Ministry of Education and Science of Ukraine
V. N. Karazin Kharkiv National University

INTENSIVE CARE AND RESUSCITATION FOR OBSTETRIC BLEEDING

Guidelines to practical training in obstetrics
for higher education students of the 5th year Medical Faculty

Kharkiv – 2020

УДК 618.2-005.1-036.882-085 (072)
I 73

Reviewers:

I. A. Vasilieva – Candidate of Medical Sciences, Associate Professor of the Department of Obstetrics and Gynecology, Medical Faculty of V. N. Karazin Kharkiv National University;

V. P. Romayeva – Candidate of Medical Sciences, Associate Professor of the Department of Perinatology, Obstetrics and Gynecology, KhMAPO.

Approved for publishing by the decision of Scientific and Methodological Council of V. N. Karazin Kharkiv National University (protocol № 4 from 24.06.2020)

Intensive care and resuscitation for obstetric bleeding : Guidelines to practical
I 73 training in obstetrics for higher education students of the 5th year Medical Faculty / comp. O. V. Grishchenko, T. O. Kozub. – Kharkiv : V. N. Karazin Kharkiv National University, 2020. – 24 p.

The methodical recommendations contain a list of theoretical questions on the given topic for independent preparation of students for practical training, a list of ecommended and additional literature, as well as an example of test tasks.

УДК 618.2-005.1-036.882-085 (072)

Навчальне видання
Грищенко Ольга Валентинівна
Козуб Тетяна Олександрівна

Інтенсивна терапія і реанімація при кровотечі в акушерстві

Методичні рекомендації
до практичних занять з акушерства для здобувачів вищої освіти
V курсу медичного факультету

(Англ. мовою)

Комп'ютерне верстання *В. В. Савінкова*
Макет обкладинки *І. М. Дончик*

Формат 60×84/16. Ум. друк. арк. 1,47. Наклад 50 пр. Зам. № 105/20.

Видавець і виготовлювач
Харківський національний університет імені В. Н. Каразіна,
61002, м. Харків, майдан Свободи, 4.
Свідоцтво суб'єкта видавничої справи ДК № 3367 від 13.01.2009
Видавництво ХНУ імені В. Н. Каразіна
Тел. 705-24-32

CONTENTS

LIST OF CONDITIONAL ABBREVIATIONS

ABS – acid-base state
ADP – adenosine diphosphate
AChT – partially activated thrombin time
AcRT – activated recalcification time
ALV – artificial lung ventilation
ARDS – acute respiratory distress syndrome
BCTA – blood clotting time is activated
BP – blood pressure
CNS – central nervous system
CVP – central venous pressure
DIC – Disseminated intravascular coagulation
EAF – embolic amniotic fluid
FDP – fibrin degradation products
HS – hemorrhagic shock
ITT – infusion-transfusion therapy
PDNLP – premature detachment of a normally located placenta
PDFF – fibrin and fibrinogen degradation products
PPH – Postpartum hemorrhage
PPEE – positive pressure at the end of exhalation
PTT – prothrombin time
TEPA – tissue endothelial plasminogen activator
TEC – thromboembolic complications
TT – thrombin time
VCB – volume of circulating blood

INTRODUCTION

Bleeding during pregnancy and childbirth occupy one of the leading places in obstetric pathology, significantly affect the maternal mortality rate, which is up to 30 %, and contribute to the development of various diseases of the female body. Globally, 14 million women develop postpartum bleeding annually worldwide. 125,000 patients die each year from PPC.

Hemorrhagic shock and DIC, as complications of obstetric bleeding, continue to be the most dangerous manifestation of diseases that lead to fatalities. In obstetric practice, hemorrhagic shock and its complications consistently occupy a leading place among the causes of maternal mortality. Usually, the cause of its development is massive blood loss, the frequency of which is 8–11 % of the total number of births.

Hemorrhagic shock – a condition of severe hemodynamic and metabolic disorders resulting from blood loss and characterized by the inability of the circulatory system to provide adequate perfusion of vital organs due to the mismatch of circulating blood volume of the vascular bed.

New approaches to the treatment of obstetric haemorrhages have emerged, the tactics of providing emergency and planned care are changing, and the approaches to conducting transfusion-infusion and intensive treatment of massive bleeding have changed significantly. As it is difficult to prevent bleeding in most cases, correct and early recognition of the causes of bleeding, timely treatment and constant readiness of the obstetrician to combat them is of particular importance.

SECTION 1

Intensive care and reanimation in obstetrics

1.1. Hemorrhagic shock

Hemorrhagic shock is an acute cardiovascular failure caused by a mismatch of circulating blood volume of the vascular bed that results from blood loss and is characterized by an imbalance between the need for tissues in oxygen and the speed of its actual delivery.

The risk of developing hemorrhagic shock occurs with blood loss of 15 FDP – fibrin degradation products 20 % VCB (0.8 FDP – fibrin degradation products 1.2 % of body weight), or 750 FDP – fibrin degradation products 1000 ml. Blood loss exceeding 1.5 % by weight, or 25 FDP – fibrin degradation products 30 % of VCB (about 1500 ml), is considered massive.

Risk factors for hemorrhagic shock in obstetrics:

1. Pathological premorbid background: hypovolemia of pregnant women; congenital defects in hemostasis; acquired hemostasis disorders.
2. Bleeding in early pregnancy: abortion; ectopic pregnancy; bubble sketch.
3. Bleeding in late pregnancy or childbirth: premature detachment of the placenta; placental presentation; uterine ruptures; embolism by amniotic fluid.
4. Bleeding after delivery: hypo- or atony of the uterus; retention of the placenta or its fragments; rupture of tissues of maternity tracts.

Pathogenesis

Whatever the cause, it did not result in massive blood loss, in the pathogenesis of hemorrhagic shock, a mismatch (disproportion) in the volume of circulating blood and the capacity of the vascular bed is a leading factor. Initially, this is manifested by impaired macrocirculation, i.e. systemic circulation, and then disorders of the microcirculation, and as a consequence, progressive disorganization of metabolism, enzymatic shifts, proteolysis develop. Acute circulating blood volume deficiency resulting from blood loss leads to a decrease in venous return to the heart, thereby reducing volume and filling the right ventricle. As a result, the stroke volume of the heart decreases, blood pressure decreases, and the syndrome of low ejection and hypo perfusion is subsequently formed. Hypovolemia, as a major factor in the pathogenesis of HS, triggers a number of diverse mechanisms of compensation, which is accompanied by activation of the sympathetic nervous system and massive release of endogenous catecholamine. This increases the heart rate, overall peripheral vascular resistance and stroke volume. In this

case, vasoconstriction does not cover all the peripheral bed evenly. It is mainly manifested in the internal organs, the innervation of which is carried out by the abdominal nerves (liver, intestines, pancreas), as well as in the kidneys, skin and muscles. In this case, the volume of blood flowing to the brain and myocardium becomes even greater than in normal conditions (circulation of blood). In this way, despite the VCB deficiency and the restriction of venous return to the heart, blood pressure and cardiac output can be maintained at a stable level for a long time in the GS compensation phase. If there is no rapid normalization of VCB, then the negative properties of vasoconstriction, manifested primarily by the violation, begin to emerge. capillary blood flow. Due to microcirculation disturbance, the supply of oxygen and energy substrates, as well as the elimination of the end products of metabolism, are insufficient. Local metabolic disorders in tissues develop, which is a sign of metabolic acidosis. The progressive accumulation of acidic products further expands the pre-capillaries while the post-capillaries remain constricted. It promotes slowing of blood flow, increase of viscosity of blood, aggregation of uniform elements. Initially, platelet aggregation occurs (white sludge phenomenon), and then erythrocytes (red sludge phenomenon). In such conditions, the capillary blood flow changes so that the formation of micro thrombi begins, or, in other words, the crisis of microcirculation leads to the development of DIC. Coagulopathy and HS are mutually reinforcing. The transition of cell activity to the conditions of anaerobic type of metabolism with the accumulation of a large number of deoxidized metabolism products and the development of lactic acidosis leads to impaired function of many organs. Including the heart, contributing to the occurrence of various arrhythmias, up to the stop of circulation. This is because under conditions of anaerobic metabolism, a pathological triad usually develops: ATP deficiency – impaired protein synthesis – impaired potassium-sodium pump. This determines the irreversibility of the shock.

Classification of hemorrhagic shock

According to LP Chepkiy's classification there are 4 degrees of severity of GS.

Table 1

Classification of hemorrhagic shock by severity
(Chepkiy LP et al., 2003)

The severity of the shock	Stage of shock	The amount of blood loss	
		% VCB	% body weight
1	Compensated	15–20	0,8–1,2
2	Subcompensated	21–30	1,3–1,8
3	Decompensated	31–40	1,9–2,4
4	Irreversible	> 40	> 2,4

The clinical picture

The clinical picture of GS in obstetric practice, in addition to the general patterns inherent in this type of shock, has its own characteristics due to the pathology that caused the bleeding. GS when presenting for the placenta is characterized by a sharp hypovolemia associated with the background on which it develops: arterial hypotension, hypochromic anemia, decreased physiological growth of VCB at the end of pregnancy. In 25 % of women formed DIC syndrome with mild thrombocytopenia, hypofibrinogenemia, increased fibrinolytic activity.

In HS, developed as a result of hypotonic bleeding in the early postpartum period, after a short period of unstable compensation rapidly comes an irreversible condition, characterized by persistent hemodynamic disturbance, respiratory failure, DIC-syndrome with profuse bleeding, and bleeding activity.

Premature detachment of a normally located placenta, as a rule, develops against the background of a long course of preeclampsia, which is characterized by chronic DIC syndrome, hypovolemia, chronic vascular spasm. HS in such a pathology is often accompanied by anuria, brain edema, respiratory distress, runs on the background of reduced fibrinolysis.

At uterine rupture, clinical symptoms of shock are characterized by symptoms of hypovolemia and shortness of breath. DIC syndrome develops infrequently.

Diagnosis

GSH is usually diagnosed without much effort, especially in the presence of significant bleeding. However, early diagnosis of compensated shock, in which the success of treatment is assured, sometimes has difficulty due to underestimation of symptoms.

The adequacy of hemodynamics should be made on the basis of the analysis of a complex of fairly simple symptoms and indicators: characterization of skin color and temperature; heart rate; determination of blood pressure; definition of the «Algover shock index»; hourly diuresis; determination of CVP; hematocrit indicators; characteristic of the ABS.

Difficulties in determining the volume of blood loss in obstetrics are due to the significant dilution of leaking blood, amniotic fluid, and the concealment of large amounts of blood in the vagina or uterine cavity.

To estimate the volume of blood loss in pregnant women, it is possible to use the modified Mooge formula:

BL = M 75 GT (ref) - GT (f) / GT (ref),

where: BL – blood loss (ml); M – body weight of the pregnant woman (kg); GT (f) – the actual hematocrit of the patient (l / l), GT (ref) – the original hematocrit of the patient (l / l).

Table 2

Criteria for the severity of hemorrhagic shock

Indicator		The degree of shock				
		0	1	2	3	4
Volume blood loss	ml	< 750	750–1000	1000–1500	1500–2500	> 2500
	% body weight	< 0,8	0,8–1,2	1,3–1,8	1,9–2,4	> 2,4
	% VCB	< 15	15–20	21–30	31–40	> 40
Pulse, beats / min		< 100	100–110	110–120	120–140	> 140 або < 40*
Sys. BP. mmHg		N	90–100	70–90	50–70	< 50**
Shock index		0,54–0,8	0,8–1	1–1,5	1,5–2	> 2
CVP, mm.w		60–80	40–60	30–40	0–30	< 0
White Spot Test		N (2c)	2–3 c	> 3 c		
Hematocrit, l / l		0,38–0,42	0,3–0,38	0,25–0,3	0,2–0,25	< 0,2
Respiratory rate		14–20	20–25	25–30	30–40	> 40
The rate of dieresis ml / h		50	30–50	25–30	5–15	0–5
Mental status		Calm down	Insigni-ficant is concerned no	Anxiety, noticeable is worrying it	Worried fear or mystery consci-ousness	Tangle consci-ousness or comma

Note * – on the main arteries;

** – The Korotkov method may not be determined.

Intensive care of hemorrhagic shock

General principles of treatment of acute blood loss:

1. Stop bleeding by conservative or surgical methods, depending on the cause of bleeding (see related methodologies).

2. Recovery of VCB.

3. Ensuring adequate gas exchange.

4. Treatment of organ dysfunction and prevention of multiple organ failure.

5. Correction of metabolic disorders.

Priority actions in case of hemorrhagic shock:

1. Assess vital functions (pulse, blood pressure, frequency and nature of breathing, mental status, diuresis).

2. Inform the responsible regular physician of obstetrician-gynecologist or deputy chief physician of medical work on the occurrence of bleeding and the development of hemorrhagic shock, mobilize staff.

3. Raise legs or foot end of bed (Trendelenburg position) to increase venous circulation to heart.

4. Return the pregnant woman to the left side to prevent the development of aorto-cavalry syndrome, reduce the risk of aspiration during vomiting and ensure a free airway.

5. Catheterize one or two peripheral veins with large diameter catheters (№№ 14–16). Provided that access to multiple peripheral veins is possible, catheterization of the central veins should not be hastened, since there is a high likelihood of complications. In case of the development of shock 3–4 degree catheterization of three veins is necessary, with one of them should be central. Preference for vein catheterization should be given to venesection v. Brachialis or puncture and catheterization by Seldinger v. Jugularis interna.

6. Collect 10 ml of blood to determine group and rhesus affiliation, cross-compatibility, hemoglobin and hematocrit content and perform a Li-White test prior to initiation of solution infusion.

7. Inhalation of 100 % oxygen at a rate of 6–8 l / min. through a nasal facial mask or nasal cannula.

Follow-up to eliminate hemorrhagic shock:

1. Intravenous intravenous infusion of crystalloids (0.9 % sodium chloride solution, Ringer's solution and others) and colloids (gelofuzin, reforman, hydroxyethylated starches) are initiated. The rate, volume and components of infusion therapy are determined by the degree of shock and the amount of blood loss (see Table 3). In the event of a 2–3 degree shock, the infusion rate should be 200–300 ml / min. Treatment of hemorrhagic shock is more effective provided that infusion therapy is started as early as possible, not later than 30 minutes. from the development of the first manifestations of shock (A).

If the blood loss is more than 2–2.5% of body weight, it is desirable to connect an artificial oxygen carrier– perfluoroan at a dose of 1.5–5 ml / kg to therapy. *Application in the program of infusion-transfusion therapy of glucose solutions is contraindicated. The use of dextran (reopolyglucine), 5 % albumin solution is not recommended.*

With a blood loss of no more than 20 % BCC, the introduction of some crystalloids (0.9 % sodium chloride solution, Ringer's solution) in a volume of 2 – C times the volume of blood loss is possible.

Indications for hemotransfusion are determined individually on a case-by-case basis, but the values of hemoglobin and hematocrit (Hb <70 g / l; Ht <0.25 l / l) should be guided.

Carriers of oxygen are erythrocytes, so it is better to transfuse the washed erythrocytes or erythrocyte mass.

Indications for transfusion of erythrocyte mass:

• only with clinical signs of hypoxia (decrease in blood's ability to carry oxygen);

• the transfusion threshold should be determined individually for each patient;

• hemoglobin concentration < 70g / l with prolonged blood loss;

• at higher hemoglobin numbers, if there is tachycardia, shortness of breath, decrease in oxygen saturation of the blood;

• if the blood loss is 1000 ml and continues, the erythrocyte mass should be easily accessible;

• Transfusion should only be given when the benefit to the woman is greater than the risk.

In a shocked state, women are not given oral fluid.

2. Stop bleeding by conservative or surgical methods, depending on the cause of the bleeding (see related guidelines).

3. Warm the woman, but do not overheat her, as this improves peripheral microcirculation, which can cause a decrease in blood supply to vital organs. Given the large volume of injected solutions, they are also heated to 36 ° C.

4. Catheterize the bladder.

5. Continue inhalation of 100 % oxygen at a rate of 6–8 l / min, if necessary – ALV.

Indications for ventilation:

• hypoxemia (PaOH < 60 mmHg at FiO_2 > 0.5);

• respiratory rate greater than 40 per minute;

• low inspiratory effort (the patient is unable to produce negative airway pressure greater than 15 cm water at maximal effort);

• blood loss of 3 % by weight or more than 35 ml / kg.

Use endotracheal tubes with high volume and low pressure cuffs. ALV under the condition of decompensated shock is carried out under the control of blood gas composition.

If lung compliance is maintained, positive end-expiratory pressure (PEP) is increased.

Assess the adequacy of cardiac output and hemoglobin level. If necessary, correct alkalosis and hypophosphatemia, which eliminates the shift of the oxyhemoglobin dissociation curve.

Criteria for discontinuation of ALV:

• stabilization of the patient's clinical condition;

• respiratory rate less than 30 per minute;

• inspiratory effort less – 15 cm water;

• RaO_2 / FiO_2 > 80 mmHg / 0.4 at PTK 7 cm water;

• the ability of the patient to double the exhaled air volume in a minute.

6. Laboratory observation: general blood count, platelet count, blood clotting time, coagulogram, electrolyte blood composition. If available – CBS and blood gases.

7. Monitor observation: non-invasive determination of blood pressure (under condition of shock development of 4 tbsp. And in the presence of equipment - invasive determination of blood pressure), heart rate, pulse oximetry, ECG, thermometry, control of hourly diuresis. With the development of shock 3–4 centuries. against infusion transfusion therapy – CVP monitoring every 30–45 minutes.

8. In the absence of signs of reduction of cardiovascular insufficiency (increase in blood pressure, decrease in tachycardia) carry out inotropic support of the myocardium with the help of vasopressors (dopamine 5–20 μg / kg / min, dobutamine 5–20 μg / kg / min).

9. In case of signs of coagulopathy, therapy of DIC syndrome is carried out depending on the stage.

10. Correction of acidosis with sodium bicarbonate provided that the blood pH is < 7.1.

After removal of the patient from the shock state, treatment is continued in the intensive care unit.

1.2. Disseminated intravascular coagulation syndrome

Disseminated intravascular coagulation (DIC) of blood – pathological syndrome, which is based on the activation of vascular – platelet or coagulation hemostasis (external or internal), resulting in the blood is initially coagulated in the microcirculatory bed, blocking the potency of the fibrous and blocking the fibrin. anti-coagulation systems lose the ability to coagulate, which is manifested by profuse bleeding and the development of syndrome of multiple organ failure.

Risk factors for developing DIC in obstetrics: embolism of amniotic fluid; shock (hemorrhagic, anaphylactic, septic); placental detachment; severe preeclampsia; eclampsia; sepsis; septic abortion; massive hemotransfusion syndrome; incompatible blood transfusions; intrauterine death of the fetus; ectopic pregnancy; caesarean section; extragenital diseases of the pregnant woman (heart defects, malignancies, diabetes mellitus, severe kidney and liver diseases).

Classification of DIC syndrome:

I. Clinical course:

- acute;
- subacute;
- chronic.

II. By stages of the course:

• stage 1 – hypercoagulation;

• stage II – hypocoagulation without generalized activation of fibrinolysis (consumption coagulopathy);

• stage III – hypocoagulation with generalized activation of fibrinolysis;

• stage IV – complete blood clotting.

Pathogenesis of DIC

From the pathogenetic point of view, DIC syndrome is a complex nonspecific pathological process caused by the flow of blood clotting activetors and platelet aggregation into the bloodstream, the increase of thrombin, the activation and subsequent depletion of plasma enzymatic systems, the convolutional, and the coronary, coronary, and clotting. the formation of numerous microthrombin in the blood and aggregates of cells that block the microcirculation, lead to hypoxia, acidosis, thrombohemorrhage, dystrophy and deep organ dysfunction, the development of secondary bleeding.

There are 2 options for activating blood clotting properties: external and internal. In obstetrics, DIC syndrome develops mainly by an external mechanism. In the outer variant of the blood coagulation mechanism is stimulated the flow of active thromboplastin into the blood (tissue thromboplates) with obstetric tissues (amniotic fluid, decidual membrane). When a woman enters the bloodstream, active thromboplates interact with prothrombin, turning it into thrombin. Thrombin is a major coagulation factor that promotes the conversion of fibrinogen to fibrin. Fibrin emboli settle on the walls of the vessels of the microcirculatory bed.

In the internal embodiment, the mechanism of activation of blood coagulation is realized without the participation of tissue thromboplastin. The main role in the internal mechanism of activation of blood coagulation belongs to the XII factor (contact factor).

In both variants high thrombinemia is created, blood clotting is activated, products of its activation are accumulated. The main anticoagulant – antithrombin III and protein C is spent to neutralize blood coagulation activation products; they have a major role to play in maintaining the blood's liquid state. With prolonged flow into the bloodstream, thromboplastin depletes the anticoagulation system, develops a deficiency of antithrombin III and there is a massive use of coagulation factors – fibrinogen. Depletion of antithrombin III ends the hypercoagulation phase and the coagulopathy phase of consumption. A significant number of microtubules, cell aggregates are formed, sweet-syndrome develops, transcapillary metabolism is disrupted, hypoxia occurs in vital organs.

As a result of massive consumption of coagulation factors under conditions of antithrombin III deficiency, the fibrinolytic system is included

in the process, and hypocoagulation develops. In the beginning, fibrinolysis is protective, promotes lysis of microtubules of fibrin and restores patency of the vascular bed. However, with prolonged action of hemostasis-activating factors, fibrinolysis becomes generalized. Generalized bleeding develops.

As a result of the above disorders in the final phase of the process can develop severe hemorrhagic syndrome, which often has the character of «uncontrollable» bleeding.

Clinic

Clinical manifestations of acute DVS syndrome are associated with ischemic and hemorrhagic lesions of the organs and tissues that manifest:

1) bleeding into the skin, into the mucous membranes;

2) bleeding from injection sites, operating wounds, uterus, etc .;

3) necrosis of some areas of the skin and mucous membranes;

4) CNS manifestations in the form of euphoria, disorientation and disturbance consciousness;

5) acute renal, hepatic, pulmonary, adrenal insufficiency. DIC syndrome runs in the form of successive stages that change each other.

Stage I is hypercoagulation. Depending on the clinic and the severity of the underlying disease at this stage of DIC, clinical signs of acute respiratory distress syndrome (ARDS) can be observed, ranging from mild stages to the most severe, in which even the use of modern methods of respiratory respiratory support .

The effects of hypercoagulation can be:

• appearance or progression of fetal-placental insufficiency;

• deepening of severity of preeclampsia;

• reduction of uterine-placental blood flow, formation of infarcted zones in the placenta and increasing the likelihood of its delamination;

• increased anemia;

• development of respiratory failure due to the progression of ARDS;

• disturbance of hemodynamics with development of symptoms of centralization of circulation;

• development of encephalopathy.

Stage II – hypocoagulation without generalized activation of fibrinolysis. Depending on the main nosological form of the disease, the clinical picture that is characteristic of this stage may be quite diverse. Characteristic: petechial type of bleeding, delayed bleeding from injection sites, posto-perative wounds and uterus. This is due to initial disorders in the hemocoagulation system.

At this stage the blood coagulates rapidly, but the clot is very fragile due to the large amount of fibrin degradation products (FDP) in it that have anticoagulant properties.

Stage III – hypocoagulation with generalized activation of fibrinolysis. In all patients there is a petechial-spotted type of bleeding: ecchymosis, petechiae on the skin and mucous membranes, bleeding from injection sites and formation in their place of hematoma, prolonged bleeding from the uterus, postoperative wounds, bleeding into the abdominal cavity and retroperitoneal space caused by hemostatic disorders. Gastrointestinal bleeding develops as a result of ischemia and impaired capillary wall penetration of the intestine and stomach. The leaking blood can still clot, but they lick quickly. There are signs of multiple organ failure syndrome. Thrombocytopenia with thrombocytopathy develops. Hypocoagulation results from the blockage of fibrinogen to fibrin transition by many fibrin degradation products. Anemia is associated with intravascular hemolysis.

Stage IV – complete blood clotting. The condition of patients is extremely severe or terminal due to the syndrome of multiple organ failure: hypotension, poorly adjusted, critical disorders of breathing and gas exchange, impaired consciousness to a comatose state, oligo or anuria on the background of massive bleeding.

Mixed-type bleeding: profuse bleeding from tissues, gastrointestinal tract, tracheobronchial tree, macrohematuria.

Table 3

The main clinical and laboratory signs of DIC

Stages DIC	**Clinical manifestations**	**Characteristics of changes coagulation properties of blood**
Stage I – hypercoagulation	Hyperemia of the skin covers from cyanosis, marbling drawing, chills, the patient's anxiety	Activation of calecrine- the kinin system, hypercoagulation, intravascular aggregation of blood cells
Stage II – hypocoagulation	Increased bleeding from genital tract, from the affected surfaces, petechial rash on skin, nasal bleeding. The flowing blood contains loose convolutions, fast licks	Exhaustion hemostatic potential, consumption VIII, V, XIII factors, fibrinogen, platelets, activation local fibrinolysis
Stage III – hypocoagulation with generalized activation of fibrinolysis	solation of liquid blood, that doesn't collapse. Generalized bleeding from places injection, operating fields, hematuria, hemorrhagic exudations in serous cavities	Sudden exhaustion factors minimize the result the formation of the great the amount of thrombin. Receipts in activator blood flow plasminogen
Stage IV – complete blood clotting.	Isolation of liquid blood, that doesn't collapse. Generalized bleeding from places injection, operating fields, hematuria, hemorrhagic exudations in serous cavities	Hypocoagulation extreme degree. High fibrinolytic and anticoagulation activity

Diagnosis of DIC

The results of laboratory tests of the hemostasis system come to the fore in the diagnosis of acute DIC.

Blood clotting time for Lee White. In a conical dry tube collect 1 ml of blood (it is preferable that it flowed from the needle on its own) and determine the clotting time at a temperature of 37 ° C.

Blood clotting time (BCT) is activated. Make 2 ml of blood into a tube of 12–16 mg of koolin. The test indicates hyper- or hypocoagulation shifts (normal – 2–2.5 minutes) and is used to control heparin therapy.

Activated partial thrombin time (APTT) – determines the deficiency of factors of the internal coagulation mechanism, such as XII, XI, IX, VII, and the presence in the blood of their inhibitor (heparin). In these cases, there is an increase in APTch. The shortening of APTT indicates hypercoagulation.

Thrombin time (TT) – characterizes the rate of transition of fibrinogen to fibrin. The increase may be due to hypo- and dysphibrinogenemia, increased FDP blood content, or the presence of direct anticoagulants.

Prothrombin time (PTT) determines the activity or deficiency of the factors of the prothrombin complex (V, VIII, X, II) of the external coagulation mechanism. Prothrombin time elongation at normal TT indicates an inhibition of the external coagulation mechanism, V and II deficiency.

Plasma fibrinogen content decreases with the progression of DIC, treatment with fibrinolytic drugs, or congenital hypo- and dysphibrinogenemia.

Fibrin degradation products (norma – 20 ng / l) increase with the progression of intravascular coagulation and activation of fibrinolysis.

Platelet count decreases with platelet depletion of hemostasis and coagulopathy consumption.

Treatment of DIC

1. Treatment of the underlying disease that caused the development of DIC (surgery, drug and infusion therapy).

2. Intravenous injection of 700–1000 ml of fresh frozen plasma containing antithrombin III heated to 37 ° C. If the bleeding does not stop, an additional injection of 1000 ml of fresh frozen plasma is required. In the next second – third day, fresh frozen plasma is used at a dose of 400–600 ml / day (C). If possible, administration of antithrombin III at a dose of 100 U / kg every 3 hours (B).

3. Given the speed of transition of the stage of hypercoagulation to the stage of hypocoagulation, the inability (in most cases due to the urgent situation) of accurate laboratory diagnosis of stage ICE syndrome from routine use of heparin should be abandoned (C).

4. Starting with stage II shows the introduction of proteolysis inhibitors.

Contrical (or other drugs in equivalent doses) is administered depending on the stage of DIC by drip intravenous infusion for 1–2 hours (C).

5. Restoration of blood coagulation factors by introduction of plasma cryoprecipitate (200 IU – II stage, 400 IU – III stage, 600 IU – IV stage). If possible, intravenous administration of male recombinant factor VII a (novoseven) is recommended – 60–90μg / kg (1–2 doses).

6. Platelet concentrate is used in the event of platelet depletion of less than 50×109 / l. The dose of platelet concentrate is selected depending on the clinical situation.

7. Local stop of bleeding from a wound surface is carried out in all cases. It is achieved by various methods and methods: coagulation, dressing of vessels, tamponade of a wound, application of local hemostatic means.

8. Treatment of multiple organ failure syndrome.

9. In extreme urgent cases (further progression of hypocoagulation, bleeding (Hb < 60 g / l; Ht < 0.25 l / l), only in case of vital indications according to the decision of the consult, the consent of the patient or his relatives (if any) and in the absence of drugs or blood components at the medical facility and at the blood transfusion station (point), it is possible to introduce warm donor blood at half dose from the volume of blood loss (C).

Prevention of hemorrhagic shock and DIC

• Adequate, timely treatment and prevention of conditions that cause the development of hemorrhagic shock and DIC.

• Timely assessment of blood loss, adequate recovery of BCC with crystalloid and colloidal solutions. Of colloidal solutions, gelatin preparations are preferred, in their absence, hydroxyethyl starch derivatives (C). Do not use reopolyglukin and 5 % albumin.

• Drugs that increase blood coagulation potential (etamzilate, epsilon-aminocaproic acid, etc.) are not systematically used (C).

• Drugs that cause thrombocytopenia or impaired platelet function (heparin, reopolyglyukin, lipiridamol, semi-synthetic penicillins) are not used without severe indications (C).

• Surgery is performed on time and in full (extirpation of the uterus) and in the shortest possible time. With continued bleeding – ligation of the internal iliac artery.

1.3. Embolism with amniotic fluid

Amniotic fluid embolism (AFE) is a critical condition associated with the ingress of amniotic fluid and its elements into the mother's bloodstream, with subsequent development of mixed genesis shock syndrome with possible cardiac arrest, acute respiratory failure, and acute syndrome.

Etiology

AFE occurs when amniotic cavity is combined with maternal circulation and the preference for pressure in the amniotic cavity is greater than the pressure in the intercostal space. By their biochemical composition, especially at the end of pregnancy, the amniotic fluid is significantly different from the blood plasma, so in the case of getting into the bloodstream of the mother, the amniotic fluid exerts a toxic-allergic effect on the maternal organism. In addition, water contains fibrinolysin and thromboxinase – like substances that cause changes in the coagulation system, fibrinolysis processes, and blood clots.

Ways of penetration of amniotic fluid to the mother's bloodstream:

• transplacental – because of placental defects in the placental area (exfoliation, presentation and manual separation of the placenta) or the fetal bladder (premature or timely rupture);

• transercervical – through damaged vessels of the cervix;

• through the vessels of any section of the uterus (transmural).

I. Increase in intrauterine pressure:

• prompt delivery;

• excessive and unsystematic stimulation of childbirth, especially against the background of prenatal discharge of amniotic fluid;

• high water;

• large fruit;

• wrong position and insertion of the fetal head;

• gross manipulation during childbirth;

• termination of pregnancy in late terms by the method of infusion of solutions over the shell.

II. Reduction of uterine contractile activity:

• weak or discoordinated delivery;

• premature detachment of the normally located placenta;

• premature discharge of amniotic fluid;

• premature birth;

• postponed pregnancy;

• childbirth in women who gave birth a lot;

• maternity fatigue;

• disharmonic conditions;

• a history of abortion;

• hypertension or hypotension during pregnancy;

• administration of oxytocin at irregular intervals or abrupt withdrawal introduction, which causes relaxation of the uterus muscles and promotes getting amniotic fluid into the mother's bloodstream.

III. Radiation of uterine vessels:

• cervical injuries;

• placental abruption and presentation;
• manual removal of litter from the uterine cavity;
• uterine rupture;
• Cesarean section.

Hypovolemia, which may be due to preeclampsia, diabetes, heart defects, the appointment of diuretics for the treatment of edema, unreasonable prescription of vascular drugs without correction of volemic disorders, should also be attributed to AFE risk factors.

Pathogenesis

The pathogenesis of AFE has not yet been fully studied, but most researchers see it as a pathological process that develops as a result of an allergic reaction of the maternal organism to the antigens of the amniotic fluid. For mechanical obstruction of the pulmonary capillaries by mechanical impurities of water (scales, hair, mucin, etc.), the lungs should be filtered about 7 liters of amniotic fluid, since 1 ml of amniotic fluid contains about 500–600 cells.

Clinic

Symptoms of AFE occur more often in the first or second periods of labor, less frequently in the postpartum and early postpartum periods. As a rule, amid heavy contractions and after spills, maternity wards suddenly complain of chills, sweating and shortness of breath. The patient is excited, she is worried about coughing, vomiting, seizures. Then cyanosis, cervical swelling, and pain behind the sternum appear. Breathing becomes superficial and arrhythmic, tachycardia (120–140 beats / min.), Pulse is weak, sharp drop in blood pressure, manifestations of shortness of breath increase sharply, the patient is sharply excited, covered with cold sweat. An increase in brain hypoxia causes seizures, loss of consciousness, to whom.

If the above symptoms are not severe or correctable, coagulopathic bleeding due to coagulopathy and thrombocytopenia occurs after 30 minutes and sometimes after 8 to 9 hours. At the same time, hypotension and atony of the uterus often occur, worsening coagulopathic bleeding.

Death occurs within minutes or 2–3 hours against the backdrop of irreversible changes caused by a combination of cardiogenic and hemorrhagic shock.

Diagnosis

Diagnosis is possible if clinical, laboratory and additional research methods are considered.

Laboratory examination reveals signs of hypocoagulation and ESR. From additional research methods it is expedient to use electrocardiography (sinus tachycardia, signs of myocardial hypoxia, acute pulmonary heart), radiological examination of the organs of the chest cavity (picture of interstitial drainage pneumonitis – «butterfly» with compaction of the image in the root root zone).

Differential diagnosis

Differential diagnostics need to be performed with many critical conditions that are often encountered by a gynecologist:

• pulmonary embolism – suddenness, general cyanosis of the face, headache, shortness of breath, chest pain, the presence of a history or risk factors for thromboembolism, on the ECG, signs of overload of the right heart;

• myocardial infarction – non-respiratory pain that irradiates the left arm, shoulder, neck, acrocyanosis, rhythm disturbance, shock and decrease in CVP, ECG changes (absent in case of fresh heart attack);

• Mendelssohn Syndrome – acid-aspiration hyperpergic pneumonitis. Occurs more often on an anesthetic anesthesia in the case of a deflated stomach;

• clinical and laboratory delineation of the EAP also requires brain hemorrhage, eclampsia, bronchial asthma, uterine rupture, premature detachment of the placenta, fat embolism, pulmonary edema, more often cardiogenic nature, and severe pneumonosomal pneumonia.

Treatment

Emergency aid

1. Transition to ventilation with positive pressure at the end of exhalation + 5 mm Hg. Indications for intubation are:

• reduction of lung capacity (less than 15 ml / kg);

• reduction in inspiratory power (below 25 mm Hg);

• reducing the partial pressure of oxygen (less than 70 mm Hg) while breathing through the mask with oxygen;

• Increasing the oxygen tension gradient of alveoli-arterioles (greater than 350 mm Hg while breathing with 100 % oxygen);

• increased ventilation of the dead space.

2. Catheterization of 2 – 3 veins.

3. Urinary catheterization.

4. Calling Reserve Donors.

5. Deployment of the operating room.

Monitoring of vital functions: non-invasive blood pressure every 15 minutes; CVP; heart rate; pulse oximetry; shock index; hourly urine output and general urine analysis; body temperature; radiography of the lungs; total blood count, HT, platelets; fibrinogen, coagulogram, blood clotting time, thromboelastogram; circulating blood volume (CBV) and minute blood volume (MBV); general peripheral counteraction; acid-base state of blood; biochemistry, blood electrolytes.

Therapeutic tactics should consist of measures aimed at:

• suppression of reactions that caused cardiopulmonary shock;

• treatment of coagulopathy;

• prevention and treatment of multiple organ failure.

Supporting vital functions of a woman's body, urgent delivery, stopping bleeding and post-syndromic drug therapy consist of many necessary points:

1. ALV at least 3–4 hours.

2. Urgent delivery should be carried out immediately after the emergency measures, depending on the condition of the maternal pathways.

3. In the presence of profuse bleeding – uterine extirpation, ligation of the internal iliac arteries.

4. Infusion transfusion therapy (ITT) (see section «Hemorrhagic shock. DIC»).

5. To support cardiac activity – cardiac glycosides

6. Membranostabilizers (hydrocortisone – up to 800–1000 mg, ascorbic acid – 500 mg, troxevazine – 5 ml, etc.).

7. Narcotic analgesics.

8. Broncholytics (euphilin – 2.4 % – 10–20 ml; alupent, bricanil – 0.5 mg).

9. Vasopressors in the absence of normalization of blood pressure (dopamine 5–10 μg / kg / min microcurrent).

10. Activators of energy processes (actovegin – 40–50 ml intravenously).

11. Prevention and treatment of DIC.

12. Disaggregators (after stopping bleeding).

13. Stimulation of gastrointestinal motility.

14. Correction of anemia.

15. Feeding through a probe.

16. Antibacterial therapy.

17. Thromboxane synthesis inhibitors (aspirin – 200 mg per day, Complin – 900 mg per day).

18. Non-specific prevention of TELA (early therapeutic exercise, elastic compression of the lower extremities).

Positive effects of treatment

The positive effects of treatment should include the following indicators:

• stop bleeding;

• systolic pressure of at least 100 mm Hg ;

• absence of disturbances of heart rhythm, cyanosis;

• stabilization of BCC (erythrocytes not less than 2 x 10 12 / l, hemoglobin not less than 70 g / l, hematocrit not less than 25 %);

• hemostasis indices : platelets not less than 70 x 10 9 / l, fibrinogen not less than 1.5 g / l, blood clotting time not more than 10 minutes, thromboelastogram – normal or hypercoagulation;

• diuresis greater than 30 ml / h.

TEST QUESTIONS

1. In the womb 1 hour after delivery, large fetal blood appeared from the vagina with blood clots. Skin and visible mucous membranes. Pulse 100 beats / min., Rhythmic, blood pressure 90/60 mm Hg. The bottom of the uterus is in the middle of the distance between the navel and the xiphoid process. The uterus is soft. In the external massage, blood from the uterus was released from the uterus. The blood loss was 700 ml. What is the diagnosis?

* A. The early postpartum period. Hypotonic bleeding.
B. Late postpartum period. Hypotonic bleeding.
C. Uterine hypotension. DIC syndrome.
D. Deep vaginal ruptures, bleeding.
E. Incomplete uterine rupture, bleeding

2. Hypotonic bleeding in the early postpartum period began in the mother of O., 16 years old. Blood loss volume is 1.6 % of body weight, heart rate is 115 beats / min, blood pressure is 80/40 mm Hg. Art., CVP – 35 mm Hp.

What is the diagnosis?
A. Hypotonic bleeding in the early postpartum period.
Hemorrhagic shock I degree.
* B. Hypotonic bleeding in the early postpartum period.
Second degree hemorrhagic shock.
C. Hypotonic bleeding in the early postpartum period.
III degree hemorrhagic shock.
D. Hypotonic bleeding in the early postpartum period.
IV grade hemorrhagic shock.

3. After bleeding during childbirth, complains of weakness, dizziness, darkening in the eyes, nausea. Objectively: BP – 80/60 mm Hg, pulse – 110 / min, hemoglobin – 74 g / l. The bleeding stopped. The diagnosis was made – hemorrhagic shock, post–hemorrhagic anemia.

What is the tactics of doing?
* A. Introduction of fresh frozen plasma and erythrocyte mass, crystalloids and colloids.
B. Direct blood transfusion, reopolyglyukin infusion, dry plasma.
C. Anti–anemic therapy using iron preparations, cyanocobalamin, dicinone.
D. Infusion therapy with crystalloid and colloid solutions.

4. Maternity O., 25 years, pregnancy 39 weeks, the main presentation. The contractions are intense, the duration of the first birth period is 2 hours. During one of the maternity contractions, she loses consciousness, heart rate up to 140 beats / min, blood pressure of 70/40 mm Hg. After 3 minutes, consciousness recovered, but there was a marked inhibition. II period – 10 minutes, III period – 5 minutes, placenta without defects. In the early postpartum period, hypotonic bleeding began. Blood released from the vaginal tract does not clot.

What is the most likely cause of bleeding?

* A. Hypotonic bleeding in the early postpartum period.

Embolism with amniotic fluid.

B. Hypotonic bleeding in the early postpartum period.

Trauma of the birth canal.

C. Hypotonic bleeding in the early postpartum period.

Premature detachment of a normally located placenta.

D. Hypotonic bleeding in the early postpartum period.

The uterine rupture.

5. Maternity A., 28, pregnancy 39 weeks, main presentation. The contractions are intense, the duration of the first birth period is 2 hours. During one of the maternity contractions, she loses consciousness, heart rate up to 140 beats / min, blood pressure of 70/40 mm. Hg. After 3 minutes, consciousness recovered, but there was a marked inhibition. II period – 10 minutes, III period – 5 minutes, placenta without defects. In the early postpartum period, hypotonic and coagulopathic bleeding began. Suspected embolism with amniotic fluid.

Which of the first aid measures is wrong?

A. Artificial lung ventilation.

* B. Immediate infusion of canned blood.

C. Deployment of the operating room.

D. Bladder catheterization.

LIST OF RECOMMENDED LITERATURE

Required

1. E. Albert Reece &John Hobbins Clinical Obstetrics of Fetus and Mother. – Third Edition. – Blackwell publishing, 2007. – 328 p.

2. Evans A. T., Niswander K. R. Manual of Obstetrics. Philadelphia, 2000. – 654 p.

3. Jennifer R., Niebyl, Henry L. Galan, Mark B. Landon, Joe Leigh Simpson, Eric R. M. Jauniaux Obstetrics: Normal and Problem Pregnancies. – Elsevier Health Sciences, 2012. – 1292 p.

4. Manual on Obstetrics / Arthur T. Evans. – Seventh Edition. – Lippincott Williams & Wilkins. – 2007. – 425 p.

5. Stuart Campbell, Christoph Lees Obstetrics by Ten Teachers. Malta, 2003. – 516 p.

Additional literature

1. Martin L., Pernoll Benson & Pernoll's handbook of Obstetrics &Gynecology – Tenth Edition, 2010. – 910 p.

2. Obstetrics – edited by Professor I. B. Ventskivska, Kyiv "Medicine", 2008. – 396 p.

3. Obstetrics & Gynecology PreTest Self-Assessment & Review, Twelfth Edition by Karen M. Schneider, Stephen K. Patrick, 2009. – 289 p.

4. Obstetrics & Gynecology PreTest Self-Assessment and Review Eleventh Edition by Karen M. Schneider, Stephen K. Patrick.-McGraw-Hill Medical Publishing Division, 2006. – 347 p.

5. Obstetrics Illustrated by Kevin P. Hanretty – 2009. – 437 p.

6. Obstetrics Illustrated by Kevin P. Hanretty. – Sixth Edition. – Churchill Livingstone, 2003. – 478 p.

7. Williams Obstetrics / M. C. Graw Hill/ 2005

Information resources

1. www.uzhnu.adu.ua - Information Package (ESTC).
2. http://www.moz.gov.ua - Ministry of Health of Ukraine.
3. http://medstandart.net/byspec/9 - Standards of medical care in Ukraine.