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MODERN TREATMENT OF ASPHYXIA IN NEWBORNS

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Abstract:

Among the many factors that damage the brain of newborns, hypoxia should be highlighted, which can be classified as a universal damaging agent. Asphyxia recorded in newborns is very often only a continuation of hypoxia, which began in utero. Intrauterine hypoxia and hypoxia in childbirth in 20-50% of cases is the cause of perinatal mortality, in 59% of cases it is the cause of stillbirth, and in 72.4% hypoxia and asphyxia become one of the main causes of fetal death in childbirth or early neonatal period

Keywords: Hypoxia, asphyxia, newborns, treatment

The term "asphyxia" is a conditional concept and is one of the most inaccurate in neonatology. Translated from Greek, the term "asphyxia" means "impulselessness", and such children are usually stillborn. A number of other authors consider the term neonatal asphyxia as the absence of gas exchange in the lungs after the birth of a child (suffocation) in the presence of other signs of live birth (heartbeat, umbilical cord pulsation, voluntary muscle movements, regardless of whether the umbilical cord has been cut and whether the placenta has separated).

In general clinical practice, obstetrician-gynecologists and neonatologists use the terms "fetal hypoxia" and "newborn asphyxia" to understand a pathological condition accompanied by a complex of biochemical, hemodynamic and clinical changes that have developed in the body under the influence of acute or chronic oxygen deficiency with the subsequent development of metabolic acidosis.

The birth rate of children in asphyxia is 1-1.5% (with fluctuations from 9% in children with a gestational age at birth of less than 36 weeks and up to 0.5% in children with a gestational age of more than 37 weeks). There are primary (congenital) and secondary (postnatal - occurs in the first hours of life) neonatal asphyxia.

Pathogenesis. Short-term or moderate hypoxia and hypoxemia cause the inclusion of compensatory adaptive mechanisms of the fetus with activation of the sympathetic-adrenal system by hormones of the adrenal cortex and cytokines. At the same time, the number of circulating erythrocytes increases, the heart rate becomes more frequent, perhaps a slight increase in systolic pressure without an increase in cardiac output. contributes to the inclusion of an energetically unfavorable way of carbohydrate metabolism - anaerobic glycolysis. The cardiovascular system responds by redistributing circulating blood with the primary blood supply to vital organs (brain, heart, adrenal glands, diaphragm), which in turn leads to oxygen starvation of the skin, lungs, intestines, muscle tissue, kidneys and other organs.

Preservation of the fetal state of the lungs is the cause of blood shunting from right to left, which leads to an overload of the right heart by pressure, and the left by volume, which contributes to the development of heart failure, increased respiratory and circulatory hypoxia. Changes in systemic hemodynamics, centralization of blood circulation, activation of anaerobic glycolysis with the accumulation of lactate contribute to the development of metabolic acidosis. In the case of severe and (or) ongoing hypoxia, a breakdown of compensation mechanisms occurs: hemodynamics, adrenal cortex function, which, along with bradycardia and a decrease in minute circulation, leads to arterial hypotension up to shock. The increase in metabolic acidosis contributes to the activation of plasma proteases, pro-inflammatory factors, which leads to damage to cell membranes, the development of dyselectrolytemia. An increase in the permeability of the vascular wall leads to slugging (gluing) of erythrocytes, the formation of intravascular thrombi and hemorrhages. The release of the liquid part of the blood from the vascular bed contributes to the development of cerebral edema and hypovolemia. Damage to cell membranes aggravates damage to the central nervous system, cardiovascular system, kidneys, adrenal glands with the development of multiple organ failure. These factors lead to a change

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in the coagulation and platelet hemostasis and can provoke DIC.

The clinical picture of asphyxia depends on its severity. With moderate hypoxia, the condition of the child after birth is usually regarded as moderately severe. In the first minutes of life, the child is lethargic, motor activity and reaction to examination are reduced. The cry is unemotional. Reflexes of the period of newborns are reduced or depressed. Auscultation of the heart - tachycardia, tones are amplified or muffled. It is possible to expand the boundaries of relative cardiac dullness. Breathing is arrhythmic, with the participation of auxiliary muscles, wired wheezing of various sizes is possible. The skin is often cyanotic, but quickly turns pink against the background of oxygenation. In this case, acrocyanosis often persists. During the first two or three days of life, these newborns are characterized by a change in the syndrome of depression to a syndrome of hyperexcitability, manifested by a small-scale tremor of the limbs, hyperesthesia, regurgitation, sleep disturbance, spontaneous Moro reflex (phase I), a decrease or inhibition of support reflexes, step, crawling, muscle hypotension, adynamia. However, changes in the physiological reflexes of newborns and muscle tone are individual.

The diagnosis of asphyxia is made on the basis of an obstetric history, the course of labor, Apgar scores, and clinical and laboratory data.

All children born with a low Apgar score are subject to monitoring.

Allocate clinical, laboratory and hardware monitoring.

Clinical monitoring: body weight control (2 times a day); dynamics of neurological and somatic status; accounting for fluid volume (nutrition, infusion) and composition (calorie content, proteins, carbohydrates, fats); accounting for the volume of all fluid losses; diuresis control; assessment of the symptom of "white spot" at each examination.

Hardware monitoring: registration of heart rate, blood pressure, respiratory rate, CVP, saturation.

Laboratory monitoring: clinical blood test + Ht, Tr, reticulocytes, color indicator; KOS and electrolytes (K, Na, Mg, Ca); biochemical blood test (glucose, total protein, CRP, bilirubin by fractions, transaminases, creatinine, urea), clinical urinalysis; osmolarity of blood and urine; coagulogram (APTT, PTI, thrombin time, bleeding time, fibrinogen); crops of biological secrets; Plain radiograph of the chest and abdomen; Ultrasound of the brain and abdominal organs; NMR.

Differential diagnosis should be carried out with intrauterine infections, intracranial and spinal birth injuries, acute adrenal insufficiency, trauma of parenchymal organs, diaphragmatic hernia, congenital heart defects, posthemorrhagic anemia.

Treatment. The most responsible step to reduce the severity of the consequences of asphyxia is the primary resuscitation in the delivery room. Strategically important during primary resuscitation should be the desire to achieve the highest possible Apgar score by 5-20 minutes of life. This is explained by the fact that the value of the Apgar score at the 5th minute has a significant impact on the severity of the child's condition in the postresuscitation period and the likelihood of developing neurological consequences of hypoxia. It takes time for a metabolic catastrophe to occur. It is usually limited to minutes or a few hours after the baby is born. The use of this short period of time, when the upcoming changes in the brain are reversible, is the key to the prevention of cerebral disorders.

Of great importance is timely and rational primary resuscitation care, which is based on strict adherence to generally accepted resuscitation principles formulated by P. Safar as A B C - resuscitation, where: A - airway - release, maintaining free airway patency. B - breath - breathing, providing ventilation - artificial (IVL) or auxiliary (IVL). C - cordial, circulation - restoration or maintenance of cardiac activity and hemodynamics.

Principle A: 1) ensuring the correct position of the newborn (the head end should be lowered by 15 degrees, with the head slightly thrown back); 2) aspiration of contents from the mouth, nose and in some cases from the trachea (amniotic fluid aspiration); 3) carrying out endotracheal intubation and sanitation of the lower respiratory tract.

Principle B: 1) tactile stimulation (in the absence of a cry within 10-15 seconds after birth, the child is transferred to the resuscitation table); 2) using a jet stream of oxygen; 3) carrying out auxiliary or artificial ventilation of the lungs (if necessary) using an Ambu, Penlon, Laerbal, Blue Cross bag and a mask or a bag and an endotracheal tube. A properly fitted mask covers the mouth, nose, and edge of the chin, but does not cover the eyes.

Principle C: 1) chest compressions; 2) the introduction of medicines. To determine the volume of primary resuscitation of newborns, the assessment of the state of the child according to the Apgar scale is traditionally used. The most informative is the determination of the parameters that make up the "cardiorespiratory" component of the scale: the number of heartbeats, the nature of breathing, the color of the skin. When assessing: heart rate - 2 points, respiration - 2 points, skin color - 1 point - resuscitation is not required. When assessing: heart rate - 2 points, respiration - 1 point, skin color - 1

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point - after a thorough sanitation of the upper respiratory tract, it is necessary to carry out auxiliary ventilation of the lungs using a 40% oxygen mask for 2-5 minutes. When assessing: heart rate - 2 (1) points, respiration - 1 point, skin color - 0 points - there is a high probability of aspiration syndrome, as evidenced by the presence of meconium content in the amniotic fluid or oropharynx.

It is necessary to conduct a control sanitation of the tracheobronchial tree (TBD) under the control of direct laryngoscopy, to assess the nature of the contents in the catheter. In the absence of contents or a meager amount of aspirate, mask ventilation can be performed. The presence of an abundant amount of amniotic fluid, blood, green staining of the contents require sanitation of the TBD and the solution of the issue of tracheal intubation and mechanical ventilation. The presence of thick meconium in the LBT, which hinders effective sanitation, requires lavage of the LBT with a warm saline solution of 0.5 ml/kg and mechanical ventilation. The multiplicity of lung lavage is determined by the nature of the resulting lavage water (without meconium admixture). When assessing: heart rate - 1 (2) points, respiration - 0 points, skin color - 0 points - tracheal intubation, sanitation of the TBD and taking the child on a ventilator are necessary.

Assessment of heart rate and tactics of action. 1. If the heart rate is less than 80-100 beats per 1 minute, a closed heart massage with lung ventilation is required. If the heart rate increases, continue ventilation. In order to maintain adequate circulation, efforts during closed lung massage should be such that the heart rate is 120 beats per minute. The ratio of ventilation and indirect massage is 1:3. 2. If there is no effect within 10 seconds against the background of ongoing resuscitation or with a heart rate of 80-60 beats per 1 minute and 100% oxygen supply, chest compressions and ventilation should be continued.

In this situation, the following are indicated: 1) tracheal intubation and artificial lung ventilation, 2) administration of drugs through the endotracheal tube: 0.01% adrenaline solution 0.1-0.3 ml / kg (1 ml of 0.1% adrenaline solution diluted in 9 ml of 0.9% sodium chloride solution or 5% glucose solution), or through the umbilical catheter: 0.01% solution of adrenaline or atropine (eliminates sinus bradycardia) at a dose of 0.1-0.3 ml / kg. When the heart rate reaches more than 80 beats per minute, closed heart massage stops. Ventilation is continued until the heart rate reaches 100 beats per minute and the newborn has spontaneous breathing. If the heart rate remains less than 100 beats per minute: 1) repeat the injection of adrenaline, if necessary, this can be done every 5 minutes, but not more than 3 injections; 2) it is necessary to introduce drugs that replenish the BCC if there are signs of hypovolemia (pallor of the skin against the background of inhalation of 100% oxygen, weak pulse with a good heart rate, arterial hypotension, muscle hypotension, a symptom of a "white spot" for 3 seconds or more, a drop in CVP, no effect from ongoing resuscitation) or acute blood loss. The dosage of the selected drug (5% albumin, 5% glucose solution, 0.9% saline, 6% infucol) is 10-15 ml/kg into the umbilical vein for 5-10 minutes. In the absence of effect - the introduction of prednisolone 1-2 mg / kg or hydrocortisone 5-10 mg / kg.

The indication for the introduction of bicarbonate is a confirmed decompensated metabolic acidosis (pH < -7.0; BE > - 12), as well as the absence of the effect of mechanical ventilation, chest compressions, adrenaline administration and BCC replenishment. Intravenously administered 4% sodium bicarbonate solution 2-4 ml/kg for 2 minutes in a 2-fold dilution of 0.9% saline or 5% glucose. When conducting primary resuscitation, if it becomes necessary to administer drugs, they should be administered in saline, because. infants born in asphyxia have hyperglycemia and lactic acidosis. Resuscitation measures are carried out with the obligatory observance of the temperature regime in the intensive care room (26-28 C), and the control of the child's body temperature (at least 36.4 to 37.0 C). Resuscitation measures in the delivery room are terminated if, during the first 20 minutes after birth, against the background of adequate resuscitation measures, the child's cardiac activity is not restored.

Apgar score ("cardiorespiratory" component of the scale): 2 2 0; 2 1 0; 1 1 0 - can often be due to the presence of CHD in a child, esophageal atresia, congenital pneumonia, aspiration syndrome. Fast and effective primary resuscitation care for a newborn with asphyxia can only be provided with ready-made equipment and well-trained, experienced personnel who have the skills to perform mechanical ventilation using a mask and modern equipment, endotracheal intubation, chest compressions. The main groups of drugs used in the acute posthypoxic (postresuscitation) period are as follows: antihypoxants and anticonvulsants (20% GHB solution 50-100 mg/kg; 0.5% seduxen solution 0.2-0.4 mg/kg, phenobarbital 5-20 mg/kg kg / day), antioxidants (5.10% oil solution of vitamin E 0.2 ml / kg, 0.1 ml / kg, respectively; aevit 0.1 ml / kg; 0.25% solution of cytochrome C or cyto-mac 1 ml /kg); corrective metabolic disorders (4% sodium bicarbonate solution at the rate = BE of the patient x m (kg) x 0.5 administered intravenously as a bolus in 2-fold dilution of 0.9% saline). To restore central and peripheral hemodynamics, they resort to titration of 0.5%

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dopamine solution, 4% dopmin solution 1-10 μg / kg / min - effects on, β - receptors; dobutamine, dobutrex 2-10 mcg / kg / min - for β - receptors). In order to stop cerebral edema, diuretics are prescribed - saluretics (1% solution of Lasix 1-2 mg / kg, veroshpiron 2-4 mg / kg / day). Given the changes in the hemostasis system and the high risk of developing hemorrhagic disorders, it is necessary to prescribe hemostatic drugs (vikasol, konakion 1-2 mg / kg; angioprotectors - 12.5% solution of dicynone, etamsylate 10-15 mg / kg; fresh frozen plasma 10-15 ml /kg).

To normalize the processes of neurometabolism and blood circulation in the central nervous system, the complex of therapeutic measures includes nootropic drugs with a sedative effect (phenibut, pantogam up to 100 mg / day in 2 doses) or a stimulating component (piracetam 50-100 mg / kg / day, picamilon 1.5- 2.0 mg/kg/day in 2 divided doses, aminalon 0.125 mg 2 times a day, encephabol 20-40 mg/kg/day); drugs that improve cerebral circulation (trental, cavinton, vinpocetine 1 mg / kg in the absence of hemorrhages, tonakan 1 cap. / kg 2 times a day); In addition, various methods of oxygen therapy are shown (nasal catheters, mask, oxygen tent, SDPPD, mechanical ventilation). In this case, it is necessary: 1) to eliminate hypoxemia and avoid the occurrence of hyperoxia, which contributes to the development of BPD and retinopathy; 2) eliminate hypercarbia, tk. an increase in CO2 in the blood leads to the development of acidosis, vasodilation of cerebral vessels and the occurrence of hemorrhages; 3) prevent hypocarbia, which is accompanied by a decrease in blood flow in the cerebral vessels, contributing to the occurrence of ischemic foci in the brain tissue. Planned infusion therapy, if necessary, is optimally started after 40-50 minutes. after birth The usual volume of infusion therapy (see Appendix) on the first day is 60-80 ml / kg (in very preterm infants it can be increased to 100-200 ml / kg). In heart failure, the infusion volume should be reduced to 50 ml/kg. From the 2nd day of life, calcium and sodium correction is carried out, from the 3rd - potassium, as well as magnesium correction (see Appendix).

CONCLUSION. The main indications for infusion therapy are: correction (normalization) of metabolic, electrolyte disorders; maintenance of carbohydrate metabolism; detoxification; normalization of peripheral and/or central hemodynamics; parenteral nutrition; replacement therapy.

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