**Methods of diagnosis of the state of fetus. Placental insufficiency. Fetal hypoxia (acute, chronic). Retardation, fetal malnutrition.**

**Aim:** to study the main methods of diagnostics, treatment and prevention of fetal hypoxia and asphyxia of the infant.

**Professional motivation:** evidence suggesting nonreassuring fetal status occuring in 5 % to 10 % of pregnancies, when there is a concern that the function of the maternal-fetal physiologic unit is so altered that fetal death or serious injury may occur.

**Basic level:**

1. Methods of fetal surveillance.

2. The main principles of immediate care (resuscitation) of the infant.

3. Emergency ventilation of the newborn infant and external cardiac massage.

**STUDENTS’ INDEPENDENT STUDY PROGRAM**

1. **Objectives for Students**

Independent Studies You should prepare for the practical class using the existing textbooks and lectures. Special attention should be paid to the following:

1. Give the definitions of such term as: "fetal hypoxia", "asphyxia of the infant", "viable infant", "dead infant".

2. Etiology and pathogenesis of fetal hypoxia and asphyxia of the infant.

3. Classification of fetal hypoxia.

4. Methods of fetal heart rate evaluation.

5. Assessment of the fetal hypoxia stages of severity.

6. Principles of treatment of fetal hypoxia.

7. Classification of asphyxia of the infant.

8. Evaluation of asphyxia severity.

9. ABC-steps of infant's resuscitation.

10. Treatment of newborn infant in postresuscitation period.

11. Prevention of fetal hypoxia and asphyxia of the infant.

**Key words and phrases:** fetal hypoxia, asphyxia of the infant, resuscitation of the infant.

**SUMMARY**

**Fetal hypoxia** is a result of dysfunction of maternal-fetal physiologic unit alteration. The term fetal distress or nonreassuring fetal status is also often used for this situation. *Causes of nonreassuring fetal status*: 1) uteroplacental insufficiency − placental edema, maternal diabetes, hydrops fetalis, Rh-isoimmunization, placental "accidents" (abruptio placenta, placenta previa), postdatism, intrauterine growth restriction, uterine hyperstimulation; 2) umbilical cord compression – umbilical cord accidents, umbilical cord knot, umbilical cord prolapse or entanglement; 3) fetal anomalies or conditions – sepsis, fetal congenital anomalies, intrauterine growth restriction, prematurity, postdatism.

**Classification of fetal hypoxia according to etiology factors**:

1) hypoxic decreased amount of oxygen in erythrocytes;

2) hemic as a result of decreased amount of erythrocytes during maternal anemia or during hydrops fetalis;

3) circulatory as a result of placental or umbilical cord disturbances;

4) tissue on the cellular level. According to duration hypoxia may be acute and chronic.

**Methods of fetal heart rate evaluation include:**

1) Assessment of fetal well-being includes maternal perception of fetal activity − the fetus is considered to be healthy when mother detects more than five fetal movements while lying comfortably and focusing on fetal activity for 30 minutes;

2) Intermittent auscultation of the fetal heart rate (FHR) after contractions;

3) Electronic fetal monitoring (EFM). Fetal heart rates EFM is described by pattern of variability.

The baseline FHR ("normal FHR") at term is defined as 120 to 160 beats per minute (bpm). Baseline fetal tachycardia is defined as > 160 bpm for 10 or more minutes, being classified mild if the baseline is between 161 and 180 bpm, and severe if more than 182 bpm. Baseline fetal bradycardia is defined as less than 120 bpm for 10 or more minutes, and is classified as moderate between 80 and 100 bpm and severe at less than 80 bpm. A sinusoidal heart rate pattern is when the rate is 120 to 160 bpm, but there is a smooth, undulating pattern of 5 to 10 bpm in amplitude and shortened short-term variability.

Fetal heart rate variability is the most reliable single EFM indicator of fetal status. The presence of good variability is highly suggestive of adequate fetal central nervous system oxygenation. Two types of variability are described: short-term and long-term variability.

Acceleration of the FHR is defined as the increase in the FHR above the baseline of at least 15 bpm, usually of 15 to 20 second duration, and is associated with an intact fetal mechanism unstressed by hypoxia and acidemia. It is reassurance and usually indication of fetal well-being.

Early deceleration is slowing of the FHR that starts as uterine contraction begins, reaches their nadir at the peak of the uterine contraction, and returns to the baseline FHR with the end of the uterine contraction. Early FHR deceleration is considered to be physiologic and is not the cause of concern. Variable FHR deceleration is slowings of the FHR that may start before, during, or after when uterine contraction begins. It is also reflexed as mediated, usually associated with umbilical cord compression. Late FHR deceleration is slowings of the FHR that begins after the uterine contraction, reaches its nadir after the peak of uterine contraction, and resolves to baseline after the uterine contraction is over. It is sometimes associated with uteroplacental insufficiency and progressive fetal hypoxia and acidemia. The most common tests that can be used are the nonstress test, the contraction stress test (called the oxytocin challenge test if oxytocin is used), and the biophysical profile;

4) Ultrasonography.

The diagnosis of neonatal asphyxia applying is characterized by significant newborn depression associated with severe hypoxia and mixed respiratory and metabolic acidosis. The American College of Obstetricians and Gynecologists has suggested that the diagnosis of neonatal asphyxia is extremely unlikely unless four criteria are met: 1) Apgar scores are less than 4 at 5 minutes of life; 2) umbilical artery pH is less than 7.00; 3) neuromuscular signs and symptoms soon after birth (including seizures, coma, hypotonia); 4) multiorgan system failure. Neonatal asphyxia is classified as moderate (Apgar score at 1'− 4−6, 5'−8−10) and severe (Apgar score at the 1'−0−3, 5'−7).

**Placental Insufficiency (PI). Fetal Development Delay (FDD)**

Placental insufficiency is a symptom complex conditioned by violations of transport, trophic, metabolic, and endocrine functions of the placenta due to structural changes in it.

Reasons: gestoses, miscarriage threat, immunoincompatible pregnancy, intrauterine infection, mother’s diseases (pyelonephritis, essential hypertension, diabetes mellitus, anemia), etc.

Classification

**I. By the term of onset:**

1) primary – develops in the terms of placenta formation (till the 16th week);

2) secondary – usually develops after the processes of placenta formation have finished.

**II. By the course: acute and chronic. Acute PI appears at cute** violation of decidual perfusion, for instance, at abruption of placenta – sharp violation of blood supply leads to fetal hypoxia or death. Chronic PI is characterised by gradual worsening of decidual perfusion as a result of the reduction of compensatory-adaptive reactions of placenta to the action of pathological conditions of the maternal organism, has a long-term course, is accompanied by disorders, chronic oxygen starvation of the fetus.

**Chronic PI** (depending on the condition of compensatory-adaptive reactions) includes:

1. Relative – compensatory-adaptive reactions are preserved in the placenta:

- compensated (the phase of persistent hyperfunctioning) develops at a threat of miscarriage and not severe forms of gestoses in cases, when these complications are successfully medically corrected;

- subcompensated (the phase of exhaustion of compensatory mechanisms, which have begun) – is more often observed in the women, in whom a complicated course of pregnancy is developing against the background of extragenital pathology.

2. Absolute (decompensated) – the severest form of PI characterised by derangement of compensatory-adaptive reactions and develops against the background of chorion ripening disorders at placenta damages of involutivedystrophic, circulatory and inflammatory character.

Diagnostics:

1. Regular clinical observation.

2. Dynamic ultrasonography in the 1st, 2nd, and 3rd trimesters.

3. Dopplerometry.

4. Investigation of the hemostasis system.

5. Detecting the content of estradiol, progesterone, chorionic gonadotropin, and α-fetoprotein in the blood serum.

6. Investigation of estradiol secretion with urine.

7. Detecting the content of oxytocinase, general and placental basic phosphatase in the blood serum.

8. Colpocytologic investigation.

Detection of the height of uterine fundus standing (HUFS) is very important in PI diagnostics, the diagnostic value of this method at the term of 32 weeks makes 76 %.

The main method of PI detection is ultrasonographic placentometry, which enables assessing placenta thickness, area, and structure. Placenta thickness from the 20th till the 36th week of pregnancy approximately equals the term of pregnancy in weeks: at 20 weeks – 20 mm, at 28 weeks – 28 mm, at 36 weeks – 36 mm, after this term the placenta does not thicken further. Placenta thinning (less than 20 mm) or

thickening (more than 50 mm) testifies to PI, which appeared as a result of intrauterine infection, immunization, etc.

At placentography there is carried out the assessment of placenta maturity by structure density singling out 4 maturity degrees (0 – 3). The 1st degree is characteristic of the 28th – 32nd week of pregnancy, the 2nd – 32nd – 37th week, the 3rd degree of placenta maturity is characteristic of the term of pregnancy of 38 – 39 weeks, if it is detected earlier, it testifies to premature placenta aging and fetoplacental insufficiency.

Ultrasonography also detects the biophysical fetal profile on the basis of its functional condition, qualitative and quantitative (in points) assessment of the indices of non-stress test, respiratory movements, motion activity, tone, amniotic fluid volume, placenta maturity degree. Normal indices of biophysical profile make 9 – 12 points.

Modern examination methods also include dopplerometric assessment of the blood flow. The essence of Doppler method consists in the fact that depending on the speed of object moving relative to the source of wave radiation the length of the wave of reflected radiation changes. Such devices are used for the qualitative assessment of blood flow in different vessels of the pelvic cavity of the pregnant woman: the uterine artery, carotid artery, umbilical artery, the descending part of the fetal aorta, medial cerebral artery. In case of necessity there are investigated the curves of speed performance of blood flow in the vessel under consideration.

Most often investigation is conducted in the umbilical artery and medial cerebral artery. Blood flow in the umbilical artery is detected by the contractile function of the fetal heart and resistance of the vessels of the fetal part of placenta, whose vascular resistance plays the main role in fetoplacental hemodynamics. The condition of blood flow in this vessel is the most informative index of the vascular resistance of the placental bloodstream.

Diagnostic criteria: Normal blood flow – a high diastolic component in the dopplerogram relative to the isoline, the ratio of systole amplitude to diastole is not more than 3.

**Pathological blood flow:**

1) decelerated blood flow – diastolic component reduction, the ratio of systole amplitude to diastole is more than 3;

2) terminal blood flow testifies to a strong probability of antenatal fetal death;

3) zero blood flow stops in the diastole phase (there is no diastolic component in the dopplerogram);

4) negative blood flow acquires reverse direction in the diastole phase.

At PI blood supply to the medial cerebral artery increases. This brain-sparing phenomenon reflects the compensatory centralization of blood supply to the essential fetal organs.

Investigation of the content of placental hormones and fetoplacental complex (estriol, placental lactogen, choriomammotropin, etc.) in biological fluids may diagnose violations of fetal condition at the presence of different pregnancy complications or extragenital pathology. The severity of fetal condition correlates with the amount of secreted hormones.

**Fetal development delay (FDD) or fetal hypotrophy is a pathological** condition, at which the newborn’s weight or biometric parameters of the fetus are not up to gestational age.

Classification:

1) symmetric – the weight and length of the fetus are proportionally reduced, all the organs are evenly reduced in size;

2) asymmetric – fetal weight reduction at normal indices of its length, unproportional dimensions of different fetal organs.

Table 1 Differential FDD Diagnostics

|  |  |  |
| --- | --- | --- |
|  | Symmetric | Asymmetric |
| Beginning | 2nd trimester | 3rd trimester |
| Fetometry | Delay of all dimensions | Delay of abdomen dimensions |
| Placental blood flow disorders | From the 24th – 25th  week | After 32 weeks |
| Amniotic fluid | Oligohydramnios | Norm |
| Malformations | Frequent | Rare |

At symmetric hypotrophy newborns have small body weight at birth, such a child can not be differentiated from a premature newborn. The symmetric form is observed at severe disorders of intrauterine development beginning from the 2nd pregnancy trimester. At asymmetric FDD newborns have a considerable weight deficit at normal body length. This from is characteristic of the fetuses, in which unfavourable development conditions began in the 3rd pregnancy trimester.

There are differentiated 3 degrees of FDD severity:

- the 1st degree – delay by 2 weks;

- the 2nd degree – from 2 to 4 weeks;

- the 3rd degree – more than 4 weeks.

FDD takes place due to the following reasons: chromosome anomalies and hereditary metabolic disorders, congenital defects caused by other factors, prenatal viral infections, action of ionizing radiation and medicinal preparations, placenta pathologies, mother’s diseases, intoxication, malnutrition. If FDD is suspected, complex examination of the pregnant woman is conducted including:

1. Detection of the height of the uterine fundus standing (HUFS) and abdomen circumference in dynamics (the weight of the woman should be taken into account). HUFS dimensions delay by 2 cm or the absence of any amount of growth during 2 – 3 weeks at dynamic observation allows suspecting FDD.

2. Sonographic fetal biometry. To asses fetal biometry there are detected the biparietal diameter of the fetal head (BDFH), diameter of the chest and abdomen, length of the fetal hip. Gestational age of the fetus is assessed by the complex of signs. If there is detected inadequacy of one or a couple of basic fetometric indices to pregnancy term, extended fetometry is conducted,

correlation of the frontooccipital and biparietal dimensions, head and abdomen circumference, biparietal dimension and hip length, hip length and abdomen circumference is calculated.

3. Assessment of the biophysical fetal profile.

4. Detection of the level of hormones in the maternal organism and amniotic fluid.

5. Dopplerometry of the blood flow speed in the umbilical artery. Treatment. Pi therapy should be begun with the treatment of the fundamental illness and prevention of unfavourable factors influence. Medicamental therapy consists in the administration of drugs, which improve the uteroplacental blood flow (sigetin), microcirculation in the placenta and rheological properties of blood (dipiridamol, actovegin, esseltiale, hofitol), have antioxidant properties (tocopherol). The increase of the uteroplacental blood flow is also promoted by hyperbaric oxygenation.

Delivery:

1. Delivery per vias naturales is conducted under cardiomonitoring control of the fetal condition at normal or decelerated blood flow in the umbilical arteries, if there is no fetal distress (BPP assessment – 6 points and less).

2. Indications to cesarean section:

- critical changes of blood flow in the umbilical arteries (zero and reverse) – urgent preterm delivery is to be conducted irrespective of the pregnancy term;

- acute fetal distress (bradycardia < 100 bpm and pathological heart rate decelerations) irrespective of blood flow type (normal or decelerated) in the umbilical arteries during pregnancy;

- pathological BPP (4 points and less) in the absence of biological maturity of the neck of uterus (after 30 weeks of pregnancy).

There is no efficient method of FDD treatment, therefore the key moment in managing such pregnant women is the clear assessment of fetal condition and timely delivery.

FDD prevention:

1. Detecting of FDD risk factors and conducting dynamic control over this group of pregnant women.

2. The pregnant woman holding to the day regimen and rational nutrition.

3. Giving up pernicious habits (tobacco smoking, alcohol consumption, etc.).

**Fetal Distress. Postnatal Asphyxia**

Presently all the violations of fetal functional condition are denoted by the term “fetal distress”. It should be noted that with the help of modern noninvasive methods of investigation it is impossible to find the true reasons for fetal cardiac dysfunction. Therefore in clinical practice one should use the term “fetal distress” instead of “chronic fetal hypoxia” and “acute fetal hypoxia”, which are not clinical.

In its turn the term “fetal hypoxia” means the state conditioned by the reasons, which lead to acute or recurrent restriction of access of oxygen to the fetus or to the violation of fetal ability to use oxygen in cellular metabolism. The notions of “fetal hypoxia” and “postnatal asphyxia” must be clearly defined. It should be noted that the term “hypoxia” is to be used in relation to an intrauterine fetus, because, in spite of significant biochemical changes shown by blood analysis, hypocapnia and not hypercapnia declares itself. Concerning newborns it is more correct to use the term “asphyxia”, which means the violation of gas metabolism with the development of hypoxia, hypercapnia, and acidosis.

Etiological factors of fetal hypoxia are divided into preplacental, placental and postplacental.

Preplacental:

1. A group of pathological conditions leading to the violation of oxygen transport to the uterus and placenta:

- violation of maternal blood oxygenation (cardiovascular and pulmonary pathology of the mother);

- hemic hypoxia of the mother – anemia of pregnancy at Hb < 100 g/L;

- generalized circulatory injury (hypotension of pregnancy, essential hypertension, preeclampsia with predominant hypertensive syndrome).

2. Circulatory injury in the uterine vessels:

- pathological changes of the spiral arterioles in the area of the placental bed as a consequence of inflammatory diseases of the endometrium and abortions in the history;

- occlusive vascular violations of the spiral arterioles in the area of the placental bed, peripheral vasoconstriction (preeclampsia, overmature pregnancy, diabetic retinal angiopathy).

Placental proper:

- primary placental insufficiency caused by a disturbance of the development and maturation of the placenta (small placenta, placenta bipartite, angioma, etc.);

- infectious-toxic injuries to the placenta in the late terms of pregnancy;

- detachment of placenta. Postplacental:

- flexure of the umbilical cord (prolapse, compression, winding, knot);

- fetal malformations and pathologies.

By the rate of development there is differentiated acute and chronic hypoxia.

The reasons for acute hypoxia: placenta detachment, umbilical factors, inadequacy of the perfusion of the intervillous lacuna of the maternal part of the placenta at acute maternal hypotension (anaphylactic shock, metrorrhexis).

All the other listed above factors lead to chronic fetal hypoxia.

The main clinical manifestations of fetal hypoxia are:

1) the change of heartbeats character (heart rate, the change of heart sounds, arrhythmia);

2) the change of fetal movements intensity;

3) the appearance of meconium in the amniotic fluid (except for the cases of pelvic presentation).

However, the diagnosis of fetal hypoxia only on the basis of these data not infrequently has erroneous results. In this connection to confirm fetal hypoxia there are detected the indices of the acid-base balance in the blood taken from the skin of the fetal head. A characteristic sign of hypoxia is evident reduction of BE, pH of blood lower than 7.20.

**Fetal Distress in the Course of Pregnancy**

For the diagnostics of fetal distress in the course of pregnancy the following methods are used:

1. Auscultation of heart function (beginning from the 20th week of pregnancy) – heart rate bigger than 170 bpm and less than 110 bpm testifies to fetal distress.

Auscultation of fetal heart function is carried out at each visit of an obstetrician-gynecologist or a midwife.

1. Biophysical fetal profile (BFP) (from the 30th week of pregnancy) – the total of the points for some biophysical parameters is evaluated:

7 – 10 points – satisfactory fetal condition;

5 – 6 points – doubtful test (to be repeated in 2 – 3 days);

4 points and less – pathological evaluation of the BFP (the question of urgent delivery is to be decided).

1. Doppler-metry of blood velocity in the umbilical artery (reflects the state of microcirculation in the fetal part of the placenta, whose vascular resistance plays the basic role in fetoplacental hemodynamics).

Diagnostic criteria are:

1. Pathological blood flow: - decelerated blood flow – reduced diastolic component; the ratio of systole to diastole makes more than 3; - terminal blood flow (testifies to a strong possibility of antenatal fetal death).

2. Zero – the blood flow in the diastole phase stops (no diastolic component in the dopplerogram).

3. Negative (reverse) – the blood flow in the diastole phase acquires reverse direction (the diastolic component below the isoline in the dopplerogram).

**Management of Pregnancy with Fetal Distress**

1. Treatment of concomitant diseases of the pregnant woman, which lead to fetal distress.

2. Staged case monitoring of the fetal condition.

3. Outpatient observation and prolongation of pregnancy is possible at normal indices of the biophysical methods of fetal condition diagnostics.

4. At decelerated diastolic blood flow in the umbilical arteries there should be conducted the investigation of the BFP:

- if there are no pathological indices of the BFP, repeated dopplerometry is to be conducted with an interval of 5–7 days;

- if there are pathological indices of the BFP, dopplerometry is to be conducted at least once in two days, BFP – daily.

5. Detection of the deterioration of blood flow indices (onset of constant zero or negative blood flow in the umbilical arteries) is an indication to urgent delivery by means of cesarean section.

Treatment:

- Till 30 weeks of pregnancy treatment of concomitant diseases of the pregnant woman, which lead to fetal distress.

- After 30 weeks of pregnancy the most effective and justified method of fetal distress treatment is timely operative delivery.

**Delivery:**

1. Is possible through the natural maternal passages (at cardiomonitor control over fetal condition) at:

- normal or decelerated blood flow in the umbilical arteries, if there is no fetal distress (6 BFP points and less).

2. Indications to urgent delivery by means of cesarean section after 30 weeks of pregnancy are:

- critical changes of blood flow in the umbilical arteries (zero and reverse);

- acute fetal distress (pathological bradycardia and heart rate deceleration) independent of the blood flow type (normal or decelerated) in the umbilical arteries during pregnancy;

- pathological BFP (4 points and less) at the absence of biological maturity of the uterine neck.

**Prevention:**

1. Detection of the risk factors of arrested fetal development and conduction of case monitoring of the patients of this group.

2. Adhering to the day regimen, rational nutrition.

3. Refusal from bad habits (smoking, alcohol consumption, etc).

**Fetal Distress in the Course of Delivery**

To diagnose fetal distress in the course of delivery the following methods are

used:

1. Auscultation of fetal heartbeats.

The technique of auscultation during delivery:

- calculation of cardiac beats is conducted for a full minute – every 15 min during the active phase and every 5 min during the second stage of delivery;

- obligatory auscultation before and after a contraction or a labor pain;

- if there are any auscultative violations of fetal heartbeats a cardiographic investigation is carried out.

2. Cardiotocography (CTG):

- at fetal distress in the course of delivery CTG usually shows one or a couple of pathologic signs: tachycardia or bradycardia, persistent rhythm monotony (recording width 5 bpm and less), early, variable, and especially late decelerations with the amplitude bigger than 30 bpm. Unfavorable prognosis is also testified to by:

- deceleration of fetal heart rate at the height of deceleration lower than 70 bpm irrespective of the type and amplitude of deceleration relative to BCSS;

- transition of late or variable decelerations to persistent bradycardia.

3. Detection of meconium in the amniotic fluid at fetal sac rupture:

- the presence of meconium in the amniotic fluid in combination with pathological changes of fetal cardiac rate is an indication to urgent delivery at cranial presentation.

**Delivery management:**

1. Avoid the dorsal position of the parturient woman.

2. Stop oxytocin introduction if it was administered earlier.

3. If the reason for pathological fetal cardiac rate is the mother’s condition, appropriate treatment is to be conducted.

4. If the mother’s condition is not the reason for pathological fetal cardiac rate, and fetal heart rate remains pathological during the last three contractions, one should carry out internal obstetric examination to determine the obstetric situation and find out possible reasons for fetal distress.

5. Fetal distress detection requires urgent delivery:

- in the first period of delivery – cesarean section; - in the second period:

* at cranial presentation – vacuum extraction or obstetric forceps;
* at breech presentation – fetal extraction by the pelvic pole.

**Postnatal Asphyxia**

Postnatal asphyxia is a syndrome accompanied by gas metabolism derangement with hypoxia, hypercapnia, and acidosis.

The reasons for asphyxia may be classified in such a way:

I. Central reasons, which are accompanied by the primary inhibition of the respiratory centers as a result of:

a) fetal hypoxia;

b) immaturity of the fetal nervous system;

c) an injury of the fetal nervous system;

d) pharmacological depressions.

II. Peripheral reasons conditioned by the violation of oxygen supply to the fetal brain right after birth:

a) airways obstruction resulting from the aspiration of the amniotic fluid, meconium, blood, fetal coat fragments;

b) anatomical or functional immaturity of fetal lungs;

c) dysfunction of the fetal cardiovascular system (congenital heart disease, hypovolemia, shock, delay of rearrangement of the fetal type of circulation into extrauterine);

d) severe fetal anemia;

e) congenital anomalies (arthresia choan, diaphragmatic hernia, etc.).

Irrespective of the reasons for fetal hypoxia, they result in the decrease of oxygen level in the fetal blood, development of respiratory and metabolic acidosis, which increases the inhibition of respiratory centres, is accompanied by further derangement of pulmonary ventilation, augmenting of hemodynamic and metabolic disorders.

Postnatal asphyxia most often results from fetal hypoxia. Therefore to the moment of birth there already exists overstrain or derangement of the adaptation mechanisms of the fetal organism in response to intrauterine hypoxia.

The degree of asphyxia is evaluated by the Apgar score on the 1st and 5th min after birth. However, if on the 5th min of life the assessment does not exceed 7 points, additional evaluations are to be conducted every 5 min up to the 20th min of life (the final decision about the inefficiency of resuscitation measures), or to double assessment of 8 and more points.

At the present stage the Apgar score is considered insufficiently informative in the prognosis of asphyxia development. More exact information is given by finding the so-called multiple organs insufficiency (MOI) caused by severe asphyxia at birth.

The main MOI criteria are: violations of the indices of the cardiovascular, respiratory, nervous, homeostasis, urinary, and digestive systems, metabolic disorders (pHa 7.1 and less; BEa 15 micromole/L and less; the level of natrium in blood plasma < 130 micromole/L or  150 micromole/L; the level of potassium in blood plasma < 3 micromole/L or  7 micromole/L; the level of glucose in blood, under the condition of complete parenteral nutrition, < 3.5 micromole/L or  12 micromole/L.

The newborns born in asphyxia are treated in three stages:

the 1st – resuscitation;

the 2nd – syndrome intensive therapy;

the 3rd – rehabilitation.

Preparation to neonatal resuscitation at a high degree of perinatal risk, and also at complicated delivery before the child’s birth: one is to prepare the place and means for resuscitation, check the availability and perfect readiness of equipment and instruments, a set of medicaments, beforehand turn on the heating system of the resuscitation table and conditioning of the breathing gas. After evaluating the fetal condition the neonatal resuscitation department should be signalled about the necessity of turning on the couveuse.

The problem of temperature rate requires special attention. The newborn supercools easily in the process of resuscitation. This is promoted by the fact that the newborn is not even wiped because of haste, and when the amniotic fluid evaporates, heat loss increases (about 540 calories are needed for the evaporation of 1 ml of water).

I.v. introduction of solutions, whose temperature is not controlled, also promotes the supercooling of the newborn. During artificial pulmonary ventilation (APV) there increases the organism’s loss of not only water, but also heat. Resuscitation and intensive therapy without any special measures concerning the optimization of temperature rates is accompanied by the increase of peripheric vessels spasm, which increases the acidosis degree.

At the present stage neonatal resuscitation is conducted by the neonatolgistresuscitator.

The basic components of the resuscitation help to the newborn are known as “ABC-steps” of resuscitation.

A. Airways patency recovery (A – airways).

B. Respiration stimulation or recovery (B – breathing).

C. Circulation support (C – circulation).

Resuscitation stages:

1. Provision of airways patency:

a) to evacuate the content of the oral cavity and pharynx from the moment the fetal head is born, not waiting for the shoulders birth;

b) to continue the suction in the “draining” position after the fetus is born;

c) at massive aspiration the toilet is to be conducted using the guidance of a laryngoscope;

d) the toilet is to be finished with the suction of stomach content to prevent recurrent aspiration after regurgitation or vomiting.

2. APV is conducted after the toilet of the airways if there is no independent breathing during 40–60 s after birth. Respiratory systems of different types are used for this. One is recommended to stick to the following rules when conducting the APV:

a) the newborn’s head is given the position of flexion, for this it is the best to put the newborn on a special table with a movable head support, or to put a roll of diapers under the head;

b) after the beginning of APV one is to conduct lungs auscultation and make sure of the full value of the toilet and efficiency of ventilation, which allows timely change of the regimen of APV in case of need. In the newborns with pulmonary vessels hypoperfusion the APV in the regimen “active inspiration – active respiration” (with negative pressure on expiration), and at excessive blood filling of the lungs and at continual atelectases the application of the APV with increased resistance on expiration of 5–6 mm Hg is administered;

c) if masked APV is ineffective during 2–3 min, trachea intubation is performed under the guidance of a laryngoscope (at a severe stage of asphyxia, massive aspiration, and even at the presence of green amniotic fluid – right after birth), the correct position of the intratracheal tube is controlled auscultatively;

d) in case of need one carries out a repeated toilet of the airways and sanation of the tracheobronchial tree through the intratracheal tube (the catheter diameter must make 2/3 of the tube’s diameter). If the aspirate is dense, isotonic natrium solution is previously introduced with a sterile syringe into the intratracheal tube and then evacuated;

e) if APV through the intratracheal tube is ineffective, in immature newborns it is indicated to use the helium-oxygen mixture containing 30 % oxygen, during 10–15 inspirations;

f) if the APV apparatus is absent or out of order, one must conduct mouth-to-mouth ventilation, being especially careful when exhaling air into the intratracheal tube.

3. Cardiac resuscitation. In case of apparent death, single heartbeats or even at heart rate < 60 bpm one must conduct closed-chest cardiac massage simultaneously with APV. The chest is pressed to the spine 2– 3 times with the tips of two fingers in such a way that the recess makes 1 cm. if cardiac function is not renewed, 0.2 ml of 0.1 % adrenaline hydrochloride solution is stream introduced into the umbilical vein, 3–5 mg of 10 % glucose solution per kg of body weight, 1–2 ml of 10 % calcium gluconate solution,

glucocorticoids (10 mg/kg of body weight or hydrocortisone 4 mg/kg – prednisolone).

If there is no effect, 0.2 ml of 0.1 % adrenaline solution and 1–2 ml of 4 % sodium bicarbonate solution are introduced into the cardial cavity, cardiac massage is continued. It is expedient to conduct APV with cardiac massage during 10 min. Correction of volemic and metabolic disorders

**TESTS**

1.By the end of the 1st period of physiological labor clear amniotic fluid came off. Contractions lasted 35-40 sec every 4-5min. Heartbeat of the fetus was 100 bpm. The BP was 140/90 mm Hg. What is the most probable diagnosis?

**A. acute hypoxia of the fetus**

B. premature labor

C. premature detachment of normally posed placenta

D. back occipital presentation

E. all of the above

2.In fetal circulation:

**A. Most of the blood entering the right atrium flows in to the left atrium**

B. oxygenated blood goes along the umbilical arteries

C. the fetal lung is bypassed by means of ductus venosus

D. the foramen ovale connects the two ventricles

E. the blood in the umbilical arteries is more oxygenated that blood in umbilical vein

3.What parts of the feto-placental complex stay in uterus after the fetal birth?

**A. placenta, membranes, umbilical cord, decidua**

B. placenta, amniotic fluids, umbilical cord

C. placenta, decidua, umbilical cord

D. placenta, amnion and chorion membranes

E. all of the above

4.What function are executed by a placenta?

**A. all the above**

B. trophic

C. excretic

D. protects an umbilical cord from the compression

E. hormonal actyvity

5.Components of biophysical profile include all of the following, EXCEPT:

**A. placental thickness**

B. fetal movement

C. fetal tone

D. fetal breathing movement

E. amniotic fluid volume assessment

6.Antenatal fetal monitoring can NOT be accomplished by:

**A. fetal scalp sampling**

B. fetal kick chart

C. non-stress test

D. obstetric U/S & Biophysical profile

E. acoustic stimulation

7.Which of the following procedures allow the earliest retrieval of DNA for prenatal diagnosis in pregnancy:

**A. chorionic Villi Sampling(CVS)**

B. fetoscopy

C. amniocentesis

D. percutaneous Umbilical Blood Sampling (PUBS)

E. fetal biopsy

8.Regarding the biophysical profile:

A**. includes fetal movement, fetal tone, fetal breathing, fetal heart rate, amniotic fluid is usually done in labor**

B. never include an non-stress test

C. includes a Doppler study

D. includes tone, movement & breathing

E. weight of fetus

9.Fetal assessment include the following EXCEPT:

**A. fetal blood sugar sample**

B. fetal biophysical profile.

C. fetal Doppler velocimetry.

D. fetal biometry.

E. fetal cardiotocography.

10.Patients with high risk pregnancy should have:

**A. fetal biophysical profile**

B. follow-up in ANC every 6 weeks

C. fetal kick chart

D. fetal maternal transfusion

E. fetal amniotomy

**SITUATIONAL TASKS**

1.A biophysical profile in which there is one or more episodes of fetal breathing in 30 minutes, three or more discrete movements in 30 minutes, opening/closing of the fetal hand, a nonreactive nonstress test (NST), and no pockets of amniotic fluid greater than 1 cm would have a total score of: 2.Which of the following statements correctly describes an abnormal contraction stress test (CST)?

3.A reactive nonstress test (NST) is characterized by a fetal heart rate increase of how many beats per minute:

4.In case of normal pregnancy width of the placenta on the 31th week of gestation is:

5. By the end of the 1st period of physiological labor clear amniotic fluid came off. Contractionsm lasted 35-40 sec every 4-5min. Heartbeat of the fetus was 100 bpm. The BP was 140/90 mm Hg. What is the most probable diagnosis?

**Students must know:**

1. Etiology and pathogenesis of fetal hypoxia and asphyxia of the infant.

2. Stages of fetal hypoxia and asphyxia of the infant severity.

3. Principles of the treatment of fetal hypoxia and infant's asphyxia.

**Students should be able:**

1. To take medical history, make general and obstetric examination.

2. To evaluate the results of electronic monitoring of the fetus, ultrasonography, colpocytological and investigations of hormones.

3. To make previous diagnosis.

4. To make plan of treatment of pregnant women and puerperants with fetal hypoxia.

5. To perform the resuscitative measures of the newborn infant.