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BLEEDING DURING PREGNANCY, CHILDBIRTH AND THE POSTPARTUM PERIOD

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BLEEDING DURING PREGNANCY, CHILDBIRTH AND THE POSTPARTUM PERIOD

MONOGRAPH

Compilied by

Kh.SH.Shavkatov

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This monograph has been prepared in accordance with the current scientific and clinical requirements in the field of obstetrics and is intended for obstetrician-gynecologists, researchers, master's degree students, clinical residents, and specialists working in the field of maternal and perinatal health. The monograph has been developed taking into account the latest achievements of modern obstetrics, the principles of evidence-based medicine, as well as the climatic, demographic, and social conditions of our Republic.

This monograph is devoted to the scientific and practical aspects of obstetric hemorrhage occurring during pregnancy, childbirth, and the early postpartum period. It provides a comprehensive analysis of the etiology, pathogenesis, classification, clinical course, modern diagnostic methods, and issues of differential diagnosis of obstetric hemorrhage.

The monograph presents the main conservative and surgical methods for the treatment and prevention of obstetric hemorrhage, emergency care algorithms, principles of intensive therapy, rehabilitation approaches, and modern management strategies based on international and national clinical guidelines. It serves as an important scientific and practical source for researchers, clinical residents, practicing physicians, and specialists in obstetrics and gynecology.

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INTRODUCTION

The main indicators of population health depend primarily on social conditions. If maternal mortality is considered separately, it is related to living conditions, the health status of pregnant women, and the level of medical care.

It is known that one of the major problems on which many scientists are working is obstetric hemorrhage. The significance of the problem is associated with the fact that obstetric hemorrhage is one of the leading causes of maternal mortality. In obstetrics, hemorrhage accounts for 25.8% as a direct cause and 78% as a secondary cause. According to the American College of Obstetricians and Gynecologists, one woman dies from bleeding every 4 minutes worldwide.

In Uzbekistan, obstetric hemorrhage has always been one of the main causes of maternal mortality, averaging 26%, and it remains an important problem of obstetric care. In addition, postpartum hemorrhage continues to occupy a leading place in the structure of causes of maternal mortality in our country.

The cause of maternal death due to bleeding is hemorrhagic shock and its complications. In this regard, the initial actions in blood loss, early cessation of bleeding, and the fight against postpartum hemorrhage are the main issues, and they have a significant influence on subsequent outcomes.

In obstetric practice, the problem of the strategy and tactics for replacing acute blood loss is particularly relevant. Depending on how this problem is solved, one can judge the qualification of medical personnel and the organization of emergency medical care in a particular obstetric institution.

The practical experience of treatment and prevention institutions in obstetrics and gynecology shows that the greatest difficulties for a physician are clinical situations requiring emergency medical care. In such cases, first of all, the issue is often saving the patient's life; therefore, incorrect or untimely actions, as well as mistakes in choosing the tactics, methods, and means of emergency care, can lead to serious and even tragic consequences. Conversely, emergency medical care that is provided correctly and in a timely manner, rationally planned, and performed using accessible methods can save not only the patient's life but also preserve her reproductive function.

A practicing physician must develop constant readiness to provide emergency care to patients with bleeding. The role of subjective factors, such as the physician's appropriate psychological attitude, sufficient theoretical training, and clear knowledge of methods for emergency diagnosis, prevention, and treatment of bleeding, cannot be ignored.

All comments and suggestions aimed at improving the structure and presentation of the material will be accepted by the authors with deep gratitude.

I. TOPOGRAPHIC ANATOMY OF THE PREGNANT UTERUS

If an incision is made in the abdominal cavity, either transverse or longitudinal, when the abdominal cavity is opened one can see a pregnant uterus of ovoid shape, located in the anterior part of the abdominal cavity and slightly to the right.

The uterus extends deeply toward the edge of the liver on the right side, 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 covers the cecum and displaces it upward and backward.

Thus, at the end of pregnancy, the cecum is located not in the right inguinal region as in non-pregnant women, but on the right side, above the umbilicus, and often near the lower border of the liver. The left border of the anterior uterine wall is covered at the level of the iliac fossa by intestinal loops pressed to the left, the fossa iliaca, which come into contact with the sharply protruding fold of the sigmoid colon anteriorly. With insufficient anesthesia, meteorism, and peritonitis, the loops of the small intestine and the S-romanum become filled with gas, protrude strongly forward, and cover the anterior surface of the uterus up to the linea alba of the abdomen.

If the woman is placed in a position with the limbs elevated, it is possible to completely free the anterior wall of the uterus and ensure that the intestine and mesentery move upward from the umbilical region. At the end of pregnancy, the lower uterine segment enters the small pelvis to a greater or lesser extent; here it is bounded laterally by the lateral pelvic walls, and anteriorly by the iliac part 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, has an arcuate or bean-shaped position. Usually, depending on the fetal head position, the bladder is situated either to the left or to the right of the fetal head.

If the position of the uterus in the abdominal cavity is analyzed, it can be concluded that it is inclined to the right and slightly rotated around its longitudinal axis from left to right. Therefore, the right adnexa and the round ligament cannot be seen. In this situation, the left round ligament lies anteriorly and slightly obliquely toward the left iliac region. A few centimeters above and behind the left round ligament, it stretches the left uterine tube and its mesentery and slightly covers the left ovary.

When the surgeon makes an incision in the lower segment of the uterus, an even more difficult situation may occur if the incision enters the bifurcation area of the left uterine artery into its ascending and descending branches in a transverse section (Figure 1).

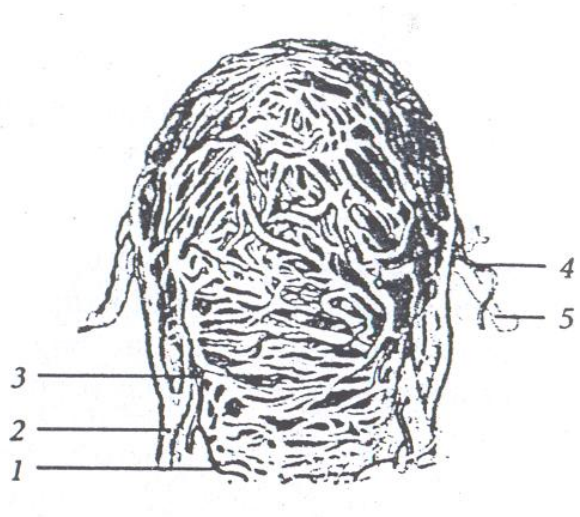
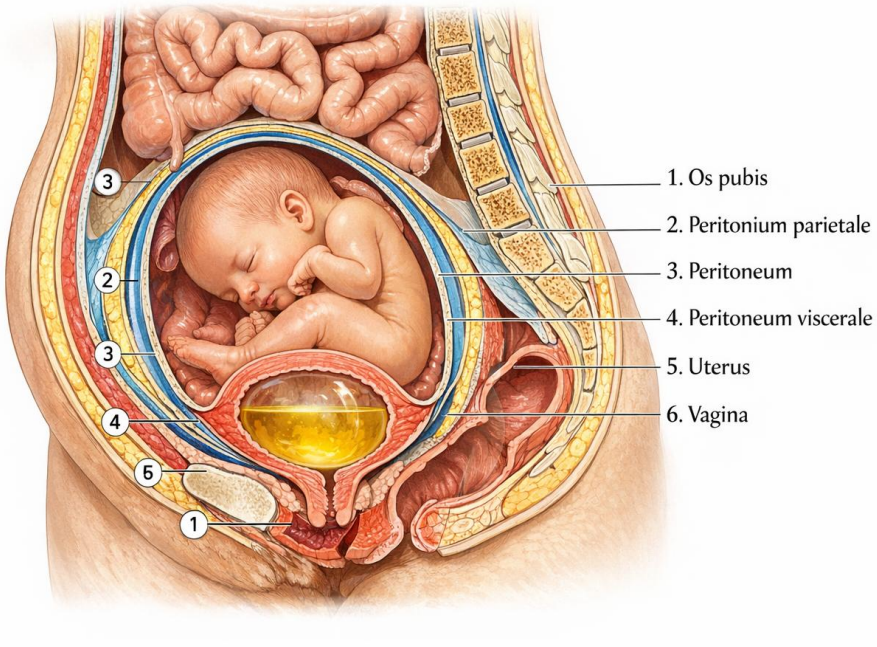


Figure 1. Blood supply of the uterus in a pregnant woman by arteries and veins: 1 - ramus descendens a. uterina; 2 - v. uterina; 3 - ramus ascendens a. uterina; 4 - a. ovarica; 5 - v. ovarica.

Most often, this mistake occurs because of insufficient knowledge of the anatomical features of the passage of the vascular bundles of the uterus during pregnancy. It is known that the lower uterine segment increases in transverse size, especially before delivery. Therefore, at the end of pregnancy, the branching of the left uterine artery occurs not at a distance of about 1 cm as in a non-pregnant woman, but near the very edge of the uterus.

Thus, uneven stretching of the lower uterine segment increases the probability of injury to the uterine artery. The trunk of the left uterine artery is located 6 cm above the pubis, whereas on the right it is located at a distance of 10 cm. Due to the physiological rotation of the uterus from left to right, the uterine artery on the right is located more deeply and posteriorly, where it is twisted and may even form loops; the left uterine artery is stretched, located closer to the anterior abdominal wall, closely approaches the uterine wall, and sharply turns upward. Such an unusual position of the left uterine artery increases the probability of its trauma during cesarean section in the lower uterine segment, especially when extracting the head of a large or giant fetus, when the incision made extends toward the left side. From the above, it is 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 layer of loose connective tissue up to 20 mm thick. This feature allows the posterior wall of the urinary bladder to be quickly separated from the anterior wall of the uterus during cesarean section performed through the abdominal cavity (Figure 2).

Homiladorlik paytida qorin pardasining topografiyasi



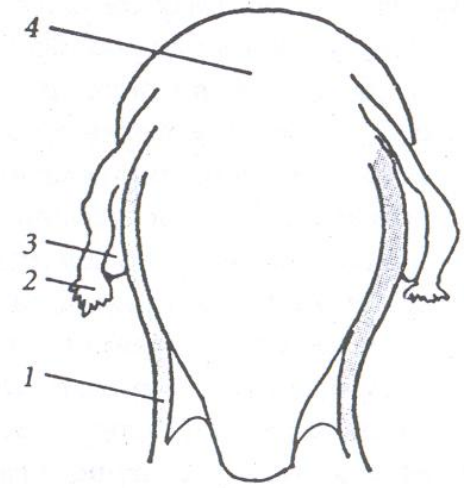
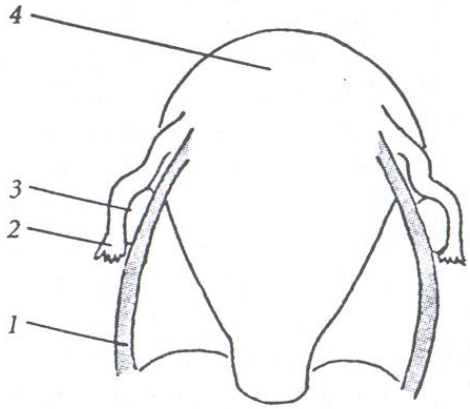
During pregnancy, the weight of the uterus reaches 1000 grams, and its length increases up to 250 mm. The uterine walls reach their maximum thickness, 30-40 mm, at the end of the first half of pregnancy. During labor, the uterus is a hollow muscle that periodically contracts and relaxes. Under the influence of contractions, the uterine walls become denser and the uterus assumes an ovoid shape. If labor pains are strong, the uterus tends to take on an ovoid form, while the uterine fundus approaches the midline and protrudes slightly forward.

Between the body of the uterus and the cervix lies the lower uterine segment. The upper border of the lower segment is considered to be the area where slight separation of the peritoneum from the uterine walls begins. It is difficult to determine the lower border of this segment, because the junction between the lower segment and the cervix has no clearly defined boundary. In addition, below the vesicouterine fold, the urinary bladder is adjacent to the lower uterine segment and is not covered by peritoneum.

In clinically narrow pelvis, when the fetal head cannot move through the maternal birth canal, the lower uterine segment rises even higher. In such cases, the upper edge of the lower segment may rise up to the level of the umbilicus, the contraction ring becomes oblique, and the risk of uterine rupture increases. It should be remembered that the lower uterine segment is easily accessible surgically only when labor has begun and the lower segment has risen from the pelvic cavity into the abdominal cavity. If the pregnant woman’s head is lowered, access to the lower uterine segment during surgery may be more convenient. This method may be used when the fetal head is fixed at the pelvic inlet.

Simultaneously with thickening of the uterine muscles, changes occur in its vascular system. Both arteries and, especially, veins expand during pregnancy; they form wide loops along the uterus, especially in its lateral parts and around the uterine tubes. In the second half of pregnancy, the uterine walls become thinner, down to 20-30 mm. During delivery, the thickness of the uterine walls is not the same in different parts: it is greatest near the fundus, 20-30 mm, much less in the lower segment, only 5 mm, and in pathological labor with a clinically and anatomically narrow pelvis it becomes even thinner, about 2 mm.

During cesarean section, it is important to know where the placenta is attached to the uterine wall. To determine the location of the placenta on the uterine wall, the signs described by Palm may be used (Figure 3).

	
Placenta attached to the posterior uterine wall (according to Palm): Lig. teres uteri. Figure 3. 1 - Lig. teres uteri; 2 - Tuba uterina; 3 - Ovarium; 4 - Uterus.	Placenta attached to the anterior uterine wall (according to Palm): Lig. teres uteri. Figure 3. 1 - Lig. teres uteri; 2 - Tuba uterina; 3 - Ovarium; 4 - Uterus.

According to the author's observations (Palm), if the placenta is located on the posterior wall of the uterus, the round ligaments become thickened and reach the size of the little finger. The ligaments attach to the uterine fundus at an acute angle and are located at a short distance from one another, so they appear thick and short. In cases where the placenta is attached to the anterior wall of the uterus, the round ligaments are stretched and extend from the organ at a right angle, more posteriorly; they are also separated from one another by a considerably greater distance.

In cases where it is necessary to stop bleeding from the uterine tube or ovary, the asymmetrical location of the lig. suspensorium ovarii should be remembered. On the right side, this lig. suspensorium ovarii extends from the pelvic wall between the

cecum and the lateral border of the m. psoas. The left lig. suspensorium ovarii is located slightly lower than the right one. To find the lig. suspensorium ovarii, the uterus should be sharply pulled to the right and the sigmoid colon to the left. In each of these ligaments, the vascular bundle consists of the ovarian artery and markedly dilated veins. The thick veins collecting blood from the tubes and ovary join the veins extending from the uterine fundus and the round ligaments, forming the plexus pampiniformis. Laterally from the adnexa, there is a plexus in the form of three veins, which unite into one common vein and drain on the right into the inferior vena cava and on the left, most often, into the renal vein.

After arising from the iliac artery, the uterine artery is directed medially and anteriorly over the fascia of the m. levator ani; it enters the base of the broad ligament and gives a branch to the urinary bladder, the ramus vesicales.

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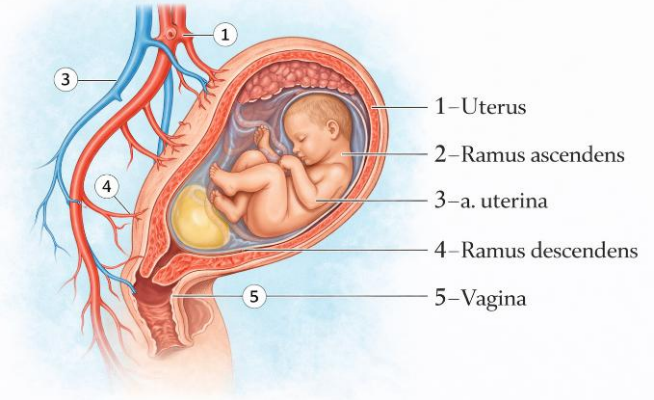


The artery passes through the periuterine tissue in front of the ureter, gives it a thin branch, approaches the uterine isthmus, and at a distance of 15-20 mm in non-pregnant women, or near the branch itself in pregnant women, divides into two branches: the descending branch, ramus descendens, and the ascending branch, ramus ascendens.

The descending branch of the uterine artery is thinner than the ascending branch and runs obliquely from above downward and from outside toward the middle. Below, additional branches arise from the main trunk of the ramus descendens and are directed downward at an acute angle toward the middle of the cervix, where they connect with similar branches of the opposite side.

Based on these anatomical data, it becomes clear why arcuate incisions with the apex directed upward are needed in the region of the uterine body and why blood loss during cesarean section is significantly greater than during abdominal operations. As the uterine fundus is approached, the arterial branches that extend obliquely from the ramus ascendens change their direction to a horizontal one. Therefore, it is advisable to make appropriate transverse incisions in this organ.

The main branch of the ramus ascendens a. uterina, having reached the origin of the ovarian ligament proper, divides into two branches. The first branch, the ramus

ovaricus, changes its direction from vertical to horizontal, goes to the upper pole of the ovary, and supplies this organ with blood through the mesovarium. It gives additional branches to the mesosalpinx, after which it anastomoses with the smaller-diameter ovarian artery closer to the lateral edge.

The second branch of the ramus ascendens a. uterina runs upward in an arcuate manner and supplies the uterine fundus with blood. From one of these branches arises the tubal branch of the uterine artery, the ramus tubarius a. uterina, which passes along the lower border of this organ from the interstitial part to the ampullary part, where it meets the ramus tubarius of the other artery. The branches of both arteries extend through the mesosalpinx to the wall of the tube, branch up to the 6th-7th order, and give rise to numerous vessels that connect with each other and form a dense plexus. It is possible that these features of uterine blood supply lead to the large blood loss that occurs during ruptured ectopic pregnancy.

The third and thinnest branch of the ascending uterine artery passes to the round uterine ligament, where it meets the branch of the inferior epigastric artery, a. epigastrica inferior.

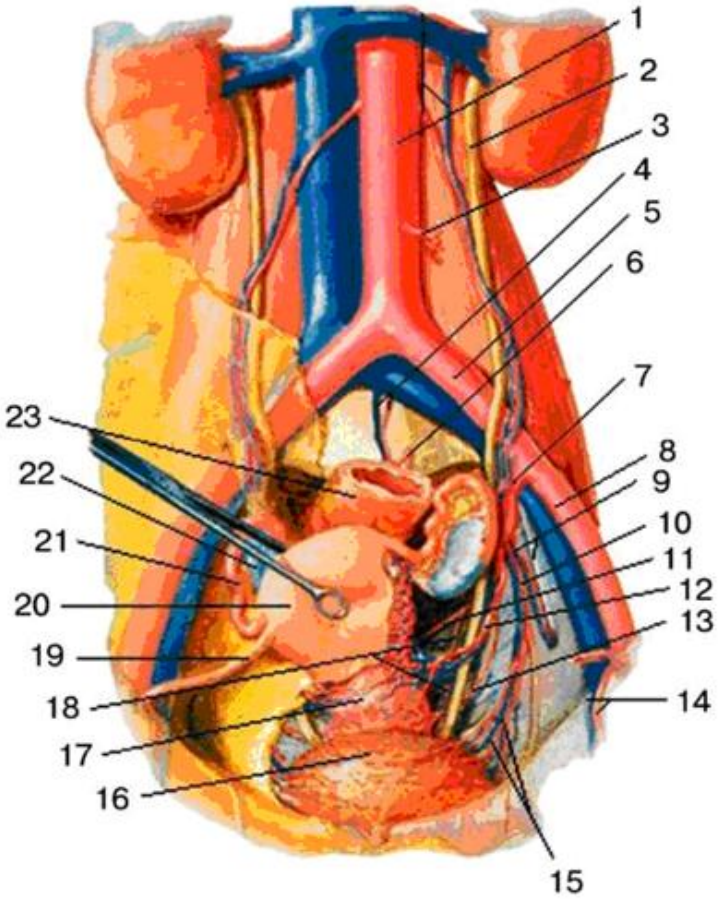


Figure 5. Arteries of the pelvic organs: 1 - aorta abdominalis; 2 - ureter; 3 - a. mesenterica inferior; 4 - v. sacralis mediana; 5 - a. iliaca communis; 6 - a. sacralis mediana; 7 - a. iliaca interna; 8 - a. iliaca externa; 9 - a. obturatoria; 10 - a. umbilicalis; 11 - a. vaginalis; 12 - a. uterina; 13 - a. vaginalis; 14 - a. rectalis

inferior; 15 - a. vesicalis superior; 16 - vesica urinaria; 17 - vagina; 18 - a. uterina descendens; 19 - lig. teres uteri; 20 - uterus; 21 - tube ovarium; 22 - ovary; 23 - rectum.

II. FEATURES OF THE REACTIONS OF SOME VITAL ORGANS IN ACUTE BLOOD LOSS IN PREGNANT AND PARTURIENT WOMEN

During normally developing pregnancy, a number of important changes occur in a woman's body, and knowledge and consideration of these changes are necessary for the diagnosis and adequate treatment of possible complications.

The total body weight of a pregnant woman increases by an average of 12 kg. In addition, 75% of this increase consists of fetal body weight, the weight of the placenta, uterus, and amniotic fluid, as well as the circulating blood volume (CBV), which begins to increase in the first trimester. In the second trimester of pregnancy, the increase in CBV reaches its maximum, and by the end of the third month it is on average 40-50% higher than the initial level, amounting to 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 increases from 2.5 L to 3.8 L by the 40th week of pregnancy, while the volume of circulating erythrocytes increases during this period from 1.4 L to 1.65 L. As a result, when hemoglobin concentration and hematocrit decrease, the phenomenon of physiological hemodilution, or dilutional anemia, occurs. The dilution of blood observed during pregnancy plays an important role in the prevention of thrombosis.

Hemodynamic changes in the cardiovascular system of a pregnant woman contribute to increased blood flow in the uterus and ensure fetal viability. Already by the end of the first trimester, due to an increase in stroke volume and heart rate, cardiac output rises from 4.5 L/min to 6.8 L/min. As a result, uterine blood flow increases from 50 ml/min at the beginning of pregnancy to 500 ml/min at the end of pregnancy, before delivery. At the same time, total peripheral and pulmonary resistance decreases, and therefore central venous pressure (CVP) remains normal. Pulmonary functions also change significantly. Pregnant women are characterized by a tendency to hyperventilation. By the end of pregnancy, the minute respiratory volume in women during labor increases on average by 50%, reaching 10 L/min due to an increase in tidal volume and respiratory rate. The increase in minute volume is accompanied by increased oxygen delivery and consumption and a decrease in the arteriovenous oxygen difference. One third of the increase in oxygen consumption is used to compensate for the intensified work of the heart, almost half is used by the kidneys, and the remaining part is consumed 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, there is a slight enlargement of the kidneys, dilatation of the calyces, and an increase in glomerular filtration from 100 to 150 ml/min/m2. At the same time, sodium reabsorption increases significantly, as a result of which total body water increases.

The changes in the blood coagulation system observed during pregnancy and childbirth deserve special attention. During uncomplicated pregnancy, the concentration of plasma coagulation factors increases. The level of factor I, fibrinogen, increases most markedly, on average from 2.5 to 3.8 g/L, as do factors VII, VIII, IX, and X. A decrease in the levels of coagulation inhibitors, in particular protein S, is observed in pregnant women. By the 12th week of pregnancy, its amount decreases by 40-50% from the initial level. Prothrombin level, activated partial thromboplastin time (APTT), bleeding time, and antithrombin III level remain normal.

A plasminogen activator inhibitor regulating fibrinolytic activity, PAI-2, is formed in the placenta and released into maternal plasma; together with another plasminogen activator inhibitor, PAI-1, it blocks tissue plasminogen activator and prevents the formation of plasmin.

Thus, a number of changes occur in the body of a pregnant woman. On the one hand, there are processes before delivery that prevent thrombus formation, such as hemodilution; on the other hand, after childbirth they ensure rapid thrombosis of the placental separation site, that is, the changes in the hemostatic system listed above. These changes are activated after childbirth when the uterus contracts and when an area of injured placental vessels appears if it does not contract.

Acute blood loss is one of the most common types of damage to the body throughout evolution; it can lead to significant impairment of vital functions, and therefore medical intervention is always necessary.

The definition of acute massive blood loss requiring transfusion intervention is associated with a large number of necessary reserves, because these reserves are precisely what give the physician the right to perform the very risky procedure of transfusing blood components.

Acute blood loss is considered massive blood loss requiring transfusion support if, within 1-2 hours, the estimated blood loss is approximately at least 30% of the initial volume. In this case, collapse of peripheral vessels, the “empty vessels” sign, a persistent decrease in blood pressure, and a decrease in hourly diuresis are observed. The body responds to massive blood loss with several standard reactions aimed, on the one hand, at creating favorable conditions for stopping bleeding, and, on the other hand, at ensuring oxygen transport to the organs and systems that make it

possible to preserve life. Oxygen transport is provided in the “order of importance”: heart, lungs, brain, liver, and kidneys. The function of the secretory glands of the gastrointestinal tract is 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 the skin and muscles remain in a state of dormancy, i.e., peripheral vasoconstriction occurs.

At the same time, lymph and interstitial fluid enter the bloodstream, and blood dilution intensifies, that is, hemodilution. The process of blood dilution is not rapid. Therefore, in the first hours of acute blood loss, its severity cannot be assessed according to hemoglobin concentration, which decreases when blood is diluted with lymph: despite considerable blood loss and marked pallor of the skin, the hemoglobin level may remain close to the initial value for several hours. Red blood cells are released into the bloodstream from the splenic depot, skin, and muscle capillaries. A form of autotransfusion of blood and maximal centralization of circulation occur. The described physiological reactions ensure replenishment of the circulating blood volume, to a certain extent replenishment of the volume of circulating red blood cells, preservation of oxygen transport, and its release to tissues.

In this process, immobility of the body plays a very important role. The area of capillaries in working muscles is approximately 20:1 compared with the area in resting muscles. Therefore, when discussing the required volume for replacing lost blood, it is necessary to take into account the sharply reduced need of the immobilized body for circulating blood and oxygen supply.

Along with the compensatory reactions listed above, an increase in oxygen release to tissues and its utilization is observed. Thus, as a physiological defense against blood loss, the maternal body responds with hemodilution, which improves blood fluidity, mobilization from the red blood cell depot, a sharp decrease after childbirth in blood volume requirements and oxygen demand due to muscle rest, and an increase in respiratory rate, cardiac output, and the work of the left ventricle of the heart.

It is known that approximately 70% of the total circulating blood volume is contained in the veins, 15% in the arteries, 12% in the capillaries, and 3% in the heart chambers.

In women, the circulating blood volume is not identical and depends on constitution: in normosthenic women it is 6.5% of body weight, in asthenic women 6%, in hypersthenic women 5.5%, and in athletic muscular women 7%. Therefore, the absolute CBV values may differ, and this must be taken into account in clinical practice.

A specific feature of obstetric hemorrhage is its massiveness 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. The specific features of pathophysiological changes in the body of pregnant women, especially in hypertensive disorders or complicated labor, lead to the rapid activation of compensatory and protective mechanisms. In addition, obstetric hemorrhage is characterized by an acute volumetric deficit of circulating blood, cardiac dysfunction, and anemic and circulatory forms of hypoxia. The main causes of hemodynamic disturbances in obstetric hemorrhage are a deficiency of circulating blood volume and a mismatch between it and intravascular capacity. Disturbances of water-electrolyte balance, acid-base balance, hormonal ratios, and enzymatic processes are present. It should be remembered that massive obstetric hemorrhage is almost always accompanied by impairment of the coagulation properties of blood, that is, disseminated intravascular coagulation (DIC) syndrome. Even with relatively small blood loss, 15-20% of CBV, two-phase changes in the hemostatic system are often observed: a short-term phase of hypercoagulation is replaced by a phase of hypocoagulation. These reactions of the body are aimed at preserving the body's viability and serve as a guideline for the physician in providing appropriate care.

III. CLASSIFICATION OF OBSTETRIC HEMORRHAGE

Bleeding during pregnancy occurs in 2.5-4% of pregnant women. This article does not discuss bleeding in pregnant women during the first and second trimesters, because such cases are interpreted as incipient abortion.

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

1. Bleeding occurring in the second half of pregnancy, including:

A) placenta previa

B) premature detachment of the placenta

B) rupture of umbilical vessels associated with varicose veins

D) cervical polyps

D) cervical erosion

E) blood diseases (Werlhof disease, APS, Schonlein-Henoch disease, etc.).

2. Bleeding during labor:

A) premature separation of the placenta

B) rupture of the lateral sinuses of the placenta

B) rupture of umbilical vessels

D) uterine rupture

3. Bleeding in the postpartum period occurs in 2.5-8% of cases, and in 20-45% of cases these hemorrhages cause maternal death (V.I. Kulakov, 2009):

A) retained placenta

B) pathological adherence of the placenta (firm attachment, true accreta)

B) incomplete separation of the placenta

D) hypotonia and uterine atony

D) trauma to the birth canal

E) hereditary and acquired disorders of hemostasis

4. Postpartum hemorrhage

A) retention of placental parts in the uterine cavity

B) hypotonia and uterine atony caused by improper management of labor, multiple pregnancy, large fetuses, and prolonged labor.

B) uterine rupture;

D) rupture of the soft tissues of the birth canal

D) hereditary and acquired disorders of hemostasis

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

Vulva - injury to varicose veins

Vagina - trauma or injury

Cervix - polyps, cervicitis, cancer, erosion, ectropion

Uterine cavity - placenta previa, early placental abruption, vasa previa.

Classification of obstetric hemorrhage according to ICD-11:

O44 - Placenta previa:

O44.0 - Placenta previa specified without hemorrhage;

O44.1 - Placenta previa with hemorrhage.

O45 - Premature detachment of the placenta

O45.8 - Other types of premature placental abruption;

O45.9 - Premature placental abruption, unspecified.

O46 - Antepartum hemorrhage, not elsewhere classified

O46.0 - Antepartum hemorrhage with coagulation disorder;

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 disorder;

O67.8 - Other intrapartum hemorrhage;

O67.9 - Intrapartum hemorrhage, unspecified.

O72 - Postpartum hemorrhage

O72.0 - Hemorrhage during the third stage of labor;
O72.1 - Other hemorrhage in the early postpartum period;
O72.2 - Late or secondary postpartum hemorrhage

IV. CLINICAL CHARACTERISTICS OF HEMORRHAGE IN OBSTETRICS

According to the World Health Organization, every year 585 thousand women die from complications of pregnancy and childbirth; according to recent data, the mortality rate has increased by 85 thousand. Maternal mortality is high in Asian countries, particularly in India, Bangladesh, Pakistan, Afghanistan, and African countries (150 thousand annually).

The causes of maternal mortality differ from country to country. In Uzbekistan, obstetric hemorrhage has occupied a 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 observed mainly during prolonged labor, when the woman in labor becomes exhausted.

At present, this indicator accounts for 3-8% of the total number of births.

In the Republic of Uzbekistan, hypo- and atonic hemorrhages account for 25% of cases, while in Samarkand Region they account for 35% of all causes of obstetric hemorrhage.

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 of time a woman may lose a large amount of blood. As a result, the compensatory capacity of the body decreases and disorders of hemodynamics and hemostasis develop, especially in women with a pathological course of pregnancy and childbirth.

According to the authors of the American journal Am Fam for 2007, the causes of bleeding were as follows: 70% of bleeding cases were due to uterine atony, 20% were due to trauma - hematomas, ruptures, and other injuries, in 10% of cases the cause of bleeding was tissue-related - retained placenta and placenta accreta, and a total of 1% were coagulopathic hemorrhages.

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 attachment of the placenta to the uterine wall, injury to the birth canal, hypoatonic bleeding, and

uterine rupture. If adequate care is not provided, these hemorrhages lead to the development of hemorrhagic shock, disorders of blood coagulation, disseminated intravascular coagulation syndrome, and subsequently multiple organ and multiple system failure. Bleeding during labor and in the early postpartum period (within 2 hours after delivery) accounts for 50% of all bleeding. In 34.7% of cases, secondary weakness of labor leads to pathological bleeding. Excessive and discoordinated uterine contractions also lead to atony.

The second group of causes of hypo- and atonic bleeding is excessive stretching of the uterine wall, as a result of which the uterus loses its contractile ability. This is observed in multiple pregnancy (twins), large fetuses, and polyhydramnios. Placental pathology - placenta previa or low-lying placenta, as well as premature detachment of a normally located placenta - causes bleeding due to weak contraction of the placental site.

Bleeding is observed in women with anatomical defects of the uterus, uterine fibromatosis, adhesions, tumors, multiple abortions, and infantilism; functional defects of the uterus occur due to a decrease in biologically active substances within it.

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

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

There are two clinical variants of early postpartum hemorrhage. In the first variant, bleeding is profuse and massive from the very beginning. The uterus is flaccid and in a state of atony; it does not respond to uterine contraction measures, external massage, or manual examination of the uterine walls. Hypovolemia develops rapidly, followed by hemorrhagic shock and disseminated intravascular coagulation syndrome. In the second variant, recurrent blood loss alternates with temporary restoration of uterine muscle tone and temporary cessation of bleeding in response to conservative treatment. Blood is lost in portions of 150-250 ml. A small volume of blood loss provides temporary adaptation of the postpartum woman to developing hypovolemia. Blood pressure remains within normal limits, the skin and visible mucous membranes become pale, and tachycardia increases. This period of temporary compensation is often overlooked. If adequate treatment is not provided in the initial, early period of uterine hypotonia, the severity of disorders increases and the volume of blood loss rises. At a certain stage, bleeding intensifies, often accompanied by deterioration of the general condition - signs of shock rapidly

increase and DIC syndrome develops. If the condition is not severe and timely assistance is provided, bleeding can be stopped within 15-30 minutes.

With regard to determining the amount of blood loss, obstetricians have certain advantages. In most cases, bleeding develops in their presence - it can be collected and measured. A separate issue is determining the volume of blood loss during bleeding that occurs outside the hospital. When assessing blood loss, interviewing witnesses (emergency physician, 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 the volume of blood loss, it is necessary to focus primarily on the percentage of the woman's known body weight and, depending on the volume of blood loss, to determine the main infusion-transfusion therapy (ITT) program. Each component of ITT must have its own indications. In obstetric hemorrhage, one of the first and main tasks of ITT is replacement of blood volume.

IV. ERRORS AND DEFICIENCIES IN PROVIDING CARE FOR OBSTETRIC HEMORRHAGE

Early detection of obstetric hemorrhage is a very important factor in treatment. Prevention, early detection, and prompt action are the key to minimizing the consequences of obstetric hemorrhage. Delay in the diagnosis of postpartum hemorrhage was identified in 68 (71%) cases, including 48 (84%) after cesarean section and 20 after vaginal delivery. The medical records contain no information on uterine tone or the amount of vaginal discharge at specified time intervals. According to the national standard for obstetric hemorrhage, routine monitoring of a woman after delivery includes assessment of uterine tone and control of vaginal discharge; monitoring of hemodynamic parameters every 15 minutes during the first hour after delivery and at the end of the 2nd, 3rd, and 4th hours; and then checking the above indicators every 4 hours during the first 24 hours. Insufficient observation in the postpartum period leads to delayed recognition of massive bleeding and, accordingly, delayed measures to stop the bleeding. In all case histories of deceased patients, there is no observation chart indicating heart rate, blood pressure, blood loss, respiratory rate, oxygen saturation, diuresis, administered drugs, and the amount of infusion at the time of observation.

It is known that the amount of blood lost can be determined by three methods:

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

In practice, however, blood loss is often determined visually; in this case errors ranging from 200 to 300 ml, and sometimes even up to 1000 ml, may occur, which negatively affects the choice of obstetric tactics.

This clinical assessment still remains the priority method in obstetric practice for determining the volume of blood loss and selecting therapy. It should be taken into account that changes in pulse and blood pressure, no matter how significant they may be, appear in the late stage of bleeding and indicate the beginning of exhaustion of compensatory mechanisms. It should also be considered that in the postoperative period fluid infusions are administered, which influence hemodynamic parameters and may prevent a marked decrease in blood pressure and tachycardia. Sudden blood loss may not be manifested by a decrease in hematocrit or a decrease in hemoglobin level during the first 4 hours or more. During normal vaginal delivery, blood loss is usually assessed visually and is often inaccurate. During delivery, the physician estimates the amount of blood loss by eye, but the true amount of blood loss is often much greater than the visual estimate. Underestimation of blood loss and insufficient monitoring of hemodynamic parameters and hemoglobin lead to delayed adequate infusion therapy and blood transfusion, which in turn leads to subsequent complications of posthemorrhagic anemia. All lost blood should be collected and measured in a graduated container or in trays with volume markings. Each institution should also know the weight of individual pads, sheets, dressing materials, including gauze of different calibers, used during surgery. When analyzing blood loss, all collected blood in graduated containers and trays should be assessed. Assessment of hemodynamics should include not only blood pressure, pulse, and diuresis, but also measurement of respiratory rate and blood oxygen saturation, which may indicate oxygen deficiency in the body. Hemoglobin and hematocrit should be assessed regularly during observation.

Delayed surgical hemostasis.

Delay in providing the necessary obstetric care. Incorrect assessment of blood loss, passivity and negligence of physicians, and refusal by the patient's relatives to consent to any manipulation lead to insufficient provision of the necessary obstetric care. The result of these measures at the first stage of care is ineffective. Analysis of medical documentation and use of questionnaires reveal the reasons for delayed surgical hemostasis:

Insufficient assessment or failure to assess the volume of blood loss:

- Staff do not have sufficient skills to perform surgical hemostasis.
- There is no protocol for performing balloon tamponade of the uterus.
- Organ-preserving technologies are not used sufficiently.

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 for bleeding is associated with hysterectomy and that the decision to remove the uterus during a first delivery is made late. Delays in surgical hemostasis also occur after cesarean section.

It should be emphasized that if stage I measures are ineffective, it is necessary to move immediately from conservative methods to surgical methods, that is, to surgical hemostasis. In any case, the time between the diagnosis of bleeding and the start of surgical control of bleeding should be minimized. After manual examination of the uterus, if incomplete uterine rupture is present, laparotomy should be started immediately. If, after manual examination of the uterus, atony is identified and blood loss continues despite bimanual uterine compression and repeated administration of uterotonics, balloon tamponade should be performed according to world practice; its effectiveness is 88-98%. If bleeding continues despite uterine balloon tamponade, laparotomy should be performed. If bleeding continues, surgical hemostasis must not be delayed, because it is the only way to save the woman. International practice recommends performing hemostatic measures according to the principle "Earlier rather than later!" Despite the recommendations of clinical guidelines, organ-preserving technologies are insufficiently used in practice. This is due to surgeons having insufficient experience and insufficient information regarding these technologies. At present, there are many methods of applying compression sutures - B-Lynch, Hayman, Cho, Pereira, Kurtser, and others. Ligation of vessels and application of compression sutures require consistent adherence to stepwise 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 left as a last resort or delayed until less radical methods, in which the surgeon has little experience, have been attempted. Subtotal hysterectomy, if there is no injury to the cervix or lower uterine segment, is the method of choice in most cases of postpartum hemorrhage requiring hysterectomy; the risk of neoplasia that may develop in the cervical stump several years later is not significant in the setting of life-threatening bleeding. Hysterectomy should be performed regardless of hemodynamic parameters and the state of hemorrhagic shock, because if the source of bleeding is not eliminated, it is used in cases where there is no possibility of stabilizing the condition.

Surgical treatment must be started under any circumstances - in hemorrhagic shock, disseminated intravascular coagulation syndrome, and other conditions - and no condition can prevent surgical control of bleeding. In severe hemorrhagic shock and when there are technical difficulties with surgical hemostasis, the principle of "damage control surgery" should be used:

Stage 1 - after laparotomy, bleeding is stopped by any method: compression, application of clamps, ligatures, tamponade, and even cross-compression of the aorta.

Stage 2 - the anesthesiologist-resuscitator stabilizes the main functions of the body, which occurs much faster and more effectively than under conditions of ongoing bleeding.
Stage 3 - after shock has been eliminated, the obstetrician-gynecologist ensures the hemostasis required for the case in a fundamentally different clinical situation.

It is known that the woman's initial condition at the time of bleeding is of great importance: hypovolemia, hypoxic-ischemic disorders in vital organs, and coagulopathy in the blood coagulation system.

To prevent these complications, ITT, in particular blood transfusion, is used and must be carried out in a timely and rational manner. In practice, however, physicians pay more attention to stopping the bleeding, while the above measures are delayed or performed inadequately. In the first 5 minutes, catheterization of two veins and initiation of crystalloid infusion were performed in only 25% of cases. In the first 15 minutes, 1000 ml of crystalloids were infused in 20% of women with major blood loss. 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. Restoration of circulating blood volume (CBV) is provided by crystalloids and colloids. In cases of major, severe blood loss of more than 1500-2000 ml, the massive transfusion protocol must be followed. Administration of blood components (plasma, red blood cells) should be started as soon as possible (within the first 2 hours), because with this volume of blood loss, infusion of more than 2000 ml of crystalloids or colloids already leads to hemodilutional coagulopathy and increases the volume of blood loss, multiple organ failure, and mortality. The reason may be a lack of blood products or even simple solutions. This may occur only due to negligence on the part of organizers of obstetric care or practitioners. According to national standards, it is necessary to calculate circulating blood volume according to the woman's weight, determine the degree of blood loss according to the volume of blood loss, and compensate blood loss according to tables. In maternity institutions, it is recommended to visually display tables on infusion programs according to the degree of blood loss, train staff to work with these tables, and organize a blood reserve in institutions with more than 2500 deliveries per year.

Insufficient antenatal care.

When studying the medical records of women who experienced massive blood loss, almost all women had antenatal risk factors: preeclampsia, uterine scar, large fetus, multiple births, placenta previa, placenta accreta (ingrowth), severe anemia, thrombocytopathies, antenatal fetal death, pneumonia with sepsis, and acute fatty liver. Despite such risk factors, in 90% of cases they were not hospitalized in a timely manner.

Deficiencies in laboratory services and diagnostic equipment.

As a rule, these deficiencies are most often found in regional maternity hospitals. The necessary elements in obstetric practice are not at the proper level: diagnosis of the blood coagulation system, thromboelastography, and coagulogram. This leads to delayed diagnosis of coagulopathic disorders, which in turn leads to incorrect differentiation of phases.

According to WHO, in addition to the above, there are the following errors and deficiencies that may lead to maternal mortality (WHO Chronicle, 2017). These are as follows:

Low level of medical knowledge among women.

No one other than the woman herself can be responsible for her health. Since a physician sees a woman once a month, or at most once a week, the woman herself and her close relatives can observe her day and night. However, knowledge of premature placental abruption, premature rupture of the amniotic membranes, antenatal fetal death, and simple warning signs indicates the need to consult a physician immediately.

Insufficient qualification of physicians.

This is especially relevant to primary care physicians, who must quickly identify life-threatening pathologies and hospitalize patients in a specialized medical institution (WHO, Geneva, 2002; Rooney S., 2012).

Poor infrastructure of medical care.

This refers to transporting a woman from the periphery to a specialized medical institution. This includes a lack of vehicles, poor roads, and poor communication (WHO/FHE/2004).

Negligent attitude of medical personnel.

Sometimes, even if a woman arrives on time, the passivity of medical personnel, delay in necessary manipulations, untimely availability of blood products or other medications, and failure to prescribe medications in time may lead to the woman's death. Every specialized obstetric hospital must 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 may lead to a reduction in maternal mortality.

V. PLACENTA PREVIA (PLACENTA PRAEVIA)

Placenta previa (PP) is an abnormal attachment of the placenta to the uterus, in which it is located in the lower uterine segment over the internal os, partially or completely covering it and lying in the pathway of the fetus. The term "previa" consists of two

parts, "prae" and "via", meaning "in the way". Normally, when the placenta is attached in the body of the uterus, its lower edge is located 7 cm or more away from the internal os.

Problems associated with placentation abnormalities:

- An increase in the number of cesarean section operations;
- Labor in these patients is accompanied by hemorrhage;
- The need to manage blood loss during surgery;
- The performance of organ-preserving surgical procedures;
- Timely diagnosis of placenta accreta spectrum disorders;

Frequency. According to an analysis of the literature, placenta previa occurs in 0.2-0.6% of cases. In reality, the frequency of this pathology is considerably higher, as confirmed by ultrasound data starting from early pregnancy. A large proportion of miscarriages in the first half of pregnancy occur as a result of low placentation. According to WHO data, the main cause of maternal mortality in placenta previa is hemorrhagic shock. Maternal morbidity is 22-25%, and preterm birth is 20-25%. Perinatal mortality ranges from 17 to 25% and is mainly associated with preterm birth, fetal immaturity, and fetal compromise during the antenatal period. Fetal death depends on the amount of blood loss. In placenta previa, preterm birth has been observed in 47% of cases. Fetal anomalies have been detected 2.5 times more often than in the general population. Fetal growth restriction has also been identified. Significant variations in statistical data are related to the following reasons: 1) in maternity hospitals where high-risk pregnant women are admitted, placenta previa is observed more often than in institutions where delivery occurs without complications; 2) there is no uniformity in views regarding the degree of cervical dilation (2-4-6 cm) at which the degree of placenta previa is finally established; 3) differences in the research methods used. At present, the most informative methods are ultrasound and MRI.

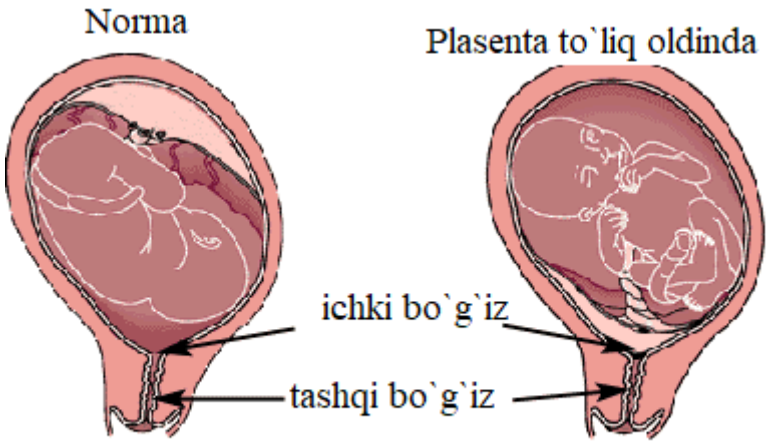


Figure 8. Normal and pathological placental location

Classification of placenta previa.

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

1. Central placenta previa (placenta previa centralis) - the internal os is covered by placental lobules, and fetal membranes are not detected in the vagina.

2. Lateral placenta previa (placenta previa lateralis) - parts of one or two placental lobules are identified in the region of 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, and only fetal membranes are present within the os.

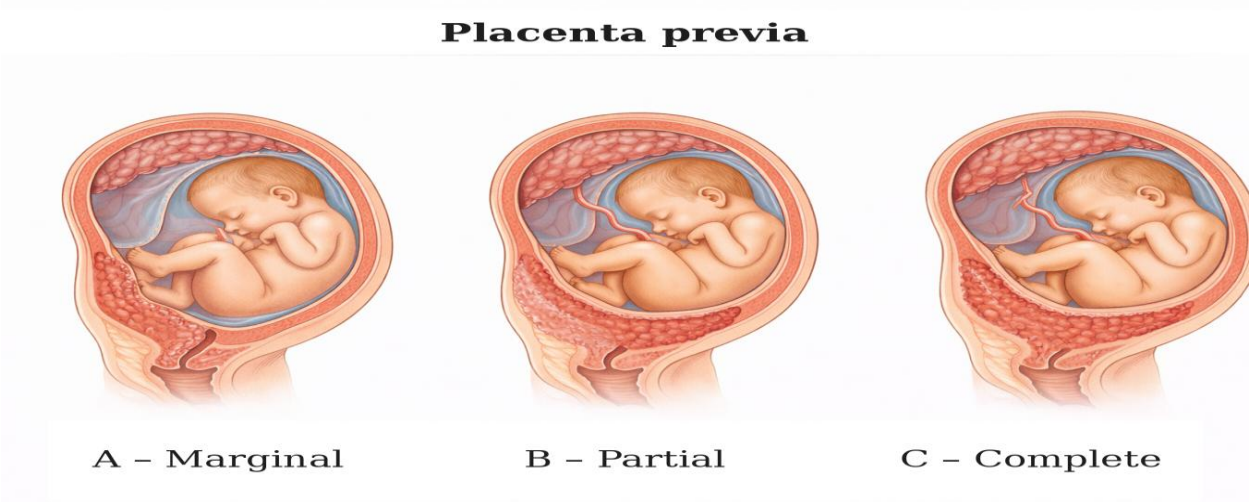
In addition, there is a simpler classification, and the following classification is used abroad (Williams Obstetrics, 2001):

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 located at the margin of the internal os.

4. Low-lying placenta - the placenta is located in the lower uterine segment, but the placental 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. Conventionally, they can be divided into 2 groups:

Fetal factor: if the fertilized ovum is positioned low or its proteolytic properties are reduced, it cannot implant in the uterine fundus in time. In such a case, implantation of the fertilized ovum occurs after it has descended into the lower parts of the uterus. A similar mechanism is possible with artificial insemination, IVF, and embryo transfer.

Uterine factor: the presence of dystrophic and atrophic changes in the uterine mucosa contributes to the development of placenta previa. In this case, the zygote cannot implant in the altered mucosa of the uterine fundus and body and descends downward.

The main causes of dystrophic and atrophic changes in the uterine mucosa are:

inflammatory processes of the uterus (chronic endometritis);

a history of multiple deliveries and frequent abortions;

postpartum and postabortion septic diseases;

uterine myoma;

deformation of the uterine cavity (uterine scars after cesarean section and other surgical interventions);

abnormal development of the uterus;

conservative myomectomy, suturing of uterine rupture, and others;

Cesarean section performed in the presence of placenta previa further increases the frequency of hysterectomy. In 25% of hysterectomies performed for placenta previa, the women had a history of two cesarean sections, whereas after a first cesarean section for placenta previa, hysterectomy was performed in only 6% of cases.

At 18 weeks of pregnancy, 25% of pregnant women had low placental location, and by the end of pregnancy it persisted in 7% of pregnant women. During ultrasound examination, low placental location (gestational age 18-20 weeks) was found in 12% of cases. If the placenta did not cover the internal os, placenta previa was not recorded. If the placenta was located over the internal os, placenta previa persisted in 40% of cases.

During transvaginal ultrasound examination at 18-23 weeks of pregnancy, placenta previa was diagnosed in 1.5% of the examined women. In only 20% of them, the placental edge at delivery was located less than 15 mm from the internal os. When it was located 25 mm or more above the os, placenta previa persisted in 40% of cases.

Table 1.

Risk factors for the development of placenta previa

No.	Factors	Relative risk
1.	Previous placenta previa in the history	2.6
2.	Previous cesarean section operations	2.5
3.	One previous cesarean section operation	2.2
4.	Two previous cesarean section operations	4.1
5.	Three previous cesarean section operations	22.4
6.	Previous termination of pregnancy	
7.	Multiparity	
8.	Advanced maternal age (> 40 years)	
9.	Multiple pregnancy	
10.	History of endometrial insufficiency: - uterine scar - endometritis - manual removal of the placenta - curettage, submucous myoma	

It has been established that in the first half of pregnancy, placental growth exceeds uterine growth. By the end of the second trimester and the beginning of the third trimester, conversely, due to the formation of the lower uterine segment, the placenta migrates upward. It may migrate over a distance of 3 to 9 cm, that is, placenta previa diagnosed in the early stages of pregnancy does not always persist until term.

Due to insufficient development of the decidua in the region of the lower uterine segment, firm placental attachment (placenta adhaerens), and sometimes true invasion (placenta increta or percreta), may occur. In placenta previa, pathological placental attachment occurred in 7% of cases. Some researchers, comparing maternal risk factors for the development of placenta previa and placental abruption, concluded that abruption is associated with conditions that occur during pregnancy, whereas placenta previa is associated with conditions that existed before pregnancy.

Clinical presentation.

Placenta previa has 2 phases:

1. "silent phase" - there are no obvious symptoms;
2. "manifest phase" - external bleeding appears, indicating separation in placenta previa.

Before the onset of bleeding, the clinical presentation of placenta previa cannot be clearly determined. In the third trimester and at the end of pregnancy, there may be a high position of the presenting fetal part, its unstable position, a high percentage of oblique and transverse positions, breech presentation, frequent signs of threatened pregnancy, and other findings. Before the use of ultrasound, the diagnosis of placenta previa was usually made only when external bleeding occurred, which is considered the main clinical symptom. This bleeding has characteristic features. The blood discharged is usually bright, and there is no pain syndrome ("painless bleeding"). Bleeding may occur without any apparent cause, at night during sleep, at rest, and so on. Another characteristic feature of the bleeding is its recurrence. A second feature is progressive anemia in the pregnant woman, because with repeated, even minor bleeding, adequate blood regeneration does not occur. Under such conditions, even relatively small bleeding during labor may cause the clinical picture of decompensated blood loss and may become life-threatening.

Uterine bleeding in placenta previa most often occurs at 30-35 weeks of pregnancy. Very rarely, it appears in the early stages of pregnancy (9-12 weeks), and rarely during the first stage of labor. The appearance of bleeding is associated with premature separation of the placenta. It is known that uterine contractions occur not only during labor, but also during normal pregnancy (Braxton-Hicks contractions), although they are not perceived by the pregnant woman. In this situation, stretching of the uterine muscle fibers occurs, especially in the isthmus region, when the lower uterine segment is being formed. The placenta does not have the ability to stretch. As a result, sliding occurs between two surface planes - the lower uterine segment and part of the placenta - whereby the maternal surface of the placenta separates from the uterine wall and bleeding from the vessels of the placental site occurs. This bleeding may stop only after contractions cease, vascular thrombosis occurs, and placental separation stops. When uterine contractions resume, bleeding also recurs.

Another explanation for the mechanism of bleeding during pregnancy is possible. At the end of the second trimester and the beginning of the third trimester, when the lower segment is forming, its length increases and the placenta lags behind in growth; as a result, placental migration is observed, which may lead to its detachment and bleeding.

Painless bleeding in the third trimester of pregnancy, regardless of its severity, should be considered as due to placenta previa until the correct diagnosis is established.

One of the factors causing bleeding in placenta previa due to the onset of labor is the tension of the membranes at the lower pole of the ovum, which holds the placental edge and does not follow the contraction of the lower uterine segment; as a result, their connection is disrupted and bleeding occurs. When the membranes rupture, the relationships change, because the placenta no longer separates following contraction of the lower segment. During labor, bleeding may stop after rupture of the membranes and mechanical compression of the placental edge by the fetal head descending into the pelvis.

In complete placenta previa, spontaneous cessation of bleeding is impossible, because during effacement and dilation of the cervix the membranes do not open and placental separation continues.

Diagnosis. When diagnosing placenta previa, it is necessary to take into account the features of the obstetric and gynecological history, including inflammatory processes of the uterus and its adnexa, postabortion and postpartum diseases, infertility, uterine anomalies, previous abortions, operations on the uterus (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 may be suspected if the fetus is in an abnormal position (transverse or oblique), has a breech presentation, or if the presenting part is located high above the pelvic inlet. When the presenting part (head) is palpated, it is felt less clearly, as if through spongy tissue (the uterine wall and placenta), and may be mistaken for the pelvic end. If the placenta is located on the posterior wall in the lower segment, the presenting part often protrudes toward the anterior aspect of the pubis; its displacement backward toward the promontory produces a feeling of resistance and may cause a decrease in the fetal heart rate.

A Doppler device can be used to successfully determine placental localization; in addition to the fetal heartbeat, it makes it possible to detect the murmur of placental vessels and determine the location of the placenta. The fetal heartbeat is usually not affected by placenta previa.

The main sign of placenta previa is the occurrence of frequently recurrent painless bleeding from the genital tract in the second half of pregnancy. Bleeding with bright red blood may be heavy or mild.

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

The following definitions are used in national guidelines on antenatal bleeding:

Spotting is the appearance of spots, streaks, or blood stains on underwear or sanitary protection products. Minor bleeding is blood loss of less than 50 ml. Major bleeding is blood loss of 50-1000 ml without clinical signs of shock. Massive bleeding is blood loss of more than 1000 ml and/or clinical signs of shock. Recurrent antenatal bleeding is a term used when bleeding episodes occur more than once.

When a pregnant woman with bleeding is hospitalized, to clarify the diagnosis, the cervix is examined with vaginal specula and a bimanual vaginal examination is performed with the operating room prepared (for cesarean section). When the cervix is examined in the specula, the source of bleeding can be identified and a number of diseases that may cause it can be excluded: a polyp of the cervical canal, cervical erosion and cancer, and varicose veins of the vagina with rupture of a node.

Bleeding from the cervical canal is also observed in premature separation of a normally located placenta, rupture of the marginal sinus of the placenta, rupture of umbilical vessels, uterine rupture, and others.

Treatment. The choice of treatment method for placenta previa depends on a number of circumstances: the time of bleeding (during pregnancy or during labor); the massiveness and volume of blood loss; the general condition of the pregnant woman or woman in labor; the condition of the birth canal (degree of cervical dilation); the type of placenta previa; gestational age; the condition and position of the fetus; and the state of hemostasis.

Management of pregnancy and labor. If ultrasound examination in the early stages of pregnancy detects placenta previa and there is no clinical manifestation of this pathology (bleeding), what should be done? It is known that bleeding may occur at any time, and not only the pregnant woman but also her relatives should be informed about this. In this case, in the woman's interests, hospitalization in a qualified hospital and examination (repeat ultrasound examination, Rh test, hemostasiogram) are recommended.

If the pregnant woman's condition is satisfactory, there is no bleeding, and the woman firmly requests discharge from the hospital, discharge is allowed only after receiving a written statement from the pregnant woman and informing close relatives about possible complications. The pregnant woman must be given clear information about which hospital to go to if bleeding occurs. Bed rest should be observed at home, and control ultrasound examinations should be performed every 2 weeks. It is recommended to prolong pregnancy until 36-37 weeks (until fetal viability), and then, depending on the specific obstetric situation, if placenta previa persists, an adequate method of delivery should be selected.

In women with placenta previa, the decisive factor is the severity of bleeding. Thus, if bleeding threatens the life of the pregnant woman, regardless of the fetal condition (nonviable or dead fetus), the only correct method is delivery by cesarean section. In all other cases, many factors must be taken into account, primarily the duration of pregnancy, the type of placenta previa, the fetal condition, and the readiness of the birth canal for delivery.

When a pregnant woman is admitted to the hospital with minor bleeding from the genital tract, if her condition is satisfactory, strict adherence to bed rest and a complete examination to clarify the diagnosis and treatment aimed at prolonging pregnancy (36-37 weeks) and assessing the fetal condition are recommended.

If bleeding continues, it is necessary to insert an intravenous catheter, measure blood pressure and pulse, determine blood group and Rh status, perform a clinical blood test, carry out a hemostasiogram, and begin intravenous fluid infusion. In the hospital, for unforeseen situations, blood of group 0 (I), Rh-negative, should be kept in reserve.

The examination primarily includes an external obstetric examination. At the same time, attention should be paid to uterine tone, protrusion and tenderness, the nature of the presenting part and its engagement at the pelvic inlet, the nature and amount of bleeding, and other findings.

To assess the fetal condition, the fetal heart rate, the character of the heartbeat, fetal motor activity are determined, Doppler examination is performed, and so on.

The most informative method is ultrasound examination, which makes it possible to determine the location of the placenta, its thickness, size, degree of maturity, and areas of separation (this usually fails, because blood flows out and does not accumulate in the areas of separation), as well as the fetal heartbeat, estimated weight (hypotrophy), umbilical cord insertion, and other parameters.

If the diagnosis of placenta previa is confirmed, the pregnant woman must remain in the hospital, where symptomatic treatment aimed at eliminating uterine excitability, strengthening the vascular wall, and other measures should be carried out. Bleeding of varying degrees may lead to the development of anemia. This

condition must be treated, because the woman is approaching delivery, which is always accompanied by some blood loss. In a premature fetus, due to the risk of developing distress syndrome at birth, administration of glucocorticoids is indicated.

In cases of partial or marginal placenta previa and in the absence of aggravating circumstances (bloody discharge, oblique or transverse fetal position, transverse presentation, multiple pregnancy, uterine scar, and others), expectant management can be followed until the spontaneous onset of labor. When labor pains are established, the head is engaged, and bleeding appears, opening of the amniotic sac is indicated. Before opening it, it is necessary to make sure that cervical dilation is present, to determine whether the fetal head is engaged or not, whether placental tissue is present or not, how much of the internal os area it covers, and whether the membranes can be identified; these are the conditions for opening. After opening the amniotic sac, if the head descends to the pelvic inlet, bleeding stops, which requires regular contractions of sufficient strength.

In this group of pregnant women, to stop bleeding it is necessary not only to open the membranes, but also to start intravenous administration of oxytocin (5 IU in 500 ml of isotonic sodium chloride solution) to strengthen uterine contractile activity. If, after opening the amniotic sac, the fetal head does not firmly press the placenta against the lower uterine segment and bleeding continues, assistance is provided by cesarean section.

The indications for cesarean section are complete, partial, or marginal placenta previa and major blood loss. According to the literature, the rate of surgery for placenta previa is 70-82%. The rationale for cesarean section is that immediate removal of the fetus and placenta allows uterine contraction and cessation of bleeding. In addition, the operation prevents the possibility of cervical trauma, which may be a serious complication in complete or partial placenta previa.

In central placenta previa, the only correct method of delivery is cesarean section, performed either electively or urgently. It is recommended to perform the operation electively when pregnancy reaches 37 weeks. This makes it possible to obtain a viable fetus, prepare for surgery, provide the necessary blood and infusion agents, prepare for reinfusion during surgery, choose an adequate method of anesthesia, and avoid prenatal blood loss.

STAGES OF MANAGEMENT AND DELIVERY IN PATIENTS WITH PLACENTA PREVIA AND PLACENTA ACCRETA:

If placenta previa (with signs of accreta) is detected after the second ultrasound examination, all patients should be referred for consultation;

1. Three screening examinations should be performed; referral for hospitalization at 35 weeks;

2. Patients living in remote areas should be accommodated from 34 weeks onward, with subsequent hospitalization.

According to researchers, the introduction of screening at 6-8 weeks of pregnancy plays an important role in identifying the formation of chorionic ingrowth in women who have undergone cesarean section.

Criteria for "placenta accreta":

thinning or absence of the uterine muscular wall in the placental area;

absence of a clear boundary between the placenta and the myometrium;

presence of placental lacunae described as "Swiss cheese" or "moth-eaten";

a clearly visible vascular network (vascularization) at the site of placenta accreta.

STAGE OF ANTENATAL HOSPITALIZATION:

1. If anemia is present (hemoglobin less than 100 g/L), treatment with parenteral anti-anemic medications;

2. Ultrasound results to determine the level of the placental edge and signs of bladder wall involvement;

3. Prevention of RDS in the newborn (taking gestational age into account);

4. Hematologist's conclusion.

Conservative hemostasis methods and blood-saving technologies

• Use of uterotonics:
  - Intravenous administration of oxytocin
  - Use of carbetocin
  - Use of misoprostol
• Antifibrinolytics (tranexamic acid)
• Normovolemic hemodilution during surgery
• Hardware-assisted reinfusion of autologous erythrocytes
• Blood components (FFP, platelets, cryoprecipitate)
• Blood coagulation factors

Carbetocin is an effective uterotonic agent. Its use during abdominal delivery in patients with a uterine scar, a large fetus, or multiple pregnancy significantly reduces the volume of blood loss compared with oxytocin. When this medication is used to prevent bleeding, additional methods for stopping hemorrhage are required less frequently. Complex clinical situations combining placenta previa and placenta

accreta do not constitute a direct indication for carbetocin if bleeding is caused by several factors and impaired uterine tone is not the leading factor. Its use in organ-preserving operations makes it possible to ensure reliable hemostasis and reduce the amount of blood loss. Carbetocin demonstrates greater effectiveness than continuous oxytocin infusion and has a similar safety profile. When administered after delivery of the placenta, a single intravenous dose of 100 micrograms of carbetocin is more effective and reliable than standard continuous oxytocin infusion in maintaining adequate uterine tone and preventing excessive blood loss during cesarean section. This helps prevent excessive bleeding aggravated by low uterine tone. Carbetocin acts rapidly and causes normal uterine contractions within 2-3 minutes after administration. Carbetocin is well absorbed and has a longer duration of action than oxytocin, lasting approximately 5 hours, whereas oxytocin lasts approximately 1 hour and 30 minutes.

Step-by-step method for surgical hemostasis

• Controlled balloon tamponade;

• Ligation of uterine vessels;

• Compression sutures on the uterus;

• Compression sutures + controlled balloon tamponade (“uterine sandwich”);

• Ligation of the internal iliac arteries;

• Hysterectomy;

• Electrosurgery (argon plasma coagulation);

• Local hemostatic sutures;

Management tactics in the first half of pregnancy

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

• etamsylate 2 ml intramuscularly 3-4 times daily until bleeding stops;

• Magne B6, 2 tablets twice daily until uterine tone normalizes and bleeding stops;

• magnesium sulfate 25% solution, 5 ml intramuscularly 1-2 times daily until uterine tone normalizes;

• dicynone 2-4 ml intramuscularly 1-2 times daily until bleeding stops;

• papaverine suppositories every night until uterine tone normalizes;

• no-spa, 1 tablet or 2 ml intramuscularly 1-2 times daily until uterine tone normalizes.

Placenta previa with severe uterine bleeding is an absolute indication for cesarean section 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), provided that bleeding is mild. In such cases, strict bed rest must be ensured. Hemostatic drugs (dicynone, vikasol) and restorative agents (40% glucose, vitamins) are prescribed.

If a pregnant woman in the second or third trimester presents to an antenatal clinic with complaints of bloody discharge from the genital tract, the physician must urgently transport her to the maternity hospital in a lying position. In the maternity hospital, in the admission department, blood group, Rh factor, and hemoglobin are determined again. An anesthesiologist is invited, the operating team is alerted, and only after this is an internal examination performed to determine the cause of bleeding from the genital tract.

Tactics of pregnancy and delivery in the second half of pregnancy.

If a pregnant woman at the end of the second trimester or in the third trimester comes to the clinic with complaints of bloody discharge from the genital tract, the physician must immediately take her to the maternity hospital, personally accompanying her in a lying position.

In complete, incomplete, and lateral placenta previa without bleeding, delivery by cesarean section is indicated electively at 37 weeks or later.

In cases of heavy bleeding, regardless of the degree of placenta previa and regardless of gestational age, surgical 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, it is possible to wait for spontaneous onset of labor, followed by amniotomy.

During cesarean section for placenta previa, some authors recommend uncitrated plasma for hemostatic purposes and, in cases of 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 intravenously once daily for 3-4 days;

aminomethylbenzoic acid 50-100 mg or 100 ml intramuscularly once daily for 4-6 days;

10% calcium gluconate solution, 5-10 ml intravenously 2-3 times daily until bleeding stops;

etamsylate IV 250 mg 3 times daily until bleeding stops;

tranexamic acid IV 1000 micrograms.

In the postoperative period, uterotonic drugs are indicated for the prevention of bleeding:

methylergometrine 0.02% solution, 2 ml in 10 ml of 5% glucose solution or 0.9% sodium chloride solution intravenously;

oxytocin IV, 15-20 units in 500 ml of 0.9% sodium chloride solution by infusion once daily for 1-2 days.

Because of the high risk of purulent-inflammatory diseases, after fetal extraction and clamping of the umbilical cord, intravenous infusion of ceftriaxone or cefepime 2 g 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 twice daily for 3-5 days;

according to indications, Doxilan 100 mg, 1 tablet twice daily for 5 days;

Metrogyl 100 ml IV infusion twice daily for 3 days.

Treatment is considered effective if it is possible to stop bleeding and preserve the life and health of the mother and her child. 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 the outpatient clinic, hospital, and by physicians) must be performed quickly and accurately.

If treatment or transportation of a pregnant woman is started late, or if hemostatic and blood replacement therapy is insufficient, the risk of hemorrhagic shock and disseminated intravascular coagulation (DIC) syndrome is high because of possible massive bleeding. This increases the risk to the life of both the mother and the fetus. In practice, the most common error is the delayed initiation of adequate hemostatic, surgical, and blood replacement therapy.

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

hexoprenaline IV, 0.005 mg in 500 mg of 0.9% sodium chloride solution or in 500 ml of 5-10% glucose solution/dextrose by infusion until uterine contractions are suppressed, followed by 0.5 mg 4-6 times daily for 2-3 weeks;

intravenous fenoterol (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 the prevention and treatment of anemia in pregnant women, the following are prescribed:

Ferretab, containing ferrous fumarate and folic acid as active ingredients. It is taken orally, 1 capsule daily for 4 weeks.

One tablet containing vitamin B1 100 mg, vitamin B6 200 mg, and vitamin B12 200 micrograms is prescribed 3 times daily. In early pregnancy, to prevent respiratory distress syndrome, large doses of glucocorticoid steroids should be used, in particular dexamethasone 8 mg intramuscularly every 12 hours, with a total dose of 24 mg.

Intraoperative stage

If injury to the bladder wall is suspected, cystoscopy and ureteral catheterization are performed.

• Normovolemic hemodilution is performed.

Anesthesia options: spinal anesthesia - 16%; intubation anesthesia - 84%.

Removal of the uterus with the fetus; determination of the method of uterine incision depending on the size of the "uterine hernia."

Corporal incision - 30%; lower incision - 43%; lower segment incision - 27%.

Extraction of the fetus.

Application of clamps or ligatures to the edges of the uterine incision.

Consequences of placenta previa.

The prognosis depends on the duration of pregnancy and timely medical care. During cesarean section, pregnant women with placenta previa have increased bleeding because of poor contractility of the lower segment. Because the lower segment area is insufficiently developed, placenta previa is often firmly attached and sometimes grows into the wall. In such cases, the uterus must be removed without delay.

VI. PREMATURE DETACHMENT OF A NORMALLY SITUATED PLACENTA (PDNSP)

Premature detachment of a normally situated placenta (separatio placentae normaliter insertae spontanea; PDNSP, Uzbek abbreviation: NJYMOA) is detachment before fetal delivery, during pregnancy or labor, usually in the first stage. This pathology is dangerous for a woman's health and sometimes for her life; it is extremely dangerous for the fetus.

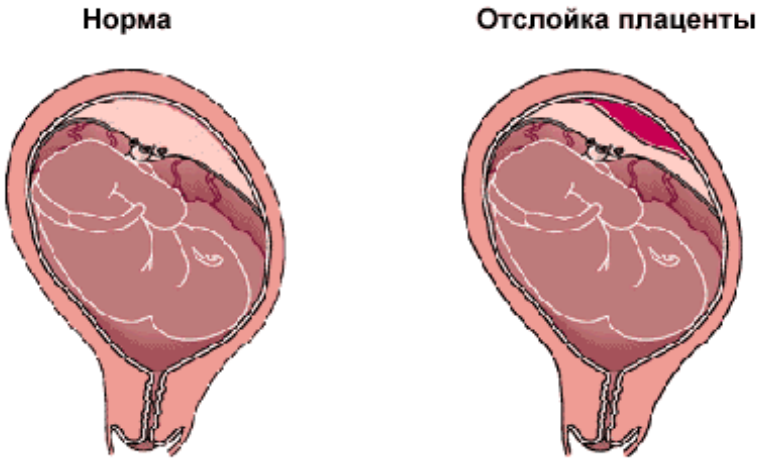


Figure. Normal placenta and premature placental detachment.

During pregnancy and labor, because of its soft and elastic structure, the placenta easily adapts to changes in intrauterine pressure and to the pressure of the uterine wall musculature closely associated with it. The pressure exerted by the uterine muscles on the placenta is balanced by intrauterine pressure, which prevents its detachment. By balancing two forces acting in opposite directions, the connection between the placenta and the uterine wall remains intact. In addition, maintenance of the connection between the placenta and uterus is facilitated by the considerable elasticity of placental tissue and by contraction of the lower placental portion of the uterus in the placental zone during labor ("progesterone block"). Any disruption of the connection between the placenta and the uterine wall during pregnancy and labor is accompanied by bleeding.

Frequency. PDNSP ranges from 0.8% to 2.5%, although in practice it is encountered more frequently. Physicians often do not pay sufficient attention to the presence of pain and bleeding in the second trimester of pregnancy, where early placental detachment may occur. Maternal mortality ranges from 2% to 15% and is caused by the consequences of this pathology: disseminated intravascular coagulation syndrome with subsequent development of multiple organ and multisystem failure, including acute renal failure in the anuric stage, cerebral and pulmonary edema, and other complications. Perinatal mortality in premature placental detachment is 25-45% and is caused by hypoxia and fetal immaturity.

Etiology and pathogenesis. The main cause of early detachment of a normally situated placenta cannot always be determined.

What are the risk factors for placental abruption?

A number of clinical and epidemiological studies have identified risk factors for placental abruption. The most predictive factor is abruption in a previous pregnancy. A large observational study conducted 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 for placental abruption 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 due to domestic violence during pregnancy, smoking, and substance abuse including cocaine and amphetamines.

Bleeding in the first trimester increases the risk of abruption later in pregnancy. A retrospective study in Denmark showed that a 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 an increased risk of placental abruption; when a retroplacental hematoma is detected by ultrasound in the first trimester, the risk of subsequent placental abruption increases.

Etiological factors include pregnancy-related hypertensive disorders, hypertension, gestosis, especially its severe forms, pyelonephritis, allergic conditions, blood diseases such as thrombocytopenia, and hypertension associated with young or advanced age in primiparous women. Placental abruption may occur in multiparous women and in the presence of developmental anomalies and uterine tumors, polyhydramnios, autoimmune conditions such as lupus antigen, and other factors.

One of the main causes of premature detachment of a normally situated placenta is preeclampsia, especially its severe forms (12.6-90%).

Hypertension in pregnant women plays a major role in placental abruption. In this condition, intravascular blood flow is reduced and, in many cases, chronic vascular disorders are present; under such conditions, placental abruption manifests in a severe form. Reduced platelet activity contributes to detachment. External trauma, a short umbilical cord, and uterine anomalies or tumors may also contribute.

Autoimmune conditions accompanied by hemostatic disorders and leading to the development of destructive changes in the placenta deserve special attention, including antiphospholipid syndrome and the presence of lupus antigen. In this case, the antigen-antibody reaction plays an important role.

The risk of placental abruption is high in subsequent pregnancies. According to some authors, recurrent placental abruption is observed in 1 of 6-8 women during subsequent pregnancy. It is difficult to predict the timing of placental abruption. Cases have been described in which normal non-stress tests and normal contraction tests were recorded 4 hours before the onset of placental abruption and rapid fetal death.

Placental abruption should not be regarded only as an acute pathology. It is the final stage of severe pathological conditions that are not always clinically detected.

Premature placental abruption is the result of vasculopathy, increased capillary permeability and fragility, and microcirculatory disorders. Similar vascular changes occur in gestosis, pyelonephritis, hypertension, and other conditions.

Early placental abruption is often observed in multiparous women, which is associated with changes in the uterine mucosa.

Many believe that placental abruption occurs as a result of gross changes at the placental site in severe gestosis, nephritis, hypertension, and heart defects.

We emphasize that the leading pathogenetic factor of early placental abruption is disseminated intravascular coagulation. Many believe that coagulopathy during early placental abruption develops through the mechanism of thrombohemorrhagic syndrome, in which two phases should be distinguished: the first, early phase is a stage of sharp intensification of intravascular coagulation due to the entry of thromboplastic substances into the bloodstream, leading to defibrination; the second is a late phase characterized by increased anticoagulant activity of the blood. In this situation, lysis of the formed fibrin occurs. Mobilization of fibrin in the area of the retroplacental hematoma and massive blood loss also play a role in blood defibrination during early placental abruption.

Placental abruption begins with hemorrhage in the decidua basalis. Then the decidua ruptures, leaving a thin layer attached to the myometrium. Subsequently, a hematoma forms in the decidual tissue, which leads to separation, compression, and destruction of the placenta adjacent to this area. Clinical signs may be absent in the initial stage. After delivery, examination of the maternal surface of the placenta may reveal changes in the form of an impression several centimeters in diameter, covered with a dark blood clot.

In some cases, decidual arteries rupture and lead to the formation of a retroplacental hematoma, which disrupts vessel integrity, separates the placenta, and increases bleeding. The area of detachment rapidly expands and may reach the edge of the placenta. Because the uterus is stretched by the enlarging hematoma, its contractile ability decreases, and the ruptured vessels at the placental attachment site are not compressed. The accumulated blood may separate the membranes from the uterine wall and flow outward, or it may collect in the uterus as a retroplacental hematoma. In some cases, blood penetrates into the thickness of the myometrium and reaches the serous membrane; less often it enters between the leaves of the broad ligament, the ovaries, and even the free abdominal cavity. This pathological condition is called uteroplacental apoplexy, first described by A. Couvelaire in 1911.

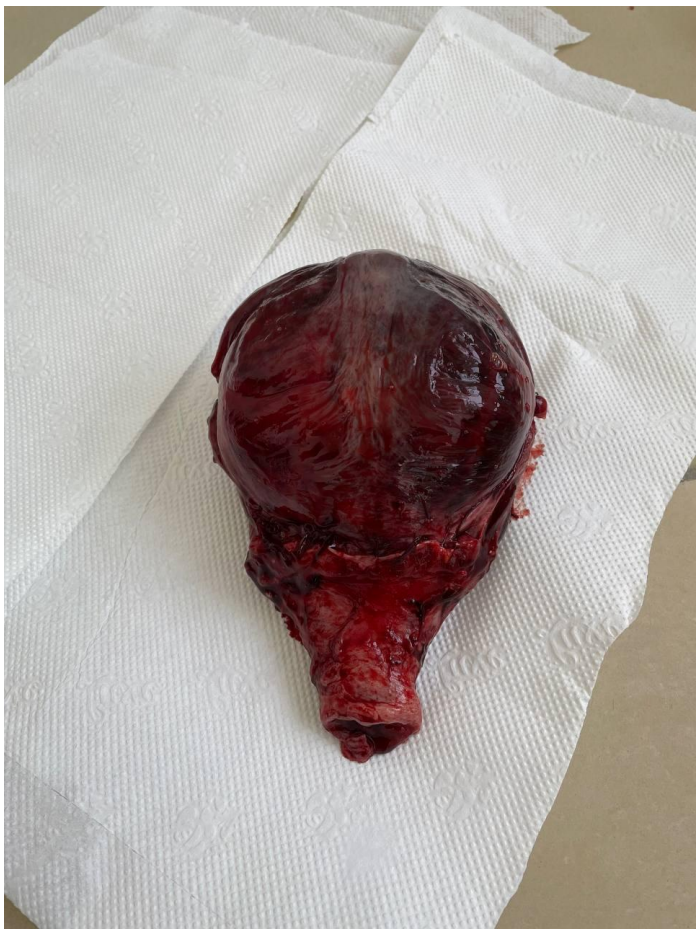


Figure 15. Uterine apoplexy (Couvelaire uterus) in premature detachment of a normally situated placenta: extensive hemorrhage into the uterine wall and beneath the peritoneum.

In this pathological condition, uterine contractility is often impaired, and severe bleeding after delivery is observed as a manifestation of DIC syndrome.

When the placenta separates, if its integrity is preserved (20%) or if it is damaged (30%), fetal blood may enter the maternal circulation (fetomaternal hemorrhage), which can be detected by examination of maternal blood smears (Kleihauer cells).

Despite numerous experimental and clinical studies, the pathogenesis of placental abruption cannot be considered fully studied. Most authors dealing with this problem reasonably believe that the most important factors in the pathogenesis are hemodynamic and microcirculatory disorders in the uteroplacental circulation, as a result of which the most important functions of the entire fetoplacental system are impaired.

In conclusion, it should be noted that PDNSP should not be considered an acute, sudden coagulopathic catastrophe, as it is often interpreted by obstetricians. Placental abruption is the result of long-standing background factors and newly emerging pathogenetic factors.

Risk factors:

• Pregnancy-induced arterial hypertension.

• History of multiple births.

• Short umbilical cord.

• Combination of arterial hypertension and a history of 3 or more deliveries.

• History of placental abruption.

• Premature rupture of the amniotic membranes.

• Smoking, especially more than 1 pack of cigarettes per day.

• Alcohol abuse and drug use.

• Pregnant woman older than 35 years.

• Polyhydramnios and multiple pregnancy.

Classification of PDNSP:

In classification, it is considered appropriate to base the division on the presence or absence of external bleeding caused by the variants of detachment (E.A. Chernukha, 2003).

In this case, the following forms can be 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.

Partial, progressive and non-progressive, as well as complete detachment of a normally situated placenta may occur. Non-progressive detachment is referred to by foreign authors as chronic when the retroplacental hematoma does not increase.

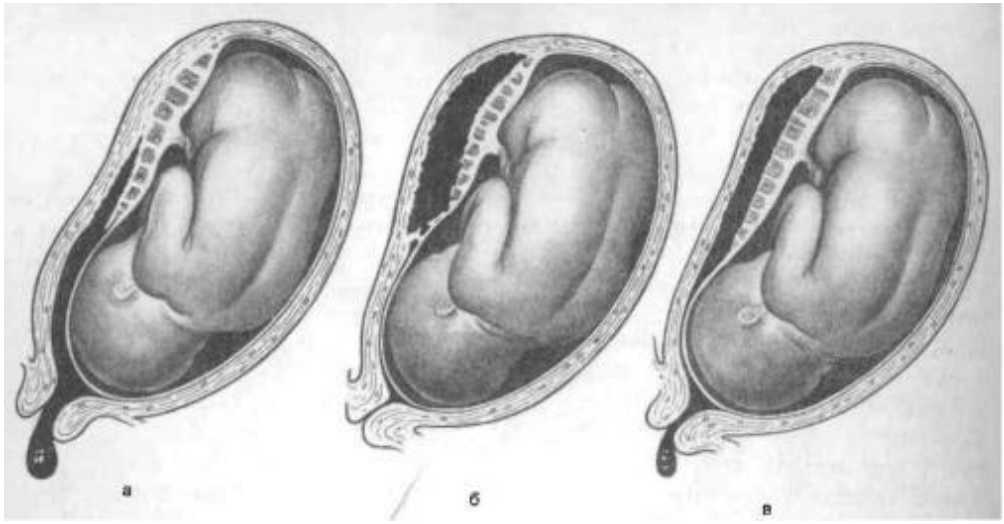


Figure 16. Variants of detachment of a normally situated placenta: a) partial detachment of the placenta with external bleeding; b) complete detachment of the placenta (retroplacental hematoma, internal bleeding); c) complete detachment of the placenta with internal and external bleeding.

Clinical presentation. The main signs of detachment are bleeding and pain. In terms of severity, they vary within very wide limits: sometimes they may be barely noticeable or even absent. In other cases, these symptoms are so pronounced that the presence of only one of them is sufficient to make the diagnosis and conclude that there is a threat to the life of the woman and fetus.

Mild detachment often does not manifest itself and is detected only after delivery when the maternal surface of the placenta is examined; small blood clots are found.

Internal bleeding (retroplacental hematoma) is much more difficult to diagnose than external bleeding. In this case, the main symptoms are pain and fetal hypoxia. In combined bleeding, the severity of internal bleeding cannot be assessed by the intensity of external bleeding. According to specialists, external bleeding prevents further separation of the placenta. If it begins as internal bleeding, it may remain concealed or may separate the membranes from the uterine wall and flow outward. In rare cases, a Couvelaire uterus forms. Very rarely, with widespread subserosal hematomas, the integrity of the peritoneum is disrupted and blood flows into the abdominal cavity.

Retroplacental hematomas may be single or multiple. The degree of bleeding depends on the site of placental detachment, being more pronounced with central detachment, the area of detachment, the severity of vascular damage, and the state of blood coagulation.

When external bleeding appears soon after detachment, the blood is usually bright red. If a certain time has passed between detachment and the appearance of blood, the blood is dark in color.

External bleeding during premature detachment of a normally situated placenta, unlike bleeding in placenta previa, does not increase during contractions; on the contrary, it stops.

Another main symptom is abdominal pain. Pain during placental separation occurs because of stretching of the uterine wall, infiltration of the uterine wall with blood, and irritation of the peritoneum.

Pain is especially pronounced with internal bleeding. However, there is no parallel between the degree of bleeding and the intensity of pain. Sometimes the pain may be so severe that it can only be compared with pain before uterine rupture or before rupture of the tube in ectopic pregnancy. The pain sometimes radiates to the symphysis and thigh and is often prolonged.

On objective examination, sharp pain is detected on abdominal palpation. The contours of the uterus are preserved, but its shape and size often change. Because of pain, palpation of the fetus is often impossible. Fetal motor activity may be pronounced or weakened; the fetal heartbeat is frequent (tachycardia) and often cannot be heard.

With slight blood loss, the woman's visible mucous membranes and skin are usually normal or pale, and the pulse is rapid but remains well filled.

With moderate blood loss, the visible mucous membranes and skin become pale, the skin is cold to the touch, sweat appears on the forehead, and the whole body becomes moist. Body temperature falls below normal. The pulse is frequent, weak, and tense. Blood pressure decreases. Breathing becomes faster.

With large blood loss, sharp abdominal pain is often disturbing and signs of shock are expressed to varying degrees. In such cases, the skin and visible mucous membranes become sharply pale, the pulse is frequent, and blood pressure is low. Dizziness, severe weakness, shortness of breath, and depression appear.

If bleeding is not stopped in time and anemia and shock are not treated, the pulse becomes thready, yawning occurs, sticky sweat appears over the entire body, and complaints of visual disturbance, thirst, and vomiting develop. Later, the patient becomes apathetic and indifferent to everything around her and gradually loses consciousness. Then coma and death occur.

In the development of shock during placental abruption, in addition to the volume of blood loss, the amount of thromboplastin entering the maternal bloodstream from the placenta plays an important role, leading to intravascular coagulation and the development of acute cor pulmonale. Experiments have shown that rapid intravenous administration of a large dose of thromboplastic substance causes severe shock.

Signs of anemia and its consequences do not always develop in parallel with blood loss. Sometimes a patient may tolerate major blood loss relatively easily, whereas in others even moderate bleeding may cause a severe reaction.

Currently, the following classification of PDNSP is used:

I. By degree of severity:

• Mild placental detachment - detachment of a small area.

• Severe degree - detachment of one-third or two-thirds of the placenta, or complete detachment.

II. By character:

• Detachment with external bleeding;

• Detachment with internal bleeding;

• Detachment with combined bleeding, that is, both external and internal bleeding are present.

III. By area of detachment:

• Partial detachment - progressive and non-progressive;

• Complete detachment of the placenta.

In the mild form, there is no pain; the uterus relaxes completely between contractions, and the fetal heartbeat does not suffer. The only sign may be a small amount of dark red discharge from the genital tract. The diagnosis is usually made after examination of the placenta, when small crater-like depressions filled with dark blood clots are found on the placental surface.

A severe form (acute placental insufficiency) is observed when one-quarter to two-thirds of the placental surface, or more than two-thirds of the placenta, is detached. Initial symptoms may develop gradually or suddenly, with constant abdominal pain and subsequent discharge of dark blood from the genital tract. Sometimes severe signs of shock are present. Uterine tone increases generally or locally, and the uterus does not relax completely between contractions. The fetus suffers from hypoxia, and intrauterine death may occur. Because of marked uterine tone, it is difficult to hear the fetal heartbeat. The onset of the disease is usually sudden, with acute abdominal pain, severe weakness, dizziness, and often fainting. The patient becomes restless and moans. The skin and visible mucous membranes are pale, the face is covered with cold sweat, breathing and pulse are accelerated, and the heartbeat is weak and tense. Blood pressure decreases. The abdomen enlarges sharply, the uterus is tense, with “local swelling,” and the small fetal parts and heartbeat cannot be determined. External bleeding from the genital tract is absent or moderate.

The severity of the condition during premature placental abruption is determined not only by the magnitude and rate of blood loss, but also by the entry of large amounts of active thromboplastins into the maternal bloodstream.

In severe cases of placental abruption, renal failure sometimes develops. In addition to major blood loss, this is primarily due to reduced renal perfusion caused by decreased cardiac output, intrarenal vasospasm, and reduced hypovolemia. Renal failure manifests as cortical glomerular necrosis. Cases of hemodialysis for placental abruption have been described. Renal dysfunction is often caused by delayed and inadequate treatment of hypovolemia. To prevent the development of renal dysfunction, administration of blood components, blood substitutes, colloid solutions, and crystalloid solutions should be started promptly.

Diagnosis. During pregnancy, a small detachment of a normally situated placenta accompanied by mild pain in the uterine area, in the absence of external bleeding, can only be suspected without the use of special research methods such as ultrasound or MRI. The final diagnosis can be made after delivery, when depressions and blood clots are found on the maternal surface of the placenta.

During examination of pregnant women, premature detachment of the placenta should be differentiated from its presentation, rupture of the marginal sinus of the placenta, rupture of umbilical vessels, uterine rupture, and other conditions (Table 2).

Differential diagnosis of bleeding in late pregnancy

Signs	Placenta previa	PDNSP	Umbilical cord presentation
Volume of blood loss	Variable	Variable	Not large
Duration of bleeding	Often stops within 1-2 hours	Usually prolonged	Stops within a short time
Abdominal pain	No	May be severe	No
Fetal heartbeat	Normal	Tachycardia, then bradycardia, later antenatal fetal death	Tachycardia, then bradycardia, later antenatal fetal death
Coagulation disorder	Sometimes	Rarely, but may accompany it; possible development of a severe form of disseminated	Not observed

		intravascular coagulation syndrome	
Features of the history	Frequent abortions, infectious complications after childbirth and abortion, hypomenstrual syndrome, infertility	Abdominal trauma, maternal hypertension, multiple pregnancy, polyhydramnios, preeclampsia in pregnant women	No

When the clinical picture of placental abruption is clearly expressed, the diagnosis is established on the basis of the history (sudden onset of the disease, acute pain in the uterine region, gestosis, hypertension, pyelonephritis, abdominal trauma, etc.) and objective examination findings. The uterus is tense, dense, asymmetric, and sharply painful in a certain area. Small fetal parts are not identified. On auscultation, fetal tachycardia or bradycardia is noted, and sometimes the fetal heartbeat is not heard. Usually, at the onset of the disease, bloody vaginal discharge is not detected.

During pregnancy, vaginal examination shows that the cervix is preserved, the external os is closed, and the presenting fetal part is located high. The variant of placental detachment is of great importance for making the diagnosis. Thus, when the placenta separates from the edge, external bleeding occurs and is usually not accompanied by pain. With central placental separation and the development of a hematoma, external bleeding is not observed even in the presence of severe pain. This is a very dangerous form that leads 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 one of the important diagnostic signs of this pathology. The leading clinical signs of this pathology are bleeding and signs of fetal hypoxia.

With placental abruption, an increase or decrease in fetal motor activity is often observed.

The pronounced clinical picture of placental abruption is characterized by changes in the hemostatic system. If hypercoagulation is observed during physiological pregnancy, then in pregnant women with premature placental abruption, isocoagulation or hypocoagulation is detected, which is associated with consumption of coagulation factors. At this stage of abruption, a decrease in the

platelet count and fibrinogen concentration occurs, with a further decrease in the level of antithrombin III and an increase in the concentration of fibrinogen degradation products.

With complete premature placental abruption and fetal death, varying degrees of hypocoagulation are observed, and a threefold increase in the concentration of antithrombin III is detected compared with the values in uncomplicated pregnancy. An increase in the content of fibrinogen degradation products indicates consumptive coagulopathy, which causes circulatory disorders and the development of hemorrhagic shock.

Even with mild placental abruption, thromboplastic substances of tissue and cellular origin enter the maternal bloodstream, resulting in hyperbilirubinemia and intravascular coagulation. The extent of this process depends on the volume of placental separation and the time over which it develops.

The most important indicator of the functioning of the hemostatic system during placental abruption is the degree of intravascular conversion of fibrinogen into fibrin. This can be assessed most accurately by determining the dynamics of antithrombin III, heparin cofactor (a natural anticoagulant), and fibrinogen degradation products in blood serum. A decrease in the level of antithrombin III in women with placental abruption is an indicator of reduced total anticoagulant activity of plasma due to intensive consumption of antithrombin III during activation of coagulation factors (XII, XI, IX, VII, V).

It has been established that the concentration of soluble complexes of fibrin monomers and fibrinogen degradation products is directly dependent on the degree of placental separation. Thus, in complete abruption, the concentration of fibrinogen degradation products is five times higher than in uncomplicated pregnancy. The severity of coagulopathic disorders also depends on the duration of premature placental abruption.

The main mechanism in the genesis of coagulation defects in placental abruption is almost undoubtedly activation of intravascular coagulation and, to a lesser extent, retroplacental coagulation. Although a corresponding amount of fibrin is usually present in the uterus in severe placental abruption and hypofibrinogenemia, it is not sufficient to enter the bloodstream. It has been found that the degree of degradation of fibrinogen products is higher in peripheral blood serum than in serum from blood located in the uterine cavity.

In the first stage of labor during placental abruption, the amniotic sac is usually tense, and sometimes a moderate amount of bloody uterine discharge with clots appears. When the amniotic sac is opened, the released fluid is markedly stained with blood. Detection of placental tissue during vaginal examination indicates placenta previa. In placental abruption, signs of anemia appear early: pallor

of the mucous membranes and skin, increased heart rate, and somewhat later a decrease in blood pressure are noted.

Ultrasound examination is an important aid in diagnosing premature abruption of a normally situated placenta, especially in cases of concealed bleeding. Longitudinal and transverse scanning makes it possible to determine the site of placental separation, the size of the retroplacental hematoma, and its structure. In marginal placental abruption with external bleeding, ultrasound examination may not reveal the pathology.

The clinical diagnosis is confirmed by ultrasound examination in only 25% of cases; therefore, negative ultrasound findings do not exclude a life-threatening placental abruption. It has been found that abruption is most often observed when the placenta is located on the anterior wall of the uterus or at the transition to one of the lateral uterine walls. When the placenta is localized in the uterine fundus, abruption is observed rarely.

If the clinical picture of placental abruption is not clearly expressed, the diagnosis is made by excluding other diseases.

Clinical recommendations

Therapy for PDNSP should be directed toward treating the factors that cause placental abruption, reducing uterine tone, correcting hemostasis, and combating anemia and shock.

The choice of treatment method for PDNSP depends on the mass and volume of blood loss, the general condition of the pregnant woman or woman in labor, the condition of the fetus, the time of bleeding (during pregnancy or labor), the type of bleeding (concealed or external), the condition of the birth canal (degree of cervical dilatation), and the state of hemostasis.

The main indicators in choosing the method of treatment for premature placental abruption are the severity of bleeding and the condition of the mother and fetus.

An important factor in choosing the method of therapy is the time of abruption, whether during pregnancy or during labor. In cases of abruption during pregnancy, taking into account the condition of the mother and fetus, if there are no conditions for rapid delivery, conservative treatment may be carried out or delivery may be performed by cesarean section.

Pregnant women suspected of premature placental abruption should be hospitalized in a maternity hospital, preferably where anesthesiology and resuscitation services, operating physicians, a laboratory, an organized blood service, and an operating unit are available.

The main task is timely control of uterine bleeding.

Management tactics for pregnancy at 34-36 weeks in PDNSP.

During pregnancy (up to 34-36 weeks), in cases of mild placental abruption, if the condition of the pregnant woman and fetus is not significantly worsened and there is no marked external or internal bleeding, conservative treatment may be performed. Bed rest, ultrasound examination, cardiotocography, monitoring of the coagulation system, administration of mild antispasmodics, multivitamins, iron preparations, and other measures are indicated.

Magne B6: 2 tablets twice daily for 1 month;

12.5% sodium etamsylate solution: 4 ml intravenously or intramuscularly, then 2 ml 4 times daily for 7-10 days;

drotaverine 2% solution: 4 ml intramuscularly, then after 4 hours 2 ml intramuscularly, 4-6 times daily for 7-10 days;

dicynone 4 ml intravenously, then 2 ml intramuscularly 3-4 times daily;

multivitamins, iron and zinc preparations (zinc sulfate solution, 1 tablespoon 4-6 times daily).

In the event of placental separation, beta-mimetics must not be used, because they relax the uterus, relieve pain and contribute to further separation, cause vasodilation, and adversely affect maternal cardiac activity.

In the presence of antiphospholipid syndrome, systemic lupus erythematosus, thrombocytopenia, and other blood diseases, the state of hemostasis should be carefully monitored and appropriate treatment should be administered.

Examination for signs of premature placental abruption includes blood tests, urine tests, biochemical blood tests, blood electrolytes, hemostasiogram, ultrasound, cardiotocography, Doppler measurements, and other investigations.

The prognosis of premature detachment of the placenta is difficult. Its further development and transition from a mild form of the disease to a severe form cannot always be predicted. Particular caution is required in the presence of small repeated bleeding episodes that indicate progression of abruption. In such cases, even if the pregnant woman's condition is satisfactory, the question of abdominal delivery should be raised.

If the clinical picture of placental abruption during pregnancy is severe (bleeding, fetal hypoxia, severe pain), urgent delivery by cesarean section is recommended. If uterine apoplexy (Couvelaire uterus) is detected during cesarean section, hysterectomy without adnexa is performed; if the adnexa are also infiltrated with blood, removal of the uterus with the adnexa is necessary because of the risk of postoperative bleeding due to uterine hypotonia and coagulopathy associated with DIC syndrome.

In our opinion, in such cases it is preferable to perform this operation with ligation of the internal iliac arteries; in this way, blood flow to the pelvis can be sharply reduced within 1.5-2 hours.

Management tactics in premature detachment of a normally situated placenta.

If a pregnant woman is in the first stage of labor, if there is no significant bleeding and the condition of the woman and fetus is satisfactory, the amniotic membranes may be opened and labor may be managed conservatively under continuous monitoring.

Stimulation of labor is not recommended; the use of oxytocin is not recommended. Increased uterine contractile activity promotes entry of thromboplastin into the maternal circulation and causes and aggravates coagulopathy. In addition, it may lead to amniotic fluid embolism. If, during careful monitoring in labor, bleeding increases, uterine tension increases, signs of fetal distress (decelerations, etc.) are noted, and there are no conditions for rapid delivery through the natural birth canal, then delivery by cesarean section is recommended.

In the second stage of labor, the main sign of premature placental abruption is acute fetal hypoxia. If the necessary conditions are present (complete cervical dilatation and the fetal head in the pelvic cavity), urgent delivery is performed by applying obstetric forceps.

In severe placental abruption, the placenta is delivered immediately after the birth of the fetus. In this case, a significant amount of blood and clots are discharged. In partial placental detachment, manual separation of the placenta and removal of the placenta are performed with a control examination of the uterine walls to check its integrity. During delivery through the natural birth canal, prophylactic administration of uterotonic agents in the third stage of labor and early postpartum period is indicated. Bleeding in the afterbirth and early postpartum periods occurs in 5.0-13.1% of cases.

In the event of premature placental abruption with a pronounced clinical picture of deterioration in the general condition of the woman in labor (shock) and the fetus, the only correct method of delivery is cesarean section.

To prevent bleeding in the early postoperative period, the use of uterotonic agents and monitoring of hemostasiogram data are indicated.

To restore blood loss, transfusion of blood (packed red blood cells), fresh frozen plasma, blood-substituting fluids, colloid solutions, and other agents is used. In bleeding, blood and physiological solutions are administered in a ratio that raises the hematocrit and maintains it at 30% or slightly higher, with diuresis of at least 30 ml/hour (preferably 60 ml/hour). Furosemide should not be used for oliguria. If intensive fluid therapy does not eliminate oliguria, central venous pressure should be determined. If early detection of pulmonary blood congestion is not possible,

other signs should be monitored, including shortness of breath, cough, and wheezing. Administration of furosemide is indicated when pulmonary congestion is present.

In newborns, trauma, prematurity, hypoxia, and sepsis predispose to intravascular coagulopathy.

Bleeding after delivery through the natural birth canal most often occurs due to disorders of blood coagulation and impaired uterine contractility. Intravenous administration of uterotonic agents (oxytocin, prostaglandins) is recommended to stop bleeding. To exclude traumatic injuries, manual examination of the uterine walls and inspection of the cervix and birth canal are necessary.

The most effective means for stopping coagulopathic bleeding are intravenous administration of fresh frozen plasma, fresh donor blood, tranexamic acid, fibrinogen, ethoxylated starch, and cryoprecipitate. In thrombocytopenia, transfusion of platelet mass is indicated to increase the platelet count to 50 thousand. After delivery, coagulation defects usually recover spontaneously within 24 hours, and the platelet count reaches normal values within 2-4 days.

Maternal mortality in premature detachment of a normally situated placenta ranges from 1.6 to 15.6%. The main causes of death are shock and bleeding.

Perinatal mortality in premature placental abruption ranges from 20.0 to 35.0%; it is caused by intrauterine hypoxia and fetal immaturity and depends on the size of the abruption and the severity of bleeding. Many newborns develop neurological damage.

In the third stage of labor and the early postpartum period, during delivery through the natural birth canal, uterotonic agents are used for prophylactic purposes (oxytocin 40 IU, methylergometrine 2 ml intravenously).

At present, plasma-substituting solutions are widely used: aminoselmin, infusol, refortan, stabizol, and others. The daily dose of these drugs is at least 1000-1200 ml intravenously. Issues of transfusion therapy and resuscitation in pregnant women, women in labor, and postpartum women with PDNSP should be coordinated with a general practitioner and especially with an anesthesiologist-resuscitator.

Therapy in the postpartum and postoperative period

If postpartum bleeding or bleeding during surgery continues, the following blood products may be administered:

- Cryoprecipitate 300-400 ml intravenously;
- Aprotinin intravenously by drip, 50-100 thousand units up to 5 times daily;
- hydroxyethyl starch derivatives;
- non-citrated plasma 150-200 ml;
- fibrinogen is administered intravenously by drip 1-2 times daily;
- platelet mass;

- Intensive therapy is continued until bleeding stops.

Surgical intervention.

Indications for surgical treatment include severe degrees of placental abruption as well as partial progressive placental detachment. Cesarean section is performed. If areas of imbibition are present in the uterus, hysterectomy is performed without delay. Unjustified conservative treatment may subsequently lead to the development of hemostatic disorders, disseminated intravascular coagulation (DIC) syndrome, and hemorrhagic shock.

Some authors propose ligation of the internal iliac arteries before removal of the uterus in order to reduce blood loss during surgery. In cases of severe placental abruption and complete uterine paralysis, we recommend performing the Porro operation, that is, removal of the uterus together with the fetus without opening its cavity. This maneuver prevents excessive blood loss.

Surgical treatment of pregnant women with premature abruption should be carried out jointly with an anesthesiologist-resuscitator, a transfusiologist, and a therapist, whose tasks include:

- complete pain relief
- support of macro- and microcirculation: controlled hemodilution using blood plasma-substituting solutions, dextrans, glucose, and other preparations
- correction of metabolic acidosis
- administration of glucocorticoids
- adequate diuresis
- mechanical ventilation
- treatment of DIC syndrome.

In the postoperative period, continue intensive therapy under clinical and laboratory control for at least 5-6 days.

The effectiveness of treatment in PDNSP consists of the following: reducing uterine tone during pregnancy, stopping bleeding, normalizing the condition of the fetus, and extracting it from the uterine cavity as quickly as possible.

Complications: development of DIC syndrome and multiple organ failure.

Prevention of premature detachment of a normally situated placenta is associated with timely diagnosis and treatment of gestosis, hypertension during pregnancy, kidney disease, antiphospholipid syndrome, and other diseases that contribute to premature placental detachment.

VII. BLEEDING DURING LABOR AND IN THE EARLY POSTPARTUM PERIOD.

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

Every year, about 140 thousand women worldwide die from postpartum hemorrhage, one woman every 4 minutes. In the Russian Federation, bleeding during pregnancy, childbirth, and the postpartum period ranks first among the causes of maternal mortality.
(approximately 17%), whereas in Uzbekistan it accounts for 38-40%. Fatal bleeding most often occurs against the background of gestosis (58%) and extragenital pathology (59%). Thus, in combination with preeclampsia, severe hypotonic bleeding causes the death of 36 women in labor, and in combination with hypertensive disorders and somatic pathology, 49%.

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

Thus, the causes of bleeding in the afterbirth and early postpartum periods are factors that disrupt the physiological process of uterine contraction and clot formation; bleeding from placental vessels is regarded as pathological blood loss. Therefore, the volume of pathological blood loss after delivery always exceeds 0.5% of body weight.

The causes of postpartum hemorrhage may be four main etiological processes called the '4 Ts' (clinical guideline for the management of patients with bleeding during labor and in the postpartum period, Tashkent, 2007).

Table 3

Etiological risk factors for postpartum hemorrhage

T	Etiological process	Clinical risk factor
T - tone (impairment of the contractile function of the uterus)	Overdistension of the uterus; 'fatigue' of the contractile ability of the myometrium; infectious process; functional/anatomical characteristics of the uterus	Polyhydramnios Multiple pregnancy Large fetus Rapid labor Prolonged labor History of a large number of

		deliveries (more than 5) Chorioamnionitis Fever during labor Uterine fibroids Placenta previa
T - tissue (retention of tissue in the uterine cavity)	Retention of placental parts; retention of blood clots in the uterine cavity	Placental defect; Operated uterus; History of a large number of deliveries; Firm placental attachment; Placenta accreta; Uterine hypotonia
T - trauma (injury of the birth canal)	Rupture of the cervix, vagina, or perineum; Traumatic uterine rupture during cesarean section; Uterine rupture; Uterine inversion.	Rapid labor Operative vaginal delivery Malposition of the fetus Low position of the presenting part Operated uterus History of a large number of deliveries Placental location in the lower uterine segment
T - thrombin (coagulation disorder)	Congenital diseases (hemophilia A, von Willebrand disease); Acquired during pregnancy: idiopathic thrombocytopathy, thrombocytopenia with preeclampsia; DIC (preeclampsia, antenatal fetal death, severe infection, amniotic fluid embolism); Treatment with anticoagulants	Hereditary coagulopathies; Liver diseases; Hematomas and/or bleeding (including at injection sites); Preeclampsia, eclampsia, HELLP syndrome; Antenatal fetal death; Antepartum bleeding; Blood clots do not form

Most obstetric hemorrhages occur in the afterbirth and early postpartum periods. The hemochorial type of placentation predetermines the loss of a certain amount of blood after separation of the placenta in the third stage of labor. This blood volume, programmed by pregnancy itself, corresponds to the volume of the intervillous space and does not exceed 300-400 ml (taking into account thrombosis

of the placental site, the amount of external blood loss is 250-300 ml), or 0.5% of the woman's body weight. From the moment the uteroplacental circulation is formed, its volume is actually not used to directly meet maternal needs, and its loss in the third stage of labor does not affect the condition of the postpartum woman. In this regard, the concept of 'physiological blood loss' exists in obstetrics.

At the same time, after separation of the placenta, the placental site is opened, which is an extensive, highly vascularized wound surface. Approximately 150-200 spiral arteries open into the placental area, and their terminal portions are devoid of a muscular membrane, creating a risk of rapid loss of a large amount of blood.

Blood loss of 500 ml or more is considered hemorrhage. The response of the female body to blood loss depends on the general condition, the presence and severity of anemia, the woman's weight, and other factors. According to many authors, the frequency of blood loss exceeding 500 ml is 5-15%.

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

1. Physiological blood loss: up to 6-8% of BCC, or up to 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 more than 1000-1200 ml.

Predisposing factors for bleeding in the afterbirth and early postpartum periods include previous abortions, multiple pregnancy, a uterine scar, uterine tumors and anomalies, severe gestosis, obesity, multiple (twin) pregnancy, a large fetus, polyhydramnios, severe maternal extragenital diseases, use of tocolytics, a dead fetus, and defects of hemostasis (von Willebrand disease, etc.).

Intrapartum risk factors for bleeding include prolonged labor, rapid labor, operative delivery, and improper (excessively active) management of the third stage of labor (use of rough manual techniques, uterine massage, attempts to separate a placenta that has not separated).

Pathology of the postpartum period.

Most often, during childbirth, the physiological course of the third stage of labor is disturbed. This is manifested by delayed placental separation and bleeding in the afterbirth period.

After the birth of the fetus, the leading pathogenetic mechanism is impairment of uterine contraction due to retention of the placenta. Therefore, the main method of treatment is to empty the uterus by manual separation of the placenta and removal of the afterbirth. In cases of bleeding, rational management of the third stage of labor is of great importance. Usually it lasts from 5 to 15 minutes. If the period is longer,

the afterbirth period should be considered prolonged, indicating the presence of possible problems.

There are active and expectant approaches to management of the third stage of labor. In our republic, active management of the postpartum period has been adopted (Order No. 500).

Delayed separation of the placenta is observed with uterine hypotonia, complete firm attachment or accretion of the placenta, and anomalies of placental structure and location (attachment in the lower uterine segment or in the uterine angles).

With uterine hypo-atony due to insufficient myometrial contractility, the placenta does not separate from the placental site. The decrease in the contractile ability of the myometrium may be congenital or acquired and may be associated with hormonal dysfunction, uterine malformations, rapid operative emptying of the uterus, prolonged labor, excessive stretching of the uterine musculature (polyhydramnios, large fetus, multiple pregnancy), and uterine fibroids. In uterine hypotonia, after fetal birth, subsequent contractions may remain weak or absent for a long time.

PATHOLOGICAL ATTACHMENT OF THE PLACENTA

Various types of pathological attachment of the placenta to the uterine wall play a special role in delaying placental separation: firm attachment (placenta adhaerens) and true accretion (placenta accreta/increta, percreta). Placental accretion may be complete (along the entire placental site) or partial.

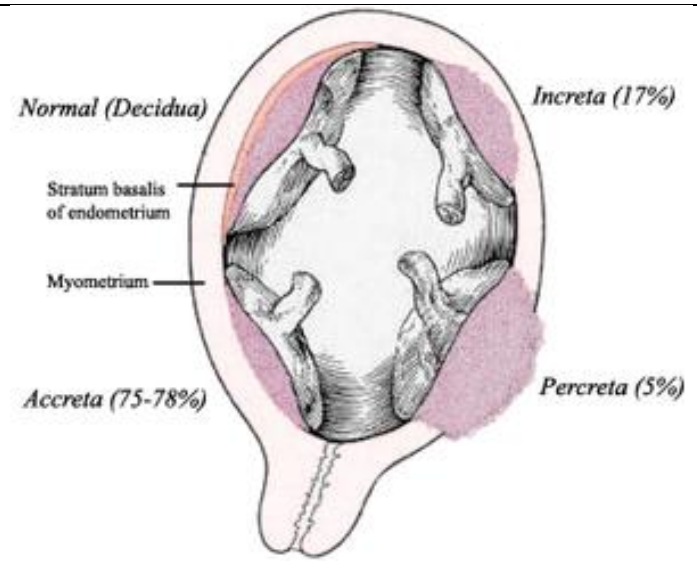


Figure 17. Pathological attachments of the placenta

The most common form of pathological placental attachment occurs when pathological changes are present in the spongy layer of the decidual membrane, where, during physiological labor, the placenta separates from the uterine wall.

In some cases, changes in the decidual membrane are so pronounced that the compact layer is underdeveloped, the spongy and basal layers are atrophic, and the Nitabuch layer (the zone of fibrinoid degeneration) is absent. Under such conditions, one or several placental cotyledons attach directly to the muscular layer of the uterus or sometimes penetrate into its thickness. In this case, true accretion develops, which occurs in one case per 10,000-15,000 deliveries.

The causes of dystrophic processes in the uterine mucosa that promote firm attachment or accretion of the placenta include postpartum and postabortion 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 probability of placenta accreta increases if it is located in the area of a postoperative scar or in sites poorly suited for implantation of the ovum, for example in the lower part of the uterus (placenta previa), as well as in uterine malformations (bicornuate uterus) and uterine neoplasms (submucosal myomatous nodes). Placenta accreta may also occur with reduced enzymatic activity of the basal layer of the mucosa, which normally prevents villi from penetrating into the uterine musculature.

The forms of pathological placental attachment can be determined only by manual examination of the uterus performed to separate the placenta. In the presence of placenta adhaerens, as a rule, all parts of the placenta can be removed manually. With placenta accreta/increta and percreta, it is impossible to separate the placenta from the wall without disrupting uterine integrity. Most often, true placental accretion is established by pathomorphological and histological examination of a uterus removed because of atony and massive bleeding in the postpartum period.

Clinical picture. Despite the different 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 rounded formation of uniformly firm consistency. The general condition of the woman in labor does not change.

The basic protocol for providing care in postpartum hemorrhage includes conservative and surgical stages of bleeding control. However, the initial stage, which includes 10 main steps, must not be forgotten. Preparation for treatment of postpartum hemorrhage is of great importance (Clinical guidelines for the management of patients with bleeding during labor and in the postpartum period, Tashkent, 2007; National standards for the provision of medical care in maternity institutions, 2015; Collection of clinical protocols, 2019). The main components of preparation for assistance can be expressed by the following points:

- Mobilize the available personnel. Without leaving the patient unattended, call an experienced obstetrician-gynecologist who fully knows surgical techniques, an anesthesiologist-resuscitator, and a laboratory assistant.
- Perform an initial assessment of the volume of blood loss: visually or using a special kidney-shaped container + 20% (when the patient is delivered by ambulance, the shock index may be used if there is no severe form of gestosis).
- Perform the initial assessment of the condition and monitor vital body functions (blood pressure, pulse, temperature, respiratory rate).
- Depending on the severity of the woman's condition, determine where care will be provided (minor or major operating room) and ensure transfer of the patient.
- If the patient's condition allows, collect an allergy history.
- Catheterize the bladder (the bladder must be empty).
- If necessary, determine the blood group and Rh factor; if necessary, take blood for compatibility testing and for performing a bedside test.
- If necessary, check the availability of blood products (according to group and Rh factor) and order them.
- If possible, determine the cause of bleeding ('4 Ts': tone, tissue, trauma, thrombin).

Measures for delayed placental separation in the absence of bleeding from the genital tract.

The woman in labor must not be moved. Separation and extraction of the placenta are performed at the place where delivery occurred.

1. Catheterization of the bladder, after which uterine contraction and placental separation occur quickly.

2. Administration of uterotonic drugs 15 minutes after expulsion of the fetus (oxytocin 5 units intravenously in 500 ml of 5% glucose solution, or 0.5 intramuscularly twice after 15 minutes, or 2 ml methylergometrine after 15-20 minutes) to increase the contractile state of the uterus.

3. Puncture or catheterization of the cubital vein and intravenous infusion of crystalloids to adequately prevent possible subsequent blood loss.

4. If signs of placental separation appear, remove the separated placenta by one of the methods for extracting the separated placenta (Abuladze, Genter, Crede-Lazarevich).

5. If there are no signs of placental separation within 20-30 minutes, against the background of administration of contracting agents, manual separation and removal of the placenta are performed under anesthesia.

After separation of the placenta and removal of the placenta from the uterus, its inner walls are carefully examined to identify accessory lobes and remnants of placental tissue and membranes. At the same time, blood clots are removed. After

removal of the placenta, the uterus usually contracts and firmly grasps the hand. If uterine tone does not recover, additional uterotonic preparations are administered and external-internal massage of the uterus on the fist is performed. After the uterus contracts, the hand is removed from the uterine cavity.

6. If true placenta accreta is suspected, attempts to separate it should be stopped and the uterus should be removed (amputation, hysterectomy). Excessive physical force when attempting manual removal of the placenta may result in massive bleeding and uterine perforation.

VIII. POSTPARTUM HEMORRHAGE

Atonic hemorrhage in the postpartum and early puerperal period deserves special attention; it is associated with impaired uterine contraction after delivery of the fetus and placenta.

Uterine hypotonia is understood as a condition in which uterine tone is markedly reduced and contractions are weakened; the uterine muscles respond to various stimuli, but the degree of these responses does not correspond to the strength of the stimulus. Hypotonia is a reversible condition.

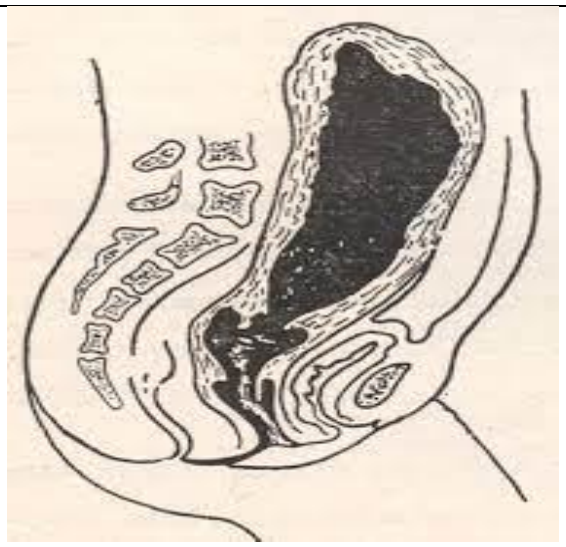


Fig. 19. Uterine atony after placental separation. The uterine cavity is filled with blood

In atony, the myometrium completely loses its tone and contractile ability. The uterine muscles do not respond to stimuli. A specific “paralysis” of the uterus develops. Atony is very rare, but it can be a source of massive bleeding.

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

- diseases or conditions that lead to uterine hypotonia or atony (diseases of the respiratory tract, heart, kidneys and liver; dysfunction of the hematopoietic organs and central nervous system; toxicoses of pregnancy; endocrine disorders; infections);

- obstetric causes (excessive uterine overdistension in polyhydramnios, multiple pregnancy, a large fetus, uterine fibromyoma, abnormalities of placental attachment, abruption of a normally situated placenta, retention of the placenta in the uterus, uterine scars after surgery, and improper management of labor).

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

Severe forms of uterine hypotonia and massive bleeding, as a rule, are combined with hemostatic disorders that occur in the form of disseminated intravascular coagulation (DIC syndrome). In this regard, special attention should be paid to bleeding that occurs after shock of various etiologies (toxic, pain-induced, anaphylactic), in amniotic fluid embolism, after collapse associated with inferior vena cava compression syndrome, or against the background of acid aspiration syndrome (Mendelson syndrome). In these pathological conditions, the cause of uterine hypotonia is blockade of the uterine contractile proteins by fibrin (fibrinogen) degradation products or by amniotic fluid.

Clinical presentation. The main sign of uterine hypo-atony is bleeding. Blood is discharged as clots of various sizes or flows out in a stream. Bleeding may have a wave-like pattern: it stops and then resumes. Subsequent contractions are rare and short. On examination, the uterus is relaxed and enlarged; its upper border reaches the umbilicus and even higher. During external uterine massage, blood clots are expelled from the uterus, after which uterine tone may be restored, but hypotonia can then recur. Relatively small fractional blood loss (150-300 ml) provides temporary adaptation of the postpartum woman to developing hypovolemia. Blood pressure remains within normal values. Pallor of the skin and tachycardia are noted. If adequate treatment is not provided in the initial period of uterine hypotonia, the severity of contractile dysfunction increases, therapeutic measures become ineffective, the volume of blood loss increases, signs of hemorrhagic shock intensify, and disseminated intravascular coagulation (DIC) syndrome develops.

In atony, the uterus is soft and doughy, and its contours are not defined. The uterus appears to spread throughout the abdominal cavity. Its fundus reaches the xiphoid process. Persistent and severe bleeding occurs. If timely assistance is not provided, the clinical picture of hemorrhagic shock develops rapidly. Pallor of the skin, tachycardia, hypotension, and cold extremities appear. The amount of blood lost after childbirth does not always correspond to the severity of the condition.

In the practical work of an obstetrician-gynecologist, the division of bleeding into hypotonic and atonic types is conditional because differential diagnosis is difficult.

The diagnosis of hypotonic hemorrhage is made on the basis of the clinical presentation.

DIC syndrome and the stage of consumption coagulopathy are assessed according to hemostatic indicators (fibrinogen and platelet deficiency, a high concentration of soluble fibrin complexes and fibrinogen-fibrin degradation products, and others).

Hypotonia and atony are usually differentiated from traumatic injuries of the soft birth canal. Severe bleeding in the presence of a large, flaccid, poorly contoured uterus through the anterior abdominal wall indicates hypotonic hemorrhage. Bleeding with a dense, well-contracted uterus indicates injury to the soft tissues of the birth canal.

The physician's tactics in hypo-atonic bleeding.

If uterine contraction is impaired in the early postpartum period and blood loss exceeds 0.5% of body weight (350-400 ml), all available means of combating this pathology must be used.

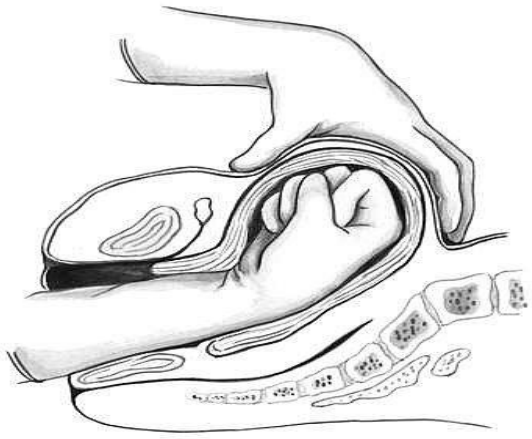
	Fig. 20. Massage of the uterus on the fist

Table 4

Algorithm of actions for early (primary) postpartum hemorrhage

Stage 1 manipulations (performed simultaneously)

Manipulation	Measures to stop bleeding
Oxygen administration by inhalation	Examination of the vulva, vagina, and cervix and, if present, suturing of soft-tissue tears of the birth canal.
Catheterization of two peripheral veins using 16G catheters	Laparotomy and suturing of uterine rupture, if present
Monitoring of vital signs (BP, pulse, respiration, oxygen saturation, diuresis)	External uterine massage
Keeping the patient warm and	Bimanual uterine compression

maintaining normothermia: intravenous administration of warm crystalloid solutions (warmed to 35-40 °C)	
Slow intravenous administration of tranexamic acid at 15 mg/kg	Aortic compression
Uterotonic therapy	

Manual examination of the uterine cavity must be performed by an experienced obstetrician-gynecologist, and repeated introduction of the hand into the uterine cavity is undesirable, useless, and very dangerous. Therefore, bimanual uterine compression is currently used (Fig. 21).

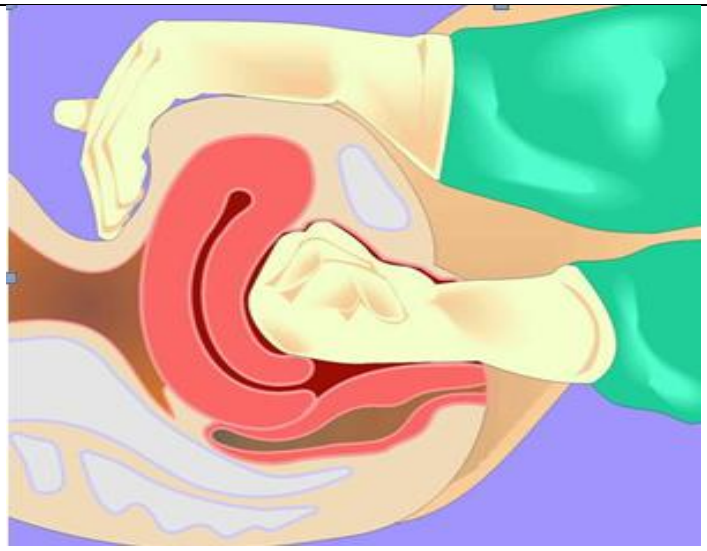


Fig. 21. Bimanual uterine compression

Conservative methods of stopping bleeding should usually be performed in two stages.

The first stage consists of emptying the bladder, external uterine massage, applying cold to the lower abdomen, and intravenous administration of uterotonic drugs (0.02% methylergometrine solution - 1 ml IV, oxytocin 40 units IV by drip).

If bleeding continues, the second stage is initiated. Manual examination of the uterine cavity is performed, followed by uterine massage on the fist under anesthesia.

If bleeding does not stop, or if it stops but resumes in small portions after 15-20 minutes, staged surgical treatment is indicated, as described below (see Chapters XI-XII).

Before placing the patient in the operating room and before preparing the surgical and anesthesiology teams, temporary clamps according to Henkel-Tikanadze or N. S. Baksheev and sutures on the cervix according to Lositskaya were

previously used to reduce blood loss before transferring the patient. At present, these methods of stopping bleeding are not used.

From the first minutes of bleeding from the genital tract, care is provided according to the principle of a multidisciplinary team with a clear distribution of tasks. The main 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 determination of its cause;
- alerting the necessary staff;
- clinical and laboratory examination;
- infusion therapy;
- uterotonic therapy;
- administration of hemostatic drugs;
- provision of the required volume of infusion and transfusion therapy.

Diagnosis, control of bleeding, and infusion therapy are carried out simultaneously with the organization of monitoring of the patient's condition. Alerting includes:

- calling a second midwife and a second obstetrician-gynecologist;
- calling an anesthesiologist-resuscitator, a transfusiologist (who must ensure the supply of fresh frozen plasma and red blood cells [erythrocytes] from the reserve), and a laboratory assistant;
- calling the duty nurse to deliver blood samples and blood components;
- appointing one member of the duty team to record procedures, infusion therapy, medications, and vital signs;
- if massive bleeding is observed, informing the admitting duty staff and the hematology consultant, calling the surgeon, and preparing the operating room.

Required manipulations:

- catheterization of a second vein (16G or 14G catheters);
- catheterization of the urinary bladder;
- oxygen administration (oxygen mask or nasal cannulae);
- monitoring of vital functions (blood pressure, heart rate, respiration, oxygen saturation, diuresis);
- slow intravenous administration of tranexamic acid at 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 their use

Uterotonic agents	Oxytocin	Methylergometrine	Misoprostol
Method of administration and dose	10 units IV in 500 ml of normal saline or Ringer's solution, 60 drops per minute.	0.2 mg IM or IV (slowly)	200 mcg
Maintenance dose	10 units IV in 500 ml of normal saline or Ringer's solution, 40 drops per minute.	Repeat 0.2 mg IM after 15 minutes; if necessary, repeat 0.2 mg IM or IV (slowly) every 4 hours	Repeat 200 mcg
Maximum dose	No more than 3 liters of solution containing oxytocin (60 IU)	5 doses (1.0 mg)	800 mcg
Contraindications	rapid administration of the drug	Arterial hypertension, preeclampsia, heart disease	Bronchial asthma

Uterotonic therapy is performed to eliminate the hypotonic component of bleeding.

Misoprostol may be used only for life-threatening bleeding when it cannot be stopped with other drugs. The decision to prescribe the drug must be made by a medical commission. The dose is 600 mcg; the route of administration is rectal.

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

To carry out infusion therapy, the following must be ensured:

- adequate intravenous access: at least two peripheral venous catheters with a diameter of 14G or 16G;
- standard monitoring (SpO2, ECG, HR, BP);
- placing the patient in a horizontal position with the foot end of the operating table (bed) elevated;

catheterization of the urinary bladder;

- equipment for warming solutions;
- equipment for warming the patient;
- equipment for rapid intravenous infusion.

Infusion therapy itself is not an indication for placement of a central venous catheter.

An important condition is warming the solutions to 35-40 °C, which helps prevent hypocoagulation and hypothermia and reduces the amount of blood loss. Infusion therapy is started immediately. As the initial solution, up to 2000 ml of warmed balanced crystalloid solutions are infused. Further replacement of the circulating blood volume (CBV) deficit may be continued by administering colloid solutions (succinylated gelatin, hydroxyethyl starch, or 5%-10% albumin solution). The volume of colloid solutions that may be administered before the start of blood transfusion is 1500 ml. When calculating the volume of infusion-transfusion therapy, it should be remembered that in massive blood loss, fresh frozen plasma (FFP) must be transfused at a volume of 12-15 ml/kg. Donor red blood cells are transfused no later than 40 minutes later, if indicated, after individual compatibility tests have been performed.

After the start of infusion therapy, blood must be taken for laboratory monitoring of the following indicators:

- hemoglobin, hematocrit, red blood cell and platelet levels;
- acid-base status (pH, BE, HCO3, SaO2, PaO2);
- electrolytes (potassium, sodium, chloride);
- glucose level;
- fibrinogen level, APTT, TT, PT.

As a result of therapy, the following indicators should be maintained:

- hemoglobin above 80 g/L;
- platelet count above 50 × 10^9/L;
- PT and APTT not more than 1.5 times the normal value;
- fibrinogen above 2 g/L (level of evidence 4).

In cases where blood loss is less than 25% of CBV (up to 1500 ml), infusion therapy is carried out in a restrictive mode, taking hemodynamic parameters into account. The basis of infusion should be balanced crystalloid solutions. Colloid solutions should be used only when signs of severe hypovolemia are present.

Patients with blood loss of 1500 ml or more, or with dysfunction of vital organs (tachycardia and hypotension), require urgent blood transfusion. In massive blood loss, the “massive transfusion” protocol should be used with the following ratio of components: packed red blood cells : FFP : platelet concentrate : cryoprecipitate = 1:1:1:1. If platelet concentrate and cryoprecipitate are unavailable,

FFP and donor red blood cells are administered in effective doses at a ratio of 1:1 or 4:6.

FFP transfusion in cases of placental abruption or amniotic fluid embolism should be started as early as possible, because these conditions are associated with early coagulopathy.

If infusion therapy is insufficiently effective, hemodynamic parameters are corrected by administering vasopressors (norepinephrine, and then dopamine if ineffective). During shock, infusion therapy is performed at the maximum rate in a volume of 30-40 ml/kg. Further infusion exceeding 200% of the calculated blood loss should be carried out only when central hemodynamic parameters can be monitored: cardiac index, stroke volume, and total peripheral vascular resistance.

Massive blood loss can lead to hyperkalemia and hypocalcemia due to red blood cell transfusion and the toxic effect of citrate.

Uncontrolled infusion therapy leads to worsening coagulopathy and fluid overload (development of pulmonary and cerebral edema). Infusion therapy must be performed under strict hemodynamic monitoring. Indicators of effectiveness include an increase in blood pressure and a decrease in tachycardia by 10% or more, with a diuresis rate of at least 0.5 ml/kg/hour. If bleeding continues, a tactic of controlled arterial hypotension should be used: mean arterial pressure is maintained above 60 mm Hg, and systolic blood pressure should not exceed 100 mm Hg. In ischemic heart disease, atherosclerotic vascular lesions, or impaired cerebral blood-flow regulation (preeclampsia, arterial hypertension), mean arterial pressure should be at least 80 mm Hg. An effective method for the prevention and treatment of obstetric hemorrhage is autologous plasma transfusion, especially in groups of pregnant women at risk of bleeding who are planning delivery.

Agents used to correct coagulation disorders include fibrinolysis inhibitors, prothrombin complex preparations, activated coagulation factor VII, cryoprecipitate, and fresh frozen plasma.

When massive blood loss develops, administration of fibrinolysis inhibitors reduces the volume of blood loss and the risk of hysterectomy (AI); it is performed only as part of complex therapy and without delaying the start of plasma transfusion.

For blood loss up to 1000 ml, tranexamic acid is administered intravenously at 15 mg/kg (on average 1000 mg) over 10 minutes; if bleeding continues, the dose may be increased to 4000 mg (40 mg/kg). Repeated administration is possible after 8 hours. A higher dose of tranexamic acid may reduce the volume of blood loss, the decrease in hemoglobin level, and the need for donor blood products. Currently, timely administration of tranexamic acid in an adequate dose helps reduce mortality, the frequency of hysterectomy, and morbidity in women with massive bleeding.

Aminomethylbenzoic acid may be used for obstetric hemorrhage at a dose of 100 mg. There is evidence for the safe use of aminomethylbenzoic acid in obstetric hemorrhage at doses up to 300 mg. Aprotinin is not recommended, because studies in patients undergoing cardiac surgery have shown a direct association with increased mortality. Large randomized controlled trials comparing tranexamic acid, aprotinin, and aminomethylbenzoic acid have not been conducted.

In the presence of hypocoagulation and/or blood loss exceeding 25-30% of CBV, FFP should be transfused. In the period before plasma transfusion begins, it is permissible to administer prothrombin complex concentrate (Octaplex or Protromplex), which contains factors II, VII, IX, and X, at a dose of up to 50 IU/kg; if there is no effect within 20 minutes, the same dose may be repeated. Data on safety and efficacy in obstetric practice are limited.

Routine use of activated factor VII for the treatment of massive blood loss in obstetrics is not recommended. Recombinant activated factor VII is used for uncontrolled bleeding at a dose of at least 90 mcg/kg. If necessary, the dose may be repeated after 3 hours. The efficacy of the drug is significantly reduced by hypothermia (below 34 °C), acidosis (pH below 7.2), thrombocytopenia (platelets below 50 × 10^9/L), and low fibrinogen (below 0.5 g/L); therefore, normal homeostasis must be maintained. The use of activated factor VII should not be delayed or regarded as a substitute for surgery.

The use of prothrombin complex concentrate, recombinant activated factor VII, and FFP requires mandatory monitoring of hemostasis parameters after 6 hours, as well as administration of a prophylactic dose of low-molecular-weight heparins after 12 hours.

Prevention of venous thromboembolic complications (VTE) should be performed in all cases of blood loss exceeding 1000 ml, provided there are no contraindications.

The efficacy of sodium etamsylate and calcium chloride as hemostatic drugs has not been proven (the latter should be used to prevent citrate intoxication and hyperkalemia during massive transfusion of red blood cells with a long storage period).

Cryoprecipitate is administered in the setting of ongoing bleeding at a dose of 4 units for every 4 units of red blood cells. The usual dose is 15-20 ml/kg; 30 ml/kg is required to reliably increase coagulation factors by 30%.

Donor platelets are administered for thrombocytopenia below 50 × 10^9/L. Initial dose: 1 adult dose (4 platelet units for 4 units of red cell mass).

The second stage in the treatment of postpartum hemorrhage is transitional.

Second-stage activities:

- continuation of infusion and transfusion therapy according to the volume of blood loss, assessment of the patient's condition, and laboratory test results;
- use of drugs that increase blood coagulation in confirmed hypocoagulation and ongoing bleeding;
- warming the patient and maintaining normothermia;
- bimanual uterine compression;
- uterine tamponade when bleeding continues despite manual examination and suturing of injuries;

If complete hemostasis is not achieved in postpartum women after vaginal delivery, preparation of the operating room should begin, anesthesia support for surgery should be provided, and repeat laboratory tests should be performed. If conditions are available, intraoperative reinfusion of autologous red blood cells should be performed.

Surgical treatment

The third stage of stopping postpartum hemorrhage is surgical treatment. Its aim is to ensure definitive cessation of bleeding by surgical methods, together with the continuation of infusion-transfusion therapy, uterotonic therapy, and normalization of the blood coagulation system.

Third-stage measures:

- transportation to the operating room;
- anesthetic management of surgery;
- repeated collection of tests to assess the dynamics of the process and the effectiveness of therapy (complete blood count, Lee-White test, hemostasiogram, urea, electrolytes);
- intraoperative reinfusion of autoerythrocytes;
- surgical treatment.

The extent of surgical treatment is determined by the clinical situation, the professional level of the physician, and the technical equipment of the institution, and in typical cases includes:

- laparotomy (if there is a uterine suture after a transverse suprapubic incision, otherwise a lower midline incision);
- ligation, tourniquet on the uterus
- compression sutures on the uterus (B-Lynch) and/or temporary complex peripheral compression of the vascular bundles;
- temporary clamping or ligation of the main uterine vessels;
- uterine artery embolization
- ligation of the internal iliac arteries;
- hysterectomy if other measures are ineffective.

The tactics of intensive care and anesthetic management should be aimed at preventing the development of hemorrhagic shock, respiratory failure (ARDS), acute kidney injury, renal failure, hepatic failure, and coagulopathy caused by acute disseminated intravascular coagulation. If possible, an additional anesthesia team should be involved in providing medical care (an anesthesiologist-resuscitator and an anesthesia nurse). Safe regional anesthesia is possible in cases of blood loss not exceeding 25% of the circulating blood volume, provided that hemodynamic parameters are stable, the patient is assessed as ASA I-III, and there are no contraindications. When performing any type of anesthesia, it is necessary to be ready to rapidly perform general anesthesia.

The following monitoring should be used in the postoperative period:

- Non-invasive blood pressure monitoring
- SpO2
- Heart rate
- ECG
- Respiratory rate
- Capnometry during mechanical ventilation (if available).
- Monitoring of diuresis rate (bladder catheterization)
- Body temperature monitoring

Before starting any type of anesthesia, venous access must be ensured. Regardless of the selected method of anesthesia, if pregnancy is present, the patient should be positioned with a left tilt of at least 15 degrees in order to prevent aortocaval compression. In hemodynamic instability, the foot end of the operating table should be elevated.

If SpO2 falls below 96% or signs of respiratory failure are present, it is recommended to immediately start oxygen therapy in the operating room through nasal cannulas or a face mask.

Correction of hypotension may be performed with ephedrine (up to a total dose of 50 mg) or bolus injections of phenylephrine 3-10 mcg, as well as by infusion of phenylephrine or norepinephrine. The initial dose should be considered 0.1-0.15 mcg/kg/min for phenylephrine and 0.04-0.06 mcg/kg/min for norepinephrine. The dose is increased until the required blood pressure level is achieved. If infusion vasopressor support is ineffective, dopamine is considered a second-line drug: the initial infusion rate is 2 mcg/kg/min (level of evidence: 4). Target hemodynamic parameters are a heart rate from 55 to 120 beats per minute and mean arterial pressure not lower than 60 mm Hg. Controlled hypotension may be used if systolic blood pressure is not higher than 100 mm Hg and mean arterial pressure is not lower than 60 mm Hg.

Premedication is performed regardless of the type of anesthesia. It should include:

- Antiemetic drugs: dexamethasone 4-8 mg and/or ondansetron 4-8 mg.
- Antacid drugs.
- Atropine 0.01 mg/kg
- Fibrinolysis inhibitors

For surgical anesthesia, spinal anesthesia or epidural anesthesia may be used if an epidural catheter is present. Performing anesthesia should not significantly prolong the preoperative period. Neuraxial blocks are contraindicated in the presence of signs of severe hypovolemia. Pre-infusion does not prevent the development of arterial hypotension against the background of a sympathetic block.

Spinal anesthesia is performed with observance of the necessary rules of asepsis and antisepsis. Puncture of the subarachnoid space is performed in the lateral position or at a level not higher than the first lumbar vertebra. Spinal needles sized 25-29G are used for the puncture. Identification of the subarachnoid space is performed by the appearance of cerebrospinal fluid in the needle hub after removal of the stylet. It is preferable to use a 0.5% hyperbaric solution of bupivacaine. The dose of anesthetic is selected individually, taking into account height, weight, general condition, and hemodynamic parameters. For anesthesia of manipulations on the cervix and perineum, administration of 5-6 mg of bupivacaine is sufficient. For anesthesia during abdominal operations, the dose of bupivacaine should be 10-12.5 mg. The use of higher doses is unjustified, as it increases the risk of hemodynamic disorders. Dose reduction may lead to an insufficient sensory block.

If an epidural catheter is installed, epidural anesthesia is performed. It is necessary to make sure that the catheter has not been displaced and to perform tension and aspiration tests. Repeated administration of a test dose of local anesthetic is not required. Injection of anesthetic into the epidural space is performed only under monitoring conditions in an equipped operating room. Ropivacaine or levobupivacaine at a concentration of 0.375% or 0.5% may be used as an anesthetic. The volume of anesthetic solution administered depends on the required level of sensory block. For anesthesia of manipulations on the cervix and perineum, 5-8 ml is sufficient; for anesthesia of abdominal operations, the anesthetic volume should be up to 15-20 ml, depending on the necessary level of sensory block and the patient's height. It should be taken into account that 15-20 minutes may be required to determine the necessary anesthetic effect.

General anesthesia is performed only when functioning equipment required to ensure airway patency, carry out mechanical ventilation, and perform CPR is available. The possibility of total intravenous anesthesia should be considered

individually, taking into account the risk of regurgitation and aspiration of gastric contents, as well as the risk of difficult airway management.

A muscle relaxant is not used during anesthesia with spontaneous breathing.

Absolute indications for mechanical ventilation:

- Circulatory arrest.
- Apnea or bradypnea.
- Weakness of the main and accessory respiratory muscles.
- Impaired consciousness due to hypoxia
- Prolonged persistent tachycardia of hypoxic origin.
- Pulmonary edema.
- Hypoxemia not corrected by oxygen therapy (PaO2 less than 60 mm Hg, SaO2 less than 90%, PaCO2 more than 55 mm Hg, vital capacity of the lungs less than 15 ml/kg).
- Ongoing bleeding, unstable hemodynamics.

Induction of general anesthesia is performed after preoxygenation for at least 3-5 minutes. Because of the presence of a 'full stomach' factor, rapid sequence induction is performed using the Sellick maneuver.

Use of general anesthesia (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 necessary)
- Propofol 2-2.5 mg/kg + fentanyl 3-5 mcg/kg; in pregnant women, the induction dose of propofol should not exceed 2.5 mg/kg. Propofol is contraindicated in severe hypovolemia.

Myoplegia:

- Succinylcholine 1.5-2 mg/kg (muscle relaxation after 20-45 seconds)
- Rocuronium bromide 0.6-1 mg/kg (muscle relaxation after 1-2 minutes)
- Cisatracurium, atracurium, vecuronium (muscle relaxation after 3-5 minutes).

After the development of sufficient muscle relaxation, orotracheal intubation is performed with a No. 7-8 tube. The position of the tube is monitored by assessing chest excursion, auscultation, and capnography. Recommended capnometry values are EtCO2 32-40 mm Hg; the calculated tidal volume is 6-8 ml/kg. If tracheal intubation is impossible, a second-generation or later laryngeal mask should be used or percutaneous access to the trachea should be performed.

If a 'full stomach' is present, the stomach is emptied using a gastric tube, after which the tube is removed.

Maintenance of anesthesia:

- **Sevoflurane, desflurane, isoflurane 0.8-1 MAC**
- **A mixture of nitrous oxide and oxygen (2:1)**

- In uncompensated shock, instead of nitrous oxide, sodium hydroxybutyrate (70 mg/kg) or ketamine (50 mg every 15-20 minutes) is used to suppress consciousness, and mechanical ventilation is performed with an air-oxygen mixture.
- Bolus injections of sodium thiopental 2-4 mg/kg or ketamine 0.3-0.5 mg/kg.
- Propofol infusion 4-12 mg/kg/hour (in the absence of pregnancy).

Analgesia: fentanyl 1-2 mcg/kg.

Provision of muscle relaxation: non-depolarizing muscle relaxants in maintenance doses.

Extubation is performed after completion of the operation when neuromuscular conduction, adequate spontaneous breathing, airway reflexes, and consciousness have recovered. Prolonged mechanical ventilation is indicated for unresolved severe anemia, an ongoing picture of hemorrhagic shock, unstable hemodynamics, and respiratory failure. Decurarization is performed in the presence of muscle relaxation by administering neostigmine methylsulfate at a dose of 10-15 mg.

Intraoperative reinfusion of autoerythrocytes (IRA) should be performed only by qualified personnel who perform it regularly and have the necessary knowledge and experience. IRA is subject to annual audit. The patient's consent must be obtained before IRA. The use of intraoperative autoerythrocyte reinfusion makes it possible to minimize the use of donor blood components in blood loss of any volume and to prevent massive transfusion syndrome; it also significantly improves the postoperative period and shortens the patient's hospital stay.

Indications for intraoperative red blood cell reinfusion during abdominal delivery: IRA is recommended for women in whom intraoperative blood loss of more than 20% of the circulating blood volume is expected.

Contraindications to intraoperative autoerythrocyte reinfusion in obstetrics.

Absolute:

- presence of purulent content in the abdominal cavity;
- presence in the shed blood of substances contraindicated for introduction into the vascular bed (hydrogen peroxide, distilled water, collagen-based hemostatic agents, etc.).

Relative:

- presence of malignant neoplasms in the patient. In obstetrics and gynecology, when autoerythrocytes are reinfused in a patient with cancer, the obtained autoerythrocytes are transfused only using leukocyte filters.

It is not a contraindication to intraoperative autoerythrocyte reinfusion in intestinal surgery, provided that preliminary evacuation of contaminated abdominal contents

is ensured, the obtained erythrocytes are additionally washed, and broad-spectrum antibiotics are used.

For IRA, Cell Saver devices, CATS autotransfusion systems, and disposable tubing sets for them are used.

Technology of using the method: the principle of the procedure is aspiration of blood shed from the surgical wound, its processing in the device, and reinfusion of the resulting autoerythrocyte suspension (Ht 60%) into the patient's vascular bed. Filling of disposable tubing sets is performed in the operating room before surgery begins. The sterile suction tip is handed to the operating nurse. Blood shed into the wound is collected by the second assistant. The negative pressure generated by the vacuum aspirator must not exceed 100 mm Hg. Blood aspirated from the wound is mixed with an anticoagulant solution, passes through a filter that retains tissue particles and blood clots, and is collected in a reservoir. When the amount of collected blood corresponds to the reservoir volume, the first stage of operation of the device begins - filling the washing bowl.

This stage consists of several steps:

- Acceleration of the centrifuge to 5600 rpm.
- Transfer of blood from the reservoir to the washing bowl using a peristaltic pump, with initiation of the centrifugation process.
- Filling of the washing bowl continues until erythrocytes fill the entire volume of the bowl (the bowl volume may be 125 ml, 175 ml, or 225 ml). During centrifugation, the separated plasma is removed together with the anticoagulant into the appropriate container. After that, the second stage starts in automatic or manual mode - washing the red blood cells in sterile physiological 0.9% NaCl solution. Washing continues until a certain volume of washing solution (1000-1500 ml in obstetrics) has fully passed through the erythrocytes. Centrifugation continues during this time.

The final stage of the device is emptying the bowl:

- The centrifuge stops and the peristaltic pump begins to rotate in the reverse direction.
- The washed red blood cells are transferred from the washing bowl into the reinfusion bag. The process is then repeated until all blood aspirated from the wound has been processed. The duration of the first cycle described is 3-5 minutes. During operation, the device display clearly reflects all necessary parameters: centrifuge rotation speed, pump rotation speed, and the amount of solution passed through. After each operating cycle, the number of collected and washed red blood cells is displayed. Reinfusion of autoerythrocytes must be performed using a leukocyte filter. Autoerythrocyte reinfusion is an effective method of restoring the globular volume during bleeding that occurs during cesarean section. The limitations of the

method are mainly related to the availability of appropriate personnel and equipment.

- Rehabilitation. After massive blood loss and hemorrhagic shock, patients are rehabilitated according to indications in departments of a level-3 multidisciplinary institution.

Within 10-20 minutes from the onset of bleeding, the following must be performed:

Continue all manipulations of the first stage:

Perform balloon tamponade; for most women in whom uterine hypotony is the main cause, it is the first-line intervention.

For refractory bleeding, uterine balloon tamponade and uterine compression sutures may be used together; an additional double-balloon uterovaginal system provides additional benefits by compressing vessels in the lower uterine segment. To reduce blood loss, balloon tamponade should be continued until hysterectomy.

If blood loss is 1000 ml and bleeding continues, or if there is a clinical picture of shock, the patient is immediately transferred to the operating room for further treatment.

If blood loss exceeds 25-30% of the circulating blood volume, surgical intervention must be performed no later than 20 minutes.

If bleeding develops during cesarean section, application of B-Lynch compression sutures is recommended (compression of the abdominal aorta before surgery helps reduce excessive blood loss).

If previous measures to stop bleeding are ineffective, the following are necessary:

- Provide anesthetic support for surgery
- Repeat the analyses

If possible, perform intraoperative reinfusion of autoerythrocytes.

Perform surgical hemostasis:

- Laparotomy or relaparotomy;
- Perform uterine devascularization, which includes ligation/temporary clamping of the main uterine vessels and/or ligation of the internal iliac arteries;

Vascular ligation. The uterine artery and vein may be ligated bilaterally either separately or together. If bleeding continues and the first ligature is ineffective, a second lower ligature may be placed to ligate the branches of the uterine artery supplying the lower segment and cervix. If this is ineffective, ligation of the utero-ovarian vessels should be performed. Ligation of the internal iliac arteries requires a high level of professionalism from the physician, as it is associated with the risk of urinary tract and vascular injury.

Angiographic embolization is an alternative to ligation of the uterine or internal iliac vessels. The procedure requires 20-30 minutes and special equipment. The effectiveness of the method varies from 58% to 98%, but hysterectomy is performed in 15% of cases.

Hysterotomy, ligation of potentially bleeding vessels of the placental bed (according to indications, in level-3 institutions with the participation of trained specialists - resection of the uterine wall from areas of placental invasion), application of compression sutures (vertical D-Lynch or horizontal), and/or temporary complex peripheral compression of the vascular bundles.

Hysterectomy is an emergency surgical intervention aimed at saving the life of the postpartum woman with uncontrolled bleeding when the previous stages of arresting bleeding have no effect. Visualization of the ureters during hysterectomy is recommended. Preservation of the uterus is permitted only if the patient is hemodynamically stable and there is no life-threatening bleeding. Relative contraindications to uterine preservation are: a large placenta with abnormal invasion (placenta accreta) or persistent bleeding from the placental site; extensive uterine damage and impossibility of reconstructive surgery; inflammatory uterine diseases.

If there is no effect from therapy, the decision on hysterectomy must be made in a timely manner and as a team.

If it is impossible to ensure the principle of staged surgical hemostasis (level-1 or level-2 obstetric hospitals), hysterectomy must be performed as early as possible as the most effective method of stopping bleeding.

For illustration, we present a specific example:

EXAMPLE No. 2

A 30-year-old multiparous woman was urgently admitted to one of the maternity hospitals for delivery with the diagnosis: Pregnancy V/I, 40-41 weeks. Labor III. OGA. First stage of labor. From the obstetric history: there had been three pregnancies, the second of which ended in term delivery, and the delivery proceeded without complications. For medical indications, there had been one abortion between deliveries, complicated by metroendometritis. For this, she was treated with antibiotics in an inpatient setting. The present pregnancy was V/I, and labor was complicated by premature rupture of the amniotic fluid and primary weakness of labor forces. Labor stimulation with Endaprost and oxytocin was administered by intravenous drip. The second stage of labor was complicated by persistent secondary weakness of labor and fetal hypoxia. Therefore, obstetric forceps were applied to the pelvic cavity. A live male fetus weighing 3400 g and measuring 53 cm was delivered. Heavy bleeding was noted in the third stage. For this reason, manual separation and removal of the placenta were performed,

followed by examination of the uterine cavity. The uterus was slightly relaxed, but after administration of methylergometrine it retracted. Wounds of the vaginal walls, vulva, and perineum were sutured. The uterus contracted and bleeding stopped. Oxytocin infusion was connected. Forty minutes after delivery, during external postpartum uterine massage, the midwife noted the discharge of 300 ml of blood clots from the uterus. The physician on duty emptied the bladder and performed repeated external uterine massage, during which another 150 ml of blood with small clots was discharged. Blood pressure decreased by 10 mm Hg (initial BP 110/70). The responsible physician was invited to repeatedly perform manual examination of the uterine cavity, followed by fist massage.

The uterus contracted and bleeding decreased. Blood pressure fell to 100/60 and then to 90/50 mm Hg. During bladder catheterization, approximately 30 ml of concentrated urine was obtained. The head of the department was called from home; after examination, he again performed manual examination of the uterine cavity. Blood pressure dropped to 80/40 mm Hg, urine output stopped, blood clotting began at 7 minutes but did not complete. Fresh frozen plasma 500 ml intravenously was connected, Gordox 200 thousand units and Contrikal 50 thousand units were administered. A small amount of liquid blood without clots was discharged from the genital tract. Total blood loss was approximately 1000 ml. Hemoglobin decreased to 50 g/L. Specialists from the regional center were called by ambulance and arrived 2 hours after the consultation; the diagnosis was made: postpartum uterine hypotony, posthemorrhagic shock stage II-III, DIC syndrome, acute renal failure in the oligoanuric stage. Surgery was performed: laparotomy revealed a hypotonic, flaccid, sharply pale uterus, with petechial hemorrhages in the area of the uterine adnexa. Extirpation of the uterus with adnexa and ligation of the internal iliac arteries were performed. The operation was performed with the participation of an experienced anesthesiologist-resuscitator, under infusion therapy, transfusion of fresh frozen plasma, red blood cells, and protease inhibitor preparations. Increased bleeding was observed during surgery, and the volume of blood loss exceeded 2 liters. Ligation of the internal iliac arteries slightly reduced bleeding, but parenchymal bleeding was noted, and the pelvis had to be tightly packed.

During suturing of the anterior abdominal wall, cardiac arrest occurred and the woman died. Death occurred due to complete exhaustion of the body's compensatory and protective capacities caused by delayed surgical tactics.

The above example shows that serious mistakes were made by the physicians when providing care to the postpartum woman (repeated external-internal uterine massage, late infusion therapy, blood transfusion, etc.). Most importantly, time for surgical treatment was lost. Therefore, the surgery was unsuccessful.

In our opinion, manual examination of the uterine cavity should be performed only under general anesthesia by an obstetrician-gynecologist. If bleeding, even in a small amount, continues after such an operation, the issue of urgent removal of the uterus should be raised, because unnecessary excessive conservatism leads to loss of time for timely surgical intervention and loss of the body's compensatory capacities.

Many such examples can be given. During manual examination of the uterus and external-internal massage surgery, a biological test for contractility is performed. For this purpose, at the end of uterine massage, a uterotonic drug is administered intravenously (methylergometrine 0.02% - 2 ml in 5 ml of physiological solution, or 1 ml of oxytocin). If there is an effective contraction that the physician feels by hand, the treatment result is considered positive, and the operation is completed by removing the remaining clots from the uterine cavity. If manual examination of the uterine cavity is performed in a timely manner and is effective, total blood loss usually amounts to approximately 600-700 ml (of which 400 ml occurred before surgery).

At the same time, repeated external-internal massage and repeated use of uterotonic drugs may be ineffective, and bleeding may continue against the background of persistent uterine hypotony. Taking into account that previous stages of drug administration, including oxytocin, methylergometrine, prostaglandins, and reflex activation of uterine contractility did not produce an effect, the state of the uterus should be considered 'shock' with loss of sensitivity to neurohumoral stimulation. Therefore, if uterine hypotony persists after manual external-internal massage and blood loss approaches 1000 ml, emergency surgical methods should be started to stop bleeding. The need for surgical intervention is also due to the fact that progressive blood loss, which usually develops against a pathological premorbid background, easily leads to hemorrhagic shock followed by development of multiple organ failure.

It is useless to use excessively large doses of contracting agents or several combinations of them, as well as repeated reflex stimulations and their repetitions. Repeated manual examination of the uterus and application of a suture according to V.A. Lositskaya only delay time, increase blood loss, and aggravate the situation.

If blood loss exceeds 1000 ml, despite the high morbidity, preference should be given to uterine extirpation as the extent of surgical intervention, because during uterine amputation the wound surface of the cervix may become a source of bleeding against the background of the developed acute form of disseminated intravascular coagulation syndrome.

Treatment of DIC syndrome in hypoatonic bleeding is a hemostasiological and resuscitation problem. To provide a reserve of time before the effect of

conservative therapy is achieved, surgical hemostasis must be ensured in the operating room. For this purpose, ligation of the internal iliac arteries is performed. This operation helps reduce pulse pressure in the pelvic vessels by 70%, which leads to a sharp decrease in blood flow, reduces bleeding from damaged vessels, and creates conditions for fixation of vascular thrombi.

To ensure surgical hemostasis during uterine removal, it is recommended to ligate the internal iliac artery in advance, that is, before removal of the uterus. Under such conditions, the operation is performed on a 'dry' background, the total amount of blood loss decreases, and the release of thromboplastin into uterine vessels is reduced. As a result, the trauma of hysterectomy and the intensity of coagulation disorders decrease.

Prevention of postpartum hemorrhage includes the following:

- active management of the third stage of labor - administration of a uterotonic after delivery of the anterior shoulder of the fetus, clamping of the umbilical cord 1-3 minutes after fetal delivery, delivery of the placenta by controlled cord traction, and uterine massage;
- management of labor with established venous access (diameter not less than 18G); administration of tranexamic acid (15 mg/kg) in the second stage of labor in women at high risk of bleeding and during cesarean section.

At present, prevention of bleeding during childbirth is carried out as follows:

- for women at low risk, oxytocin 2 ml (10 IU) is administered intramuscularly into the lateral thigh when the fetal shoulder is delivered;
- or slow intravenous administration of oxytocin (5 units in 50 ml of physiological solution), starting from the end of the second stage of labor by infusion pump at a rate of 1.8 ml/hour, increasing the rate to 16.2 ml/hour (27 mU/min) after delivery of the fetal shoulder; use of a dropper at a rate of 40 drops/min is also possible. Higher doses of the drug provide no benefit.

For cesarean section, prophylaxis consists of administering uterotonics after the surgical intervention:

- **oxytocin 1 ml (5 IU) is administered slowly intravenously (over more than 1-2 minutes) after fetal delivery.**
- or carbetocin 1 ml (100 mcg) intravenously immediately after delivery;
- or an oxytocin solution (1 ml [5 units] in 50 ml of physiological solution) is administered intravenously through an infusion pump at a rate of 16.2 ml/hour (27 mU/min) after fetal delivery. It is also possible to use a dropper at a rate of 80 drops/min (5 units in 500 ml of physiological solution). It should be taken into account that there are three methods of preventing postpartum hemorrhage: a combination of ergometrine and oxytocin; carbetocin; and a combination of

misoprostol and oxytocin, which have shown higher effectiveness than the standard use of oxytocin.

Oxytocin primarily acts in the upper part of the uterus and has a vasodilatory effect on vascular smooth muscles, which may cause hypotension, especially in patients with severe cardiovascular failure.

Ergometril acts on the upper and lower parts of the uterus. It is the drug of choice for hypotony of the lower uterine segment. It has a vasoconstrictor effect on blood vessels, which leads to an increase in blood pressure.

Ergometril is contraindicated for use in patients with hypertension. It may also cause spasm of the coronary arteries, which can lead to myocardial infarction in at-risk patients. Nausea and vomiting are observed 20-25 times. It may also cause retention of placental parts, requiring manual separation.

Misoprostol is inferior in effectiveness to parenteral uterotonics, but it is the only drug for oral, rectal, and vaginal use. Side effects include hyperthermia and chills. The disadvantages of the combination of ergometrine and oxytocin are hypertension and vomiting, and of misoprostol and oxytocin - a high body temperature.

Carbetocin has the lowest frequency of side effects. In the risk group for postpartum hemorrhage, additional prophylactic measures may be used - uterine or combined uterovaginal tamponade.

Each department (maternity ward, emergency room, postpartum unit, etc.) should have a detailed interdisciplinary algorithm for actions in postpartum hemorrhage (PPH) placed in a visible area.

A hemorrhage kit should be available in all departments where there may be a pregnant woman, a woman in labor, or a postpartum woman.

All personnel working in the field of maternal care should be trained in obstetric emergencies, including the management of postpartum hemorrhage.

Training in providing care for postpartum hemorrhage should be multidisciplinary and should include the acquisition of teamwork skills. All cases of postpartum hemorrhage associated with blood loss of more than 1500 ml should undergo a formal clinical review.

Thus, in order to reduce the frequency of obstetric hemorrhage during pregnancy, childbirth, and the postpartum period, and to prevent severe hemorrhagic complications and maternal mortality, the following measures should be taken:

- Identify a risk group for the development of bleeding during pregnancy, childbirth, and the postpartum period on the basis of a complete examination of pregnant women:
- women with infantilism, ovarian hypofunction, disorders of the ovarian-menstrual cycle, a burdened obstetric history (abortions, post-abortion infectious

complications, etc.), multiparous women, polyhydramnios, a prolonged complicated course of previous delivery, the presence of extragenital diseases (pyelonephritis, diabetes, anemia, rheumatism, etc.), and preeclampsia.

- Carry out a complete examination in clinical hospitals and regional centers, improve their health status, and prepare them for pregnancy and childbirth.
- If pregnancy occurs in women from the high-risk group, place them under strict supervision with the involvement of appropriate specialists and strengthen their health.
- Regularly examine the hemostasis system in order to identify and correct disorders predisposing to pathological blood loss. If signs of maladaptation of the hemostatic system are found in pregnant women or signs of its decompensation are observed in women during labor, specific correction of the disorders is indicated (daily fresh frozen plasma 200-300 ml and Contrikal 80-100 thousand units). Depending on the nature of the hemostatic defect in pregnant women, small doses of heparin may be used, 2500-5000 units subcutaneously 3-4 times a day; Dicynone 500 mg every 4-6 hours; prednisolone 90-120 mg. After delivery, treatment with heparin is discontinued; depending on hemostasis parameters, heparin therapy is resumed 8-12 hours after delivery.
- Pregnant women at risk of placenta previa and premature placental abruption should be referred to regional maternity centers for examination and ultrasound. If this pathology is suspected, hospitalization in the pregnancy pathology department is recommended.
- Manage the first and second stages of labor rationally, avoid prolonged use of labor-stimulating drugs, and if unfavorable conditions appear in the physiological course of labor, urgently raise the question of surgical delivery. When prescribing medications during labor, take into account their effect on uterine tone and contractility.
- From the end of the second stage of labor, establish a peripheral intravenous infusion system using venous catheterization; prevent bleeding with methylergometrine when the parietal part of the fetal head emerges; carefully manage the third stage of labor according to the principle of expectant-active tactics (waiting for signs of placental separation and expulsion).

IX. BIRTH TRAUMA

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

Birth trauma is damage to the soft tissues of the birth canal (external genital organs, vagina, perineum), the cervix and the body of the uterus, which occurs during a pathological course of labor, or as a result of untimely or unskilled obstetric care.

Superficial and shallow asymptomatic injuries heal spontaneously and may remain unrecognized.
Sometimes injuries to the soft birth canal are so significant that they cause serious complications, the consequences of which are detected during labor and in the postpartum period. Some injuries of the birth canal are life-threatening for the woman and may lead to death. In some cases, they lead to long-term loss of working capacity and disability.
Ruptures of the soft tissues of the birth canal are most often observed in primiparous women, whereas uterine rupture is mainly observed in multiparous women.
According to various authors, cervical rupture occurs in 6–15% of deliveries.

Causes of birth trauma:

- incorrect protection of the perineum;
- delivery of a large, giant or post-term fetus;
- rapid labor;
- prolonged labor, especially the second stage;
- incorrect position of the fetal head;
- anatomically narrow pelvis;
- breech presentation of the fetus;
- rigidity of tissues, cicatricial or inflammatory changes in tissues;
- operative delivery (obstetric forceps and vacuum extraction of the fetus).

The extensibility of the soft tissues of the birth canal has certain limits. The part of the fetus that advances through the birth canal exerts increasing pressure on the surrounding structures, stretches them and may create a risk of injury and subsequently tissue rupture.

In obstetric practice, the following types of maternal birth trauma are encountered:

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

Vulvar ruptures.

Vulvar ruptures. They usually occur in the area of the labia minora and clitoris and represent superficial or deep fissures or tears. They are mainly observed in women who have previously had colpitis and vulvovaginitis of various origins.
Vulvar ruptures are divided into superficial and deep ruptures.

Clinical presentation and diagnosis. Ruptures of the vulva, especially of the clitoris, are often accompanied by heavy bleeding. The diagnosis is made on the basis of a complete examination of the external genital organs and the presence of bleeding from the rupture sites.

Treatment. Ruptures in the area of the labia minora are sutured with thin catgut using continuous sutures or separate interrupted sutures without involving the underlying tissues, in order to prevent bleeding from the cavernous bodies. When suturing clitoral ruptures, it is recommended first to insert a urinary catheter. Suturing of tears may be performed under local infiltration anesthesia; however, intravenous anesthesia is more appropriate, because after repair of vulvar ruptures the remaining parts of the birth canal must be examined instrumentally.

Vaginal ruptures and hematomas of the soft tissues of the birth canal.

According to etiology, they are divided into:

- spontaneous;
- forced (associated with vaginal operative delivery).

According to localization:

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

According to the depth of injury:

- superficial;
- deep.

Most often, vaginal ruptures, if they are located in the lower third, are combined with perineal rupture. Ruptures in the upper third sometimes extend into the vaginal fornix and are then combined with cervical rupture. The middle third of the vagina, due to its extensibility, is injured less often. Sometimes rupture and crushing of tissues involve only the deep submucosal layers of the vagina, while the elastic mucosa remains intact; a hematoma may develop at the site of the submucosal rupture. Most often, vaginal ruptures are combined with perineal rupture when they are localized in the lower third. Ruptures in the upper third sometimes extend to the vaginal fornix and subsequently combine with cervical rupture. The middle third of the vagina is damaged less often because of its distensibility. Sometimes tissue rupture and crushing involve only the deep submucosal layers of the vagina, while the elastic mucosa remains intact, and a hematoma may develop at the site of the submucosal rupture.

A hematoma of the soft tissues of the birth canal is a limited accumulation of blood that occurs with closed injuries, accompanied by rupture of blood vessels and leakage of blood into the surrounding tissues.

According to localization, hematomas of the birth canal are divided into:

- in the area of the labia majora;
- in the perineal area;
- in the area of the vaginal wall;
- in the paravaginal tissues;
- in the area of the gluteal muscles.

Clinical presentation and diagnosis. Clinically, vaginal tears manifest as bleeding from the injured wall or, in submucosal rupture with damage to venous and sometimes arterial vessels, as formation of a hematoma that protrudes from the lateral wall of the vagina and considerably increases in size; it may also markedly enlarge the labia on one side. This condition causes the postpartum woman to feel a sense of fullness. The size of the hematoma depends on the caliber of the injured vessel.

In our practice, there was a case in which the hematoma was large and extended toward the gluteal muscles. The patient complained of a feeling of tension and severe pain in the area of the left buttock, which was enlarged and had a bluish-purple color. Hemoglobin decreased and blood pressure fell to 80/40 mm Hg. The patient was covered with cold sweat. The picture of hemorrhagic shock developed.

It was necessary to open the hematoma under general anesthesia: the incision was made from both sides, from the vaginal side and from the buttock side. The source of bleeding from the vagina was eliminated, and the hematoma was managed by creating a counteropening between the vagina and the left buttock.

The main clinical signs of a hematoma are:

a bluish-purple tumor-like formation; absence of clear boundaries; doughy or elastic consistency; tissue tension over the area of hemorrhage and pain; deformity of the vulva in the presence of a significant hematoma; signs of anemia in the presence of significant bleeding.

The diagnosis of hematoma is made on the basis of the clinical picture of the disease and examination of the cervix using a vaginal speculum.

Laboratory monitoring is very important: determination of hemoglobin level, the number of red blood cells, color index, hematocrit, and blood coagulation time according to Sukharev.

Treatment. The bleeding vaginal wall is sutured with separate interrupted or continuous catgut sutures; the hematoma is opened and emptied, and the bleeding vessels are sutured together with the underlying tissues. Small hematomas usually resolve without any intervention.

If the rupture is localized in the upper third and extends into the vaginal fornix, manual examination of the uterus is necessary to exclude rupture of the uterus in the area of its lower segment.

If the hematoma rapidly increases in size and anemia worsens, surgical intervention under general anesthesia is indicated: the capsule of the hematoma is incised, the accumulated blood is removed, and the bleeding vessel is ligated. The cavity is tightly sutured and drainage is left in place.

Cervical rupture.

Cervical rupture most often occurs in an upward direction, that is, from the external os toward the internal os.

Classification. There are three degrees of cervical rupture: Grade I – rupture of the cervix on one or both sides not exceeding 2 cm in length; Grade II – ruptures longer than 2 cm but not reaching the vaginal fornix by 1 cm; Grade III – ruptures that reach or extend into the vaginal fornix. A Grade III rupture is a severe type of obstetric injury, in which extension of the cervical rupture into the lower uterine segment is difficult to exclude; therefore, manual examination of the uterine cavity is required.

Clinical features and diagnosis. Shallow tears of about 1 cm usually do not cause symptoms. Deeper cervical tears are accompanied by bleeding of varying intensity. When the descending cervical branch of the uterine artery is damaged, bleeding is profuse and begins immediately after birth of the child. With the placenta delivered and the uterus well contracted, blood flows as a bright red stream.

When cervical tissue is crushed due to prolonged compression of the head against the pelvic bones, bleeding may be absent, although in this case the cervical injury may be very extensive.

In the absence of bleeding, cervical rupture can be detected only by examining all its edges with a vaginal speculum and soft clamps. Examination should be performed in all postpartum women during the first 2 hours after delivery. In the presence of bleeding, the examination must be performed immediately after separation and inspection of the placenta.

At present, according to Order No. 500 of the Ministry of Health of the Republic of Uzbekistan, instrumental examination of the birth canal must be carried out strictly in cases of bleeding.

In our opinion, examination of the birth canal for tears with mirrors should be performed in all primiparous and multiparous women who had colpitis outside pregnancy or during pregnancy, as well as vulvovaginitis of any infectious etiology. Neglecting this rule may have serious consequences.

The following example illustrates this:

EXAMPLE No. 3

A 34-year-old multiparous woman N. M. was admitted to one of the maternity hospitals in Samarkand region for certain reasons, and an experienced physician of the highest category from the department decided to manage the delivery. From the

anamnesis: this was the fourth pregnancy; several years earlier, two pregnancies had ended in urgent deliveries and one in an abortion outside a hospital. This was her second marriage, and pregnancy occurred in the third year of regular sexual activity. Before her second marriage she had suffered from gonorrhea, vulvovaginitis and cervical erosion. She had been treated repeatedly by different physicians, had undergone electrocoagulation of the cervix, and later biopsy of the damaged cervical area was performed. This labor proceeded physiologically, with adequate analgesia using analgesics and antispasmodics. There were no complications during labor. A full-term male fetus weighing 3,800 g and measuring 56 cm was delivered. In the postpartum period, in the delivery room, the physician noted increased bleeding. Under general anesthesia, it was decided to perform manual separation and removal of the placenta, which was done. During this operation, the physician blindly examined the condition of the cervix and other soft tissues of the birth canal with the fingers and stated that everything was intact. Cold was applied to the lower abdomen, and 2 ml of methylergometrine was administered intravenously. However, the bleeding continued. A consultation of physicians was convened and the diagnosis was made: postpartum period, hypoatonic bleeding. The postpartum woman was taken to the operating room and laparotomy was performed. Supravaginal amputation of the uterus without appendages was carried out. However, the bleeding did not stop. It was decided to remove the cervix. But before this operation, the cervix was examined with mirrors, and a second-degree rupture was found, from which a branch of the cervical artery was pulsating. Two catgut sutures were placed, and the bleeding stopped. The laparotomy operation was limited to supravaginal amputation of the uterus.

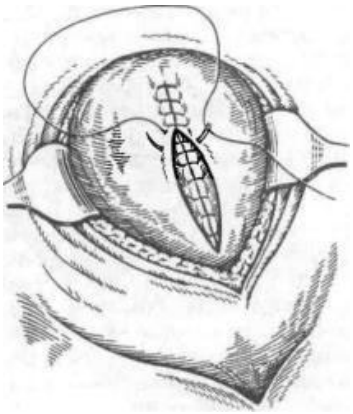


Figure 26. Two-row suture technique

Treatment. Immediately after the cervix is examined and the injury is identified, U-shaped catgut sutures should be placed on the tear in two rows. The first row is applied to the cervical mucosa and the muscular layer of the cervical canal. The second row of sutures is placed on the mucosa of the vaginal portion of the cervix

and on the remaining part of the underlying muscular layer. A single row of catgut sutures may also be applied in order to include 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 severe bleeding, and may lead to maternal death and, often, the birth of a dead fetus. Every case of uterine rupture in a pregnant woman or a woman in labor is analyzed in order to determine the circumstances that led to severe obstetric injury.

According to domestic authors, the frequency of uterine rupture ranges from 0.05 to 0.1%. Currently (2000), cases of uterine rupture in our country amount to hundredths of a percent (0.16 per 1,000 births, according to data from the Ministry of Health of the Republic of Uzbekistan).

According to foreign authors, uterine rupture occurs in 0.03–0.005% of cases.

Classification. In our country, the classification proposed by L. S. Persianinov (1954) has become widely used. In this classification, uterine rupture is divided according to a number of features.

I. According to the time of occurrence:

1. Rupture during pregnancy.
2. Rupture during labor.

II. According to pathogenetic features:

3. Spontaneous uterine rupture:

- a) typical: mechanical (in the presence of a mechanical obstacle to the birth of the fetus);
- b) atypical: histopathic (with pathological changes in the uterine wall);
- mechanohistopathic (a combination of mechanical obstruction and changes in the uterine wall).

4. Violent uterine rupture:

- a) pure (rough intervention during labor in the absence of hyperextension of the lower segment or accidental injury);
- b) mixed (external influences in the presence of hyperextension of the lower segment).

III. According to the clinical condition:

5. Threatening rupture.
6. Beginning rupture.
7. Complete rupture.

IV. According to the nature of the injury:

8. Fissure (tear).

9.Incomplete rupture (not penetrating into the abdominal cavity).
10. Complete rupture (penetrating into the abdominal cavity).

V. According to localization:
11. Rupture of the uterine fundus.
12. Rupture of the uterine body.
13. Rupture of the lower segment.
14. Separation of the uterus.

Uterine rupture

The most common uterine rupture is observed in the lower segment, along its anterior or lateral surface. Rupture in the body and fundus of the uterus, as a rule, occurs in the area of an old scar after a previous surgical intervention.

Etiology and pathogenesis.

It is customary to distinguish two causes of uterine rupture during labor. In the last century, Bandl (1875) proposed the mechanical theory of uterine rupture, which is still supported today. He explained uterine rupture during labor by disproportion between the size of the fetus and the pelvic part of the mother.

Table 7

Classification of causes of uterine rupture

According to Williams Obstetrics (2001)

Uterine injury or developmental abnormalities before the onset of pregnancy	**Uterine injury or complications during pregnancy**
1. Surgical interventions on the uterus: - cesarean section or hysterotomy; - previous suturing of uterine rupture; - history of myomectomy; - resection of the tubal angle of the uterus; - metroplasty. 2. Previous uterine injury: - instrumental abortion; - sharp or blunt trauma; - accident, gunshot or stab wound; - history of asymptomatic uterine rupture. 3. Congenital anomalies:	1. Before delivery: - persistent intense uterine contractions; - stimulation of labor with oxytocin and prostaglandins; - intra-amniotic administration of solutions or prostaglandins; - perforation of the uterus by a catheter used to measure intrauterine pressure; - external trauma (sharp or blunt); - external version of the fetus; - excessive stretching of the uterus (polyhydramnios, multiple pregnancy). 2. During labor: - internal version;

- pregnancy in a rudimentary uterine horn.	- application of forceps in difficult operations; - extraction of the fetus by the pelvic end; - fetal developmental anomaly (hydrocephalus); - strong labor pains during labor; - difficult manual removal of the placenta. 3. Rare causes: - placenta increta or percreta; - trophoblastic disease; - adenomyosis; - incarceration of the uterus in retroversion.

If there is an obstacle to expulsion of the fetus, strong labor develops; the upper uterine segment contracts more and more, and the fetus gradually moves into the thin-walled, stretched lower segment. If the cervix (usually the anterior lip) has not retracted beyond the fetal head and is compressed between it and the pelvic walls, overstretching and rupture of the uterus occur easily.

The ring rises higher and higher, reaching the level of the umbilicus, and the uterus assumes the shape of an "hourglass." As labor continues, overstretching and thinning of the lower uterine segment reach their limit, and rupture occurs.

When the uterus ruptures completely and large vessels are damaged, bleeding into the abdominal cavity begins. When the uterus ruptures in the anterior part of the lower segment or is separated from the vaginal fornix, the urinary bladder may sometimes be involved in the rupture. 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 serous covering of the uterus, or in the prevesical tissue.

At the beginning of the twentieth century, N. Z. Ivanov (1901) and Ya. F. Verbov (1911) proposed another theory of the origin of uterine rupture. According to this theory, rupture occurs as a result of histopathic changes in the myometrium caused by inflammatory and degenerative processes, cicatricial changes after cesarean section, reconstructive operations on the uterus, enucleation of myomatous nodes, abortion, complicated labor, multiple births, and traumatic injury to the uterus. It has now been established that during prolonged labor there are pronounced metabolic

disturbances accompanied by accumulation of toxic compounds that damage tissues — “biochemical injury of the uterus.” The uterine muscle becomes flaccid and ruptures easily. Uterine rupture occurs against the background of weak or discoordinated contractions.

At present, the most common cause of uterine rupture is a scar on the uterus, which is associated with the expansion of indications for abdominal delivery and the more frequent performance of reconstructive plastic operations on the uterus. The adequacy of the scar depends on the location of the uterine incision, the method of cesarean section, the suture material, the postoperative course, and other factors.

In women with a uterine scar, rupture during labor occurs in 0.2–1.5% of cases when the lower segment was incised transversely, in 1–7% of cases when the lower segment was incised longitudinally, and in 4–9% of cases after corporal and T-shaped uterine incisions. Approximately one third of uterine ruptures occurred before labor.

The frequency of uterine rupture is influenced by the number of cesarean sections in the anamnesis. With one previous cesarean section, uterine rupture was observed in 0.6% of cases, and with two previous cesarean sections in 1.8%; according to other studies, the figures were 0.8% and 3.7%, respectively.

It is necessary to identify violent factors that lead to uterine rupture. These include application of the Kristeller maneuver and performance of obstetric operations without the necessary conditions and with violation of the surgical technique, such as destructive fetal operations, obstetric forceps and vacuum extraction. Uterine rupture may occur when attempting to turn a fetus in an advanced transverse lie, when the birth canal is not fully dilated, during extraction of the fetus by the pelvic end, while releasing the arms thrown back, or when extracting the aftercoming unrotated arms.

Uterine rupture may occur in pregnant women exposed to external mechanical forces, such as a blow to the abdomen, a traffic accident or an earthquake.

Prolonged stimulation of the uterus with an excessive dose of oxytocin (10 units), especially in women older than 30 years, in multiparous women, in multiple pregnancy, in the presence of a large fetus and in a narrow pelvis, contributes to rupture.

In our opinion, uterine rupture is related not to the use of contractile agents itself, but to incorrect assessment of the condition of the uterus, especially in the presence of a scar, and to delayed diagnosis of deviation from the normal mechanism of labor, such as a clinically narrow pelvis and other conditions.

At present, histopathic changes in the myometrium are leading in the genesis of uterine rupture. In this situation, the greatest importance is attached to an incompetent scar on the uterus after cesarean section, the diagnosis of which is very

difficult. A morphologically confirmed incompetent uterine scar is found in 48–75% of women.

Clinical picture and diagnosis.

The clinical picture of uterine rupture is variable. Threatening, beginning and completed rupture should be distinguished, because the outcome for the mother and fetus depends on this.

Threatening uterine rupture in the presence of clinical disproportion is associated with excessive stretching of the lower segment. This stage of rupture is characterized by intense and increasing labor pains. Over time, the contractions become convulsive and the pauses between them shorten. The behavior of the woman in labor becomes restless: she cries out, rushes about, holds her abdomen with her hands and asks for help. Her face is frightened, the eyes are wide open, the lips and tongue become dry, the pulse accelerates, and body temperature often rises. Urination becomes difficult due to compression of the bladder and urethra.

On examination and objective assessment, sharp pain is detected on palpation of the lower uterine segment; the Vasten sign is usually positive. A deep groove is identified below the umbilicus or at the level of the umbilicus, running transversely or obliquely across the uterus (the contraction ring). This represents a clearly defined boundary between the contracted uterine body and the excessively stretched lower uterine segment. The uterus assumes the shape of an “hourglass.”

Because of abdominal tension and frequent contractions, it is difficult to hear the fetal heart rate. Acute fetal hypoxia develops and may lead to fetal death, because strong and prolonged uterine contractions cause impairment of placental circulation. During vaginal examination, complete dilatation of the cervix and a caput succedaneum of the fetal head, or an edematous cervix compressed between the fetal head and the pelvic bones, are often found.

With atypical uterine rupture (in the presence of a scar), the clinical picture is less pronounced. Contractions often remain weak; pain is noted when palpating the lower uterine segment in the scar area, thinning is found, uterine tone is usually increased, and signs of fetal hypoxia are often observed. Ultrasound examination may reveal scar insufficiency.

Most often, the threat of uterine rupture is observed at the end of pregnancy in women with a uterine scar. Characteristic signs are pain in the epigastric region, nausea, pain during fetal movements, pain and thinning of the uterine scar, and increased tone. Ultrasound examination detects thinning or deformation of the uterine scar.

The clinical picture of beginning uterine rupture is determined by disruption of the integrity of the tissues of the lower segment, rupture of blood vessels and formation of a hematoma in the uterine wall. The general condition of the woman in labor is

characterized by loud crying and a strong feeling of fear accompanied by dilatation of the pupils.

Contractions become convulsive and the uterus does not relax between them. Painful bearing-down efforts occur despite the lack of fetal descent, high location of the fetal head and complete dilatation of the cervix. Bloody or blood-stained discharge may appear from the genital tract, and blood may be present in the urine. Fetal cardiac activity is disturbed; active fetal movements may occur, followed by sudden death.

If there is a uterine scar after cesarean section or other operations, the signs of uterine rupture are less pronounced clinically than in a clinically narrow pelvis. Pain appears in the epigastric region, nausea and possibly vomiting occur, along with dizziness and weakness. Later, pain is detected in the lower abdomen, in the upper third of the vagina and in the area of the uterine scar; its thinning and bleeding are noted. Bloody discharge from the genital tract may occur, and signs of fetal hypoxia are recorded. Ultrasound examination reveals thinning and irregular contours of the scar.

Characteristic signs of uterine rupture along a scar during the second stage of labor include restlessness of the woman in labor, pain in the lower abdomen and sacrum, painful bearing-down efforts, bloody discharge from the vagina and acute fetal hypoxia.

The clinical picture of uterine rupture is accompanied by a number of characteristic signs: sharp abdominal pain at the height of one of the contractions, sudden cessation of contractions (a sudden “calm after the storm”), signs of the torpid phase of shock and internal bleeding. The woman in labor becomes indifferent, assumes a forced position, and pain increases when the position is changed. The general condition worsens; the skin becomes pale, the pulse accelerates and becomes poor, blood pressure rapidly falls to a critical level, cold sweat, nausea, vomiting and hiccups appear. Palpation and percussion reveal abdominal pain, especially in the lower abdomen, and the Shchetkin-Blumberg sign is positive. After uterine rupture, meteorism rapidly appears and increases as a result of intestinal atony; vaginal bleeding is observed, which increases when the presenting part moves upward. If the fetus was alive up to that moment, it dies. When the fetus enters the abdominal cavity, the abdomen assumes an irregular shape, and small fetal parts are easily palpable through the anterior abdominal wall. The uterus, displaced to the side and well contracted, is palpated.

During vaginal examination, the presenting part, previously pressed tightly against the pelvic inlet, moves upward and becomes mobile. Blood in the urine is often detected due to compression and venous congestion of the bladder mucosa or injury to its wall (tear or rupture). When the uterus ruptures completely, percussion of the abdomen often reveals fluid (blood) in the abdominal cavity.

Incomplete uterine rupture is usually localized in the lower segment along the scar, with formation of a hematoma between the layers of the broad ligament or under the peritoneum. The hematoma may spread upward and reach the region of the liver. Its formation is accompanied by pain in the lower abdomen, sometimes very severe, sometimes radiating to the sacrum and leg.

With incomplete uterine rupture, labor may not stop, and in some cases even spontaneous delivery may occur. Signs of shock and internal bleeding may be mild or sometimes absent. External bleeding may be absent. In the case of major blood loss, the woman's condition worsens, the skin becomes pale, the pulse becomes frequent and weak, and blood pressure decreases.

In all unclear cases in which incomplete uterine rupture is suspected, after birth of the fetus it is necessary to perform manual examination of the uterus and examination of the cervix and vagina with mirrors.

During vaginal-abdominal examination, if there is a hematoma in the parametrium, deviation of the uterus in the direction opposite to the rupture is detected; on its side, a formation without clear contours, which may reach the pelvic walls, is palpated. Ultrasound examination helps to diagnose incomplete uterine rupture.

In some cases, incomplete uterine rupture manifests several days after delivery with the following signs: pallor of the skin, increased heart rate, decreased blood pressure, pain in the lower abdomen with irradiation to the sacrum and leg, meteorism and increased body temperature.

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

The clinical picture of uterine rupture along a scar in the lower segment usually differs from rupture after cesarean section. In the latter case, bleeding is greater than with rupture in the lower segment.

In incomplete rupture of the uterus along a scar (with intact peritoneum), significant bleeding is usually absent. This pathology can be recognized by manual examination of the uterus and by ultrasound examination.

Separation of the uterus from the fornix is a very rare complication that occurs during labor. The main cause is clinical disproportion between the presenting part of the fetus and the size of the pelvic cavity. Clinically, it manifests either by violent labor or, conversely, by prolonged labor. The woman in labor experiences severe pain in the lower abdomen and sacral region. The presenting part of the fetus does not advance and remains fixed at the pelvic inlet. The lower uterine segment is painful on palpation. Urinary retention is present, and rarely there is blood in the urine. Moderate or significant bleeding from the genital organs is observed. During vaginal

examination the cervix is edematous and not completely dilated. A defect of the vaginal fornix extending into the parametrium is detected.

The diagnosis of uterine rupture is made on the basis of the combination of the clinical signs listed above.

Differential diagnosis is performed with clinically narrow pelvis, premature detachment of a normally situated placenta, acute appendicitis and amniotic fluid embolism.

During pregnancy (usually in the third trimester), symptoms of uterine rupture along a scar often resemble the picture of acute appendicitis, with nausea, vomiting and pain in the lower abdomen. In appendicitis the tongue is covered with a white coating, marked leukocytosis is present, and signs of peritoneal irritation in the lower abdomen on the right side are detected. To clarify the diagnosis, it is necessary to determine from the anamnesis the indications for cesarean section, the location of the uterine incision and the postoperative course; ultrasound examination should be performed with complete examination of the abdominal wall, measurement of uterine wall thickness and assessment of the structure of the suspicious scar area.

Differential diagnosis of threatening and beginning uterine rupture is difficult, because many symptoms are the same. It is known that Grade III clinical disproportion, without timely intervention, ends in uterine rupture.

Cephalopelvic disproportion is characterized by:

- the appearance of painful bearing-down efforts when the presenting part remains high;
- insertion of the head that is typical for the existing form of pelvic narrowing or, more often, unusual;
- marked molding of the head, or absence of molding in post-term pregnancy, with pronounced birth swelling;
- absence of descent of the head (or pelvic end) despite complete dilatation of the cervix and strong labor;
- appearance of signs of fetal hypoxia;
- positive Vasten sign;
- severe signs of bladder compression.

The above signs are also observed when there is a risk of uterine rupture.

Detachment of a normally situated placenta, especially in the second stage of labor, is difficult to differentiate from beginning uterine rupture in a morphologically altered uterus, because the following symptoms appear: abdominal pain, increased uterine tone, frequent occurrence of bleeding of varying intensity from the birth canal, and acute fetal hypoxia. In this situation, ultrasound examination is of great help.

Completed uterine rupture must be differentiated from amniotic fluid embolism, which is often observed at the end of the first stage and during the second stage of labor. With embolism, shock develops rapidly, with signs of acute cardiovascular failure, a sharp fall in blood pressure, cyanosis, tachypnea and shortness of breath.

Treatment.

A condition manifested by uterine rupture during pregnancy is an indication for laparotomy and cesarean section. During labor, when there is a threat or onset of uterine rupture, delivery must be completed urgently under deep anesthesia. Cesarean section is then performed. When the abdominal cavity is opened, serous fluid, swelling of the bladder and hemorrhages on the serous membrane of the uterus may be found. The lower segment is usually thinned. The incision in the lower uterine segment must be performed carefully, without damaging the circular fibers, because the incision may extend into the area of the vascular bundle.

When opening the uterus, care must be taken not to injure the child. In addition, the fetus must be removed carefully in order not to injure or extend the incision. After removal of the fetus and placenta, the uterus must be examined carefully.

If the fetus is dead and conditions are available, an operation for extraction of the dead fetus is performed. However, if the physician does not know the technique, it is better to resort to cesarean section because of the risk of uterine rupture. If the fetus is in a transverse lie, cesarean section is advisable regardless of fetal condition (alive or dead). Because of the risk of uterine rupture, such operations are strictly contraindicated: turning the fetus followed by extraction by the foot, application of obstetric forceps and vacuum extractor. Because of excessive stretching of the lower uterine segment, if there is a scar in the lower segment, the incision is made along the scar; before suturing the uterine wound, scar tissue must be excised.

When the diagnosis of uterine rupture is made, emergency surgery is indicated. Transfusion of blood components and blood substitutes must begin before surgery and continue during the operation and in the postoperative period until hemodynamic parameters stabilize, and also depending on the amount of blood loss. In the chain of therapeutic measures, antibiotics, cardiotonic drugs, nutrition and patient care are of great importance.

The choice of surgical method for uterine rupture must be decided individually, taking into account the time of rupture onset, signs of infection, the nature of the rupture and other factors.

After opening the abdominal cavity and uterus, the fetus and placenta are removed; sometimes they are located in the abdominal cavity. If the uterine rupture is small and linear, or if its torn edges can be easily excised, and also if the rupture is recent and the risk of infection is low, the rupture may be sutured. In extensive rupture,

especially with crushed tissues and the presence of infection, extirpation of the uterus and tubes is usually performed.
In the terminal condition of the patient, the operation is performed in two or three stages, with temporary interruption of surgery after hemostasis, during which measures to combat shock are continued.
When the uterus is separated from the vaginal fornix, hysterectomy is indicated. If rupture of the bladder occurs simultaneously with uterine rupture, it is sutured from the abdominal cavity.
In incomplete uterine rupture, the peritoneum over the hematoma is first separated and liquid blood and clots are removed. Bleeding is stopped by ligation of the injured vessels and bleeding tissues. After hemostasis, the rupture is examined and either sutured or managed as a complete uterine rupture.
During surgery, care must be taken not to damage the iliac vessels or injure the ureter.
If it is impossible to stop the bleeding, the internal iliac artery must be ligated.
In uterine rupture, anesthesia is one of the main components of emergency medical care, because it protects the woman's body from pain and from the additional trauma of surgery, and helps prevent severe shock states.
The outcome, even with timely obstetric care, remains unfavorable for the fetus in cases of complete uterine rupture, because this is associated with placental detachment. For the mother, the outcome depends on the amount of blood loss, the severity of shock and the urgency of qualified care.

X. HEMORRHAGIC SHOCK IN OBSTETRIC PRACTICE.

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

The risk of developing hemorrhagic shock occurs when blood loss amounts to 15-20% of the BCC (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 the BCC is considered massive.

In pregnant women, women in labor, and postpartum women, the causes of hemorrhagic shock are 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 concept of "shock" is not a specific diagnosis, but a general name for a number of pathological conditions of various etiologies. Depending on the cause, they are divided into traumatic, hemorrhagic, bacterial-toxic, cardiogenic, hypovolemic, hypo-normo-hypovolemic shock, and others.

Traumatic shock is one of the main forms observed in obstetric practice and is associated with the presence of a pain factor during labor, insufficient anesthesia, as well as various manipulations (obstetric forceps, manual examination of the uterine cavity, suturing of birth canal tears, etc.) and hemorrhagic shock.

Birth trauma and blood loss are the 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 so on. The concept of "shock" informs the physician about the patient's serious condition.

It is a signal for immediate treatment and the need for appropriate medical care.

From a pathophysiological point of view, "shock" is a crisis of microcirculation, an inability to ensure adequate tissue metabolism, to satisfy tissue requirements for oxygen and energy products, and to remove toxic metabolic products.

The pathophysiological changes occurring in the body in response to blood loss are called "hemorrhagic shock."

In obstetric practice, trauma and bleeding often occur together; therefore, they simultaneously form a pathophysiological process. For this reason, they should always be considered as a single whole. It should also be taken into account that many pregnant women have so-called "readiness for shock" because of chronic circulatory, metabolic, and dystrophic changes against the background of somatic diseases, gestosis, and other complications of pregnancy.

Complicated labor accompanied by exhaustion, trauma, and pain reduces the woman's body's resistance to blood loss, which contributes significantly to the development of hemorrhagic shock.

Pathogenesis of hemorrhagic shock

In the pathogenesis of hemorrhagic shock, the main role is played by a mismatch between a decrease in circulating blood volume (BCC deficiency) and vascular capacity. BCC deficiency, in turn, leads to a decrease in venous return to the right heart, a decrease in stroke and minute cardiac output, and a decrease in blood pressure (BP).

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

- Redistribution of blood in the vessels;
- Redistribution of fluid from the interstitial space into the bloodstream;
- Reflex peripheral spasm due to activation of the vasomotor center and the release of vasoactive substances into the bloodstream - catecholamines (adrenaline, norepinephrine, vasopressin, etc.), which leads to spasm of peripheral blood vessels.

In the event of blood loss, the first two mechanisms can compensate for blood loss of up to 20% of the BCC (800-1000 ml). Blood from the vessels of the digestive tract, muscles, and subcutaneous tissue enters the central circulation. Blood dilution also occurs as extracellular fluid enters the bloodstream (autohemodilution), the hemoglobin concentration decreases, and the number of red blood cells decreases. If bleeding continues and the volume of blood lost reaches 25-30% of the BCC (1000-1200 ml), these mechanisms are insufficient for compensation. In this case, the leading protective reaction of the body is the third mechanism - spasm of the peripheral vessels, which maintains central hemodynamics and blood pressure.

Here, constriction of the venous vessels plays an especially important role, since they contain up to 70% of the blood volume.

Prolonged vascular spasm subsequently becomes one of the causes of the development of irreversible shock. It constantly contributes to the following:

- opening of arteriovenous shunts;

expansion of the vascular lumen by opening additional capillaries that did not function during the physiological process of labor (10-15%). This leads to an even greater discrepancy between blood volume and vascular capacity. As a result, progressive slowing of blood flow occurs in the peripheral vascular unit, tissue hypoanoxia develops, and accumulation of incompletely oxidized products (histamine) occurs. At the same time, central hemodynamics deteriorates. Venous return decreases again, cardiac minute volume decreases further, and arterial blood pressure falls;

- development of DIC syndrome and subsequent progression of irreversible shock.

Hypotension is the main symptom indicating decompensation of circulation.

During decompensation of blood circulation in the tissues of vital organs, metabolic acidosis develops due to anaerobic glycolysis (glycogen breakdown), which leads to electrolyte imbalance, intracellular edema, subsequent membrane destruction, and cell death. These events promote the entry into the bloodstream of large amounts of acidic products with toxic properties and high concentrations of aggressive polypeptides. The described disturbances of blood circulation and

metabolism (metabolic processes), if treatment is not carried out in time, may not disappear on their own even after bleeding is stopped. Therefore, timely assistance to stop bleeding and appropriate transfusion therapy are of great importance for normalizing blood circulation in vital organs. Otherwise, as blood loss continues, hemodynamic and metabolic disorders progress.

In hemorrhagic shock, different organs are affected differently. First of all, the blood supply to the lungs is impaired. Breathing becomes frequent and shallow (shock lung). Later, the kidneys, liver, and pituitary gland are affected. Prolonged spasm of the renal vessels creates conditions for the development of hypoxia and subsequent necrosis. This later manifests as the development of acute renal failure. Prolonged spasm of the hepatic microvasculature leads to centrilobular necrosis, followed by the development of liver failure. The pituitary gland is among the first to respond to impaired or decreased circulation in the central nervous system. Prolonged spasm of the pituitary microvessels leads to hypoxia and subsequent development of necrosis (Sheehan syndrome. In hemorrhagic shock and hypoxia, the central nervous system is the first to react. Prolonged spasm of the pituitary microvessels leads to hypoxia and subsequently to its necrosis).

Because microcirculation is impaired as a result of capillary spasm, placental blood flow is disturbed and the microcirculatory vessels of the placenta become obstructed by thrombi and aggregates. There is blockage of the placental perfusion reserve, which leads to a decrease in fetoplacental blood flow and fetal hypoxia. This process subsequently leads to edema of the uterine muscle cells, which then lose their contractile activity. Such a uterus does not respond to the administration of uterotonics, i.e., the syndrome of "uterine shock" develops. The final stage of this syndrome is complete paralysis of the uterus, or the so-called "Couvelaire uterus."

In hemorrhagic shock, the functions of the central nervous system and the cardiovascular system are preserved for a more or less prolonged period. In patients in shock, loss of consciousness occurs when arterial blood pressure remains below 60 mmHg for more than 2 hours. This indicates a terminal condition.

Clinical picture and diagnosis of hemorrhagic shock.

According to the clinical picture and depending on the volume of blood loss, there are four degrees of hemorrhagic shock: I - compensated, II - subcompensated, III - decompensated, IV - irreversible.

To objectively assess the severity of bleeding, the following set of clinical and laboratory data should be taken into account:

- The color of the skin and mucous membranes, respiratory and heart rates, blood pressure and central venous pressure levels, shock index (the ratio of heart rate to blood pressure);

Minute diuresis, relative density of urine;

- Hematocrit value, number of red blood cells, hemoglobin content, indicators of the acid-base state and blood gas composition, and the state of water-electrolyte and protein metabolism;
- Lee-White clotting time, number and aggregation of erythrocytes, fibrinogen, antithrombin III concentration, content of fibrin/fibrinogen degradation products, and paracoagulation tests.

In grade I shock, blood loss is compensated by changes in cardiovascular activity. In obstetric bleeding, this stage does not last long. The patient remains conscious, the skin is pale, tachycardia is up to 100 beats/min, blood pressure is normal or slightly decreased, the shock index is 0.5 or higher, central venous pressure equals 5-15 mm water column, hemoglobin content is 100 g/l, hematocrit is decreased, oliguria is present, and iso- or hypercoagulation is observed.

Grade II shock is characterized by progression of circulatory and metabolic disorders. A decrease in systolic blood pressure to a critical level (80 mmHg) comes to the forefront; pulse rate rises up to 120 beats/min, respiratory rate is 20 or more per minute, shock index is up to 1.5 or higher, and central venous pressure is less than 5 mm water column. Hemoglobin content is 80 g/l, hematocrit is decreased, oliguria is present, and Lee-White clotting time is more than 10 minutes.

Decompensated hemorrhagic shock develops with large blood loss. It is characterized by a severe course: systolic blood pressure falls to a critical level (<80 mmHg), the patient's consciousness is impaired, rapid pallor of the skin and a marbled skin color are noted, pulse rate is 120 beats/min, respiratory rate is more than 30 per minute, shock index is 2 or higher, and central venous pressure is close to 0. Hemoglobin content is 80 g/l or less, hematocrit is reduced, anuria is observed, and Lee-White clotting time is more than 15 minutes.

If the period of decompensation lasts more than 12 hours, despite treatment, the process may become irreversible. Blood loss is very large (more than 50-60% of blood volume).

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

- preagonal state - the pulse is determined only on the carotid and femoral arteries or by the number of heartbeats, blood pressure is not determined, breathing is shallow and frequent, and consciousness is confused;
- agonal state - consciousness is lost, heartbeat and blood pressure are not determined, and severe respiratory distress is present;
- clinical death - cardiac and respiratory arrest for 5-7 minutes.

A specific feature of obstetric bleeding is its suddenness and massive nature. Therefore, the stages of development of hemorrhagic shock cannot always be clearly defined. A pronounced clinical picture of shock can be observed in acute premature placental abruption and uterine rupture, when a severe, even agonal, condition may

develop within a short time (5-10 minutes). At the same time, with prolonged bleeding against the background of uterine hypotonia, especially if it recurs in small portions, it is difficult to determine the body's transition from the stage of compensation to decompensation. Sometimes the apparent, relative well-being of patients misleads the doctor, and he may suddenly face the fact of a critical blood volume deficit.

Diagnosis usually does not cause major difficulties, especially in the presence of external bleeding. However, early diagnosis of compensated shock, which ensures successful treatment, is sometimes missed by physicians because existing symptoms are insufficiently assessed. The severity of shock should not be evaluated only on the basis of blood pressure values or the amount of blood lost during external bleeding.

The adequacy of hemodynamics should be assessed by a number of simple signs and indicators:

- color and temperature of the skin, especially of the extremities;

heart rate and arterial blood pressure;

- the "shock index" according to Algover (the ratio of heart rate per minute to the systolic pressure value; normally 0.5-0.8);
- hourly diuresis (normally 50-70 ml/hour);
- level of central venous pressure (normally 50-120 mm water column);
- hematocrit (normally 40-45%);
- acid-base state of the blood.

The color and temperature of the skin are indicators of peripheral blood flow. Warm and pink skin and pink color of the nail bed, even with decreased blood pressure, indicate good peripheral blood flow. Cold pale skin with normal or even slightly elevated blood pressure indicates centralization of circulation and impaired peripheral blood flow; this reflects spasm of the cutaneous microvessels. Marbling of the skin and acrocyanosis are already consequences of deep impairment of peripheral circulation, vascular paresis, and the approaching irreversibility of the condition.

Pulse rate serves as a simple and important indicator of the patient's condition only when compared with other symptoms. Thus, tachycardia during bleeding may indicate hypovolemia and acute heart failure. These conditions can be differentiated by measuring central venous pressure.

During hypovolemic shock, a simple and highly informative indicator of the degree of hypovolemia is the Algover shock index. When BCC decreases by 20-30%, it increases to 1.0; with a 30-50% loss, the BCC corresponds to 1.5. With a shock index of 1.0, the patient's condition causes serious concern, and if it rises to 1.5, the patient's life is in danger.

Hourly diuresis is an important indicator characterizing organ blood flow. A decrease in diuresis to 30 ml indicates insufficiency of peripheral circulation, and less than 15 ml indicates the approach of irreversible decompensated shock.

Central venous pressure is an important indicator in the comprehensive assessment of the patient's condition. A decrease below 50 mm water column indicates severe hypovolemia requiring urgent replacement. If, against the background of infusion therapy, blood pressure remains low, an increase in central venous pressure to 140 mm water column indicates decompensation of cardiac activity and determines the need for cardiac therapy. In the same situation, low central venous pressure values indicate the need to increase the infusion rate.

The hematocrit value is a decisive test indicating whether the body's circulation is adequate or inadequate. A hematocrit value below 30%, especially when compared with an Algover shock index approaching 1.5, is a dangerous sign; a value below 25% is even more alarming. With further increase of the shock index, it characterizes a severe degree of blood loss. An increase in hematocrit together with a progressive increase in the Algover index indicates the irreversibility of the course of shock.

Determination of the acid-base state (according to Zinggaard-Andersen, by Astrup's micromethod) is a necessary investigation when removing the patient from shock. It is known that hemorrhagic shock is characterized by metabolic acidosis, which may be combined with respiratory acidosis: plasma pH is below 7.38. At the same time, alkalosis may develop in the final stage of metabolic disorders: plasma pH is above 7.45 together with excess bases.

Difficulties in determining the volume of blood loss are related to significant hemodilution of the blood flowing out with amniotic fluid, as well as retention of a large amount of blood in the abdominal cavity or uterine cavity. For approximate determination of the volume of blood loss in pregnant women, the modified Moore formula can be used:

BL = Mx75x0.42 - Ht f/0.42,

where BL is blood loss (ml); M is the pregnant woman's body weight (kg); Ht f is the patient's actual hematocrit.

Treatment of hemorrhagic shock.

Treatment of hemorrhagic shock must be comprehensive, pathophysiologically justified, and timely. The purpose of emergency medical care and intensive therapy is to supply vital organs (brain, myocardium, lung tissue, etc.) with sufficient oxygen necessary to maintain metabolic processes in them.

Prehospital care.

In outpatient settings (medical point, antenatal clinic), pregnant women with signs of hemorrhagic shock should be kept in a horizontal position, with the patient's

legs elevated, warmed, and quickly (mandatorily) transported to the nearest maternity hospital. In the ambulance, a team of medical workers (physician, midwife, paramedic) must ensure the patient's breathing and circulation; it is advisable to warn the on-duty staff of the maternity hospital by radio.

Inpatient care.

In the emergency department of the maternity hospital, if the woman's condition allows, blood group, Rh factor, hemoglobin, and coagulation should be determined immediately. Otherwise, the pregnant woman or woman in labor must be quickly taken to the operating room, where resuscitation and surgical care are performed in parallel.

The main elements of treatment of hemorrhagic shock (according to M.O. Repina) are:

- Urgent arrest of uterine bleeding and elimination of other sources.
- Rapid compensation of blood loss and elimination of central hemodynamic disorders through adequate and timely blood transfusion and infusion therapy.
- Correction of hemostasis in the development of disseminated intravascular coagulation syndrome.
- Elimination of respiratory failure and provision of general protection of the body from pain and additional trauma - artificial ventilation and full anesthesia;
- Prevention and treatment of the consequences of microcirculatory disorders (acute renal and hepatic failure, myocardial and cerebral hypoxia).

Excluding any one of the indicated links in intensive therapy for the treatment of shock worsens the prognosis for recovery.

The success of treatment of hemorrhagic shock depends on the time of treatment. If the optimal time for stopping bleeding and restoring circulation is missed, subsequent, even the most powerful, therapy will be ineffective. Elimination of the source of bleeding must include a complex of effective methods. Repeated instrumental examination of the birth canal, manual examination of the uterine cavity, application of clamps and sutures to the cervix, and similar repeated manipulations should not be resorted to.

Stopping bleeding must include a set of effective methods. In hypotonic bleeding and if the measures taken are ineffective (external uterine massage, administration of uterotonic agents, manual examination of the uterus with gentle external-internal massage), the issue of internal iliac artery ligation or removal of the uterus should be raised in a timely manner. In progressive abruption of a normally located or previa placenta, and in the absence of conditions for vaginal delivery, proceed immediately to cesarean section. If there are signs of disseminated intravascular coagulation in a Couvelaire uterus, hysterectomy is performed. In case of uterine rupture, urgent surgery with removal of the uterus or repair of the rupture

is indicated. All manipulations and operations must be performed under adequate analgesia with mechanical ventilation. After surgery, prolonged mechanical ventilation is required until the function of vital systems and hemostasis is fully restored.

Elimination of the consequences of bleeding and compensation of blood loss are carried out through infusion therapy. When performing this procedure, the rate, volume, and composition of the solutions are of decisive importance.

Taking into account the great importance of the time factor for successful treatment of hemorrhagic shock, in the initial stage of therapy it is necessary to use colloid solutions that are always available and have sufficiently high osmotic and oncotic activity, combining them with crystalloid blood substitutes. These solutions help mobilize the body's compensatory capabilities by attracting fluid into the bloodstream from the interstitial space. Thus, they provide time to prepare for subsequent transfusion of blood components, which should be started as soon as possible but in compliance with all rules and indications.

Thus, it is rational to begin ITT with intravenous administration of colloid plasma-substituting solutions capable of normalizing central and peripheral hemodynamics. Such drugs include hydroxyethyl starch (volekam, stabizol, refortan), dextran, and gelatin derivatives.

A rapid increase in BCC can be achieved using polyglucin and reopolyglucin. These plasma substitutes improve the rheological properties of blood, eliminate stasis and aggregation of formed elements, optimize peripheral blood flow, and lead to redistribution of blood. The average daily dose of dextrans is 500-1000 ml. However, because of the possibility of increased bleeding, the use of dextrans in DIC is undesirable.

Today, effective colloid plasma substitutes include drugs such as refortan, infuzol, and 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 of body weight. Subsequently, if indicated, it should not exceed 50 ml per day.

Because of severe hemodilution, increasing the dose of these drugs leads to a decrease in hematocrit, hemoglobin, and plasma protein. Therefore, when the concentration of total protein decreases during hemorrhagic shock, mandatory transfusion of albumin is indicated, followed by additional administration of washed red blood cells or packed red blood cells.

Thus, blood substitutes should be considered the first-line treatment of hemorrhagic shock in acute blood loss.

To eliminate hypovolemia, infusion therapy for hemorrhagic shock includes gelatin and polyethylene glycol preparations, which provide a hemodilution effect

without affecting vascular-platelet and coagulation hemostasis. The average dose of these drugs is 500-1000 ml.

Blood and its components should be used in the second stage of treatment, when the source of bleeding has been eliminated.

However, the use of any colloid preparation capable of restoring hemodynamic disorders cannot ensure the oxygen-transport function of blood, which is performed by red blood cells. In modern medical practice, this problem is solved with erythrocyte-containing products: red blood cell mass, washed red blood cells, and erythrocyte concentrate. Red blood cell mass must be of the same group; its storage period must not exceed 3 days; before infusion it must be warmed to 37°C. If blood loss exceeds 1500 ml, the infused red blood cell mass should constitute 0.5-0.8% of the volume of blood loss.

To maintain a controlled hemodynamic regimen, transfusion of erythrocyte-containing agents should be combined with administration of colloid and crystalloid solutions (Disol, Acesol, Trisol) in a ratio of 1:1 or 1:2. For this purpose, any solution available to the physician may be used. Blood-substituting solutions improve the rheological properties of blood, reduce aggregation of formed elements, thereby returning deposited blood to active circulation and improving peripheral circulation.

At present, transfusion of whole blood is not recommended because of possible serious complications. Such complications may include:

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

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

If erythrocyte-containing media of the required group and Rh factor are unavailable, a plasma-substituting drug based on perfluoroorganic compounds - perftoran - may be used to increase oxygen transport.

Perftoran has a multifunctional effect:

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

Perftoran is used at a dose of 5-30 ml/kg. It can be combined with solutions of albumin, glucose, and isotonic saline solutions. However, it cannot be used in the same system with polyglucin, reopolyglucin, or hydroxyethyl starch. If the coagulation properties of blood are impaired, perftoran must under no circumstances be used, which significantly limits its practical application.

Adequate treatment of hemorrhagic shock requires not only large volumes of infusions, but also a significant rate of administration, i.e., a volumetric infusion rate. In severe stages of hemorrhagic shock, the volumetric infusion rate should be at least 250-300 ml/min. Stage II shock requires infusion at a rate of 100-150 ml/hour. This rate is possible with central venous catheterization. To avoid losing time, infusion therapy should be started by puncturing the cubital vein, and catheterization of a large vein (usually the subclavian) should be started immediately, which allows infusion therapy to be carried out for a long time. The infusion rate must be monitored according to the patient's general condition: skin color and temperature, pulse, blood pressure, hourly diuresis, central venous pressure, acid-base state, and ECG. The duration of infusion therapy must be strictly individual.

Stabilization of the patient's condition is expressed by the following:

- disappearance of cyanosis, severe pallor and sweating of the skin, restoration of blood pressure (systolic pressure not less than 90 mmHg),
- normalization of pulse filling, disappearance of dyspnea, achievement of hourly diuresis of at least 40-50 ml without forced diuresis, and an increase in hematocrit to 28-30%. After this, one can switch to drip administration of red blood cells and fluid in ratios of 2:1 or 3:1. Drip administration of solutions should continue for one day or longer until all hemodynamic parameters and the woman's general condition are fully stabilized.

To improve oxidation-reduction processes, administration of 200-300 ml of 10-20% glucose solution with an adequate amount of insulin, 100 mg cocarboxylase, and vitamins B and C is indicated to normalize the acid-base balance and gas composition of the blood. Antihypoxants help restore cellular metabolism and glucose utilization.

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

After elimination of hypovolemia against the background of improvement in the rheological properties of blood, an important component of normalization of microcirculation is the use of drugs that relieve peripheral vasoconstriction. A good effect is achieved by administering 100-150 ml of a 0.5% novocaine solution with 20% glucose solution and insulin (at a rate of 1 unit per 5 g of dry substance). Peripheral vascular vasoconstriction can be eliminated with antispasmodics (papaverine hydrochloride 2% - 2-4 ml, No-Spa 2% - 2-4 ml, aminophylline 2.4% -

5-10 ml) or with ganglion blockers such as pentamine (0.5% - 1 ml per 200 ml physiological solution) and benzohexonium (1 ml of 2.5% solution in drip infusion).

To reduce the resistance of renal vessels and increase blood flow in them, dopamine is required (5 ml - 0.5% solution), diluted in 150 ml of 5% glucose solution at a rate of 5-10 drops/min. To improve renal blood flow, administration of 400 ml of 10% mannitol solution is indicated. The use of these drugs must be carried out under monitoring of blood pressure, central venous pressure, and diuresis. If necessary, Lasix is prescribed.

Important components of therapeutic measures include the use of antihistamines (2 ml of 1% diphenhydramine solution, 2 ml of 2% suprastin solution, 2 ml of 2.5% pipolphen solution);

corticosteroid hormones (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 solution).

In hemorrhagic shock, disorders of the coagulation properties of blood, such as DIC syndrome, are almost always present. If in mild cases increased blood coagulation is observed, in moderate and severe cases a decrease in coagulability may develop according to the type of consumption coagulopathy and activation of fibrinolysis, i.e., deficiency coagulopathy.

Taking this into account, blood coagulation capacity must be restored. Many authors have developed principles for including drugs in intensive infusion therapy for hemorrhagic shock aimed at restoring coagulopathic disorders:

- use of protease inhibitors (contrical, gordox) to suppress excessive fibrinolysis;
- refusal to use heparin to prevent intravascular coagulation;
- replacement therapy through administration of large amounts of fresh frozen plasma (at least 1 liter per day);
- stimulation of the vascular-platelet component of hemostasis (dicynone, etamsylate, ATP);
- use of tranexamic acid at a dose of 500-750 mg per 500 ml NaCl solution, which suppresses plasmin activity, stabilizes coagulation factors, and prevents fibrin degradation.

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

Thus, the time factor is of decisive importance in the treatment of hemorrhagic shock. The earlier treatment is started, the less effort is required to remove the patient from shock, and the better the immediate and long-term results will be.

Removing the patient from the severe condition associated with hemorrhagic shock is the first stage of treatment. In the following days, therapy continues, aimed at eliminating the consequences of massive bleeding and preventing new complications. During this period, medical actions are directed toward monitoring kidney, liver, and heart function, normalizing water-salt and protein metabolism, treating anemia, and preventing infectious complications.

XI. MAIN PRINCIPLES OF TREATING OBSTETRIC HEMORRHAGE

Therapeutic measures adopted for obstetric hemorrhage during pregnancy, labor, the placental stage and the postpartum period, that is, regardless of the time and cause of the event, must begin with the main task: stopping the bleeding and eliminating its cause. In cases of progressive acute premature placental abruption and placenta previa, cesarean section must be performed in a timely manner. In hypotonic and atonic hemorrhage, if conservative measures are ineffective, these include external-internal massage with manual examination of the uterine cavity and massage of the uterus on the fist, administration of uterotonic drugs, balloon tamponade, and other measures.

If the patient's blood loss is 800-1000 ml, the question of surgical intervention must be raised: injection of prostenon into the uterine muscle, achieving uterine ischemia by applying clamps and ligatures to the vascular bundles of the uterine and ovarian arteries, application of hemostatic compression sutures according to B-Lynch and Pereira, and ligation of the internal iliac arteries.

If the blood loss exceeds 1500 ml, immediate ligation of the internal iliac arteries, uterine amputation or extirpation is recommended. In cases of birth canal rupture, the ruptures must be sutured urgently; in cases of uterine rupture, the rupture area must be sutured or the uterus must be removed.

Algorithm for providing care in postpartum hemorrhage (Gordeeva G.D., 2008).

Stage 1 - initial assessment

Identification of the cause:

- assessment of uterine tone
- examination of the birth canal
- assessment of the condition of blood clots

Actions of the resuscitation specialist:

- catheterization of one or two veins
- monitoring of blood pressure, pulse, respiratory rate and diuresis
- catheterization of the bladder with an indwelling catheter
- oxygen mask
- assessment of blood oxygen saturation (SpO_2).

Laboratory tests:

- complete blood count with platelet count
- hematocrit
- coagulogram
- blood group and Rh factor

Stage 2 - hypotonic hemorrhage: search for the cause according to the "4T" principle

"Tissue" - retained placental tissue:

- manual examination of the uterine cavity
- curettage

"Tone" - hypotonic state of the uterus:

- external uterine massage
- manual examination of the uterus
- use of uterotonic drugs

"Trauma" - rupture of the soft birth canal or the uterus:

- diagnosis of traumatic injuries and their repair

"Thrombin" - pre-existing or acquired coagulation disorders:

- restoration of circulating blood volume (CBV)
- restoration of coagulation factors

Stage 3 - massive postpartum hemorrhage

Actions of the resuscitation specialist:

- intensive therapy and resuscitation
- anesthetic support
- monitoring of blood pressure, pulse, respiratory rate and diuresis

Preparatory measures:

- manual compression
- preparation for surgery

Stage 4 - surgical treatment

Actions of the resuscitation specialist:

- intensive therapy and resuscitation
- anesthetic support
- monitoring of blood pressure, pulse, respiratory rate and diuresis

Surgical treatment:

- compression sutures on the uterus
- bilateral ligation of the uterine arteries
- bilateral ligation of the internal iliac arteries
- hysterectomy

Stage 5 - bleeding after hysterectomy

Actions of the resuscitation specialist:

- intensive therapy and resuscitation

• anesthetic support
• monitoring of blood pressure, pulse, respiratory rate and diuresis

Surgical treatment:

• ligation of the internal iliac arteries, if it has not been performed
• tight tamponade of the vagina and abdominal cavity in the pelvic region
• the abdominal cavity should not be sutured until bleeding has stopped.

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

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

The success of hemorrhage treatment depends on the level of organization of work in the obstetric hospital and is based on a clearly developed system of staff actions. It is necessary to identify risk groups that allow timely implementation of preventive measures that reduce the frequency of bleeding and the severity of its consequences. Early connection of an intravenous infusion system for women in labor ensures timely administration of oxytocin and methylergometrine when the parietal tuberosities of the fetal head appear, and also makes it possible to begin infusion therapy immediately at the first signs of pathological blood loss.

Among other organizational preventive measures for providing comprehensive care, the following are important: constant readiness of sterile systems for intravenous infusions; availability of sets for catheterization of peripheral and central veins; preparedness of the anesthetic nurse in the operating room; availability of an anesthesiologist, resuscitation specialist and transfusiologist; and adequate provision of infusion and transfusion agents.

The main task of the therapeutic measures being carried out is solved in the process of infusion-transfusion therapy. The main purpose of infusion-transfusion therapy is rapid restoration of capillary perfusion, which prevents disruption of compensatory mechanisms with the development of shock changes. For this purpose, it is necessary to create an effective circulating blood volume and ensure normalization of microcirculation. After solving this problem, normalization of the respiratory function of the blood and prevention and treatment of coagulation, water-electrolyte, metabolic and other disorders are initiated.

Infusion and transfusion therapy must correspond to the rate, volume and quality of blood loss. As mentioned above, hemorrhage in obstetric practice is characterized by sudden onset and massiveness; therefore, the time factor is crucial when providing care. Delay in starting surgery and a delay of the infusion rate behind the rate of blood loss are decisive factors in the development of hemorrhagic shock. The infusion rate depends on the rate and volume of blood loss and on the woman's

condition. In severe cases, when clinical signs of hemorrhagic shock and a fall in blood pressure are present, the infusion rate should reach at least 250-400 ml per minute. To ensure a high infusion rate, infusion therapy should be performed simultaneously through two peripheral veins or a central (subclavian) vein, strictly under the control of central venous pressure, which is normally 80-120 mm H_2O.

An important organizational point in initiating infusion therapy for obstetric hemorrhage is blood testing to determine the blood group and Rh status, compatibility with donor blood, clinical analysis and coagulogram. These data are necessary to determine the nature of the bleeding, including coagulopathy, and to monitor the dynamics of the process and the effectiveness of therapy.

To date, it has been proven that the use of stored blood as the first and leading component of infusion therapy is unjustified. Donor blood is a transplant. Compatibility between the donor's and the patient's blood is determined only by the ABO system and Rh factor. The second aspect of this problem is the high risk of infection during transfusion, including hepatitis, HIV infection, syphilis and others. The functional properties of stored blood are low. During the first two days of storage, leukocytes and platelets die. By days 3-4, the gas-transport function of erythrocytes 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 occlude the pulmonary capillary system, which worsens its function. In addition, approximately one third of the transfused blood accumulates in the microcirculatory system and is excluded from active circulation.

Based on the above, it can be stated that transfusion of stored blood in the initial stages of treating obstetric hemorrhage is not an effective and safe method. Elimination of the consequences of blood loss is achieved through infusion therapy, in which the composition of the drugs used, the rate of their administration and their 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, erythrocyte count, diuresis and blood clotting time.

When performing infusion therapy, systolic blood pressure should not be below 80-90 mmHg; central venous pressure should not be below 30-40 mm H_2O or above 120 mm H_2O; hematocrit should not be below 25%; erythrocytes should not be fewer than 2 million; blood clotting time should not exceed 8-10 minutes; and diuresis should be more than 30 ml per hour. When CBV is restored and oliguria persists, diuresis should be stimulated by administration of Lasix and furosemide. If there is no effect from stimulation of diuresis and anuria persists, ureteral ligation during hysterectomy must be excluded. In doubtful cases, it is better to perform relaparotomy to rule out urinary tract obstruction.

Rapid replenishment and restoration of circulation in parenchymal organs is achieved by administration of plasma-substituting solutions such as Refortan, Stabisol, Infuzol and ethoxylated starch (Volecam). These drugs are similar to human glycogen and are broken down by blood amylase.

High-molecular-weight dextrans can be widely used, including Polyglyukin, Reopoliglyukin, Reogluman, Gelatinol and 5% albumin solution. Polyglyukin rapidly increases blood volume and helps restore hemodynamics. It is advisable to use the low-molecular-weight dextran Reopoliglyukin after the source has been eliminated, because it reduces the aggregating capacity of blood cells, helps restore blood flow in small capillaries and improves microcirculation in organs. All of the listed drugs have high colloid osmotic pressure, attract fluid into the vascular bed and retain interstitial fluid within it for a long time. In this way, by activating the autohemodilution reaction, vascular filling is accelerated, blood viscosity and the disaggregating effect are reduced, which leads to rapid restoration of microcirculation and prevents the development of disseminated intravascular coagulation in the blood.

Table 15

Approximate infusion volume based on blood loss

Blood loss volume relative to body weight	Blood volume (ml)	Total infusion volume relative to blood loss	Of these, 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 is of great importance in the treatment of obstetric hemorrhage. At present, fresh frozen plasma and cryoprecipitate are used at the beginning of treatment to replace blood loss. Fresh frozen plasma is administered at a dose of 15 ml/kg body weight per day. After the plasma is warmed to 37 °C, 60% of the daily dose is administered intravenously, and the remaining part is infused by drip.

To arrest DIC and prevent it, protease inhibitors such as Gordox and Contrical should be used simultaneously with infusion therapy. The daily dose of Contrical should be at least 200,000-300,000 units, and Gordox at least 2,000,000 units. The

initial dose of Contrical should be 10,000 units (10 ampoules), and the initial dose of Gordox should be at least 500,000 units. If coagulation is not restored within 1 hour after the initial dose, the dose may be repeated. After coagulation occurs, the remaining drugs should be administered every 4-6 hours. The duration of use should be at least 2-3 days.

Hemodilution resulting from administration of solutions and stimulation of capillary circulation leads to a decrease in hemoglobin. However, the human body has a safety reserve in hemoglobin that allows it to tolerate the loss of more than half of its volume, whereas a reduction in plasma volume by one third leads to irreversible consequences.

At the same time, the advantages of restoring microcirculation under conditions of hemodilution and the possibility of using the erythrocytes remaining in the circulation make it possible to maintain the required hemoglobin level for a certain period of time.

Therefore, after stabilization of hemodynamic and microcirculatory parameters, including stable blood pressure, adequate diuresis, clear consciousness, absence of respiratory failure and other signs, an important stage of treatment is elimination of a dangerous degree of anemia. For this purpose, when blood loss exceeds 1% of body weight, transfusion of erythrocyte mass or a suspension of washed erythrocytes in an amount of 25-30% of the lost blood volume should be used; the remaining part should consist of plasma-replacing solutions and dextrans.

An important component of therapy for obstetric hemorrhage is the use of glucocorticoid hormones, which are prescribed at the beginning of infusion therapy in cases of major blood loss. For this purpose, hydrocortisone 200-250 mg or prednisolone 120-150 mg is administered intravenously.

Thus, when bleeding is stopped, correct and timely use of infusion therapy makes it possible to prevent severe hemodynamic disorders and restore impaired functions of vital organs.

The basic care protocol includes conservative and surgical stages of stopping bleeding.

Conservative stage:

- Manual examination of the uterine cavity and bimanual compression of the uterus. Manual examination of the walls of the uterine cavity and bimanual compression should be performed as carefully as possible;
- Administration of uterotonics: oxytocin, methylergometrine and prostaglandin F2α;
- Intermediate measures between the conservative and surgical stages: intrauterine hemostatic balloon and compression of the abdominal aorta.

At the conservative stage, the main type of intervention depends on the cause of bleeding. In most cases, manual examination of the uterine cavity walls should be performed. If the integrity of the placenta and uterine walls is confirmed, bimanual compression is recommended. Examination of the birth canal makes it possible to exclude bleeding from damaged tissues. Exclusion of the main causes against the background of ongoing bleeding allows primary or secondary hypocoagulation to be suspected.

Manual examination of the walls of the uterine cavity and bimanual compression have their advantages and disadvantages. The main advantage of manual examination of the uterine cavity is the possibility of checking uterine integrity and excluding retained placental remnants. When bimanual compression is performed, there is no risk of infection and anesthesia is not required. The use of uterotonics must be a mandatory measure in the treatment of obstetric hemorrhage. The first-line drug should be oxytocin, because other groups of uterotonic drugs have side effects. Ergometrine preparations are contraindicated in hypertensive conditions and heart disease. Prostaglandins should not be used in hypertension, glaucoma or bronchial asthma. However, in the absence of contraindications, any uterotonics are indicated for bleeding. Attention should be paid to the tablet preparation containing prostaglandins, misoprostol (Mirolut, Cytotec), which by its properties is the drug of choice for developing countries.

The effectiveness of intermediate measures between the conservative and surgical stages should be taken into account, including insertion of an intrauterine balloon and, if possible, compression of the abdominal aorta according to Biryukova (Figure 29).

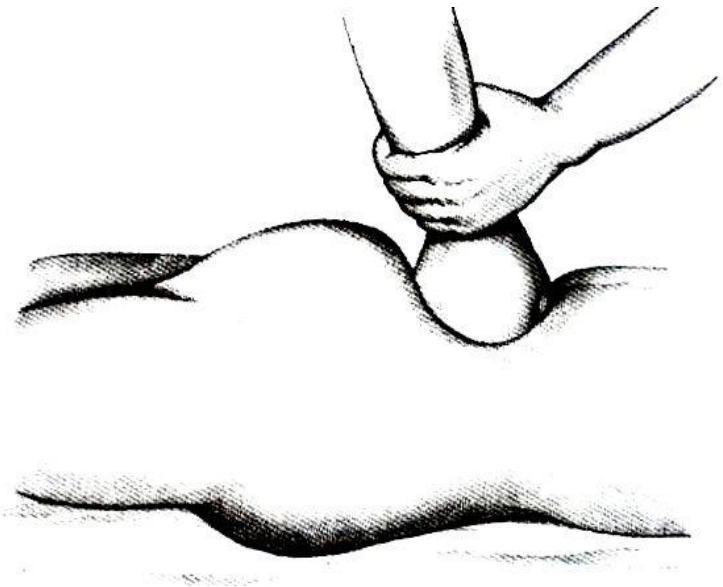


Figure 29. Manual compression of the aorta

An intrauterine balloon is the simplest device, consisting of the balloon itself, a reservoir and a hollow tube connecting them. Balloon tamponade has a number of

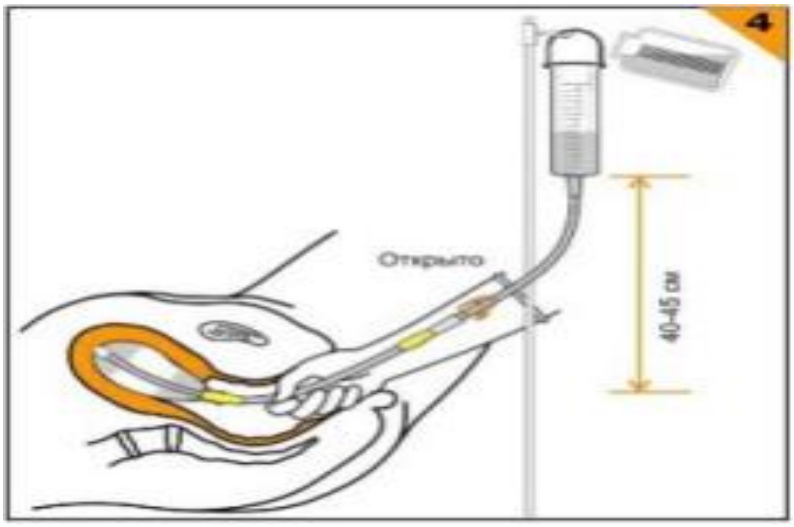
advantages. These include ease of use, rapid application, ease of monitoring the situation, sterility, atraumatic character and no need for anesthesia. In addition, the balloon can be applied by an obstetrician, which saves time between the conservative and surgical stages.

Algorithm of controlled balloon tamponade.

The main indications for balloon tamponade in postpartum hemorrhage are the following: hypotonic bleeding after delivery or during cesarean section; vaginal tamponade for vaginal rupture or hematoma, as a temporary measure. For prophylactic purposes, balloon tamponade can be used during delivery by cesarean section in placenta previa, placenta accreta, multiple pregnancy, polyhydramnios, large fetus and hypotonic bleeding. At present, two types of balloons are available for controlled tamponade for the prevention and treatment of obstetric hemorrhage: a uterine balloon catheter and a vaginal balloon catheter. Each of these catheters may be used as an independent method or in combination.

Stopping hypo-atonic bleeding after childbirth.

Uterine balloon - 10 steps. 1. Place the reservoir 45-50 cm above the level of the uterus. 2. Fill the reservoir with warm sterile 0.9% sodium chloride solution. 3. Close the clamp on the tube. 4. Expose the cervix in mirrors, fix it with clamps and, under aseptic conditions and visual control, insert the balloon catheter into the uterine cavity up to the fundus. 5. Maintaining contact of the distal end of the catheter with the uterine fundus, connect its open end to the reservoir tube. 6. Open the tube clamp. 7. Add solution to the reservoir, maintaining its level in the middle of its volume. 8. Add solution until the level in the reservoir stabilizes, while holding the uterine catheter against the uterine fundus with the hand. In most cases, 350-400 ml of solution is sufficient. It is recommended to use ultrasound to confirm expansion of the balloon in the uterine cavity. 9. If the hemostatic effect is insufficient, raise the reservoir by another 10-15 cm to increase the pressure of the balloon wall at the placental site. 10. The balloon helps restore uterine tone and contractile activity.



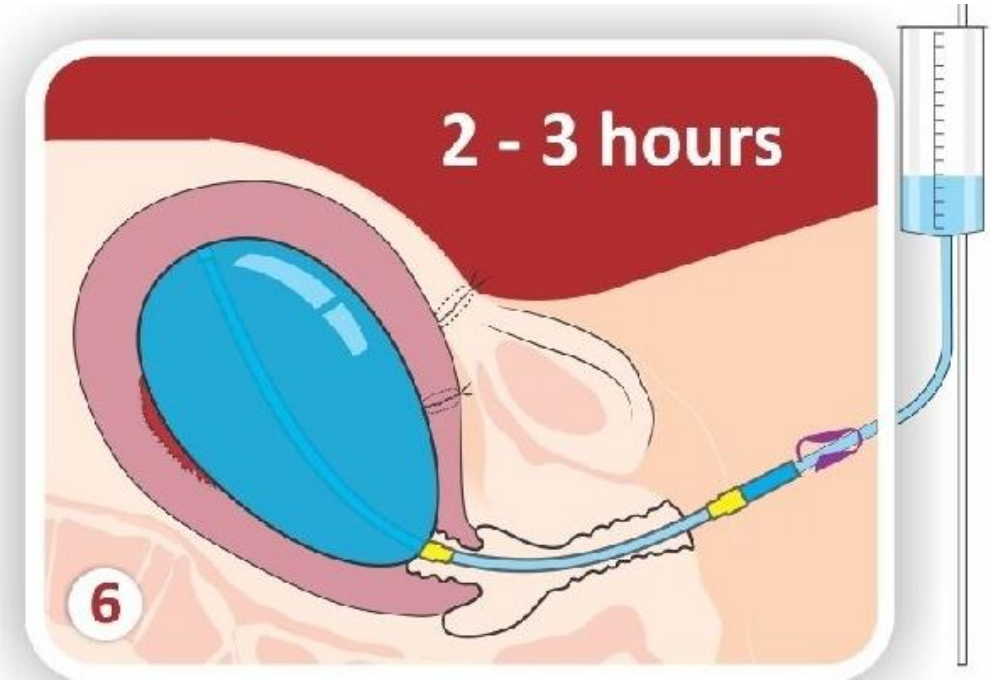


Figure 30. Filling an inserted uterine balloon for postpartum hemorrhage

Therefore, usually within 15-60 minutes, the solution from the balloon begins to be displaced into the cylinder; this can be judged by the rise in the fluid level in the container. After bleeding stops, the technique of controlled balloon tamponade can be considered completed when the balloon is expelled on its own into the vagina or passes onto the catheter within the uterus; after this, the catheter can be removed from the uterine cavity. In the treatment of postpartum hemorrhage with a closed cervix, the effectiveness of balloon tamponade exceeds 95%.

Stopping hypotonic bleeding after childbirth.

Two-balloon technique. This increases the effectiveness of tamponade and prevents premature expulsion of the uterine balloon (Figure 27):

- Insertion of the uterine balloon.
- Placement of the vaginal catheter in the vaginal cavity along the balloon catheter.
- Filling the vaginal catheter with a syringe (150-180 ml).
- Filling the uterine catheter using the method described above.
- Monitoring the amount of genital tract discharge and the fluid level in the reservoir.
- After 4-6 hours, the fluid is removed from the vaginal cuff.
- The reservoir level is lowered, and the uterine balloon is emptied and removed.

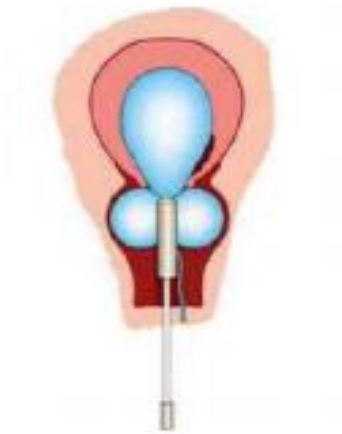


Figure 31. Two-balloon technique of controlled uterine tamponade

- Place the reservoir on the wall 50 cm above the level of the uterus, close the tube and fill it with warm sterile saline solution.
- The surgeon inserts the uterine catheter retrogradely through the hysterotomy incision using a guide. The balloon catheter is advanced in the cranial direction until it touches the uterine fundus.
- Suture the uterine wound without obstructing the balloon.
- While holding the balloon, the assistant removes the guide, connects the balloon to the reservoir tube, opens the clamp on the tube and fills the balloon with 300-400 ml.
- At the end of the operation, transfer the patient to the intensive care unit with the tube clamp closed.
- Open the tube clamp and monitor the fluid level in the cylinder. When the level rises by 2 divisions, lower the balloon to the same height; repeat the maneuver until a height of 10-15 cm is reached.
- Remove the balloon after 2-3 hours or after reaching a height of 10-15 cm above the level of the uterus.

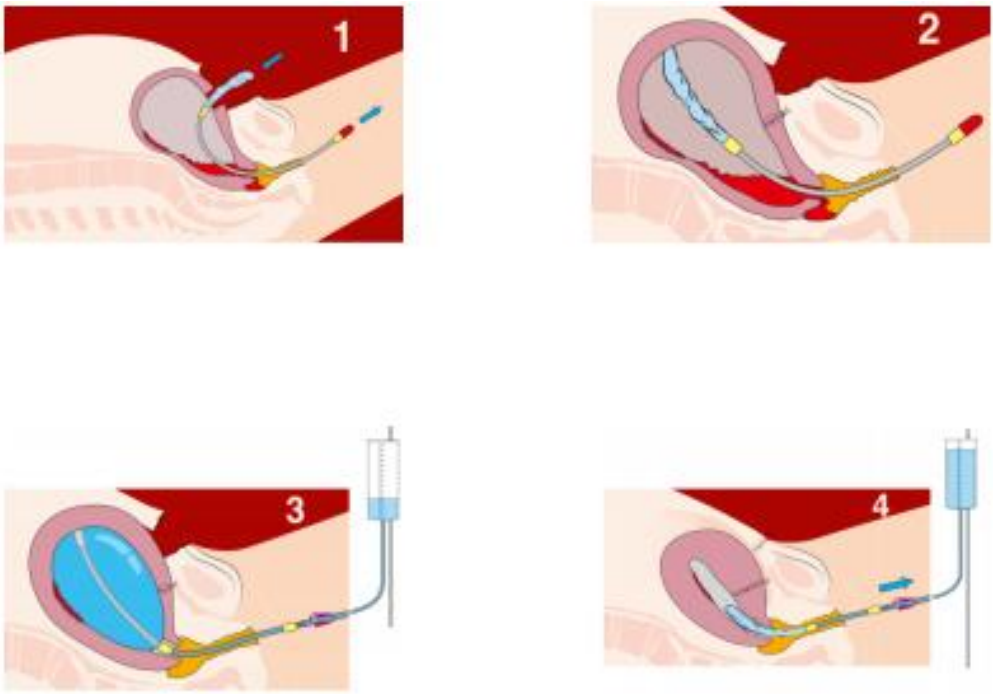


Figure 32. Technique of uterine balloon tamponade during cesarean section

Two-balloon controlled tamponade technique during cesarean section.

Preoperative stage:

- Insertion of the vaginal module;
- Insertion along the vaginal axis up to the cervix, minus 1 cm;
- Connection of the prepared syringe without filling the cuff.

Stage II during cesarean section: immediately after placental separation.

Filling the cuff of the vaginal module with warm physiological solution (150-180 ml).

Stage III during cesarean section: placement of the uterine catheter using the method described above.

Stage IV after cesarean section

- Monitoring 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 confidence in achieved hemostasis, the fluid is removed from the cuff of the vaginal module, the reservoir level is lowered, and the uterine and vaginal balloons are emptied and removed.

Vaginal ruptures and hematomas after childbirth.

When it is not possible to achieve complete arrest of bleeding after surgical measures, such as suturing wounds or suturing the hematoma bed, a vaginal balloon is used as a temporary or additional measure. When the filled balloon is located in the vagina, tamponade is performed for 24-36 hours depending on the severity of postpartum vaginal trauma, with monitoring of genital tract discharge and continuous bladder catheterization.

Modern practitioners perform compression of the lateral uterine walls according to N.S. Baksheev. They state that this method can be used as the definitive method of stopping bleeding in 75% of cases. In our practice, we do not use this method because its application does not exclude injury to the ureters and venous plexuses, especially with prolonged compression for 2 hours.

For rapid assessment of the hemostatic state, a bedside test is recommended. A test tube containing blood taken from the patient's vein is warmed in the hand. If after 7 minutes no clot forms, or a loose, easily disintegrating clot appears, hypocoagulation should be considered.

Surgical stage (laparotomy)

- Injection of prostenon into the uterine muscle;
- Prevention or achievement of uterine ischemia by applying clamps and ligatures to the vascular bundles of the uterine and ovarian arteries;
- Application of hemostatic compression sutures according to B-Lynch, Pereira and others;
- Ligation of the internal iliac arteries. If blood loss exceeds 1500 ml, immediate ligation of the internal iliac arteries is recommended;
- Angiographic embolization may be performed if appropriate equipment and personnel are available;
- Performance of amputation or hysterectomy.

The problem of hypotensive bleeding after cesarean section is somewhat different. The frequency of this complication is 3-5 times higher than after spontaneous delivery.

If conservative measures are ineffective, including uterotonics, external uterine massage, manual examination of the uterine walls and gentle external-internal massage, and application of Baksheev clamps, which is possible only when blood loss does not exceed 1.5% of the woman's body weight, this is a reason to proceed to the surgical stage of stopping bleeding. Previously, doctors believed that continued bleeding against the background of the measures listed above was an indication for laparotomy and hysterectomy.

Our experience allows us to critically evaluate this recommendation. We believe that the above statement can be used as an indication for laparotomy, but not for hysterectomy, especially in primiparous women. Let us emphasize two important aspects. First, it is important to ensure the integrity of the birth canal tissues. Second, the desire to preserve the uterus and reproductive function should not be considered erroneous or even controversial.

Before the surgical stage of stopping bleeding, almost one goal is set: to achieve final hemostasis in the shortest possible time, regardless of the contractile ability of the uterus.

The surgical stage of stopping bleeding has a clear algorithm of actions. If uterine bleeding is detected, prostaglandins, such as dinoprostone 0.25 mg, are administered intramuscularly.

If the volume of blood loss exceeds 1500 ml, ligation of the internal iliac arteries is indicated first. If blood loss is between 1000 and 1500 ml, or if there are no conditions for ligation of the iliac arteries, and also when blood loss exceeds 1500 ml but such ligation cannot be performed, the uterine vessels, including the uterine artery and ovarian artery, should be ligated and hemostatic compression sutures applied according to B-Lynch, Pereira, Hayman or Cho.

The undeniable advantages of uterine ischemia by applying ligatures to the ovarian arteries and the ascending branch of the uterine artery include simplicity and speed of performance. It is very important to bring the uterine body with its ligamentous apparatus into the wound so that, when using diaphanoscopy of the cardinal and round ligaments, the uterine and ovarian arteries can be clearly sutured and ligated.

The next measure is hemostatic compression sutures on the uterus, the effectiveness of which has been confirmed by many researchers. The use of these sutures is widespread abroad, whereas in Uzbekistan this method receives very little attention. The principle of their application is to place absorbable sutures in one plane of subserosal wrapping or transversely, with compression of the uterine body. A thick thread, 1-2 USP, on an atraumatic needle is used. After bleeding stops and the uterus contracts, the sutures are absorbed.

The advantages of the B-Lynch suture method are speed of application and the absence of need for special instruments. Application of the Pereira suture method also does not require special instruments and can be performed quickly. In addition, the sequence of applying the thread, from the lower part toward the lower segment, and the possibility of dosing the load on the suture when tightening each thread create the best conditions for hemostasis.

The application of reinforcing sutures has a number of disadvantages. For example, performing the Pereira ligature in the lower uterine segment is associated with risk of injury to the uterine venous plexus, and the possibility of ureteral injury cannot be excluded. With the B-Lynch method, the difficulty of dosing the load on the threads is determined either by insufficient tension, or by cutting of the uterine muscle in the area of the knot, or by thread rupture. In addition, the use of the B-Lynch suture requires obligatory assistance from an assistant. In cases of recurrent bleeding, relaparotomy with a more radical intervention is required, which increases the frequency of possible adverse consequences.

The tactics for treating obstetric hemorrhage must always be based on the principle of organ preservation. It is abnormal for a woman admitted to a maternity hospital to leave without a reproductive organ. Of course, there are exceptions to any rule,

but today there is no doubt that organ-preserving tactics should be a priority in the treatment of obstetric hemorrhage.

The most effective organ-preserving method is ligation of the internal iliac arteries or, if technically possible, their embolization. It should be noted that these manipulations should be performed only by a highly qualified specialist or even by a vascular surgeon.

Some researchers consider ligation of the internal iliac arteries as an alternative to hysterectomy in massive obstetric hemorrhage. This method currently has the highest effectiveness in stopping bleeding while preserving the organ, exceeding 95%. In addition, if previous methods mainly make it possible to control bleeding of hypotonic and atonic origin, ligation of the internal iliac arteries is also used for coagulopathic blood loss and the development of disseminated intravascular coagulation syndrome, naturally as part of complex treatment. In massive bleeding that occurs immediately after placental separation, this method may be used alone or as the first manipulation.

Prevention of obstetric hemorrhage includes the following:

- Timely treatment of women with inflammatory diseases of the genital organs outside pregnancy and with extragenital diseases, and the fight against abortion.
- Rational management of pregnancy in women at risk of bleeding.
- Prevention of development of hypertensive disorders and other pregnancy complications, timely hospitalization and treatment in a qualified medical institution.
- Rational management of labor, correct assessment of obstetric situations, optimal regulation of labor, pain relief during labor and timely decision-making regarding operative delivery.
- Active management: artificial stimulants that induce uterine contractions, such as ergometrine, oxytocin or equivalents, may be administered intramuscularly to the mother after birth of the anterior shoulder or after birth of the baby. This leads to shortening of the third stage of labor and reduction of bleeding. This process is called active management of the third stage of labor. With active management, the placenta and membranes can be delivered by controlled cord traction, but before traction on the cord, it is necessary to make sure that the placenta has separated. Active management shortens the third stage of labor and reduces the risk of postpartum hemorrhage.
- Rational management of the placental and postpartum periods, beginning from the end of the fetal expulsion period, including the first 2 hours of the early postpartum period, with timely, not premature, use of uterotonic drugs.
- After birth of the child, the bladder must be emptied, cold applied to the lower abdomen, periodic external uterine massage performed, and the placenta

thoroughly examined, including the water-air test. If a defect is present, it must be removed in a timely manner under anesthesia, with accurate accounting of the amount of lost blood and assessment of the general condition of the postpartum woman.

Real methods for the prevention of hemostatic disorders and coagulopathic bleeding in obstetrics include the following:

- Timely prevention and timely treatment of pregnancy complications, including hypertensive disorders, premature detachment of a normally located placenta and anemia;
- Timely prevention of obstetric hemorrhage and, if it occurs, replacement of blood loss using active therapy and autoplasma donation;
- Introduction of mandatory hemostasiological monitoring of the ongoing pregnancy.

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