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AEROBIC METABOLISM IN PREMATURE NEWBORNS WITH HYPOXIC-ISCHEMIC LESION OF CNS

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All forms of oxygen deficiency are accompanied by the development of hypoxia and energetic deficiency, which leads to tension of metabolic processes of the organism. Metabolic effect of hypoxia includes stark reduce of mitochondrial activity due to a significant inhibition enzymes of the Krebs cycle: succinate dehydrogenase (SDH).

Research purpose: to increase the efficiency of diagnosis of hypoxic-ischemic CNS lesions in premature infants by determining the activity of SDG in early neonatal period.

The activity of SDH was determined in 15 conventionally healthy preterm infants (CHPI), which made the comparison group and 64 premature babies with hypoxic-ischemic CNS lesions on 1-7 day of life. They which were divided into three groups: I group – 26 premature children with mild CNS lesions; II group – 20 premature children with severe hypoxic-ischemic lesions and low birth weight; III group – 18 premature newborns with severe damage of central nervous system and extremely low birth weight.

In the premature newborn metabolic effect of hypoxia occurs in the early neonatal period through severe inhibition of mitochondrial respiratory activity, as testified by reduce activity of the main aerobic enzyme – SDH.

In infants with mild perinatal CNS injury in the early neonatal period the total number of granule of formazan into cells decreased on 33% relative to the comparison group ($p < 0,001$). In same time for infants in second and third groups were quantity 1,7 and 2,0 times lower respectively than in comparison group ($p < 0,001$).

Along with a decreased of the total number of formazan granules, were decreased number of cells containing these granules, and the average number of granules per cell.

Reduced activity of SDH indicates the initial stages of the process of decompensation, which is accompanied by disturbance of energy metabolism and leads to the formation of tissue hypoxia. At severe CNS injury further decrease activity of this enzyme shows the progression of decompensation and the formation of severe energetical disorders.

Thus, the process of aerobic glycolysis occurs in the mitochondria of brain cells with enzymes of Krebs cycle, the main of which is SDH, only in case of normal diffusion oxygen from intercellular space into neurons. So as markers of severity of perinatal hypoxic-ischemic CNS lesions in premature infants in early neonatal period can use activity of SDH.

COURSE OF ASPIRATION SYNDROME IN NEWBORN INFANTS

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Aspiration syndrome of newborn children is one of reasons of development of the decompensated respiratory insufficiency that requires realization of artificial ventilation of lungs (ALV). In the department of intensive therapy new-born (ICU) of the Sumy regional child's clinical hospital for period 2009-2014 acted 1179 babies, 200 (17%) from them were hospitalized with a diagnosis neonatal aspiration. All babies were transported in the conditions of departure neonatology brigade from maternity establishments of region. During transporting ALV was conducted all children. Middle age of children at entering in the ICU was $17 \pm 0,6$ h., the 95% newborns were translated during the first twenty-four hours of life. In the ICU high-frequency ALV was conducted all children. All children by type of neonatal aspiration have been divided into two groups.

The first group included 126 infants with pure aspirating amniotic fluids (PAF), which accounted for 63% of the total number of aspiration syndromes. The second group included 74

patients with meconium aspirating syndrom (MAS), which accounted for 37% of the total number of aspiration syndromes.

In the analysis of the duration of ALV, the average number of bed-days no significant differences between the groups was not. There was a trend to increase in these indicators in children with meconium aspiration syndrome.

Radiological examination in children with meconium aspiration pneumonia were detected significantly more frequently, air leak syndromes and atelectasis.

Restoring of pneumatization of lung tissue in children of this group has been slower than in other neonatal aspiration syndromes. Four children were full-term, one child - premature, gestational age 35 weeks. Fatal outcomes were registered in 5 children (0.4%). Sectional data on children in three main diagnosis was septic process of different etiology, in two children intrauterine infection of chlamydia etiology. Competing diagnoses in one child registered with congenital heart disease, and two children - aspiration pneumonia.

ANAEROBIC METABOLISM IN PREMATURE NEWBORNS WITH HYPOXIC-ISCHEMIC LESION OF CNS

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The brain, as the main target organ in case of hypoxia, is very sensitive to hypoxia. Energy supply of the brain caused most of all by aerobic mechanisms. Hypoxia causes an energy stress that activates compensatory anaerobic way of glucose utilization, for which is high specific increase activity of anaerobic enzymes, especially lactate dehydrogenase (LDH).

Research purpose: to increase the efficiency of diagnosis of hypoxic-ischemic CNS lesions in premature infants by determining the activity of LDG in early neonatal period.

The activity of LDG was determined in 15 conventionally healthy preterm infants (CHPI), which made the comparison group and 64 premature babies with hypoxic-ischemic CNS lesions on 1-7 day of life. They which were divided into three groups: I group – 26 premature children with mild CNS lesions; II group – 20 premature children with severe hypoxic-ischemic lesions and low birth weight; III group – 18 premature newborns with severe damage of central nervous system and extremely low birth weight.

Research LDH levels in serum of premature infants with perinatal hypoxic-ischemic CNS lesions found that in case of oxygen deficiency in children of all groups was significant increase level of this enzyme, and thus activation of anaerobic glycolysis. In the early neonatal period in children with mild hypoxic lesions enzyme concentration increased 2,5 times relative to the comparison group ($p < 0,001$). This shows the maximum tension of compensatory adaptive mechanisms aimed to the effective utilisation of energy substrates to prevent the energy deficiency.

In case of severe hypoxia observed certain exhaustion of compensatory mechanisms of anaerobic glycolysis activation, but even among newborns of second and third groups with severe hypoxic injury LDH level in serum was 1,7 times greater ($p < 0,001$ and $p < 0,01$, respectively) than in neonatal comparison group.

Hypoxia dramatically reduces the activity of aerobic glycolysis. Metabolism in brain cells becomes anaerobic with the activation of the corresponding enzyme (LDH). Functional brain activity is suppressed, which is manifested clinically by progressive impairment of consciousness. Hypoxia blocks include pyruvic acid into Krebs cycle, that's why piruvate isn't oxidized and converted to lactic acid. Increasing the concentration of the latter cause acidosis, this is a factor of destruction of cell membranes of neurons.

Thus, as markers of severity of perinatal hypoxic-ischemic CNS lesions in premature infants in early neonatal period can use activity of LDH.