgical management

Indications for surgery include severe placental abruption and progressive partial abruption. Cesarean section is performed. If uterine imbibition areas are present, hysterectomy is performed without delay. Unjustified conservative treatment can lead to hemostatic failure, DVS syndrome, and hemorrhagic shock.

Some authors recommend internal iliac artery ligation before hysterectomy to reduce intraoperative blood loss. In severe abruption with complete uterine paralysis, **Porro operation** is recommended (removal of the uterus with fetus without opening the uterine cavity), reducing additional blood loss.

Surgical treatment for women with early abruption should be done jointly with anesthesiologist-resuscitator, transfusiologist, and therapist. Their tasks include:

- complete analgesia/anesthesia
- support of macro- and microcirculation (controlled hemodilution with plasma substitutes, dextrans, glucose, etc.)
- correction of metabolic acidosis
- glucocorticoid administration
- adequate diuresis
- mechanical ventilation
- DVS syndrome treatment

Postoperative intensive therapy should continue for at least 5–6 days under clinical and laboratory monitoring.

Treatment effectiveness in premature separation of a normally located placenta is based on: reducing uterine tone during pregnancy, stopping bleeding, normalizing fetal condition, and timely delivery from the uterus.

Complications: development of DVS syndrome and multiple organ failure.

Prevention: timely diagnosis and treatment of gestosis, gestational hypertension, kidney disease, antiphospholipid syndrome, and other conditions that contribute to premature separation of a normally located placenta

VI. BLEEDING DURING LABOR AND THE EARLY POSTPARTUM PERIOD

Among various obstetric complications occurring during labor and the early postpartum period, bleeding continues to occupy one of the leading positions. It is customary to distinguish between bleeding during labor and bleeding in the early postpartum period. Postpartum hemorrhage is not a diagnosis, but a symptom.

Each year, about 140,000 women die worldwide from postpartum hemorrhage—one woman every 4 minutes. In the Russian Federation, bleeding during pregnancy, labor, and the postpartum period ranks first among causes of maternal mortality (about 17%); in Uzbekistan, this figure is 38–40%. Most often, fatal hemorrhage occurs against the background of gestosis (58%) and extragenital pathology (59%). Thus, when combined with preeclampsia, severe hypotonic bleeding causes maternal death in 36 cases; when combined with hypertensive disease and somatic pathology, in 49%.

Assessment of the quality of medical care shows that the main factors leading to death in massive obstetric hemorrhage are: insufficient examination, inadequate assessment of women's condition during labor and the postpartum period, and delayed/incomplete intensive therapy. In this regard, prevention and intensive treatment of hemorrhage remain urgent issues in modern obstetrics.

Thus, the cause of bleeding during labor and in the early postpartum period is a set of factors that disrupt the physiological processes of uterine contraction and clot formation in placental vessels; therefore, pathological postpartum blood loss is any blood loss exceeding 0.5% of body weight.

The causes of postpartum hemorrhage may be grouped into four main etiological processes known as the **"4Ts"** (Clinical guideline for management of patients with bleeding during labor and the postpartum period, Tashkent, 2007).

Table 3

Etiological Risk Factors of Postpartum Hemorrhage

T Category	Etiological Process	Clinical Risk Factors
T – Tone (uterine contractility disorder)	Overdistension of the uterus; exhaustion of myometrial contractility; infectious process; functional/anatomical uterine features	Polyhydramnios; Multiple pregnancy; Fetal macrosomia; Rapid labor; Prolonged labor; Grand multiparity (>5 deliveries); Chorioamnionitis; Intrapartum fever; Uterine fibroids; Placenta previa
T – Tissue (retained intrauterine tissue)	Retained placental fragments; Retained blood clots in the uterine cavity	Placental defect; Previous uterine surgery; History of multiple deliveries; Abnormally adherent placenta; Placenta accreta; Uterine hypotonia
T – Trauma (birth canal injury)	Cervical, vaginal, and perineal lacerations; Traumatic uterine rupture during cesarean section; Uterine rupture; Uterine inversion	Rapid labor; Operative vaginal delivery; Malpresentation; Low presenting part; Previous uterine surgery; History of multiple

T Category	Etiological Process	Clinical Risk Factors
		deliveries; Low placental implantation
T – Thrombin (coagulation disorders)	Congenital disorders (Hemophilia A, von Willebrand disease); Acquired during pregnancy: idiopathic thrombocytopenia; Thrombocytopenia associated with preeclampsia; Disseminated intravascular coagulation (preeclampsia, antenatal fetal death, severe infection, amniotic fluid embolism); Anticoagulant therapy	Hereditary coagulopathies; Liver diseases; Hematomas and/or bleeding (including at injection sites); Preeclampsia, eclampsia, HELLP syndrome; Antenatal fetal death; Antepartum hemorrhage; Failure of clot formation

Most obstetric hemorrhages occur during labor and in the early postpartum period. The hemochorial type of placentation predetermines a certain amount of blood loss in the third stage of labor after placental separation. This volume, programmed by pregnancy itself, corresponds to the volume of the intervillous space and does not exceed 300–400 ml (taking placental site thrombosis into account, external blood loss is 250–300 ml), or 0.5% of the woman's body weight. From the moment the uteroplacental circulation is established, its volume is not directly used to meet maternal needs, and its loss in the third stage of labor does not affect the postpartum woman's condition. Therefore, obstetrics uses the concept of **"physiological blood loss."**

At the same time, after placental separation, the placental bed is exposed—a large, richly vascularized wound surface. About 150–200 spiral arteries open into the placental area; their terminal segments lack a muscular layer, creating a risk of rapid massive blood loss.

Blood loss of **500 ml or more** is considered hemorrhage. The maternal response to blood loss depends on many factors: general condition, presence and severity of anemia, body weight, etc. According to many authors, the frequency of blood loss over 500 ml ranges from 5% to 15%.

In obstetric practice, the following categories of blood loss are distinguished:

1. **Physiological blood loss** — up to 6–8% of BCC, or 0.3–0.5% of body weight, or 250–400 ml.
2. **Pathological blood loss** — 15% or more of BCC, or 0.5–1.5% of body weight, or 500–900 ml.
3. **Massive blood loss** — more than 20% of BCC, or more than 1.5% of body weight, or over 1000–1200 ml.

Predisposing factors for bleeding in the postpartum and early postpartum periods include prior abortions, multiple pregnancy, uterine scar, uterine tumors and anomalies, severe gestosis, obesity, multiple gestation (twins), large fetus, polyhydramnios, severe maternal extragenital diseases, tocolytic use, stillbirth, and hemostatic defects (e.g., von Willebrand disease).

Intrapartum risk factors include prolonged or precipitous labor, operative delivery, and improper (overly aggressive) management of the third stage of labor (rough manual techniques, uterine massage, attempts to separate an unseparated placenta).

Postpartum pathology

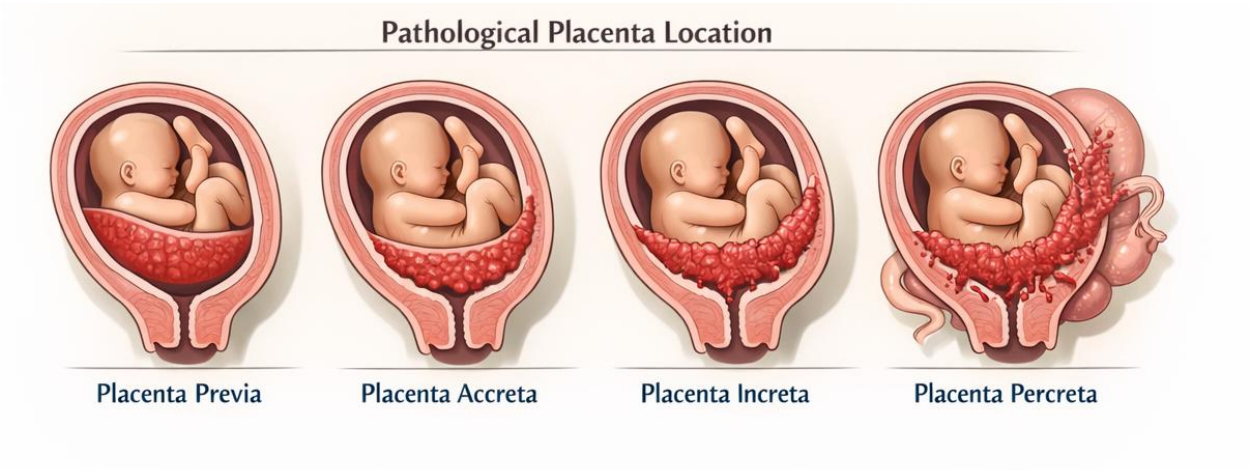
Most often, the physiological process of the third stage of labor is disturbed, presenting as delayed placental separation and bleeding in the afterbirth period.
After fetal delivery, the leading pathogenetic mechanism is impaired uterine contraction due to retained placenta. Therefore, the main treatment is manual placental separation and uterine emptying by removal of retained placental tissue. In hemorrhage, rational management of the third stage of labor is crucial. Usually, it lasts 5 to 15 minutes. If longer, it should be considered prolonged, indicating potential complications.

There are active and expectant approaches to third-stage management. In our republic, active management of the postpartum period is adopted (Order No. 500).
Delayed placental separation is observed in uterine hypotonia, complete dense placental attachment or invasion, and abnormalities of placental structure/localization (attachment in the lower uterine segment or uterine angles).

In uterine hypo-/atony, due to insufficient myometrial contractility, the placenta does not separate from the placental site. Reduced myometrial contractility may be congenital or acquired and may result from hormonal dysfunction, uterine malformations, rapid operative uterine emptying, prolonged labor, uterine overdistension (polyhydramnios, large fetus, multiple pregnancy), and uterine fibroids. In uterine hypotonia, after fetal birth, postpartum contractions may remain weak or absent for a long time.

VII. PATHOLOGICAL PLACENTAL ATTACHMENT

In delayed placental separation, various forms of pathological placental attachment to the uterine wall play a special role: dense attachment (placenta adhaerens) and true invasion (placenta accreta/increta/percreta)**.** Placental invasion may be complete (along the entire placental bed) or partial.



The most common form of pathological placental attachment occurs when pathological changes are present in the spongy layer of the decidual membrane, in which case, during physiological labor, the placenta separates from the uterine wall.In some cases, the changes in the decidual membrane are so markedly expressed

that the compact layer does not develop, the spongy and basal layers are atrophied, and the Nitabuch layer (zone of fibrinoid degeneration) is absent. Under such conditions, one or more cotyledons of the placenta directly adhere to the muscular layer of the uterus or sometimes penetrate into its thickness. In this case, true placental invasion develops, which occurs in approximately 1 in 10,000–15,000 deliveries.The causes of dystrophic processes in the uterine mucosa that contribute to dense placental attachment or invasion include postpartum and post-abortion septic processes (atrophic endometritis), specific endometrial lesions (gonorrhea, tuberculosis), excessive curettage, and the presence of postoperative scars, for example after conservative myomectomy or cesarean section. The likelihood of placenta accreta increases if it is located in the area of a postoperative scar or in areas less suitable for implantation of the ovum, such as the lower uterine segment (placenta previa), as well as in cases of uterine malformations (bicornuate uterus) or uterine neoplasms (submucosal myomatous nodes). Placenta accreta may also occur due to decreased enzymatic activity of the basal layer of the endometrium, which normally prevents chorionic villi from penetrating into the uterine muscle. The diagnosis of forms of pathological placental attachment can only be established during manual examination of the uterus for placental separation. In cases of placenta adhaerens, as a rule, all parts of the placenta can be removed manually. In placenta accreta/increta/percreta, it is impossible to separate the placenta from the uterine wall without compromising uterine integrity. Often, true placental invasion is confirmed by pathomorphological and histological examination after hysterectomy performed due to uterine atony and massive postpartum hemorrhage.Clinical presentation. Despite various causes of delayed placental separation, the clinical course is characterized by common symptoms — absence of signs of placental separation for 15 minutes or more, as well as bleeding. At the same time, the uterus is palpated as a large, round formation of uniformly firm consistency. The general condition of the woman in labor remains unchanged.The main protocol for the management of postpartum hemorrhage includes conservative and surgical stages of bleeding control. However, we should not forget the initial stage, which includes 10 main steps. Preparation for the management of postpartum hemorrhage is of great importance (Clinical guidelines on the management of bleeding during labor and the postpartum period, Tashkent 2007; National Standards for Medical Care in Maternity Institutions, 2015; Collection of Clinical Protocols, 2019). The main components of preparation for assistance can be expressed as follows:

• Mobilization of available personnel. Without leaving the patient unattended, call an experienced obstetrician-gynecologist fully proficient in surgical techniques, an anesthesiologist-resuscitator, and a laboratory specialist.

•Preliminary assessment of blood loss volume: visually or using a special kidney-shaped container + 20% (when the patient is transported by ambulance, if there is no severe preeclampsia, the shock index may be used).

•Initial assessment of the condition and monitoring of vital functions (blood pressure, pulse, temperature, respiratory rate).

•Depending on the severity of the woman's condition, determine where assistance will be provided (minor or major operating room) and ensure patient transfer.
•If the patient's condition allows, collect an allergy history.
•Catheterization of the bladder (the bladder must be empty).
•If necessary, determine blood group and Rh factor; take blood for compatibility testing ("bedside test") if required.
•If necessary, check the availability of blood products (according to blood group and Rh factor) and place an order.
•If possible, determine the cause of bleeding ("4T": tone, tissue, trauma, thrombin).
Management in cases of delayed placental separation without genital bleeding.
The woman in labor must not be transferred. Separation and removal of the placenta should be performed at the place where delivery occurred.

1. Catheterization of the bladder, after which uterine contraction and placental separation may occur promptly.
2. Administration of uterotonic drugs 15 minutes after fetal expulsion (oxytocin 5 IU intravenously in 500 ml of 5% glucose solution, or 0.5 ml intramuscularly twice after 15 minutes, or 2 ml of methylergometrine after 15–20 minutes) to enhance uterine contractility.
3. Venipuncture or catheterization of the cubital vein and intravenous administration of crystalloids to adequately compensate for possible blood loss.
4. If signs of placental separation appear, remove the separated placenta using one of the methods (Abuladze, Genter, Crede-Lazarevich).
5. If within 20–30 minutes there are no signs of placental separation despite administration of contractile agents, manual separation and removal of the placenta should be performed under adequate anesthesia.

After separation and removal of the placenta from the uterus, its internal walls should be carefully examined to identify additional lobes, retained placental tissue, and membrane remnants. Blood clots are removed simultaneously. After placental removal, the uterus usually contracts and firmly grasps the hand. If uterine tone does not recover, additional uterotonics are administered and bimanual uterine massage is performed. Once uterine contraction is achieved, the hand is removed from the uterine cavity.

6. If true placental invasion is suspected, attempts at separation should be stopped and the uterus should be removed (amputation or hysterectomy). Excessive physical force during attempts at manual removal may result in massive bleeding and uterine perforation.

VIII. POSTPARTUM HEMORRHAGE

Postpartum hemorrhage, particularly early postpartum atonic bleeding, deserves special attention, as it is associated with impaired uterine contraction after the delivery of the fetus and placenta.
Uterine hypotonia refers to a significant decrease in uterine tone accompanied by weakened contractions. In this condition, the uterine muscles respond to various

stimuli; however, the intensity of the response does not correspond to the strength of the stimulus. Hypotonia is considered a reversible condition.

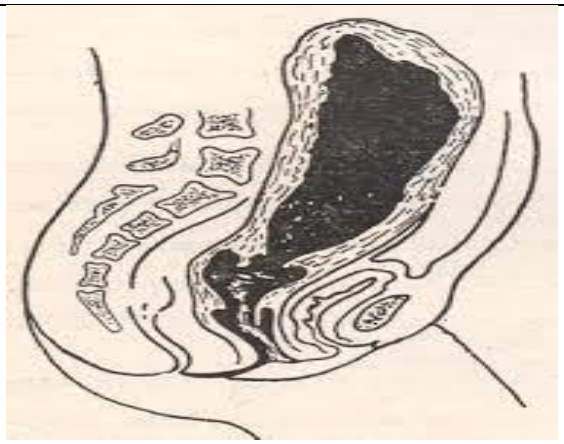


Figure 19. Uterine Atony After Placental Separation – Uterine Cavity Filled with Blood

Uterine Atony and Hypotonic Postpartum Hemorrhage

In uterine atony, the myometrium completely loses its tone and contractile ability. The uterine muscles do not respond to stimulation. A characteristic "paralysis" of the uterus develops. Although atony is relatively rare, it can be a source of massive hemorrhage.

There are two main groups of causes of hypotonic (atonic) hemorrhage:

1. Medical conditions leading to uterine hypotonia or atony:

- Diseases of the respiratory, cardiovascular, renal, and hepatic systems
- Dysfunction of hematopoietic organs
- Central nervous system disorders
- Pregnancy toxicosis
- Endocrine diseases
- Infections

2. Obstetric causes:

- Uterine overdistension (polyhydramnios, multiple pregnancy, fetal macrosomia)
- Uterine fibromyoma
- Abnormal placental attachment
- Premature separation of a normally located placenta
- Retained placenta
- Uterine scarring after surgery
- Mismanagement of labor

Additional contributing factors include rapid or excessively prolonged labor, premature rupture of membranes, and untimely medical interventions.

Severe forms of uterine hypotonia and massive hemorrhage are usually associated with hemostatic disorders, particularly disseminated intravascular coagulation (DIC syndrome). In this regard, bleeding that develops after various types of shock (toxic, painful, anaphylactic), amniotic fluid embolism, inferior vena cava compression

syndrome, or acid aspiration syndrome (Mendelson syndrome) occupies a special place. In these pathological conditions, uterine hypotonia may result from blockade of uterine contractile proteins by fibrin (fibrinogen) degradation products or amniotic fluid components.

Clinical Presentation

The main sign of uterine hypo-atony is bleeding. Blood may be discharged in clots of varying size or flow continuously. The bleeding may have a wave-like pattern, temporarily stopping and then recurring. Uterine contractions are infrequent and short-lived.

On examination, the uterus is enlarged, flaccid, and its upper border may reach or exceed the level of the umbilicus. External uterine massage may result in expulsion of clots and temporary restoration of tone; however, hypotonia often recurs.

Fractional blood loss of relatively moderate volume (150–300 mL) may allow temporary compensation for developing hypovolemia. Blood pressure may remain within normal limits. Pallor and tachycardia are observed.

If adequate treatment is not initiated in the early stages of uterine hypotonia, the severity of contractile dysfunction increases, therapeutic measures become ineffective, blood loss progresses, hemorrhagic shock develops, and DIC syndrome may occur.

In uterine atony, the uterus is soft, doughy, and poorly contoured. It appears diffusely enlarged within the abdominal cavity, and its fundus may reach the xiphoid process. Severe and persistent bleeding develops. Without timely intervention, clinical signs of hemorrhagic shock rapidly appear: pallor, tachycardia, hypotension, and cold extremities. The amount of blood lost does not always correlate directly with the clinical severity.

In clinical obstetric practice, differentiation between hypotonic and atonic bleeding is often conditional due to diagnostic complexity.

Diagnosis of hypotonic hemorrhage is based primarily on clinical findings.

DIC syndrome, particularly the stage of consumption coagulopathy, is diagnosed based on hemostatic parameters such as:

- Decreased fibrinogen
- Thrombocytopenia
- Elevated soluble fibrin complexes
- Increased fibrinogen–fibrin degradation products

Hypotonia and atony must be differentiated from traumatic injuries of the soft birth canal.

A large, flaccid, poorly contoured uterus with heavy bleeding suggests hypotonic hemorrhage. In contrast, heavy bleeding with a firm, well-contracted uterus indicates soft tissue trauma of the birth canal.

Management Tactics for Hypo-Atonic Hemorrhage

If uterine contractility is impaired in the early postpartum period and blood loss exceeds 0.5% of body weight (approximately 350–400 mL), all available therapeutic measures to control postpartum hemorrhage must be initiated immediately.

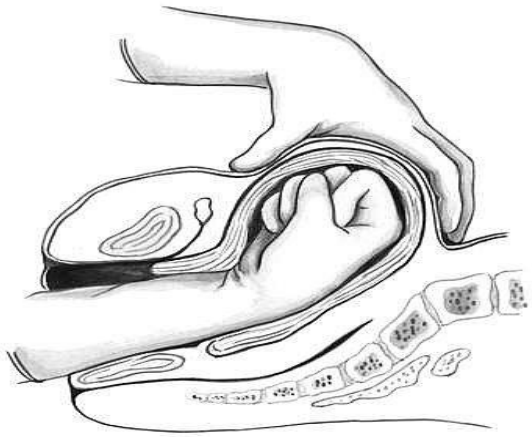


Figure 20. Bimanual Uterine Compression (Fist Massage of the Uterus)

Table 4

Algorithm of Actions for Early (Primary) Postpartum Hemorrhage

Manipulation	Measures to Stop Bleeding
Oxygen administration by inhalation	Examination of the vulva, vagina, and cervix; if present, suturing tears of the soft tissues of the birth canal
Catheterization of 2 peripheral veins using a 16G catheter	Laparotomy and, if present, suturing of uterine rupture
Monitoring vital signs (BP, pulse, respiration, blood oxygen saturation, urine output)	External uterine massage
Warming the patient and maintaining normothermia: intravenous infusion of warmed crystalloid solutions (heated to 35–40°C)	Bimanual uterine compression
Slow intravenous administration of tranexamic acid 15 mg/kg	Aortic compression
—	Uterotonic therapy

Manual exploration of the uterine cavity should be performed only by an experienced obstetrician-gynecologist. Repeated insertion of the hand into the uterine cavity is undesirable, ineffective, and potentially very dangerous. For this reason, bimanual uterine compression is currently recommended (Figure 21).

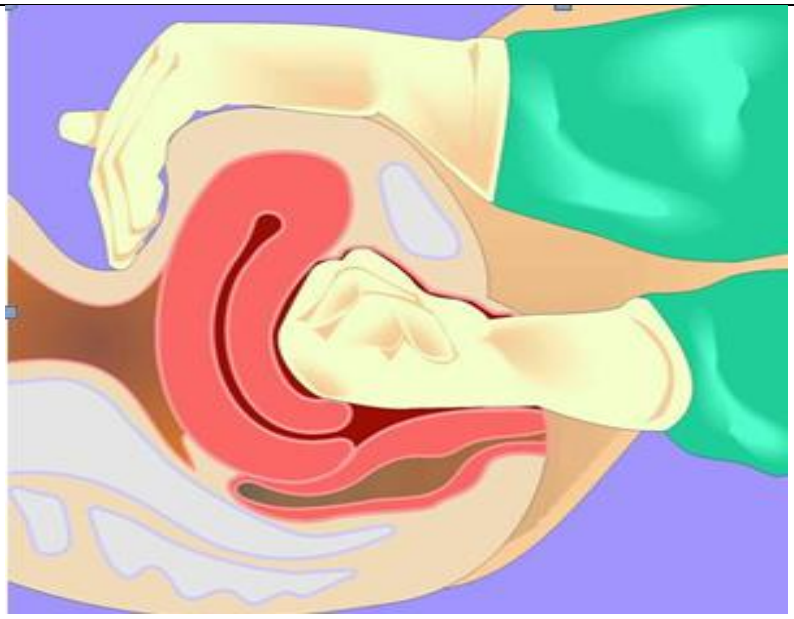


Figure 21. Bimanual Uterine Compression

Conservative methods to stop bleeding should usually be carried out in 2 stages.
The first stage includes: emptying the bladder, external uterine massage, applying cold to the lower abdomen, and intravenous administration of uterotonic drugs (methylergometrine 0.02% solution – 1 ml IV, oxytocin 40 units IV drip).
If bleeding continues, proceed to the second stage. A manual examination of the uterine cavity is performed, followed by uterine massage "on the fist" under anesthesia.
If the bleeding does not stop, or stops but recurs in small portions after 15–20 minutes, stepwise surgical management is indicated (see Chapters XI–XII below).
Before transfer to the operating room and preparation of the surgical/anesthesia team, temporary clamping methods according to Genkel-Tikanadze or N.S. Baksheev, and cervical sutures according to Lositskaya, were previously used to reduce blood loss before patient transfer. At present, these bleeding-control methods are not used.
From the first minutes of genital bleeding, management is carried out according to a multidisciplinary team principle with clear task distribution.
The primary task is to stop the bleeding and, at

the same time, determine its cause.
The first stage consists of conservative measures. Their goals are:

- diagnosis of bleeding and identification of its cause;
- alert/activation of response;
- clinical and laboratory examination;
- infusion therapy;
- uterotonic therapy;
- administration of hemostatic drugs;
- provision of the required volume of infusion and transfusion media.

Diagnosis, bleeding control, and infusion therapy are performed simultaneously with organizing patient monitoring.
The alert/activation process includes:

- calling a second midwife and a second obstetrician-gynecologist;
- calling an anesthesiologist-resuscitation specialist, transfusiologist (who must ensure availability of fresh frozen plasma and red blood cells [erythrocytes]), and a laboratory assistant;
- calling the duty nurse to deliver blood tests and blood components;
- assigning one member of the duty team to record interventions, infusion therapy, medications, and vital signs;
- in case of massive bleeding, notifying the receiving duty staff, hematology consultant, calling a surgeon, and preparing the operating room.

Required manipulations:

- catheterization of a second vein (16G or 14G catheters);
- urinary bladder catheterization;
- oxygen supply (oxygen mask or nasal cannula);
- monitoring of vital functions (blood pressure, heart rate, respiration, oxygen saturation, urine output);
- slow intravenous administration of tranexamic acid 15 mg/kg;
- administration of crystalloid solutions (warmed to 35–40°C).

Uterotonic agents and methods of their use are presented below (Table 5).

Table 5

Uterotonic Agents and Methods of Administration

Uterotonic Agent	Route and Dose	Maintenance Dose	Maximum Dose	Contraindications / Cautions
Oxytocin	10 IU diluted in 500 mL of normal saline or Ringer's solution, administered IV at 60 drops/min	10 IU in 500 mL normal saline or Ringer's solution, IV at 40 drops/min	Not more than 3 liters of solution containing oxytocin (total up to 60 IU)	Rapid bolus administration is contraindicated
Methylergometrine	0.2 mg IM or slow IV	Repeat 0.2 mg IM after 15 minutes if necessary; may repeat 0.2 mg IM or slow IV every 4 hours	5 doses (total 1.0 mg)	Arterial hypertension, preeclampsia, heart disease
Misoprostol	200 mcg	Repeat 200 mcg if required	800 mcg	Bronchial asthma

Uterotonic therapy is carried out to eliminate the hypotonic component of bleeding.

Misoprostol may be used only in life-threatening bleeding, when it cannot be stopped with other medications. The decision to prescribe it should be made by a medical commission. Dose: 600 mcg**,** route: rectal**.**

Carbetocin is used only during cesarean section and for prevention of postpartum hemorrhage. It is not used for treatment of bleeding after cesarean section.

To provide infusion therapy, the following must be ensured:

- Adequate intravenous access: at least two peripheral venous catheters, 14G or 16G;
- Standard monitoring (SpO_2, ECG, HR, BP);
- Horizontal positioning with the foot end of the surgical table (bed) elevated;
- Bladder catheterization;
- Solution-warming equipment;
- Patient warming equipment;
- Equipment for rapid intravenous infusion.

Performing infusion therapy is not by itself an indication for central venous catheter placement.

An important condition is warming solutions to 35–40°C**,** which helps prevent hypocoagulation and hypothermia and reduces blood loss. Infusion therapy should begin immediately. As initial therapy, up to 2000 ml of warmed balanced crystalloids is infused. Further correction of circulating blood volume (CBV) deficit may continue with colloids (succinylated gelatin, hydroxyethyl starch, or 5–10% albumin solution). The volume of colloids that may be infused before blood transfusion is 1500 ml. When calculating infusion-transfusion volume, remember that with major blood loss, FFP transfusion is required at 12–15 ml/kg**.** Donor red blood cells should be transfused no later than 40 minutes**,** when indicated, after individual compatibility testing.

After starting infusion therapy, blood should be taken for laboratory monitoring of:

- Hemoglobin, hematocrit, red blood cells, platelets;
- Acid-base parameters (pH, BE, HCO_3, SaO_2, PaO_2);
- Electrolytes (potassium, sodium, chloride);
- Glucose level;
- Fibrinogen, APTT, TT, PT.

Therapy should maintain:

- Hemoglobin > 80 g/L
- Platelets > 50×10^9/L
- PT and APTT not more than 1.5× normal
- Fibrinogen > 2 g/L (evidence level 4)

If blood loss is <25% of CBV (up to 1500 ml), infusion therapy is carried out in a restrictive mode, considering hemodynamic parameters. Balanced crystalloids should be the basis of infusion. Colloids should be used only when there are signs of severe hypovolemia.

Patients with blood loss ≥1500 ml or dysfunction of vital organs (tachycardia and hypotension) require immediate transfusion. In major blood loss, a massive transfusion protocol should be used with a component ratio of: packed RBCs : FFP : platelet concentrate : cryoprecipitate = 1:1:1:1. If platelet concentrate and

cryoprecipitate are unavailable, FFP and donor RBCs are used at 1:1 or 4:6 in effective doses.
FFP transfusion should start as early as possible in placental abruption or amniotic fluid embolism because these conditions are associated with early coagulopathy.
If infusion therapy is insufficient, hemodynamic parameters are corrected with vasopressors (norepinephrine, then dopamine if ineffective). At peak shock, infusion therapy is performed at maximum speed in a volume of 30–40 ml/kg**.** Further infusion exceeding 200% of calculated blood loss should be done only when central hemodynamic monitoring is available: cardiac index, stroke volume, total peripheral vascular resistance.
Major blood loss may cause hyperkalemia and hypocalcemia due to red cell transfusion and citrate toxicity.
Uncontrolled infusion therapy can worsen coagulopathy and cause fluid overload (pulmonary and cerebral edema). Infusion therapy must be carried out under strict hemodynamic monitoring. Effectiveness indicators: increased BP, and reduction of tachycardia by 10% or more, urine output at least 0.5 ml/kg/h**.**
If bleeding continues, controlled arterial hypotension may be used: mean arterial pressure maintained above 60 mmHg**,** systolic BP not above 100 mmHg**.** In ischemic heart disease, atherosclerotic vascular lesions, or impaired cerebral autoregulation (preeclampsia, arterial hypertension), mean arterial pressure should be at least 80 mmHg**.**
An effective method for prevention and treatment of obstetric hemorrhage is autoplasma transfusion, especially in pregnant women planning delivery and belonging to high bleeding-risk groups.
Agents used to correct coagulation disorders include fibrinolysis inhibitors, prothrombin complex concentrates, activated factor VII, cryoprecipitate, and fresh frozen plasma.
With major blood loss, use of fibrinolysis inhibitors reduces blood loss volume and risk of hysterectomy (AI), and should be used only as part of complex therapy without delaying plasma transfusion.
For blood loss up to 1000 ml**,** tranexamic acid is given IV over 10 minutes at 15 mg/kg (average 1000 mg**).** If bleeding continues, dose may be increased up to 4000 mg (40 mg/kg). Repeat administration is possible after 8 hours. Timely adequate-dose tranexamic acid may reduce mortality, hysterectomy rate, and morbidity in women with massive bleeding.
Aminomethylbenzoic acid may be used in obstetric bleeding at 100 mg**;** there is evidence of safe use up to 300 mg.
Aprotinin is not recommended, as cardiac surgery studies have shown an association with increased mortality. Large randomized controlled trials comparing tranexamic acid, aprotinin, and aminomethylbenzoic acid have not been conducted.
In hypocoagulation and/or blood loss >25–30% of CBV, FFP transfusion is required. Before plasma transfusion starts, administration of a prothrombin complex concentrate (Octaplex or Prothromplex) is acceptable at up to 50 IU/kg for factors II, VII, IX, X; if no effect within 20 minutes, repeat the same dose. Safety and efficacy data in obstetrics remain limited.

Routine use of activated factor VII for major obstetric hemorrhage is not recommended. Recombinant activated factor VII may be used for uncontrolled bleeding at at least 90 mcg/kg, repeatable after 3 hours if needed. Efficacy decreases significantly with hypothermia (<34°C), acidosis (pH <7.2), thrombocytopenia (<50 × 10^9/L), and low fibrinogen (<0.5 g/L), so normal homeostasis must be maintained. Activated factor VII should not delay or replace surgery.
Use of PCC, recombinant activated factor VII, and FFP requires mandatory reassessment of hemostasis after 6 hours and prophylactic low-molecular-weight heparin after 12 hours.
VTE prophylaxis should be performed in all cases of blood loss >1000 ml when there are no contraindications.
The effectiveness of sodium etamsylate and calcium chloride as hemostatic agents has not been proven (the latter should be used to prevent citrate intoxication and hyperkalemia during large-volume transfusion of long-stored red blood cells).
Cryoprecipitate is used against ongoing bleeding at 4 units per 4 units**;** usual dose is 15–20 ml/kg**,** and 30 ml/kg is needed to reliably increase clotting factors by 30%.
Donor platelets are administered for thrombocytopenia < 50 × 10^9/L. Initial adult dose: 1 dose (4 platelet units per 4 units of RBC mass).

Second stage of postpartum hemorrhage treatment (transitional phase)

Second-stage actions:

- Continue infusion and transfusion therapy according to blood loss volume, patient condition, and laboratory findings;
- Use procoagulant medications in confirmed hypocoagulation with ongoing bleeding;
- Warm the patient and maintain normothermia;
- Perform bimanual uterine compression;
- Perform uterine tamponade if bleeding continues despite manual uterine exploration and suturing of trauma.

If complete hemostasis is not achieved after vaginal delivery in postpartum patients, begin operating room preparation, provide anesthesiology support for surgery, and repeat laboratory tests. If conditions allow, perform intraoperative reinfusion of autoerythrocytes.

Surgical Management

The third stage of postpartum hemorrhage control is **surgical**.
Its goal is to achieve definitive hemostasis by surgical methods while continuing infusion-transfusion therapy, uterotonic treatment, and correction of coagulation disorders.

Third-stage measures

- transfer to the operating room;
- anesthetic management of surgery;

- repeat laboratory testing to assess process dynamics and treatment efficacy (CBC, Lee-White clotting test, hemostasiogram, urea, electrolytes);
- intraoperative autologous red blood cell reinfusion;
- surgical treatment.

The extent of surgical treatment is determined by the clinical situation, physician expertise, and institutional technical capacity. Standard options include:

- laparotomy (transverse suprapubic incision if a uterine scar is present; otherwise lower midline);
- uterine tourniquet/banding;
- uterine compression sutures (B-Lynch) and/or temporary complex peripheral compression of vascular pedicles;
- temporary clamping or ligation of major uterine vessels;
- uterine artery embolization;
- ligation of internal iliac arteries;
- hysterectomy if other measures are ineffective.

Intensive care and anesthetic tactics should aim to prevent:

- hemorrhagic shock;
- respiratory failure (ARDS);
- acute kidney injury/renal failure;
- liver failure;
- coagulopathy due to acute disseminated intravascular coagulation (DIC).

If possible, an additional anesthesia team (anesthesiologist-resuscitator and nurse anesthetist) should be involved. Safe regional anesthesia is possible in hemodynamically stable patients (ASA I–III), without contraindications, and with blood loss not exceeding 25% of circulating blood volume (CBV). For any anesthesia type, readiness to rapidly convert to general anesthesia is mandatory.

Postoperative monitoring should include

- noninvasive blood pressure monitoring;
- SpO2;
- heart rate;
- ECG;
- respiratory rate;
- capnometry during mechanical ventilation (if available);
- urine output monitoring (bladder catheterization);
- body temperature control.

Before any anesthesia, venous access must be established.
If pregnancy is ongoing, the patient should be positioned with at least a 15° left tilt to prevent aortocaval compression. If hemodynamically unstable, elevate the foot end of the operating table.
If SpO2 falls below 96% or signs of respiratory failure are present, oxygen therapy via nasal cannula or face mask should be initiated immediately in the OR.
Hypotension correction may include:

- ephedrine (total up to 50 mg), or
- phenylephrine boluses 3–10 mcg,
- infusion of phenylephrine or norepinephrine.

Initial infusion rates:

- phenylephrine: 0.1–0.15 mcg/kg/min,
- norepinephrine: 0.04–0.06 mcg/kg/min, titrated to target BP. If vasopressor support is ineffective, dopamine is second-line (initial 2 mcg/kg/min; evidence level 4).

Target hemodynamics:

- HR 55–120/min,
- MAP ≥60 mmHg. Controlled hypotension may be used with systolic BP ≤100 mmHg and MAP ≥60 mmHg.

Premedication **(regardless of anesthesia type) should include:**

- antiemetics: dexamethasone 4–8 mg and/or ondansetron 4–8 mg;
- antacids;
- atropine 0.01 mg/kg;
- fibrinolysis inhibitors.

For surgical anesthesia, spinal or epidural anesthesia may be used if an epidural catheter is present. Anesthesia should not significantly delay surgery. Neuraxial block is contraindicated in severe hypovolemia; pre-infusion does not reliably prevent hypotension under sympathetic block.

Spinal anesthesia

Performed under strict asepsis/antisepsis. Subarachnoid puncture is done in lateral or sitting position, not above L1 level, using 25–29G spinal needles. Correct placement is confirmed by CSF return after stylet removal.

Preferred local anesthetic: **0.5% hyperbaric bupivacaine**. Dose should be individualized (height, weight, condition, hemodynamics).

- For cervical/perineal procedures: 5–6 mg bupivacaine is usually sufficient.
- For abdominal surgery: 10–12.5 mg.

Higher doses increase hemodynamic risk; lower doses may provide inadequate sensory block.

Epidural anesthesia

If an epidural catheter is already placed:

- verify catheter position;
- perform traction and aspiration checks;
- repeat test dose is not mandatory.

Administer local anesthetic only in monitored OR conditions. Possible agents: ropivacaine or levobupivacaine 0.375% or 0.5%.

Typical volumes:

- 5–8 ml for cervical/perineal manipulations,
- 15–20 ml for abdominal surgery (depending on required sensory block level and patient height).

Note: effective block may require 15–20 minutes.

General anesthesia

Only when functional equipment is available for airway control, mechanical ventilation, and CPR.

Total intravenous anesthesia should be considered carefully due to aspiration/regurgitation risk and possible difficult airway.

Without spontaneous breathing, muscle relaxants are required.

Absolute indications for mechanical ventilation

- circulatory arrest;
- apnea or bradypnea;
- failure of primary/auxiliary respiratory muscles;
- altered consciousness due to hypoxia;
- prolonged hypoxic tachycardia;
- pulmonary edema;
- hypoxemia not corrected by oxygen therapy (PaO2 <60 mmHg, SaO2 <90%, PaCO2 >55 mmHg, vital capacity <15 ml/kg);
- ongoing bleeding with unstable hemodynamics.

General anesthesia induction is performed after 3–5 minutes of preoxygenation. Because of "full stomach" risk, rapid sequence induction with Sellick maneuver is used.

Induction options (after premedication)

- thiopental sodium 4–8 mg/kg + fentanyl 3–5 mcg/kg (contraindicated in severe hypovolemia);
- ketamine 1.0–1.5 mg/kg (± fentanyl 1–2 mcg/kg if needed);
- propofol 2–2.5 mg/kg + fentanyl 3–5 mcg/kg (in pregnancy propofol induction should not exceed 2.5 mg/kg; contraindicated in severe hypovolemia).

Muscle relaxation

- succinylcholine 1.5–2 mg/kg (onset 20–45 sec),
- rocuronium bromide 0.6–1 mg/kg (onset 1–2 min),
- cisatracurium / atracurium / vecuronium (onset 3–5 min).

After adequate relaxation, perform orotracheal intubation (tube size 7–8). Confirm position by chest excursion, auscultation, and capnography.

Recommended capnometry target: EtCO2 32–40 mmHg.

Tidal volume: 6–8 ml/kg.

If intubation is impossible, use second-generation laryngeal mask airway or establish percutaneous airway access.

In "full stomach" situations, decompress stomach with gastric tube, then remove.

Maintenance

- sevoflurane/desflurane/isoflurane 0.8–1 MAC;
- nitrous oxide + oxygen (2:1);

- in uncompensated shock, for hypnosis use sodium oxybutyrate (70 mg/kg) or ketamine (50 mg every 15–20 min) instead of nitrous oxide, with ventilation by air-oxygen mixture;
- boluses: thiopental 2–4 mg/kg or ketamine 0.3–0.5 mg/kg;
- propofol infusion 4–12 mg/kg/h (if not pregnant).

Analgesia: fentanyl 1–2 mcg/kg. Muscle relaxation: maintenance doses of nondepolarizing relaxants.
Extubation is performed after restoration of neuromuscular conduction, adequate spontaneous respiration, airway reflexes, and consciousness.
Prolonged ventilation is indicated in unresolved severe anemia, ongoing hemorrhagic shock, unstable hemodynamics, and respiratory failure.
Decurarization can be done with neostigmine methylsulfate 10–15 mg when residual blockade is present.

Intraoperative autologous RBC reinfusion (IAR)
Should be performed only by trained, experienced staff and is subject to annual audit.
Patient consent is required. IAR reduces use of donor blood products, decreases risk of massive transfusion syndrome, improves postoperative recovery, and shortens hospital stay.
Indication in obstetrics
Expected intraoperative blood loss >20% of CBV during abdominal delivery.
Contraindications in obstetrics
Absolute:
- purulent intra-abdominal content;
- prohibited substances in shed blood (hydrogen peroxide, distilled water, collagen-based hemostatics, etc.).

Relative:
- malignant neoplasms (if reinfusion is performed, leukocyte filters are mandatory).

In bowel surgery, contamination is not an absolute contraindication provided contaminated contents are evacuated first, RBCs are additionally washed, and broad-spectrum antibiotics are used.
Equipment: Cell Saver/CATS systems with disposable tubing sets.
Method principle: aspiration of shed blood from surgical field → machine processing → reinfusion of autologous RBC suspension (Ht ~60%).
Negative pressure should not exceed 100 mmHg.
Escalation timing
Within 10–20 minutes from hemorrhage onset, continue all stage-1 measures, including:
- **Balloon tamponade** as first-line intervention in most women with uterine hypotonia.
- In refractory bleeding, combine balloon tamponade with uterine compression sutures; a two-balloon utero-vaginal system may additionally compress lower segment vessels.

- Balloon tamponade may be continued until hysterectomy to reduce blood loss.

If blood loss reaches 1000 ml and bleeding continues or shock signs appear, transfer immediately to OR.
With blood loss >25–30% of CBV, surgery should be initiated within 20 minutes.
If bleeding develops during cesarean section, B-Lynch sutures are recommended (manual abdominal aortic compression before surgery may reduce blood loss).
If prior measures fail

- provide anesthesia support for surgery;
- repeat analyses;
- perform intraoperative autologous reinfusion if available;
- perform surgical hemostasis:
    - laparotomy/relaparotomy;
    - uterine devascularization (ligation/temporary clamping of major uterine vessels and/or internal iliac ligation).

Vascular ligation sequence

- bilateral ligation of uterine artery and vein (separately or together);
- if persistent bleeding, place a second, lower ligature to include branches supplying lower segment/cervix;
- if still ineffective, ligate utero-ovarian vessels;
- internal iliac ligation requires high surgical skill due to risk of ureter and vascular injury.

Angiographic embolization

Alternative to uterine/internal iliac ligation; requires 20–30 minutes and special equipment.
Reported efficacy: 58–98%, but hysterectomy is still required in ~15% of cases.

Other procedures

- hysterotomy;
- ligation of placental bed bleeding vessels;
- in selected cases and tertiary centers with trained staff: resection of uterine wall at placental invasion site;
- compression sutures (vertical D-/B-Lynch or horizontal) and/or complex temporary peripheral compression of vascular pedicles.

Hysterectomy

Life-saving emergency procedure for uncontrolled postpartum hemorrhage when previous steps fail. Ureter visualization during hysterectomy is recommended. Uterine preservation is acceptable only if patient is hemodynamically stable and there is no life-threatening bleeding.
Relative contraindications to uterine preservation:

- major placenta accreta spectrum with ongoing placental bed bleeding;
- extensive uterine injury not amenable to reconstruction;
- inflammatory uterine disease.

Decision on hysterectomy must be timely and team-based.
If staged surgical hemostasis is not feasible (level 1–2 obstetric facilities), hysterectomy should be performed as early as possible as the most effective method of hemorrhage control.

To illustrate, let us provide a specific example:

CASE No. 2

A 30-year-old multiparous woman was urgently admitted to a maternity hospital for delivery with the diagnosis: Pregnancy V, para I, 40–41 weeks. Labor III. Burdened obstetric-gynecologic history. Stage I of labor.

From the obstetric history: she had three pregnancies; the second ended in a term delivery without complications. Between deliveries, she had one abortion for medical indications, complicated by metroendometritis, for which she received inpatient antibiotic treatment. In the current pregnancy, labor was complicated by premature rupture of membranes and primary uterine inertia. Labor stimulation with Endaprost and oxytocin was administered intravenously by infusion. The second stage of labor was complicated by persistent secondary weakness of labor and fetal hypoxia. Therefore, abdominal obstetric forceps were applied. A live male infant was delivered, weighing 3400 g and 53 cm in length. In the third stage, heavy bleeding was noted. For this reason, manual separation and removal of the placenta were performed, followed by uterine cavity examination. The uterus was somewhat relaxed, but after methylergometrine administration, uterine contraction improved. Injuries of the vaginal wall, vulva, and perineum were sutured. The uterus contracted and bleeding stopped. Oxytocin infusion was started. Forty minutes after delivery, during postpartum external uterine massage, the midwife recorded expulsion of 300 ml of blood clots from the uterus. The duty physician emptied the bladder and repeated external uterine massage, during which an additional 150 ml of blood with small clots was expelled. Blood pressure decreased by 10 mmHg (initial BP 110/70). A responsible physician was called to perform repeat manual uterine exploration followed by fist massage. The uterus contracted and bleeding decreased. BP dropped to 100/60 and then 90/50 mmHg. During bladder catheterization, approximately 30 ml of urine was obtained. The department head was called from home; after assessment, he repeated manual uterine exploration. BP dropped to 80/40 mmHg, urine output stopped, clotting started at 7 minutes and remained incomplete. Fresh frozen plasma 500 ml IV was started; Gordox 200,000 units and Contrical 50,000 units were given. A small amount of non-clotted liquid blood continued from the genital tract. Total blood loss was approximately 1000 ml. Hemoglobin decreased to 50 g/L. Specialists were called from the regional center by emergency transport and arrived within 2 hours after consultation. Diagnosis: postpartum uterine hypotonia, posthemorrhagic shock stage II–III, DIC syndrome, acute renal failure in the oligoanuric stage. Surgery was performed: laparotomy showed a hypotonic, dull, sharply pale uterus with petechial rash in the adnexal area. Uterine extirpation with adnexa and ligation of internal iliac arteries was performed. Surgery was carried out with an experienced anesthesiologist-intensivist, under infusion therapy, fresh frozen plasma, packed red blood cells, and protease inhibitor transfusion. During surgery, bleeding increased; blood loss exceeded 2 liters. Internal iliac artery ligation reduced bleeding only slightly, but parenchymal bleeding persisted, and tight pelvic packing was required.

During closure of the anterior abdominal wall, cardiac arrest occurred and the woman died. Death resulted from complete exhaustion of compensatory and protective mechanisms due to delayed surgical tactics.

The above example shows that serious errors were made in postpartum care (repeated external/internal uterine massage, delayed infusion therapy, delayed blood transfusion, etc.). Most importantly, time for surgical treatment was lost; therefore, the surgery was unsuccessful.

In our opinion, manual uterine cavity examination should be performed only under general anesthesia by an obstetrician-gynecologist. If bleeding continues even in small amounts after such intervention, urgent hysterectomy should be considered, because unnecessary excessive conservatism leads to loss of time for timely surgical intervention and loss of compensatory reserves.

Many such examples can be cited. During manual uterine exploration and external-internal massage, a biological contractility test is performed. At the end of uterine massage, a uterotonic drug is given intravenously (methylergometrine 0.02% in 5 ml saline or oxytocin 1 ml + 2 ml saline). If an effective contraction is palpated by the physician, the result is considered positive, and the procedure ends with removal of residual clots from the uterine cavity. If manual exploration is timely and effective, total blood loss is usually around 600–700 ml (about 400 ml before the procedure). However, repeated external-internal massage and repeated uterotonic administration may be ineffective, and bleeding may continue in persistent uterine hypotonia. If previous pharmacologic methods (oxytocin, methylergometrine, prostaglandins) and reflex stimulation of uterine contractility have failed, the uterus should be considered in "shock" due to loss of sensitivity to neurohumoral stimulation. Therefore, if uterine hypotonia persists after manual external-internal massage and blood loss approaches 1000 ml, urgent surgical hemostatic methods should be initiated. Excessively large doses of uterotonics, combining multiple contraction agents, repeated reflex stimulation, repeated manual uterine exploration, and replacement sutures according to V.A. Lositskaya are ineffective; they only delay treatment, increase blood loss, and worsen the condition.

When blood loss exceeds 1000 ml, despite higher morbidity, uterine extirpation is preferred over amputation, since in uterine amputation the cervical stump wound surface may become a bleeding source in acute DIC syndrome.

Management of DIC in hypotonic hemorrhage is primarily a hemostatic and resuscitation challenge. To gain time before conservative treatment effects appear, surgical hemostasis in the operating room is required. For this purpose, internal iliac artery ligation is performed. This procedure reduces pelvic pulse pressure by about 70%, resulting in a sharp decrease in blood flow, reduced bleeding from damaged vessels, and better conditions for thrombus fixation.

For surgical hemostasis during hysterectomy, pre-ligation of internal iliac arteries before uterine removal is recommended. Under such conditions, surgery is performed in a "drier" field, total blood loss is reduced, and thromboplastin release into uterine vessels decreases. As a result, hysterectomy-related trauma and severity of coagulation disorders are reduced.

Prevention of postpartum hemorrhage includes:

- Active management of the third stage of labor: uterotonic administration after delivery of the anterior shoulder, cord clamping 1–3 minutes after birth, controlled cord traction for placental delivery, uterine massage.
- Labor management with established venous access (diameter not less than 18G).
- In high-risk women and during cesarean section: tranexamic acid (15 mg/kg) in the second stage of labor.

Current prevention during vaginal delivery:

- Oxytocin 2 ml (10 IU) IM into the lateral thigh at delivery of the anterior shoulder in low-risk women;
  or
- Slow IV oxytocin (5 IU in 50 ml saline) from the end of second stage via infusion pump at 1.8 ml/h, increasing to 16.2 ml/h (27 mU/min) after delivery of the anterior shoulder; drop infusion at ~40 drops/min may also be used. Higher doses provide no additional benefit.

Cesarean section prophylaxis:

- Oxytocin 1 ml (5 IU) IV slowly (over >1–2 min) after fetal delivery;
  or
- Carbetocin 1 ml (100 mcg) IV immediately after delivery;
  or
- Oxytocin solution (5 IU in 50 ml saline) via infusion pump at 16.2 ml/h (27 mU/min) after fetal delivery; alternatively drip 5 IU in 500 ml saline at 80 drops/min.

Three effective prevention strategies have been identified:

1. Ergometrine + oxytocin
2. Carbetocin
3. Misoprostol + oxytocin (more effective than standard oxytocin alone)

Pharmacologic notes:

- **Oxytocin** primarily acts on the upper uterus; it may cause vasodilation and hypotension, especially in severe cardiovascular insufficiency.
- **Ergometrine** acts on both upper and lower uterine segments; it is useful for lower segment hypotonia. It causes vasoconstriction and may increase BP.
- Ergometrine is contraindicated in hypertension and may provoke coronary spasm and myocardial infarction in high-risk patients. Nausea/vomiting occur frequently and retained placental fragments may occur.
- **Misoprostol** is less effective than parenteral uterotonics but is available orally, rectally, and vaginally. Side effects: hyperthermia and chills.
- **Carbetocin** has the lowest frequency of adverse effects.
- In high-risk groups, additional prevention may include uterine or combined utero-vaginal tamponade.

Every unit (delivery ward, emergency room, postpartum unit, etc.) should have a visible interdisciplinary postpartum hemorrhage algorithm and hemorrhage kits available in all relevant departments. All maternity care staff should be trained in

obstetric emergencies, including postpartum hemorrhage management. Training should be multidisciplinary and team-based. All postpartum hemorrhage cases with blood loss >1500 ml should undergo formal clinical review.

Therefore, to reduce obstetric hemorrhage frequency and prevent severe hemorrhagic complications and maternal mortality:

- Identify high-risk groups through full antenatal evaluation;
- Provide full examination and preconception preparation in specialized hospitals/regional centers;
- Closely monitor high-risk pregnancies with relevant specialists;
- Regularly assess and correct hemostatic disturbances;
- Refer women at risk of placenta previa or placental abruption to regional centers for US and assessment;
- Rationally manage first and second stages of labor; avoid prolonged use of labor stimulants; if labor becomes unfavorable, promptly decide on operative delivery;
- Establish peripheral venous access by the end of second stage; use prophylactic methylergometrine at crowning; carefully conduct the third stage using expectant-active tactics (waiting for signs of placental separation and descent).

IX. BIRTH TRAUMA

One of the common causes of bleeding during labor, in the early postpartum period, and in the postpartum stage is **birth trauma**.

Birth trauma is damage to the soft tissues of the birth canal (external genitalia, vagina, perineum), cervix, and uterine body that occurs in a pathological course of labor or due to delayed/inadequate obstetric care.

Superficial and shallow asymptomatic injuries may heal spontaneously and can remain unrecognized.

In some cases, soft birth canal injuries are significant and cause serious complications, the consequences of which become evident during labor and postpartum. Some birth canal injuries are life-threatening and may be fatal. In certain cases, they lead to long-term loss of work capacity and disability.

Soft tissue tears of the birth canal are more common in primiparous women, while uterine rupture is mainly observed in multiparous women.

According to various authors, cervical tears occur in **6–15%** of deliveries.

Causes of birth trauma

- improper perineal protection;
- delivery of large, giant, or post-term fetus;
- precipitous labor;
- prolonged labor, especially the second stage;
- malposition of the fetal head;
- anatomically narrow pelvis;
- breech presentation;
- tissue rigidity, scarring, or inflammatory tissue changes;

- operative vaginal delivery (obstetric forceps, vacuum extraction).

The extensibility of soft tissues of the birth canal has physiological limits. As the presenting fetal part advances, pressure on surrounding structures increases, which may lead to injury and eventually tissue rupture.

Types of maternal birth trauma in obstetric practice

- vulvar tears;
- vaginal tears;
- hematomas of soft tissues of the birth canal;
- cervical tears;
- perineal tears;
- uterine rupture.

Vulvar Tears

Vulvar tears usually occur in the region of the labia minora and clitoris and may present as superficial or deep fissures/lacerations. They are more common in women with a history of colpitis or vulvovaginitis of various etiologies.

Vulvar tears are classified as:

- superficial;
- deep.

Clinical features and diagnosis

Vulvar tears, especially clitoral tears, are often accompanied by severe bleeding. Diagnosis is based on complete examination of the external genitalia and identification of active bleeding from tear sites.

Treatment

Tears in the area of the labia minora are sutured with fine catgut using continuous or interrupted sutures without involving deep tissues, to avoid injury/bleeding from cavernous tissue.

When suturing clitoral tears, urinary catheterization is recommended first. Repair may be performed under local infiltration anesthesia; however, intravenous anesthesia is preferable because after repair of vulvar tears, instrumental examination of the remaining birth canal is required.

Vaginal Tears and Hematomas of Soft Birth Canal Tissues

By etiology, they are divided into:

- spontaneous;
- forced (during vaginal operative delivery).

By localization:

- upper third of vagina;
- middle third of vagina;
- lower third of vagina.

By depth of injury:

- superficial;
- deep.

Most often, tears in the lower third of the vagina are associated with perineal tears. Tears in the upper third may extend into the vaginal fornix and then be associated with cervical tears. The middle third is less frequently damaged due to greater

distensibility. Sometimes tissue rupture/crushing affects only deep submucosal layers while the mucosa remains intact, and a submucosal hematoma forms at the rupture site.
A hematoma of soft birth canal tissues is a localized collection of blood resulting from closed injury with vessel rupture and blood extravasation into surrounding tissues.

Localization of birth canal hematomas

- region of the labia majora;
- perineal region;
- vaginal wall region;
- paravaginal tissue region;
- gluteal muscle region.

Clinical features and diagnosis

Clinically, vaginal tears manifest as bleeding from the injured wall or, in submucosal rupture, as hematoma formation when venous (sometimes arterial) vessels are injured. A lateral vaginal wall hematoma may enlarge significantly and increase the size of one labium. The woman clearly perceives this postpartum.
Hematoma size depends on caliber of the injured vessel.
In clinical practice, large hematomas may extend toward gluteal muscles. A patient may complain of tension and severe pain in an enlarged bluish-purple buttock area. Hemoglobin may decrease; BP may drop to 80/40 mmHg; cold sweating appears; a picture of hemorrhagic shock develops.
In such cases, under general anesthesia, the hematoma is opened (from both vaginal and gluteal sides), the bleeding source is eliminated, and drainage/contraperture is established if needed.

Main clinical signs of hematoma

- bluish-purple tumor-like mass;
- indistinct borders;
- doughy or elastic consistency;
- tissue tension and pain over the lesion;
- vulvar deformity in large hematomas;
- signs of anemia in significant bleeding.

Diagnosis is based on clinical findings and cervical/vaginal examination with speculum.
Laboratory monitoring is essential:

- hemoglobin;
- erythrocyte count;
- color index;
- hematocrit;
- blood clotting time (Sukharev method).

Treatment

- suturing of bleeding vaginal wall with interrupted or continuous catgut sutures;
- opening and evacuation of hematoma;
- ligation/suturing of bleeding vessels together with surrounding tissue.

Small hematomas often resolve without intervention.
If tear is located in the upper third and extends into the vaginal fornix, manual uterine exploration is required to exclude lower uterine segment rupture.
If hematoma enlarges rapidly, anemia worsens, surgical intervention under general anesthesia is indicated: incision and evacuation of hematoma, ligation of bleeding vessel, tight closure, and drainage.

Cervical Tears

Cervical tears most often occur from below upward, i.e., from the external os toward the internal os.

Classification

There are 3 degrees of cervical tears:

- **Grade I**: unilateral or bilateral tears up to 2 cm in length;
- **Grade II**: tears >2 cm but not reaching the vaginal fornix (at least 1 cm short);
- **Grade III**: tears reaching or extending into the vaginal fornix.

Grade III tear is a severe obstetric injury; extension into the lower uterine segment is difficult to exclude, therefore manual exploration of the uterine cavity is required.

Clinical features and diagnosis

Shallow tears up to 1 cm are usually asymptomatic. Deeper tears are accompanied by bleeding of varying intensity. If the descending cervical branch of the uterine artery is injured, bleeding is profuse and starts immediately after childbirth. After placental delivery, if the uterus is well contracted, bright red blood flows in a stream. If cervical tissue is crushed due to prolonged compression of the head against pelvic bones, bleeding may be absent despite severe injury.
In the absence of bleeding, cervical tears can only be diagnosed by thorough examination of all cervical edges using vaginal speculum and atraumatic forceps.
All postpartum women should be examined within the first 2 hours after delivery. If bleeding is present, examination should be performed immediately after placental separation and inspection.
According to Order No. 500 of the Ministry of Health of the Republic of Uzbekistan, instrumental examination of the birth canal should be performed strictly in bleeding cases. In our opinion, speculum examination for birth canal tears should also be performed in primiparous and multiparous women, as well as in women with pre-pregnancy or pregnancy-associated colpitis/vulvovaginitis of any infectious etiology. Ignoring this rule can lead to serious consequences.

CASE No. 3

A 34-year-old multiparous woman, N.M., was admitted to one of the maternity hospitals in the Samarkand region for specific reasons. An experienced senior obstetrician decided to manage the delivery.

From the anamnesis: this was her fourth pregnancy. Several years ago, two pregnancies ended in emergency vaginal deliveries, and one ended in an out-of-hospital abortion. This is her second marriage, and the pregnancy occurred in the third year of regular sexual activity. Before her second marriage, she had suffered from gonorrhea, vulvovaginitis, and cervical erosion. She was treated multiple times by various physicians, underwent electrocoagulation of the cervix, and later had a biopsy of the damaged cervical area.

This labor progressed physiologically. Pain relief was adequately achieved with analgesics and antispasmodics. No complications occurred during labor. A full-term male infant weighing 3800 g and measuring 56 cm in length was delivered.

In the early postpartum period, the physician noted increased bleeding in the delivery room. Under general anesthesia, manual removal of the placenta and uterine cavity exploration were performed. During this procedure, the physician blindly examined the cervix and other soft tissues of the birth canal with his fingers and concluded that everything was intact.

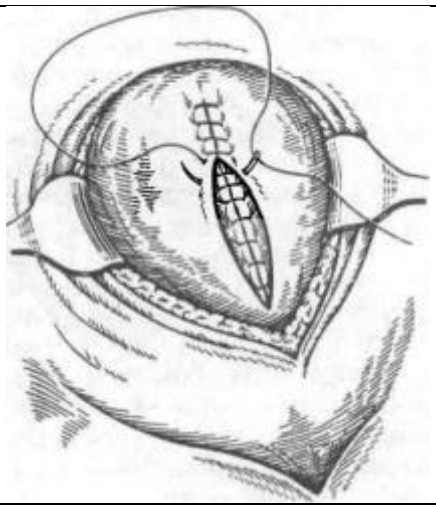
Cold was applied to the lower abdomen, and 2 ml of methylergometrine was administered intravenously. However, the bleeding continued.

A medical consultation was convened, and the diagnosis was made: Early postpartum period. Hypotonic postpartum hemorrhage.

The patient was transferred to the operating room, and a laparotomy was performed. A supravaginal hysterectomy (subtotal hysterectomy) without adnexal removal was carried out. However, the bleeding did not stop. It was decided to remove the cervix as well.

Before proceeding with this surgery, the cervix was examined using speculums. A second-degree cervical tear was identified, from which a branch of the cervical artery was pulsating. Two catgut sutures were placed, and the bleeding stopped.

The laparotomy was therefore limited to supravaginal hysterectomy of the uterus.

	Two-layer suturing technique

Treatment

After examination of the cervix and identification of the injury, **two-layer U-shaped catgut sutures** should be applied to the tear immediately.

- The **first layer** is placed through the cervical mucosa and the muscular layer of the cervical canal.
- The **second layer** is placed through the mucosa of the vaginal portion of the cervix and the remaining deeper muscular layer.

Alternatively, **single-layer catgut sutures** may be used if they adequately approximate all layers of cervical tissue.

Uterine Rupture

Uterine rupture is one of the most severe complications in obstetrics. It most often occurs during labor, is accompanied by massive bleeding, and may result in maternal death and, frequently, stillbirth. Every case of uterine rupture in a pregnant or laboring woman must be analyzed to identify factors that led to severe obstetric trauma.

According to local authors, the incidence of uterine rupture ranges from **0.05% to 0.1%**.

By national data (around 2000), the rate was approximately **0.16 per 1,000 births** (Ministry of Health of the Republic of Uzbekistan).

Foreign authors report an incidence of **0.03–0.005%**.

Classification

In our country, the classification proposed by **L.S. Persianinov (1954)** is widely used. It divides uterine rupture by several criteria:

I. By time of occurrence

1. Rupture during pregnancy
2. Rupture during labor

II. By pathogenetic features

1. **Spontaneous uterine rupture**
   a) **Typical (mechanical):** in the presence of a mechanical obstruction to fetal delivery
   b) **Atypical:**
      - **Histopathic:** due to pathological changes in the uterine wall
      - **Mechanohistopathic:** combination of mechanical obstruction and uterine wall pathology
2. **Violent (traumatic) uterine rupture**
   a) during labor due to грубое intervention without hyperextension of the lower uterine segment or accidental injury
   b) mixed type (external impact in the presence of lower-segment hyperextension)

III. By clinical stage

1. Threatened rupture
2. Beginning rupture
3. Complete rupture (established rupture)

IV. By extent/type of damage

1. Fissure (tear)

2. Incomplete rupture (does not penetrate into the abdominal cavity)
3. Complete rupture (penetrates into the abdominal cavity)

V. By localization

1. Rupture of the uterine fundus
2. Rupture of the uterine body
3. Rupture of the lower uterine segment
4. Uterine avulsion (detachment)

The most common site of uterine rupture is the lower uterine segment**,** typically along its anterior or lateral wall.

Rupture of the uterine body and fundus, as a rule, occurs in the area of a previous surgical scar.

Etiology and Pathogenesis

Traditionally, two main causes of uterine rupture during labor are distinguished.

In the 19th century, **Ludwig Bandl** (1875) proposed the mechanical theory of uterine rupture, which remains supported to this day.

According to this theory, uterine rupture during labor is explained by a disproportion between the fetal size and the maternal pelvic dimensions (cephalopelvic disproportion), leading to excessive stretching and eventual tearing of the uterine wall.

Table 7
Classification of Causes of Uterine Rupture
According to *Williams Obstetrics* (2001)

Uterine Injury or Developmental Abnormalities Before Pregnancy	**Uterine Injury or Complications During Pregnancy**
1. Previous Uterine Surgical Interventions:	**1. Before Delivery:**
- Cesarean section or hysterotomy	- Persistent excessive uterine contractions
- Previous suturing of uterine rupture	- Labor stimulation with oxytocin and prostaglandins
- History of myomectomy	- Intra-amniotic infusion of solutions or prostaglandins
- Resection of the uterine tubal angle	- Uterine perforation during catheter placement for intrauterine pressure monitoring
- Metroplasty	- External trauma (sharp or blunt)
	- External cephalic version

Uterine Injury or Developmental Abnormalities Before Pregnancy	Uterine Injury or Complications During Pregnancy
	- Excessive uterine overdistension (polyhydramnios, multiple pregnancy)
2. Previous Uterine Trauma:	**2. During Labor:**
- Instrumental abortion	- Internal version
- Sharp or blunt trauma	- Use of forceps in difficult operative deliveries
- Accident, gunshot, or stab wound	- Breech extraction
- History of asymptomatic uterine rupture	- Fetal developmental anomaly (hydrocephalus)
	- Strong excessive labor pains
	- Difficult manual removal of the placenta
3. Congenital Anomalies:	**3. Rare Causes:**
- Pregnancy in a rudimentary uterine horn	- Placenta increta or percreta
	- Trophoblastic disease
	- Adenomyosis
	- Uterine torsion in retroversion

If there is an obstruction to fetal expulsion, strong labor activity develops: the upper uterine segment contracts more and more, and the fetus gradually descends into a thin-walled, overstretched lower uterine segment. If the cervix (usually the anterior lip) has not passed beyond the fetal head and becomes compressed between it and the pelvic walls, excessive uterine overdistension and rupture may occur more easily.

The constriction ring rises progressively higher and may reach the level of the umbilicus; the uterus acquires an “hourglass” shape. As labor continues, maximal overstretching and thinning of the lower uterine segment is reached, and rupture occurs.

When the uterus ruptures completely and large vessels are damaged, intra-abdominal bleeding begins. When the uterus ruptures in the anterior part of the lower segment or separates from the vaginal fornices, the bladder may also be involved. In incomplete uterine rupture, when the peritoneum remains intact, a hematoma forms depending on the site of injury: between the leaves of the broad ligament, under the serosal covering of the uterus, or in the prevesical tissues.

At the beginning of the 20th century, N.Z. Ivanov (1901) and Ya.F. Verbov (1911) proposed another theory of uterine rupture genesis, according to which rupture results from inflammatory and degenerative processes, scar changes after cesarean section, reconstructive uterine surgeries, the capsule of myomatous nodes, trauma during abortion, complicated labor, and uterine injuries in multiparous women, leading to histopathological changes in the myometrium. It has now been established that during prolonged labor, pronounced metabolic disturbances occur, accompanied by accumulation of toxic compounds that damage tissues ("biochemical injury of the uterus"). The uterine muscles become flaccid and rupture easily. Uterine rupture may occur against a background of weak or uncoordinated uterine contractions.

At present, the most common cause of uterine rupture is a uterine scar, due to expanded indications for abdominal delivery and the increasing frequency of reconstructive plastic uterine surgeries. The quality (integrity) of the scar depends on the location of the uterine incision, the cesarean technique, suture material, postoperative period, and other factors.

In women with a uterine scar, rupture during labor occurs in 0.2–1.5% of cases after a transverse incision in the lower segment, in 1–7% after a longitudinal incision in the lower segment, and in 4–9% after corporal or T-shaped uterine incisions. Approximately one-third of uterine ruptures occur before the onset of labor.

The frequency of uterine rupture increases with the number of previous cesarean sections. With a history of one cesarean section, rupture occurs in 0.6% of cases; with two cesareans, in 1.8%; according to other studies, the rates are 0.8% and 3.7%, respectively.

Violent (iatrogenic) factors leading to uterine rupture should be distinguished; these include the use of the Kristeller maneuver and the performance of operative obstetric procedures (destructive operations, forceps delivery, vacuum extraction) when conditions are inappropriate and/or surgical technique is violated. Uterine rupture may occur during attempts to rotate the fetus in an advanced transverse lie, if the cervix is not fully dilated, during breech extraction, during release of arms extended behind the head, or during extraction of unrotated arms.

Uterine rupture may also occur in pregnant women exposed to external mechanical force (abdominal trauma, road-traffic accident, earthquake).

Overdosage of oxytocin (over 10 IU), especially in women older than 30 years, multiparous women, multiple pregnancy, macrosomia, and prolonged stimulation in the presence of a contracted pelvis, may contribute to rupture.

In our opinion, uterine rupture is not caused by the use of uterotonic agents per se, but rather by incorrect assessment of uterine condition (especially in the presence of a scar) and failure to diagnose deviations from the normal labor mechanism in time (clinically contracted pelvis, etc.).

At present, histopathological changes in the myometrium play a leading role in the genesis of uterine rupture. The greatest significance in this context is a defective uterine scar after cesarean section, the diagnosis of which is very

difficult. Morphologically confirmed defective uterine scars are found in 48–75% of women.

Clinical features and diagnosis

The clinical presentation of uterine rupture varies. Threatened, incipient (beginning), and completed rupture should be distinguished, since the maternal and fetal outcome depends on the stage.

In cases of clinical cephalopelvic disproportion, threatened uterine rupture is associated with excessive stretching of the lower uterine segment. This stage is characterized by intense, progressively strengthening uterine contractions; over time the contractions become more frequent, and the pauses between them shorten. The woman in labor becomes restless, screams, may attempt to move about, holds her abdomen, and asks for help. Her face is fearful, pupils dilate, lips and tongue become dry, pulse rate increases, and body temperature often rises. Due to compression of the bladder and urinary tract, urination becomes difficult.

On examination, sharp pain is elicited on palpation of the lower uterine segment; the segment is tense, and the Vasten sign is usually positive. Below the umbilicus or at the level of the umbilicus, a deep groove is detected running transversely or obliquely across the uterus (the retraction/constriction ring), representing a clearly defined boundary between the contracted uterine body and the excessively overstretched lower segment. The uterus assumes an “hourglass” shape.

Because of constant abdominal wall tension and frequent contractions, fetal heart sounds may be difficult to auscultate; acute fetal hypoxia develops and may lead to fetal death, since strong and prolonged uterine contractions disrupt uteroplacental circulation.

Vaginal examination often reveals complete cervical dilation and a pronounced caput succedaneum, or an edematous cervix compressed between the fetal head and pelvic bones.

In atypical uterine rupture (in the presence of a scar), the clinical picture is less pronounced. Contractions often become weak; there is pain on palpation of the lower segment in the scar area, thinning of the segment, usually increased uterine tone, and signs of fetal hypoxia are frequently observed. Ultrasound may detect scar insufficiency.

Most often, threatened uterine rupture is observed at the end of pregnancy in women with a uterine scar. Characteristic signs include epigastric pain, nausea, pain during fetal movements, tenderness of the uterine scar with thinning, and increased uterine tone. Ultrasound demonstrates thinning or deformation of the uterine scar.

The clinical picture of incipient rupture is determined by disruption of tissue integrity in the lower segment, vascular tearing, and hematoma formation in the uterine wall. The general condition worsens; the woman may cry out loudly and experience intense fear accompanied by pupillary dilation.

Contractions become paroxysmal, and the uterus does not relax between contractions. Painful bearing-down efforts may occur when the fetal head

remains high and the cervix is fully dilated. Bloody discharge may appear from the genital tract; hematuria may occur. Fetal heart activity becomes abnormal; increased fetal movements may occur followed by sudden fetal death.

If there is a uterine scar after cesarean section or other surgery, signs of rupture are often less pronounced than in clinically contracted pelvis. Epigastric pain appears, nausea, possible vomiting, dizziness, weakness; then pain develops in the lower abdomen, pain in the upper third of the vagina, pain in the area of the scar with thinning, and bleeding. Bloody discharge from the genital tract may occur; signs of fetal hypoxia are noted. Ultrasound shows thinning of the scar and irregular contours.

In the second stage of labor, typical signs of scar rupture include maternal anxiety, lower abdominal and sacral pain, painful straining, bloody vaginal discharge, and acute fetal hypoxia.

Completed uterine rupture is accompanied by several characteristic signs: acute abdominal pain at the peak of a contraction, sudden cessation of contractions ("calm after the storm"), signs of the torpid phase of shock, and internal bleeding. The woman becomes apathetic, assumes a forced position; attempts to change position increase pain. The condition deteriorates, the skin becomes pale, pulse becomes rapid and weak, blood pressure rapidly falls to critical levels, cold sweat appears, nausea, vomiting, and hiccups may occur. On palpation and percussion, abdominal tenderness is present, especially in the lower abdomen; the Shchetkin–Blumberg sign is positive. After rupture, meteorism develops rapidly and increases due to intestinal atony; vaginal bleeding occurs and increases when the presenting part rises. The fetus, if still alive, dies. If the fetus enters the abdominal cavity, the abdomen becomes irregular in shape and fetal small parts are easily palpated through the anterior abdominal wall. A well-contracted uterus displaced to the side of the fetus can be palpated.

During vaginal examination, the presenting part, previously tightly pressed at the pelvic inlet, rises and becomes mobile. Hematuria is often detected due to compression and venous congestion of the bladder mucosa or due to bladder wall injury (tear, rupture). With complete rupture, percussion may reveal free fluid (blood) in the abdominal cavity.

Incomplete rupture is usually localized in the lower segment, often along a scar, with hematoma formation between the leaves of the broad ligament or under the peritoneum. The hematoma may spread upward, reaching the hepatic region. Its formation is accompanied by lower abdominal pain, sometimes severe, radiating to the sacrum and leg.

With incomplete rupture, labor may not stop; in some cases, spontaneous delivery still occurs. Signs of shock and internal bleeding may be mild or sometimes absent. External bleeding may be absent. With significant blood loss, the woman's condition worsens: pallor, frequent weak pulse, and decreased blood pressure.

In all doubtful cases of suspected incomplete rupture, after delivery it is necessary to perform manual uterine exploration and examine the cervix and vagina with specula.

During vaginal–abdominal examination, if a parametrial hematoma is present, the uterus is displaced in the direction opposite to the rupture; on the side, an ill-defined mass may be palpated reaching the pelvic walls. Ultrasound helps establish the diagnosis of incomplete rupture.

In some cases of incomplete rupture, symptoms appear several days after delivery: pallor, tachycardia, hypotension, lower abdominal pain radiating to the sacrum and leg, meteorism, and fever.

Signs of rupture occurring in a morphologically altered uterine wall (scar) are similar to those of mechanical rupture, but they develop gradually and are mainly manifested by signs of hemorrhagic shock.

The clinical picture of incomplete rupture along a scar in the lower segment usually differs from rupture after cesarean section; in the latter, bleeding is generally more pronounced than in rupture of the lower segment.

With incomplete rupture along a scar (with intact peritoneum), significant bleeding is usually absent. This pathology can be detected by manual uterine exploration and ultrasound.

Separation of the uterus from the vaginal fornices is a very rare obstetric complication. The main cause is clinical mismatch between the presenting part and pelvic dimensions. Clinically, it manifests either as precipitous labor or, conversely, prolonged labor. The woman experiences severe pain in the lower abdomen and sacral area. The presenting part does not advance and is fixed at the pelvic inlet. The lower uterine segment is painful on palpation. Urinary retention is present; rarely, hematuria occurs. Moderate or heavy genital bleeding is observed. Vaginal examination shows an edematous cervix with incomplete dilation. A defect is detected in the vaginal walls extending into the parametrium.

The diagnosis of uterine rupture is based on a combination of the clinical signs listed above.

Differential diagnosis is made with clinically contracted pelvis, placental abruption of a normally located placenta, acute appendicitis, and amniotic fluid embolism.

During pregnancy (usually in the third trimester), symptoms of scar rupture may mimic acute appendicitis (nausea, vomiting, lower abdominal pain). In appendicitis, the tongue is coated, marked leukocytosis is present, and signs of peritoneal irritation are found in the right lower abdomen. To clarify the diagnosis, it is necessary to determine from the history the indications for cesarean section, the location of the uterine incision, and the postoperative course; perform ultrasound with complete evaluation of the abdominal wall; measure uterine thickness and assess the structure of the suspicious scar area. Threatened and incipient rupture are difficult to differentiate, since many symptoms overlap. It is known that grade III clinical disproportion, without timely intervention, ends in uterine rupture.

Cephalopelvic disproportion is characterized by:

- Painful bearing-down efforts with a high presenting part;
- Engagement of the head characteristic of the type of pelvic narrowing or often atypical;
- Marked head configuration or, in the postpartum period, lack of configuration, pronounced caput succedaneum;
- Complete cervical dilation and strong contractions without descent of the head (to the pelvic floor);
- Development of fetal hypoxia;
- Positive Vasten sign;
- Severe signs of bladder compression.

The above signs may also be present when there is a risk of uterine rupture.

Abruption of a normally located placenta, especially in the second stage of labor, is difficult to distinguish from incipient rupture in a morphologically altered uterus, because similar symptoms occur: abdominal pain, increased uterine tone, frequent bleeding of varying intensity from the birth canal, and acute fetal hypoxia. In such situations, ultrasound is very helpful.

Completed uterine rupture must be differentiated from amniotic fluid embolism, which most often occurs at the end of the first stage and in the second stage of labor. In embolism, shock develops rapidly with signs of acute cardiovascular failure, a sharp drop in blood pressure, cyanosis, tachypnea, and dyspnea.

Treatment

During pregnancy, uterine rupture is an indication for immediate delivery by cesarean section. During labor, if rupture is threatened or beginning, labor must be terminated urgently under deep anesthesia, followed by cesarean section. When opening the abdomen, serous fluid, swelling of the bladder, and hemorrhages in the uterine serosa may be found. The lower segment is usually thinned. The incision in the lower segment must be made carefully, without damaging the circular fibers, because it may extend into the vascular bundles. When opening the uterus, care must be taken not to injure the fetus. The fetus should be extracted carefully to avoid damaging or extending the incision. After removal of the fetus and placenta, the uterus must be carefully inspected. If the fetus is dead and conditions permit, destructive fetal extraction may be performed. However, if the physician is not skilled in this technique, cesarean section is preferable due to the risk of rupture. In transverse lie, regardless of fetal viability, cesarean section is advisable. Because of the risk of rupture, the following procedures are strictly contraindicated: internal version with extraction, application of forceps, and vacuum extraction. With extreme overdistension of the lower segment, if there is a scar in the lower segment, the incision is made along the scar; before repairing the uterine wound, scar tissue should be excised.

When uterine rupture is diagnosed, emergency surgery is indicated. Transfusion of blood components and blood substitutes should be started before surgery and continued intraoperatively and postoperatively until

hemodynamic stabilization, depending on the volume of blood loss. In the treatment chain, antibiotics, cardiotonic agents, nutrition, and patient care are of great importance.
The choice of surgical approach must be individualized, taking into account the time of rupture onset, signs of infection, the nature of rupture, and other factors.
After opening the abdomen and uterus, the fetus and placenta are removed; sometimes they are located within the abdominal cavity. If the rupture is small, linear, the torn edges can be easily refreshed, the rupture is recent, and the risk of infection is low, the rupture can be sutured. With extensive rupture, especially with crushed tissues and infection, extirpation of the uterus with tubes is usually performed.
In terminal condition, surgery may be performed in two or three stages: bleeding control, temporary surgical pause, and continuation of anti-shock measures.
When the uterus separates from the vaginal cavity, hysterectomy is indicated. If bladder rupture accompanies uterine rupture, it is repaired via the abdominal approach.
With complete rupture, first the peritoneum over the hematoma is incised and liquid blood and clots are evacuated. Bleeding is controlled by ligation of damaged vessels and suturing of injured tissues. After hemostasis, the rupture is examined and repaired, or the case is managed as a complete rupture.
During surgery, care must be taken to avoid injury to the iliac vessels and the ureter.
If bleeding cannot be controlled, ligation of the internal iliac artery is required.
In uterine rupture, anesthesia is one of the main components of emergency care, since it protects the woman from pain and additional trauma due to surgery, and helps prevent severe shock.
Even with timely obstetric care, fetal outcome in complete uterine rupture remains unfavorable, since it is associated with placental separation. Maternal outcome depends on the volume of blood loss, severity of shock, and the urgency and quality of skilled care.

X. HEMORRHAGIC SHOCK IN OBSTETRIC PRACTICE

Hemorrhagic shock is a complex of changes that develops in response to pathological blood loss and is characterized by progression of low blood flow syndrome, tissue hypoperfusion (decreased capillary blood flow), and the development of multiple system and multiple organ failure.

The risk of hemorrhagic shock arises when blood loss reaches 15–20% of circulating blood volume (CBV) (0.8–1.2% of body weight) or 750–1000 ml. Blood loss exceeding 1.5% of body weight or more than 25–30% of CBV is considered massive.

In pregnant women, women in labor, and postpartum women, the causes of hemorrhagic shock include bleeding in the following conditions:

- premature placental abruption,

- placenta previa,
- rupture of the uterus and soft tissues of the birth canal,
- true placenta accreta,
- uterine hypotonia,
- amniotic fluid embolism,
- intrauterine fetal death, and others.

The term “shock” is not a specific diagnosis, but a general name for a number of pathological conditions of various etiologies. Depending on the cause, shock is classified as traumatic, hemorrhagic, bacterial-toxic, cardiogenic, hypovolemic, hypo-/normo-/hypovolemic shock, and others.

Traumatic shock is the most common in obstetric practice and is associated with the presence of pain during labor, inadequate analgesia, as well as various manipulations (obstetric forceps, manual exploration of the uterine cavity, suturing of birth canal tears, etc.), and hemorrhagic shock.

Birth trauma and blood loss are two decisive factors determining the development of obstetric shock. In general, the term “shock” should be used only to describe severe conditions associated with trauma, blood loss, burns, infection, and similar causes. The concept of “shock” informs the physician about the severity of the patient’s condition. It is a signal for urgent treatment and the need for appropriate medical care.

From a pathophysiological standpoint, “shock” is a microcirculatory crisis in which microcirculation can no longer provide adequate tissue metabolism, meet tissue needs for oxygen and energy substrates, or remove toxic metabolic products.

Pathophysiological changes in the body in response to blood loss are referred to as hemorrhagic shock.

In obstetric practice, trauma and bleeding often occur together and simultaneously form a single pathophysiological process; therefore, they should always be considered as a whole. It should also be taken into account that many pregnant women have so-called “shock readiness” due to chronic circulatory, metabolic, and dystrophic changes associated with somatic diseases, preeclampsia, and other pregnancy complications.

Exhaustion, trauma, and pain during complicated labor reduce the woman’s tolerance to blood loss and significantly contribute to the development of hemorrhagic shock.

Pathogenesis of Hemorrhagic Shock

The key role in the pathogenesis of hemorrhagic shock is played by a decrease in circulating blood volume (CBV deficiency) and the mismatch between CBV and vascular capacity. CBV deficiency, in turn, contributes to reduced venous return to the right heart, decreased stroke volume and cardiac output, and a decline in arterial blood pressure (BP).

As a result, the following physiological adaptive reactions are activated:

- redistribution of blood within the vascular system;
- redistribution of fluid from the interstitial space into the bloodstream;

- activation of the vasomotor center and release of vasoactive substances into the bloodstream—catecholamines (adrenaline, norepinephrine, vasopressin, etc.), causing reflex peripheral vasospasm.

In blood loss, the first two mechanisms may compensate for up to 20% of CBV loss (800–1000 ml). Blood shifts from the vessels of the gastrointestinal tract, muscles, and subcutaneous tissue into the central circulation. Hemodilution also occurs due to entry of extracellular fluid into the bloodstream (autohydremia), leading to decreased hemoglobin concentration and reduced red blood cell count. If bleeding continues and blood loss reaches 25–30% of CBV (1000–1200 ml), these mechanisms become insufficient. In this situation, the leading defensive reaction is the third mechanism—peripheral vasospasm, which maintains central hemodynamics and blood pressure.

Venous constriction is particularly important, as veins contain up to 70% of the blood volume.

Prolonged vasospasm subsequently becomes one of the causes of irreversible shock. It consistently contributes to:

- opening of arteriovenous shunts;
- expansion of vascular capacity by opening additional capillaries that are normally inactive during physiological labor (10–15%). This leads to an even greater mismatch between blood volume and vascular capacity. As a result, progressive slowing of blood flow occurs in the peripheral vascular unit, tissue hypoanoxia develops, and poorly oxidized products (histamine) accumulate. At the same time, central hemodynamics deteriorate: venous return decreases again, cardiac output falls further, and arterial blood pressure declines.
- development of disseminated intravascular coagulation (DIC) syndrome and subsequent progression to irreversible shock.

Hypotension is the main symptom indicating decompensation of circulation.

When circulation decompensates in vital organs, metabolic acidosis develops due to anaerobic glycolysis (glycogen breakdown), leading to electrolyte imbalance, intracellular edema, subsequent membrane destruction, and cell death. These processes promote entry into the bloodstream of large amounts of acidic products and high concentrations of aggressive polypeptides with toxic properties. The described circulatory and metabolic disturbances may not resolve spontaneously even after bleeding has stopped if treatment is not initiated in time. Therefore, timely hemostasis and appropriate transfusion therapy are critically important for normalization of circulation in vital organs. Otherwise, as bleeding continues, hemodynamic and metabolic disorders progress.

Different organs are affected differently in hemorrhagic shock. First, pulmonary perfusion is impaired. Breathing becomes frequent and shallow (“shock lung”). Subsequently, the kidneys, liver, and pituitary gland are affected. Prolonged renal vasospasm leads to hypoxia and later necrosis, manifested as acute renal failure. In the liver, prolonged microvascular spasm leads to centrilobular necrosis and subsequent hepatic failure. The pituitary gland responds early to impaired or

reduced cerebral circulation. Prolonged pituitary microvascular spasm leads to hypoxia and subsequent necrosis (Sheehan syndrome).

Due to capillary spasm and microcirculatory impairment, placental blood flow is disrupted, and placental microvessels become occluded by thrombi and aggregates. A blockade of placental perfusion reserve occurs, leading to reduced fetoplacental blood flow and fetal hypoxia. This process later causes edema of uterine muscle cells, resulting in loss of contractile activity. Such a uterus does not respond to uterotonics, i.e., the "uterine shock" syndrome develops. The final stage of this syndrome is complete uterine paralysis, or the so-called "Couvelaire uterus."

In hemorrhagic shock, the functions of the central nervous system and the cardiovascular system are preserved for a relatively long time. Loss of consciousness in patients in shock occurs when arterial blood pressure falls below 60 mmHg for more than 2 hours. This indicates a terminal condition.

Clinical Presentation and Diagnosis of Hemorrhagic Shock

Clinically, depending on the volume of blood loss, four degrees of hemorrhagic shock are distinguished:

I — compensated,
II — subcompensated,
III — decompensated,
IV — irreversible.

To objectively assess the severity of blood loss, the following set of clinical and laboratory data should be considered:

- skin and mucosal color, respiratory and heart rate, blood pressure and central venous pressure (CVP), shock index (ratio of heart rate to systolic BP);
- minute urine output (diuresis), urine specific gravity;
- hematocrit, red blood cell count, hemoglobin level, acid–base status and blood gas values, water–electrolyte and protein metabolism;
- Lee–White clotting time, erythrocyte count and aggregation, fibrinogen, antithrombin III concentration, fibrin/fibrinogen degradation products, paracoagulation tests.

Grade I shock

Blood loss is compensated by changes in cardiovascular activity; in obstetric bleeding it is usually short-lived. Consciousness is preserved; pallor is present; tachycardia up to 100/min; blood pressure normal or slightly decreased; shock index ≥0.5; CVP 5–15 mmHg; hemoglobin ~100 g/L; hematocrit decreased; oliguria; iso- or hypercoagulation.

Grade II shock

Characterized by worsening circulatory and metabolic disorders. A fall in systolic blood pressure to a critical level (≈80 mmHg) is prominent; heart rate increases up to 120/min; respiratory rate ≥20/min; shock index ≥1.5; CVP <5 mmHg; hemoglobin ~80 g/L; hematocrit decreased; oliguria; Lee–White clotting time >10 minutes.

Grade III (decompensated) shock

Develops with large blood loss and is severe: systolic BP critically low (<80 mmHg), impaired consciousness, rapidly increasing pallor and "marbled" skin, heart rate ≥120/min, respiratory rate >30/min, shock index ≥2, CVP near 0. Hemoglobin ≤80 g/L, hematocrit decreased, anuria, Lee–White clotting time >15 minutes.

If decompensation persists for more than 12 hours, despite therapy, the process may become irreversible. Blood loss is extremely large (more than 50–60% of blood volume).

If there is no therapeutic effect, a terminal condition develops:

- preagonal state — pulse detected only in carotid/femoral arteries or by heart rate count; BP not measurable; breathing shallow and rapid; confusion;
- agonal state — loss of consciousness; heart rate and BP not measurable; severe respiratory distress;
- clinical death — cessation of cardiac and respiratory activity for 5–7 minutes.

A specific feature of obstetric hemorrhage is its sudden and massive nature. Therefore, the stages of hemorrhagic shock cannot always be clearly delineated. A pronounced clinical picture can be observed in acute placental abruption or uterine rupture, where within a short time (5–10 minutes) a severe, even agonal state may develop. In contrast, with prolonged bleeding due to uterine hypotonia, especially if recurrent in small portions, it may be difficult to identify the transition from compensation to decompensation. Sometimes an apparent "relative well-being" misleads the physician, and a critical deficit of blood volume may develop suddenly.

Diagnosis usually does not pose major difficulties, especially in the presence of external bleeding. However, early diagnosis of compensated shock—which ensures successful treatment—is sometimes overlooked due to underestimation of existing symptoms. Severity should not be assessed solely by blood pressure values or the amount of externally lost blood.

Hemodynamic adequacy should be evaluated using simple indicators:

- skin color and temperature, especially of the extremities;
- heart rate and blood pressure;
- Algower shock index (heart rate per minute divided by systolic BP; normal 0.5–0.8);
- hourly diuresis (normally 50–70 ml/hour);
- central venous pressure (normally 50–120 mm H_2O);
- hematocrit (normal 40–45%);
- acid–base status.

Skin color and temperature reflect peripheral blood flow. Warm, pink skin and pink nail beds, even with low BP, indicate adequate peripheral perfusion. Cold, pale skin with normal or slightly elevated BP indicates centralization of circulation and impaired peripheral perfusion—manifestation of microvascular spasm. Marble skin and acrocyanosis indicate deep impairment of peripheral circulation, vascular paresis, and an approaching irreversible state.

Pulse rate is an important indicator only when compared with other signs. Tachycardia during bleeding may reflect hypovolemia or acute heart failure; these conditions can be differentiated by measuring CVP.

Algower shock index is a simple and informative measure of hypovolemia severity: with a 20–30% decrease in CBV, it rises to 1.0; with 30–50% loss, it reaches 1.5. At an index of 1.0, the condition is alarming; at 1.5, life-threatening.

Hourly diuresis reflects organ perfusion. A decrease to 30 ml suggests peripheral circulatory insufficiency; below 15 ml indicates progression toward irreversible decompensated shock.

CVP is important for comprehensive assessment. A drop below 50 mm H_2O indicates severe hypovolemia requiring urgent volume replacement. If BP remains low during infusion therapy while CVP rises to 140 mm H_2O, this indicates cardiac decompensation and the need for cardiotonic therapy. Conversely, persistently low CVP suggests the need to increase infusion rate.

Hematocrit is a key test of adequacy of circulating volume. Against a shock index approaching 1.5, a hematocrit below 30% is an alarming sign; below 25% is dangerous. With further increase in shock index, it reflects severe blood loss. Rising hematocrit in combination with progressive increase in Algower index indicates irreversibility.

Acid–base evaluation (Zinggaard–Andersen, Astrup micromethod) is important in shock management. Hemorrhagic shock is characterized by metabolic acidosis, which may be combined with respiratory acidosis: plasma pH <7.38. In the terminal stage, alkalosis may develop: plasma pH >7.45.

Difficulties in estimating blood loss may be due to significant hemodilution of blood mixed with amniotic fluid, and due to retention of blood in large volumes in the uterus. For approximate assessment of blood loss in pregnant women, a modified Moore formula may be used:
$BL = M \times 75 \times 0.42 - Ht\_f / 0.42$,
where BL — blood loss (ml); M — body weight (kg); Ht_f — actual hematocrit.

Treatment of Hemorrhagic Shock

Treatment must be comprehensive, pathophysiologically grounded, and timely. The goal of emergency care and intensive therapy is to provide vital organs (brain, myocardium, lung tissue, etc.) with sufficient oxygen to maintain metabolic processes.

Pre-hospital care

In outpatient settings (e.g., antenatal clinic), pregnant women with signs of hemorrhagic shock should be kept in a horizontal position with legs elevated, warmed, and urgently transported to the nearest maternity hospital. In the ambulance, the medical team (physician, midwife, paramedic) must ensure breathing and circulation; notifying the duty staff of the maternity hospital by radio is advisable.

In-hospital care

In the emergency department of the maternity hospital, if the woman's condition permits, blood group, Rh factor, hemoglobin, and coagulation should be determined immediately. Otherwise, the woman in labor should be urgently transferred to the operating room where resuscitation and surgical care are provided in parallel.

Main elements of hemorrhagic shock management (according to M.O. Repina):

1. urgent control of uterine bleeding and elimination of other sources;
2. rapid compensation of blood loss and correction of central hemodynamic disorders by adequate and timely transfusion and infusion therapy;
3. correction of hemostasis in DIC syndrome;
4. correction of respiratory failure and general protection from pain and additional trauma—mechanical ventilation and full anesthesia;
5. prevention and treatment of consequences of microcirculatory disorders (acute renal and hepatic failure, myocardial and cerebral hypoxia).

Excluding any of these components from intensive therapy worsens the prognosis.

Success depends on timing. If the optimal time for bleeding control and restoration of circulation is missed, subsequent therapy, even aggressive, may be ineffective.

Bleeding source control must include an effective set of measures. Repeated instrumental examinations of the birth canal, repeated manual uterine explorations, repeated application of cervical clamps and sutures, etc., should be avoided.

If hypotonic bleeding persists and measures are ineffective (external uterine massage, uterotonics, gentle bimanual massage during manual uterine exploration), timely consideration should be given to ligation of the internal iliac artery or hysterectomy. In progressive placental abruption, placenta previa, and absence of conditions for vaginal delivery, immediate cesarean section is required. In Couvelaire uterus with DIC signs, hysterectomy is performed. In uterine rupture, urgent laparotomy with hysterectomy or repair is indicated. All manipulations and surgeries must be performed under adequate anesthesia with mechanical ventilation. After surgery, prolonged ventilation is required until full restoration of vital systems and hemostasis.

Correction of consequences of bleeding and replacement of blood loss are achieved through infusion therapy, where the rate, volume, and composition of solutions are decisive.

Given the time factor, initial therapy should always include colloid solutions with sufficiently high osmotic and oncotic activity combined with crystalloid plasma substitutes. These mobilize compensatory mechanisms by drawing fluid into the bloodstream from the interstitial space and provide time for preparation of blood components transfusion, which should be initiated as soon as possible in compliance with all rules.

Therefore, a rational approach is intravenous administration of colloid plasma substitutes capable of normalizing central and peripheral hemodynamics. Such agents include hydroxyethyl starch (e.g., volekam, stabizol, refortan), and derivatives of dextran and gelatin.

Using polyglucin and reopolyglucin, rapid increase in CBV can be achieved. These improve rheological blood properties, reduce stasis and aggregation of formed elements, optimize peripheral blood flow, and promote redistribution of blood. The

average daily dose of dextrans is 500–1000 ml. However, due to the risk of increased bleeding, dextrans are undesirable in DIC.

Currently, effective colloid plasma substitutes include refortan, infuzol, stabizol, which successfully support centralized circulation during the first 4–6 hours of hemorrhagic shock. The daily dose during the first day is 20–25 ml/kg; subsequently, if indicated, it should not exceed 50 ml/day.

Because excessive dosing may cause severe hemodilution with reduced hematocrit, hemoglobin, and plasma protein, if total protein decreases, mandatory albumin transfusion is indicated, followed by washed red blood cells or packed red blood cells.

Thus, plasma substitutes should be considered first-line therapy in acute blood loss with hemorrhagic shock.

To correct hypovolemia, infusion therapy also includes gelatin and polyethylene glycol preparations, which provide hemodilution without affecting platelet-vessel and coagulation hemostasis. The average dose is 500–1000 ml.

Blood and its components should be used at the second stage, after the bleeding source has been eliminated.

However, any colloid cannot replace the oxygen-carrying function provided by red blood cells. In modern practice, this is addressed with RBC-containing products: red cell mass, washed red cells, packed red blood cells, or red cell concentrate. Red cell mass should be of the same blood group, stored no longer than 3 days, and warmed to 37°C before infusion. If blood loss exceeds 1500 ml, the volume of infused RBC mass should be 0.5–0.8 of the blood loss volume.

For a controlled hemodynamic regime, transfusion of RBC-containing products should be combined with colloid and crystalloid solutions (Disol, Acesol, Trisol) in a ratio of 1:1 or 1:2. Any available solutions may be used. Plasma substitutes improve rheology, reduce aggregation, return pooled blood to active circulation, and improve peripheral perfusion.

Due to serious potential complications, whole blood transfusion is currently not recommended. Possible complications include:

- immunological conflicts;
- infection with hepatitis B and C viruses, herpes simplex, HIV, syphilis, etc.;
- intoxication due to metabolic disorders during blood storage.

In addition to RBC-containing products, other blood components are used. Concentrated albumin solutions (10% and 20%) are used to restore plasma colloid-oncotic pressure. Fresh frozen plasma is used to prevent or compensate for loss of coagulation factors.

If RBC-containing products of the required group and Rh are unavailable, a perfluorocarbon-based plasma substitute—perftoran—may be used to increase oxygen transport.

Perftoran has multifunctional effects:

- optimizes oxygen and carbon dioxide transport at low hemoglobin levels;
- restores central and peripheral hemodynamics;
- has a protective effect on the myocardium;
- acts as a membrane stabilizer.

Perftoran is administered at 5–30 ml/kg. It may be combined with albumin, glucose, and isotonic saline solutions, but must not be administered in the same infusion line with polyglucin, reopolyglucin, or hydroxyethyl starch. Under no circumstances should perftoran be used in coagulation disorders, which significantly limits its practical use.

Adequate therapy requires not only large infusion volumes but also high infusion rates. In severe stages, volumetric infusion rate should be at least 250–300 ml/min. Stage II shock requires infusion at 100–150 ml/hour. Such rates are possible via central venous catheterization. To avoid time loss, infusion should be started via cubital vein puncture while immediately initiating catheterization of a large vein (usually subclavian) to allow prolonged therapy. The infusion rate must be monitored by: skin color and temperature, pulse, BP, hourly diuresis, CVP, acid–base status, and ECG. Duration is strictly individualized.

Stabilization is characterized by:

- disappearance of cyanosis, severe pallor, and sweating; restoration of BP (systolic ≥90 mmHg),
- normalization of pulse filling, resolution of dyspnea, achieving hourly diuresis ≥40–50 ml without forced diuresis, increase of hematocrit to 28–30%.

After that, one may transition to drip infusion of RBCs and fluids in a 2:1 or 3:1 ratio. Drip infusion should continue for one day or longer until all hemodynamic parameters and the general condition are fully stabilized.

To improve redox processes, administration of 200–300 ml of 10–20% glucose solution with adequate insulin (1 unit per 5 g dry substance), 100 mg cocarboxylase, vitamins B and C, and normalization of acid–base balance and blood gases are indicated; antihypoxants support restoration of cellular metabolism and glucose utilization.

To reduce platelet aggregation by inhibiting thromboxane A2 synthesis, Thrombo ACC 50 mg is prescribed once daily in the postoperative period.

After correction of hypovolemia and improvement of rheology, an important component of microcirculation normalization is the use of agents that relieve peripheral vasoconstriction. A good effect is achieved by administering 100–150 ml of 0.5% novocaine solution with 20% glucose solution and insulin (1 unit per 5 g dry substance). Peripheral vasoconstriction can be eliminated with antispasmodics (papaverine hydrochloride 2% — 2–4 ml; no-spa 2% — 2–4 ml; eufillin 2.4% — 5–10 ml) or ganglion blockers such as pentamine (1 ml for 200 ml of 0.5% saline) and benzohexonium (1 ml of 2.5% solution) in drip infusion.

To reduce renal vascular resistance and increase renal blood flow, dopamine is indicated (5 ml of 0.5% solution diluted in 150 ml of 5% glucose) at 5–10 drops/min. To improve renal perfusion, 10% mannitol solution in a volume of 400 ml is indicated. These agents must be used under control of BP, CVP, and diuresis. If needed, Lasix is prescribed.

Important components of therapy include antihistamines (2 ml of 1% diphenhydramine; 2 ml of 2% suprastin; 2 ml of 2.5% pipolfen), corticosteroids (hydrocortisone 125–250 mg, prednisolone 120–150 mg), and cardiotonic drugs

(strophanthin 0.5–1 ml of 0.05% solution or 1 ml of 0.06% corglycon solution with 10–20 ml of 40% glucose).

In hemorrhagic shock, coagulation disorders are almost always present, such as DIC syndrome. Mild hypercoagulation may occur, while in moderate and severe cases consumptive coagulopathy and activation of fibrinolysis develop, i.e., deficiency coagulopathy.

Therefore, coagulation must be restored. Many authors have developed principles for including medications into intensive therapy aimed at correcting coagulopathy:

- use of protease inhibitors (contrical, gordox) to suppress excessive fibrinolysis;
- refusal to use heparin to prevent intravascular coagulation;
- replacement therapy by administering large volumes (at least 1 liter/day) of fresh frozen plasma;
- stimulation of the vascular-platelet component of hemostasis (dicynone, etamsylate, ATP);
- use of tranexamic acid 500–750 mg per 500 ml of NaCl solution to suppress plasmin activity, stabilize coagulation factors, and prevent fibrin degradation.

It should be emphasized that hemorrhagic shock treatment must be carried out under mandatory monitoring of the hemostasis system with the participation of a hemostasiologist.

Thus, the time factor is decisive in hemorrhagic shock management. The earlier treatment begins, the less effort is required to reverse shock, and the better the immediate and long-term outcomes.

Removal of the patient from a severe condition associated with hemorrhagic shock is the first stage of treatment. In subsequent days, therapy continues to eliminate the consequences of massive blood loss and prevent new complications. Medical measures during this period aim to monitor kidney, liver, and cardiac function; normalize water-salt and protein metabolism; treat anemia; and prevent infectious complications.

XI. BASIC PRINCIPLES OF TREATMENT OF OBSTETRIC HEMORRHAGE

Therapeutic measures undertaken for obstetric hemorrhage—during pregnancy, labor, the placental stage, and the postpartum period—regardless of the timing and cause of the event, must begin with the main priority: stopping the bleeding and eliminating its cause. In progressive acute placental abruption and placenta previa, a cesarean section must be performed in a timely manner. In hypotonic and atonic hemorrhage, when conservative measures are ineffective (external–internal uterine massage with manual exploration of the uterine cavity and fist massage of the uterus, administration of uterotonic drugs, balloon tamponade, etc.), escalation is required.

If blood loss is 800–1000 ml, the question of surgical intervention should be raised: injection of prostenon into the uterine musculature; achieving uterine ischemia by applying clamps and ligatures to the vascular bundles of the uterine and

ovarian arteries; application of a hemostatic agent; compression sutures according to B-Lynch and Pereira; ligation of the internal iliac arteries.

If blood loss exceeds 1500 ml, it is recommended to immediately perform ligation of the internal iliac arteries, uterine amputation or extirpation, urgent repair of birth canal tears, and in cases of uterine rupture—suturing of the rupture site or hysterectomy.

Algorithm for Assistance in Postpartum Hemorrhage (Gordeeva G.D., 2008)

Stage 1 — Initial assessment

Identify the cause:

- assess uterine tone
- examine the birth canal
- assess the condition of blood clots

Actions of the anesthesiologist-resuscitator:

- catheterize one or two veins
- monitor blood pressure, pulse, respiratory rate, diuresis
- bladder catheterization with an indwelling catheter
- oxygen mask
- assess oxygen saturation (StO_2)

Laboratory tests:

- complete blood count with platelet count
- hematocrit
- coagulation profile (coagulogram)
- blood group and Rh factor

Stage 2 — Hypotonic bleeding: search for the “4T” causes

“Tissue” — retained placental fragments:

- manual exploration of the uterine cavity
- curettage

“Tone” — uterine hypotonia:

- external uterine massage
- manual uterine examination
- administration of uterotonics

“Trauma” — tears of the soft birth canal / uterine rupture:

- diagnosis of traumatic injuries and repair

“Thrombin” — congenital or acquired coagulation disorders:

- restore circulating blood volume (CBV)
- restore coagulation factors

Stage 3 — Massive postpartum hemorrhage

Actions of the anesthesiologist-resuscitator:

- intensive therapy and resuscitation
- anesthetic management
- monitoring BP, pulse, respiratory rate, diuresis

Preparatory measures:

- manual compression
- preparation for surgery

Stage 4 — Surgical treatment

Actions of the anesthesiologist-resuscitator:

- intensive therapy and resuscitation
- anesthetic management
- monitoring BP, pulse, respiratory rate, diuresis

Surgical measures:

- uterine compression sutures
- bilateral ligation of uterine arteries
- bilateral ligation of internal iliac arteries
- hysterectomy

Stage 5 — Bleeding after hysterectomy

Actions of the anesthesiologist-resuscitator:

- intensive therapy and resuscitation
- anesthetic management
- monitoring BP, pulse, respiratory rate, diuresis

Surgical measures:

- ligation of internal iliac arteries (if not performed)
- tight vaginal and abdominal pelvic packing (tamponade)
- the abdominal cavity should not be closed until bleeding stops

Therapeutic measures performed in parallel with bleeding control must be based on three main principles:

- timely initiation of treatment;
- an integrated approach;
- intensity of therapy proportional to the severity of the pathological process.

The success of hemorrhage management depends on the level of organization of obstetric hospital work and on a clearly developed system of staff actions. Risk groups must be identified to allow timely preventive measures that reduce the frequency of hemorrhage and the severity of its consequences. Early connection of an intravenous line in women in labor ensures timely administration of oxytocin and methylergometrine at the moment of delivery of the parietal tubercles of the fetal head, and also enables immediate initiation of infusion therapy at the first signs of pathological blood loss.

Among other organizational preventive measures important for comprehensive care are: constant readiness of sterile IV infusion sets; availability of kits for peripheral and central venous catheterization; preparedness of the anesthesia nurse in the operating room; availability of an anesthesiologist, resuscitator, and transfusiologist; and ensuring adequate supplies of infusion and transfusion agents.

The key tasks of ongoing therapeutic measures are largely solved through infusion–transfusion therapy. The main goal of infusion–transfusion therapy is rapid restoration of capillary perfusion, preventing failure of compensatory mechanisms with the development of shock changes. This requires creation of an effective circulating blood volume and normalization of microcirculation. After this, attention is directed to normalization of the respiratory function of blood and to prevention and treatment of coagulation, water–electrolyte, metabolic, and other disorders.

The rate, volume, and quality of infusion–transfusion therapy must correspond to the clinical situation. As noted above, obstetric hemorrhage is characterized by sudden onset and massive volume; therefore, time is critical. Delayed onset of surgery and an infusion rate lagging behind the rate of blood loss are decisive factors in the development of hemorrhagic shock. The infusion rate depends on the speed and volume of blood loss and the woman's condition; in severe cases with clinical signs of hemorrhagic shock and falling blood pressure, it should reach at least 250–400 ml per minute. To ensure high infusion rates, therapy should be administered simultaneously through two peripheral veins or central access (subclavian), strictly under central venous pressure control (normally 80–120 mm H_2O).

An important organizational point at the start of infusion therapy in obstetric hemorrhage is collecting blood for determining blood group and Rh status, compatibility with donor blood, complete blood count, and coagulation profile. These data are necessary to determine the nature of bleeding (coagulopathy), monitor dynamics, and assess treatment effectiveness.

To date, it has been proven unjustified to use stored whole blood as the first and leading component of infusion therapy. Donor blood is essentially a transplant. Compatibility between donor and patient blood is determined only by the ABO system and Rh factor. Another major issue is the high risk of infection during transfusion (hepatitis, HIV infection, syphilis, etc.). Stored blood has reduced functional properties: during the first 2 days, leukocytes and platelets die; by days 3–4, the oxygen-transport function of red blood cells decreases sharply, partial hemolysis occurs due to reduced hemoglobin affinity for oxygen. Microclots up to 200 μm form in stored blood; during passive transfusion they can obstruct pulmonary capillaries, worsening function. In addition, about one third of transfused blood pools in the microcirculatory bed and is excluded from active circulation.

Based on the above, at the initial stages of obstetric hemorrhage management, transfusion of stored whole blood is neither an effective nor a safe method. Correction of the consequences of blood loss is achieved via infusion therapy, in which the composition of administered agents, their infusion rate, and total volume are of great importance.

The volume of infusion is determined by the following indicators: blood pressure level, heart rate, central venous pressure, hemoglobin, hematocrit, red blood cell count, diuresis, clotting time.

During infusion therapy, systolic BP should not be below 80–90 mmHg, CVP should not be below 30–40 mm H_2O and not above 120 mm H_2O, hematocrit should not be below 25%, red blood cells should not be below 2 million, clotting time should not exceed 8–10 minutes, and diuresis should be at least 30 ml/hour. When CBV is restored but oliguria persists, diuresis should be stimulated by administration of Lasix and furosemide. If there is no response and persistent anuria remains, ligation/injury of the urinary tract during hysterectomy must be excluded. If in doubt, relaparotomy is preferable to exclude ureteral obstruction.

Rapid replenishment and restoration of circulation in parenchymal organs is achieved by administering plasma substitutes such as refortan, stabizol, infuzol,

ethoxylated starch (volecam). These agents are similar to human glycogen and are degraded by blood amylase.

High-molecular-weight dextrans may be widely used—polyglucin, reopolyglucin, reogluman, gelatinol, and 5% albumin solution. Polyglucin rapidly increases blood volume and helps restore hemodynamics. After elimination of the bleeding source, the use of the low-molecular-weight dextran reopolyglucin is advisable because it reduces aggregation of blood cells, restores microcirculation in small capillaries, and improves organ perfusion. All these agents have high colloid osmotic pressure, draw fluid into the vessels, and retain intravascular fluid for a prolonged time. In this way, by activating the autogemodilution response, vascular filling accelerates, blood viscosity decreases, and a disaggregating effect develops, which leads to rapid restoration of microcirculation and prevents the development of disseminated intravascular coagulation

Table 15
Approximate Volume of Infusion Therapy Based on Blood Loss

Blood Loss as % of Body Weight	Blood Volume (ml)	Total Infusion Volume (% of blood loss)	Of Which: Erythrocyte Mass
0.6 – 0.8%	400–600 ml	80–100%	—
0.8 – 1%	600–800 ml	130–150%	—
1 – 1.5%	800–1200 ml	150–180%	30% (300–400 ml)
1.5 – 2%	1200–1600 ml	180–200%	40–50% (600–800 ml)
2 – 2.5%	1800–2000 ml	220–250%	50–60% (800–1200 ml)
More than 2.5%	>2000 ml	250–300%	70–80% (≥1500 ml)

Plasma plays a major role in the treatment of obstetric hemorrhage. Currently, fresh frozen plasma (FFP) and cryoprecipitate are used at the beginning

of therapy to replace blood loss. Fresh frozen plasma is administered at a dose of 15 ml/kg of body weight per day. After warming to 37°C, 60% of the daily dose is given intravenously as a bolus, and the remaining volume is infused by drip.

To stop and prevent disseminated intravascular coagulation (DIC), protease inhibitors such as **Gordox** and **Contrical** should be administered simultaneously with infusion therapy. The daily dose of Contrical should be at least 200,000–300,000 units, and Gordox — 2,000,000 units. The initial dose of Contrical is 10,000 units (10 ampoules), and Gordox — at least 500,000 units. If coagulation is not restored within 1 hour after the initial dose, the dose may be repeated. After coagulation is achieved, the remaining doses are administered every 4–6 hours. The duration of therapy should be at least 2–3 days.

As a result of hemodilution caused by infusion and stimulation of capillary circulation, hemoglobin levels decrease. However, the human body has a hemoglobin reserve that allows it to tolerate the loss of more than half of its volume, whereas a one-third reduction in plasma volume may lead to irreversible consequences.

At the same time, under conditions of hemodilution, the restoration of microcirculation and utilization of the remaining circulating erythrocytes allows maintenance of adequate hemoglobin levels for a certain period.

Therefore, after stabilization of hemodynamic and microcirculatory parameters (stable blood pressure, adequate diuresis, clear consciousness, absence of respiratory failure, etc.), the next important stage of treatment is correction of severe anemia. For this purpose, when blood loss exceeds 1% of body weight, transfusion of packed red blood cells or washed erythrocyte suspension in an amount of 25–30% of the lost blood volume is indicated. The remaining volume should be replaced with plasma-substituting solutions and dextrans.

An important component of therapy for obstetric hemorrhage is the administration of **glucocorticoid hormones**, which are prescribed at the beginning of infusion therapy in cases of massive blood loss. Hydrocortisone 200–250 mg or prednisolone 120–150 mg is administered intravenously.

Thus, timely and adequate infusion therapy after bleeding control prevents severe hemodynamic disturbances and helps restore the function of vital organs.

Basic Emergency Management Protocol

The protocol includes both conservative and surgical stages of bleeding control.

Conservative Stage:

- Manual exploration of the uterine cavity and bimanual uterine compression. These procedures should be performed as carefully as possible.
- Administration of uterotonics (oxytocin, methylergometrine, prostaglandin F2α).
- Intermediate measures between conservative and surgical stages (intrauterine hemostatic balloon tamponade, compression of the abdominal aorta).

During the conservative stage, the choice of intervention depends on the cause of bleeding. In most cases, manual exploration of the uterine cavity is necessary. If the integrity of the placenta and uterine wall is confirmed, bimanual compression is

recommended. Examination of the birth canal allows exclusion of bleeding from traumatized tissues. Persistent bleeding after exclusion of the main causes suggests primary or secondary hypocoagulation.

Manual exploration of the uterine cavity and bimanual compression have both advantages and disadvantages. The main advantage of manual exploration is verification of uterine integrity and exclusion of retained placental fragments. Bimanual compression carries no infection risk and does not require anesthesia.

The use of uterotonics is mandatory in the treatment of obstetric hemorrhage. The first-line drug should be **oxytocin**, as other uterotonics have more side effects. Ergometrine preparations are contraindicated in hypertensive conditions and heart disease. Prostaglandins are contraindicated in hypertension, glaucoma, or bronchial asthma. However, in the absence of contraindications, any uterotonic may be used.

Particular attention should be paid to oral prostaglandin preparations such as **misoprostol (Mirolut, Cytotec)**, which, due to its properties, is considered a drug of choice in developing countries.

Intermediate measures between conservative and surgical stages include intrauterine balloon tamponade and, if possible, abdominal aortic compression according to Biryukova (Fig. 29).

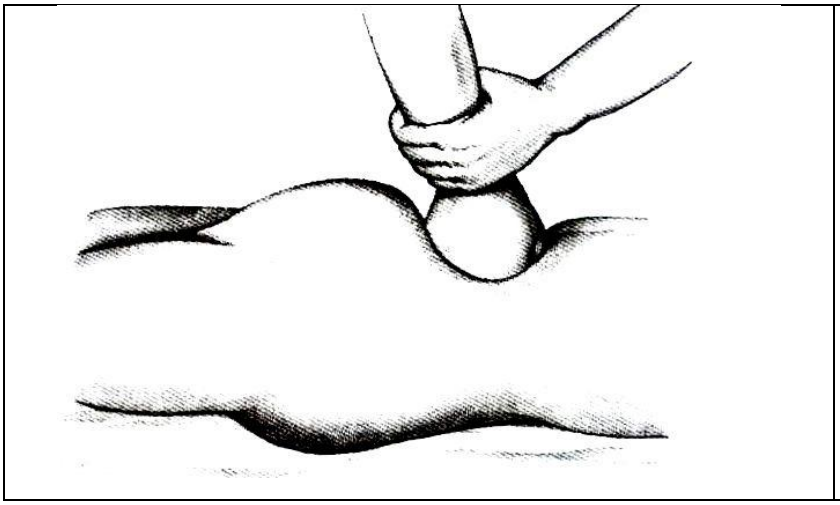


Figure 29. Manual Compression of the Aorta

The intrauterine balloon is a simple device consisting of the balloon itself, a reservoir, and a hollow connecting tube between them. Balloon tamponade has several advantages: ease of use, rapid application, convenient monitoring of the situation, sterility, atraumatic nature, and no need for anesthesia. In addition, the balloon can be inserted by an obstetrician, which saves time between the conservative and surgical stages of management.

Algorithm for Controlled Balloon Tamponade

The main indications for balloon tamponade in postpartum hemorrhage are:

- Hypotonic bleeding after vaginal delivery or during cesarean section
- Vaginal tamponade in case of vaginal laceration or hematoma (as a temporary measure)

Balloon tamponade may also be used prophylactically in the following situations:

- During cesarean section for placenta previa
- Placenta accreta
- Multiple pregnancy, polyhydramnios, large fetus
- Hypotonic bleeding

Currently, two types of balloons are available for controlled tamponade in the prevention and treatment of obstetric hemorrhage:

- **Uterine balloon catheter**
- **Vaginal balloon catheter**

Each of these catheters can be used independently or in combination.

Control of Hypotonic–Atonic Postpartum Hemorrhage
Uterine Balloon – 10 Steps

1. Position the reservoir 45–50 cm above the level of the uterus.
2. Fill the reservoir with warm, sterile 0.9% sodium chloride solution.
3. Clamp the tube.
4. Expose the cervix using a speculum, grasp it with forceps, and under aseptic conditions and visual control insert the balloon catheter into the uterine cavity up to the fundus.
5. Maintain contact of the distal end of the catheter with the uterine fundus; connect its open end to the reservoir tubing.
6. Open the clamp.
7. Fill the reservoir with solution, maintaining the fluid level at mid-volume.
8. Continue adding solution until the level stabilizes in the reservoir, while holding the uterine catheter in contact with the fundus. In most cases, 350–400 ml of solution is sufficient. Ultrasound is recommended to confirm balloon expansion within the uterine cavity.
9. If the hemostatic effect is insufficient, raise the reservoir by an additional 10–15 cm to increase pressure of the balloon wall in the placental bed area.
10. The balloon promotes restoration of uterine tone and contractile activity.

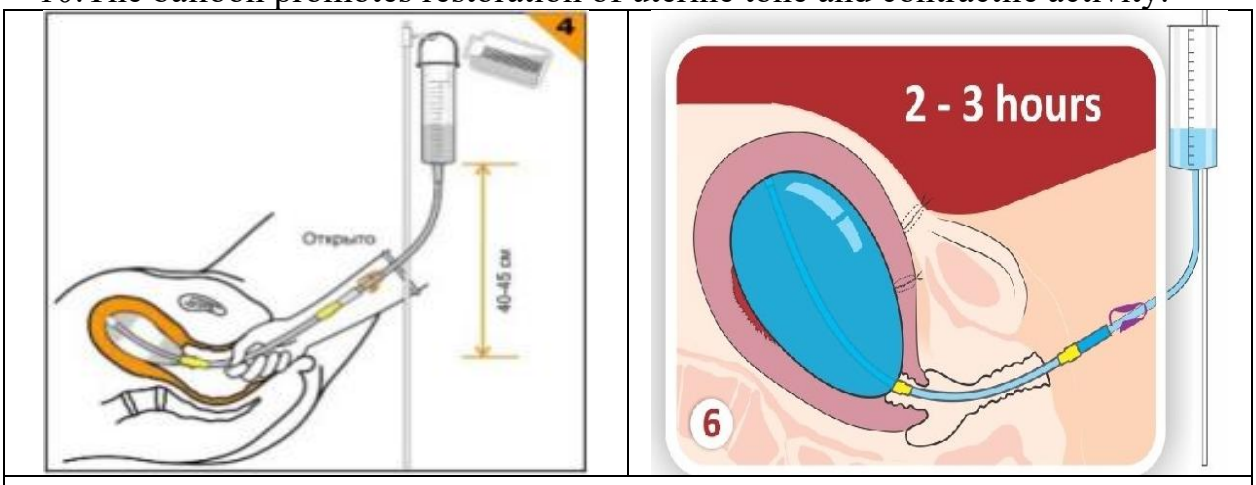


Figure 30. Filling of the Uterine Balloon Inserted for Postpartum Hemorrhage

Therefore, usually within **15–60 minutes**, the solution from the reservoir begins to flow into the balloon. This can be assessed by observing the rise of the fluid level in the container.

After bleeding has stopped, and when the balloon either spontaneously descends into the vagina or remains inside the uterine cavity along the catheter, the controlled balloon tamponade procedure may be considered complete. After this, the catheter can be removed from the uterine cavity.

When treating postpartum hemorrhage (PPH) with a closed cervix, the effectiveness of balloon tamponade exceeds **95%**.

Control of Hypotonic Postpartum Hemorrhage
Double-Balloon Technique

(Increases tamponade effectiveness and prevents premature expulsion of the uterine balloon)

(Figure 27)

- Insert the uterine balloon.
- Insert the vaginal catheter into the vaginal cavity along the balloon catheter.
- Inflate the vaginal catheter using a syringe (150–180 ml).
- Inflate the uterine catheter using the method described above.
- Monitor the amount of genital tract discharge and the fluid level in the reservoir.
- After 4–6 hours, remove the fluid from the vaginal cuff.
- Lower the reservoir level, deflate the uterine balloon, and remove it.

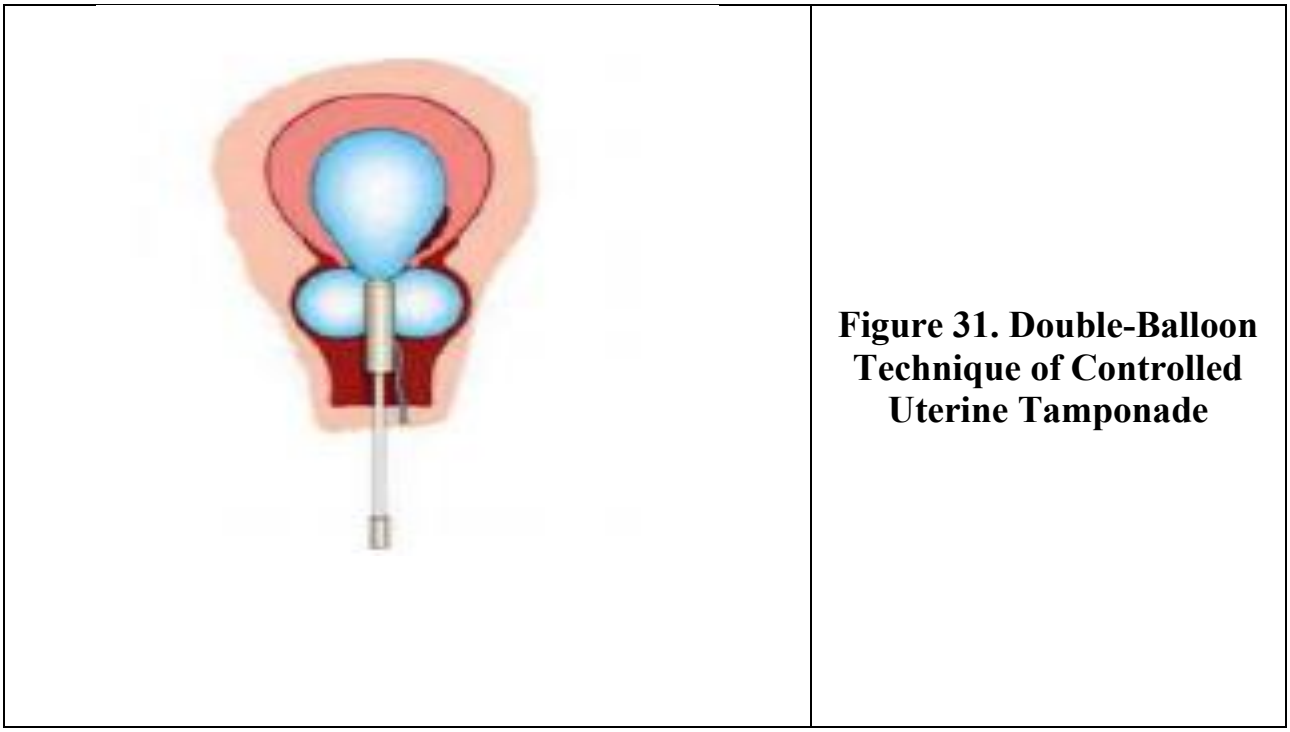


Figure 31. Double-Balloon Technique of Controlled Uterine Tamponade

 Place the reservoir on the wall **50 cm above the level of the uterus**, clamp the tube, and fill it with warm sterile saline solution.

 The surgeon inserts the uterine catheter retrogradely through the hysterotomy incision using a guide. Advance the balloon catheter in a cranial direction until it contacts the uterine fundus.
 Suture the uterine incision carefully, ensuring that the balloon is not punctured.
 While holding the balloon in place, the assistant removes the guide, connects the balloon to the reservoir tubing, opens the clamp, and fills the balloon with **300–400 ml** of solution.
 At the end of surgery, transfer the patient to the ICU with the clamp closed.
 Open the clamp and monitor the fluid level in the reservoir. When the level rises by two divisions, lower the balloon to the same height. Repeat this maneuver until a height of 10–15 cm is reached.
 Remove the balloon after 2–3 hours or once it reaches a height of 10–15 cm above the uterine level.

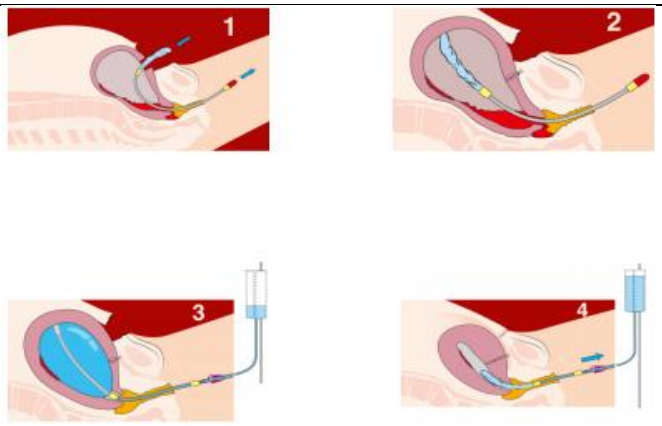


Figure 32. Technique of Uterine Balloon Tamponade During Cesarean Section

Here is the English translation of your text, preserving the academic and clinical style:
Controlled Double-Balloon Tamponade Technique for Cesarean Section
Preoperative stage:

- Insert the vaginal module;
- Advance it along the vaginal axis to the fornix (minus 1 cm);
- Connect the prepared syringe without inflating the cuff.

Stage II during cesarean section: immediately after placental separation

- Inflate the vaginal module cuff with warm saline solution (150–180 ml).

Stage III during cesarean section:

- Insert the uterine catheter using the method described above.

Post-cesarean stage:

- Monitor the amount of genital tract discharge and the fluid level in the reservoir;
- After 4–24 hours (depending on the severity of the condition and confirmed hemostasis), remove the fluid from the vaginal module cuff, lower the reservoir level, deflate both uterine and vaginal balloons, and remove them.

Postpartum Vaginal Tears and Hematomas

After surgical management, when complete hemostasis cannot be achieved (suturing of lacerations, hematoma evacuation), a vaginal balloon may be used as a temporary or adjunctive measure. When inflated and positioned in the vagina, tamponade is maintained for 24–36 hours depending on the severity of the vaginal injury. It is necessary to monitor genital tract discharge and ensure continuous bladder catheterization.

Modern practitioners also use lateral uterine wall compression according to N.S. Baksheev. According to published data, this method may serve as a definitive hemostatic procedure in up to 75% of cases. However, in our practice, we do not use this technique due to the risk of ureteral and venous plexus injury, especially during prolonged compression (up to 2 hours).

Rapid Hemostasis Assessment

For quick evaluation of hemostasis, a "bedside clot test" is recommended. A test tube containing venous blood from the patient is warmed manually. If no clot forms within 7 minutes, or if the clot is loose and easily fragmented, hypocoagulation should be suspected.

Surgical Stage (Laparotomy)

- Injection of prostaglandins into the uterine muscle (e.g., Prostenon);
- Induction of uterine ischemia by applying clamps and ligatures to the uterine and ovarian vascular bundles;
- Application of hemostatic compression sutures (B-Lynch, Pereira, etc.);
- Bilateral internal iliac artery ligation (recommended immediately if blood loss exceeds 1500 ml);
- Angiographic embolization, if equipment and trained personnel are available;
- Uterine amputation or hysterectomy.

Post-Cesarean Hypotonic Hemorrhage

The incidence of hypotonic bleeding after cesarean section is 3–5 times higher than after spontaneous vaginal delivery.

If conservative measures are ineffective (uterotonics, external uterine massage, manual exploration with gentle bimanual compression, Baksheev clamps — acceptable only if blood loss does not exceed 1.5% of body weight), transition to the surgical stage is indicated.

Previously, continued bleeding under these conditions was considered an absolute indication for hysterectomy. Our experience suggests a more critical approach: laparotomy may be indicated, but hysterectomy — especially in primiparous women — should not be automatic.

Two essential principles must be considered:

1. Ensure integrity of the birth canal tissues.
2. Preserve uterine and reproductive function whenever possible.

The primary goal of the surgical stage is rapid and definitive hemostasis, regardless of uterine contractility.

If uterine bleeding is confirmed, prostaglandins (dinoprostone 0.25 mg IM) are administered.

If blood loss exceeds 1500 ml, internal iliac artery ligation is indicated first. If blood loss is 1000–1500 ml or iliac ligation is not feasible, uterine vessel

ligation (a. uterina, a. ovarica) and application of compression sutures (B-Lynch, Pereira, Hayman, Cho) are performed.

Uterine ischemia via ovarian and ascending uterine artery ligation is relatively simple and fast. Exteriorization of the uterus facilitates accurate ligation using diaphanoscopy of the round and cardinal ligaments.

Hemostatic Compression Sutures

These sutures are widely used internationally but remain underutilized locally.

Principle: placement of absorbable sutures around the uterine body in longitudinal or transverse planes to achieve compression.

Advantages of B-Lynch:

- Rapid application
- No special equipment required

Advantages of Pereira:

- Also rapid and equipment-free
- Allows controlled tension distribution
- Improved hemostatic conditions

Disadvantages:

- Risk of venous plexus or ureter injury (Pereira)
- Technical difficulty in tension control (B-Lynch)
- Possible uterine wall cutting or suture rupture
- Requires an assistant
- Risk of re-bleeding requiring relaparotomy

Organ-Preserving Principle

Management of obstetric hemorrhage must prioritize uterine preservation whenever possible. A woman leaving the maternity hospital without her reproductive organ should be an exception, not a routine outcome.

The most effective organ-preserving method is bilateral internal iliac artery ligation or embolization (if technically feasible). These procedures must be performed by highly qualified specialists.

Some researchers consider internal iliac artery ligation an alternative to hysterectomy for massive obstetric hemorrhage, with effectiveness exceeding 95%. Unlike other methods that mainly address hypotonic bleeding, iliac ligation is also effective in coagulopathic bleeding and DIC syndrome.

Prevention of Obstetric Hemorrhage

- Timely treatment of genital inflammatory diseases and prevention of abortions;
- Rational management of high-risk pregnancies;
- Prevention and treatment of hypertensive disorders;
- Proper labor management and timely operative decisions;
- Active management of the third stage of labor (oxytocin or ergometrine IM after anterior shoulder delivery);
- Proper postpartum monitoring (bladder emptying, uterine massage, placental inspection);
- Accurate blood loss estimation and adequate analgesia.

Prevention of Coagulopathic Hemorrhage

- Early prevention and treatment of pregnancy complications (hypertension, placental abruption, anemia);
- Timely management of obstetric hemorrhage and autologous plasma donation;
- Mandatory hemostasiological monitoring during pregnancy.

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