COMPARATIVE ANALYSIS OF NEWBORNS ADAPTATION TO INTRAUTERINE HYPOXIA AND CEREBRAL ISCHEMIA DEPENDING ON THE LEVEL OF ROS ACTIVITY AND ANTIOXIDANT DEFENSE SYSTEM

Diagnostics of hypoxic-ischemic encephalopathy (HIE) that is based mainly on clinical and neurosonographic criteria should be added with the research of biochemical parameters of the generation of reactive oxygen species (ROS) and ROS-scavenger enzymes activity in umbilical blood which can be useful. It is revealed, that the level of ROS generation at women from the group of risk (with an anemia and FPI) before giving a birth and in umbilical blood has an identical direction of changes. In chronic hypoxia caused by an anemia of mothers, the increase of catalase activity and decrease of SOD activity are detected both in blood of mother, and umbilical blood. Chronic hypoxia at FPI is accompanied by the oppression of activity both SOD and catalase.

According to the condition of mother it is possible to predict the current of early neonatal period at newborn, according to the condition of SOD activity and catalase in umbilical blood, and also it is possible to judge about the intensity of protective systems of the organism on activation of ROS generation that accompanies hypoxia/re-oxygenation and to determine the approaches to the directed antioxidant therapies.

Keywords: Chronic hypoxia, reactive oxygen species, ROS-scavenger.

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Introduction

Nowadays successes of neonatology provide the raising of the opportunities of nursing prematurely and maturely born children with heavy perinatal damages of the brain that reduce a level of mortality (Perlman, 2006). At the same time, the problem of early diagnostics, definition of a condition and early intervention at children of risk groups on development of heavy hypoxic defeats of CNS possess the big urgency as the increase of physical inability and deep neurologic complications at survived patients (Hannah et al, 2009).

The studying of the processes occurring in brain tissue at the hypoxia-ischemia with the help of biochemical and immunoenzymatic research methods allowed to receive the new information about the changes in cells on molecular level that promoted the expansion of opportunities of early detection of cerebral injury (Chavez, 2006).

Hypoxic defeat of a brain can be developed at fetus (an anemia at mother, fetus-placental insufficiency), at acute anoxia, in early neonatal period. Despite of variety of the reasons of hypoxia development, the changes occurring in brain cells, include the following mechanisms: switching on anaerobic metabolism, ATP deficiency, the disorder of the

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work of ionic pumps, neuronal membranes depolarization, release of excitatory amino acids, the increase of production of reactive oxygen species (ROS), oxidative stress, cell destruction by apoptosis and necrosis mechanism (Mir Ahamed Hossain, 2008).

In a cerebral cortex and basal ganglia excitatory amino acids, glutamate and aspartate, interacting with specific receptors such as kainate, N-methyl-D-aspartate (NMDA), and amino-3-hydroxy-5-methyl-4 isoxazole propionate (AMPA), start the glutamate-Ca\(^{2+}\) cascade, stimulate generation of ROS, provoking membrane destruction and apoptosis. Interaction between kainate and NMDA receptors determines the duration of excitation wave and efficiency of reorganization of a metabolism of a nervous cell under influence of Ca\(^{2+}\). Excitotoxicity of glutamate also assumes that excitation of NMDA receptors leads to the increase of ROS production, the distribution of defeat, vasoconstriction that aggravates hypoxia and ROS production. The second important mechanism of damage of neurons is disorder of ionic pump Na\(^+/\)K\(^+\)-ATPase as a result of surplus generation of ROS and oxidation of components of cellular membranes. It is quite probable that ROS and excitatory amino acids interfere with factors which normally operate as apoptosis that increases the rate and expressiveness of process programmed death of a cell (Mir Ahamed Hossain, 2005).

ROS includes hydrogen peroxide, hydroxyl-radical and superoxide. In norm ROS are formed in a respiratory chain of mitochondrion, at hypoxia there is an inadequate saturation of mitochondrial cytochromoxidase by oxygen, damage of electrons transport on a respiratory chain and hyper production of superoxide, and in the subsequent -hydrogen peroxide and superoxide -radical (at the presence of metals of variable valence such as iron). ROS, in an excess amount, have a toxic effect. Increase of concentration of endocellular Ca\(^{2+}\), activates NO-syntase, cyclooxigenase, lipooxigenase that increases the synthesis of ROS. NO production under the influence of nNOS is increased at the excitation of NMDA receptors. NO-radical, in this case, has damaging effect on neuronal DNA, and its interaction with superoxide leads to the formation of peroxinitrit and hydroxyl-radical. ROS hyperproduction leads to the destruction of cells via apoptosis and necrosis mechanism.

It is impossible separately to speak about necrosis and apoptosis, the combination of these two variants of cells destruction to prevalence of one of them in various phases hypoxic defeats of a brain always takes place. Special vulnerability of olygodendroglia at prematurely-born children is caused by easier accumulation of free radicals in it by their greater toxicity in the attitude immature olygodendroglia cells in comparison with mature. Differentiated neuroglia cells are very sensitive to the deficiency of oxygen in the neonatal period, its defeat in the specified period leads to the disorders of myelinisation. Selective necrosis of neurons is determined by various sensitivity of brain cellular elements to hypoxia. In a mature brain the neurons are more vulnerable, and less vulnerable ones are olygodendroglia, astrocytes and microglia (Weidemann, 2009).

A micro and macroglia cells, neurons, endothelium, cerebral vessels are damaged by hypoxia. Glutamate under the influence on NMDA-receptors in microglia promotes the increasing generation of ROS that leads to the damaging and losing of astrocytes and gliosis, apoptosis and membrane destruction. Activation of kainite and AMPA-receptors occurs in olygodendrocytes leading to the damaging and losing of olygodendroglia. Both processes promote the spongy formation of the white matter in brain tissue. Local distinctions in a rate of damage can be explained that excitatory amino acids selectively influences on CA1 in area of hippocamp, on olygodendroglia and neuronal cells on border of periventricular areas, especially in a developing brain. It can be a morphological substratum for the disorder of cognitive abilities and memory at children who had underwent hypoxic-ischemic encephalopathy (HIE) in infancy (Yvonne et al., 2006).

Without dependence on etiology of HIE, at the same patient some types of damage can be found out. The important condition in development of child’s brain is degree of brain maturity at the moment of action of damaging factor. However, even in absence of
obvious neurologic symptoms in the neonatal period, further at such children the functional deviations can be observed. In group of the schoolboys who have moderate HIE, 15-20% have significant difficulties of studying, even in absence of obvious symptoms of damage of a brain. Because of it all children who have a cerebral ischemia, should continue the same supervision at the neurologist, as well as at infantile age.

Despite on significant success of neonatology and neonatal neurology frequency of cerebral ischemia and its consequences does not decrease. Probably, it is caused by that predictive value of clinical and biochemical markers are variable. There are no the trouble-shooting tests, finally proving, that the certain expressiveness anoxia causes the certain neurologic defect. Above-stated dictates necessity of search of markers of the structurally functional insufficiency of brain describing a degree of adaptation newborn to a pathological condition of hypoxia and reflecting in interrelation of change in system a mother - fetus, allowing to predict complications in neonate in early neonatal period.

The purpose of this work is revealing dependence of clinical current of a cerebral ischemia from a degree of adaptation of an organism to intra-uterine hypoxia by studying a level of ROS generation and activity of system antiradical and antiperoxidant protection in blood at mothers from group of risk on hypoxia of fetus.

**Materials and methods**

In the course of clinical observations, the patients were analyzed on the basis of Neonatal Intensive Care Unit at Scientific Medicine Center of Obstetrics and Gynecology, Tashkent Maternity Complex No.6, TashPMI Hospital. There were observed 147 children (pairs mother/newborn) from the neonatal period till one year old. From them 20 children were mature, born from healthy mothers and during the neonatal period and the first year of life were practically healthy. They have made control group.

18 children were born in acute anoxia, 126 children had attributes of a cerebral ischemia (P 91.0 on IDK-10) a various degree or were born from mothers from group of risk on chronic intra-uterine hypoxia (mother with anemia, fetus-placenta insufficiency (FPI) and the burdened somatic status), that also, agrees IDK-10, is the basis for diagnosing hypoxic-ischemic encephalopathy II degree. Thus, all surveyed have formed 3 groups: I - 126 mature newborn with a cerebral ischemia of a various degree owing to transferred chronic intra-uterine hypoxia, II - 18 mature newborn with a cerebral ischemia of a various degree owing to transferred acute anoxia, III - 20 healthy newborn (control). Diagnosis anoxia was established at рН 7.0 and is lower, deficiency of the bases 16 mmol/l or more in umbilical blood or blood of the child at the first hour after birth, the estimation on Apgar scale 5 and lower on 10th minute of life.

At pregnant women it was carried out clinical observation, ultrasonic, biochemical researches of blood. At newborns multi spectrum researches of clinical current of HIE and the analysis of the basic clinical symptoms and syndromes in dynamics were carried out.

Neurosonologic research was done on Ultrasound equipment DC7 (MINDRAY) using phasing, convex transducers with 3.5-5.0-7.5-10 MHz. Intra-periventricular hemorrhages were classified by Levine and ischemic lesions by De Vries.

Biochemical research of umbilical blood is carried out. Concentration of malondialdehyde (MDA) in the blood serum is defined by Stalnaja method (1977). The method is based on interaction of 2-tiobarbiturate acid with MDA at high temperature with production of the colored trimethin complex which has a characteristic wavelength absorption spectrum showing maxima at 532nm. Molar coefficient of extinction of this complex Е532=1.56x105cm⁻¹M⁻¹. For definition of intensity of spontaneous ROS generation the incubatory mixture consist of 0.2 ml of blood cerum and 0.8 ml TRIS HCl buffers. A mixture incubated at 37°C during 30 mines, reaction stopped by addition of 2 ml of 17% threchloracetat (TCA), then probes were centrifugated for 15 minutes, 2 ml of supernatant was separated and added to 1 ml of 0.5% of a solution 2-TBA. Then probe was placed in
a boiling water bath for 10 minutes for development of coloring. After cooling probe was spectrometred. As comparison probe used a sample containing all components of incubation mixture, but not exposed at 37°C.

Catalase activity was measured by S.M.Zubkova with permanganatometry. SOD activity was investigated on method of Mirsa P.H., Fridovich S. in modification of O.S. Brusov, 1976. The method is based on inhibition the enzymes of autooxidation of adrenaline in the alkaline environment (pH 10.2) and formation of adrenochrome, registered on change of optical density at 480 nm. Registered initial speed of free autooxidation of adrenaline in adrenochrome (control test), then initial speed of autooxidation of adrenaline at presence of SOD of researched tests. For unit of activity of enzyme there was accepted such amount of SOD which is required for inhibition of autooxidation of adrenaline in the specified conditions on 50%. Articles were identified through a search of the PubMed database.

Results and discussion

The data of observation of pairs the mother/child among mature newborn with attributes of a cerebral ischemia of a various degree (P 91.0), acute anoxia (n=18) and chronic hypoxia (n=126) are given in comparative aspect. From 126 mature newborn with the cerebral ischemia of a various degree at the majority of mother were from group of risk of development chronic intra-uterine hypoxia: women with iron-deficit anemia was 59; with FPI - 30; with the burdened anamnesis and extra genital diseases - 17, practically healthy - 20. In blood serum of group of women with an anemia the contents of hemoglobin was below similar parameter of blood of control group in 1.23 times (Table 1).

In blood of mothers the level of oxidizing stress was the greatest at mothers with FPI (in 4.4 times above norm), and much less expressed at mothers who have given birth to children with acute anoxia (in 1.5 times there is more than norm). Activity of enzymes antiperoxide and antiradical protection at lying women with an anemia and FPI authentically differed from similar parameters of norm. Remarkable there was a fact of that SOD activity was oppresssed, and catalase activity - was increased. So, catalase activity at mothers with an anemia was higher than norm in 1.7 times, whereas SOD activity was lower than norm in 1.4 times. At the women who have given birth to children with acute anoxia, catalase activity on the contrary went down in 1.2 times concerning the control, and SOD activity - authentically did not differ from normal parameters. Such different character of changes of components of antioxidative system (AOS) is possibly explained by the duration of hypoxia, accompanying an anemia and FPI. It specifies that

### TABLE 1. INTENSITY OF OXIDATIVE STRESS AND ACTIVITY OF ROS-SCAVENGER ENZYMES IN BLOOD OF MOTHERS

<table>
<thead>
<tr>
<th>Group of patients</th>
<th>MDA, nmol/mg of protein x min</th>
<th>Hb, g/l</th>
<th>Catalase, mmol H2O2/min RBC</th>
<th>SOD, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Healthy - the control (n=20)</td>
<td>0.51 ± 0.01</td>
<td>114.0 ± 1.2</td>
<td>40.62 ± 0.87</td>
<td>77.1 ± 2.2</td>
</tr>
<tr>
<td>Mothers with an anemia (n=59)</td>
<td>1.46 ± 0.04</td>
<td>93.0 ± 1.6*</td>
<td>69.56 ± 0.74*</td>
<td>56.1 ± 3.2*</td>
</tr>
<tr>
<td>Mothers with FPI (n=30)</td>
<td>2.25 ± 0.06*</td>
<td>108.2 ± 1.4</td>
<td>33.6 ± 1.2*</td>
<td>41.9 ± 3.7*</td>
</tr>
<tr>
<td>Mothers who have given birth to children with acute anoxia (n=18)</td>
<td>0.75 ± 0.09*</td>
<td>106.0 ± 2.4</td>
<td>32.60 ± 0.81*</td>
<td>71.8 ± 1.2</td>
</tr>
<tr>
<td>Mothers with burdened anamnesis and extra genital diseases (n=17)</td>
<td>0.87 ± 0.09*</td>
<td>108.0 ± 2.9</td>
<td>30.5 ± 0.32*</td>
<td>63.2 ± 1.1*</td>
</tr>
<tr>
<td>Practically healthy mothers who have given birth to children with HIE (n=20)</td>
<td>0.9 ± 0.12*</td>
<td>110.0 ± 2.1</td>
<td>37.5 ± 0.82</td>
<td>73.2 ± 1.4</td>
</tr>
</tbody>
</table>

Note: * - it is authentic concerning the control.

Apparently from Table 1, the level of oxidizing stress in blood was the greatest at mothers with FPI (in 4.4 times above norm), and much less expressed at mothers who have given birth to children with acute anoxia (in 1.5 times there is more than norm). Activity of enzymes antiperoxide and antiradical protection at lying-in women with an anemia and FPI authentically differed from similar parameters of norm. Remarkable there was a fact of that SOD activity was oppresssed, and catalase activity - was increased. So, catalase activity at mothers with an anemia was higher than norm in 1.7 times, whereas SOD activity was lower than norm in 1.4 times. At the women who have given birth to children with acute anoxia, catalase activity on the contrary went down in 1.2 times concerning the control, and SOD activity - authentically did not differ from normal parameters. Such different character of changes of components of antioxidative system (AOS) is possibly explained by the duration of hypoxia, accompanying an anemia and FPI. It specifies that
at acute anoxia influence of adverse factors to a lesser degree mentions an organism of mother, rather than at an anemia and FPI. It is remarkable, that in umbilical blood the changes similar revealed in blood of lying-in women were found out, but they, as against parameters of parent blood, characterize a status of a fetus and its adaptable opportunities (Table 2) in the greater degree.

### Table 2. Intensity of oxidizing stress and activity AOS in umbilical blood

<table>
<thead>
<tr>
<th>Group of patients</th>
<th>MDA, nmol/mg of protein x mines</th>
<th>Hb, g/l</th>
<th>Catalase, mmol H2O2/mln RBC</th>
<th>SOD, T %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Healthy adults</td>
<td>0.51 ± 0.01</td>
<td>114.0 ± 1.2</td>
<td>40.62 ± 0.87</td>
<td>77.1 ± 2.2</td>
</tr>
<tr>
<td>Healthy newborn (n=20)</td>
<td>1.71 ± 0.01</td>
<td>184.0 ± 4.8</td>
<td>60.93 ± 0.90</td>
<td>88.6 ± 1.3</td>
</tr>
<tr>
<td>Newborn from mother with an anemia (n=59)</td>
<td>5.46 ± 0.14*</td>
<td>172.0 ± 3.8</td>
<td>69.84 ± 4.74</td>
<td>46.5 ± 8.7</td>
</tr>
<tr>
<td>Newborn from mother with FPI (n=30)</td>
<td>7.25 ± 0.30*</td>
<td>169.0 ± 3.8</td>
<td>54.5 ± 3.2</td>
<td>49.8 ± 5.9</td>
</tr>
<tr>
<td>Children who have been given birth in acute anoxia (n=18)</td>
<td>2.75 ± 0.2*</td>
<td>171.0 ± 5.6</td>
<td>39.14 ± 0.81</td>
<td>29.7 ± 1.7</td>
</tr>
<tr>
<td>Newborn from mother with burdened anamnesis and extra genital diseases (n=17)</td>
<td>1.87 ± 0.19*</td>
<td>168.0 ± 1.9</td>
<td>48.5 ± 0.62*</td>
<td>53.2 ± 1.1*</td>
</tr>
<tr>
<td>Children with HIE from rather healthy mothers (n=20)</td>
<td>4.33 ± 0.11</td>
<td>170.2 ± 3.5</td>
<td>53.1 ± 0.8</td>
<td>49.5 ± 2.2</td>
</tr>
</tbody>
</table>

If acute anoxia is the short-term hypoxia an organism of newborn, the anemia and FPI in mothers are the influence of long term chronic hypoxia of fetus. Probably, the given approach in treatment of the received data allows interpreting them from the point of view of adaptive opportunities of an organism. Children who have been given birth in acute anoxia and from mothers from group of risk on intra-uterine chronic hypoxia had the difference of compensation-adaptive mechanisms reflected in parameters AOS.

Analyzing the MDA level and the AOS enzymes activity in umbilical blood at healthy newborn, it would be desirable to note the high level of MDA and high intensity of oxidative stress at healthy newborn, no less than activity of enzymes of protection from ROS. We shall emphasize, that at studying processes of lipid peroxidation and efficiency of mechanisms AOS are matter not so much absolute sizes of parameters, how many a ratio between them. At healthy newborn it is revealed increased in 3.35 times in comparison with adults level of MDA, however at them in conditions of activation free-radical processes reliable enough functioning mechanisms AOS is marked.

At newborn with intra-uterine hypoxia, the misbalance between ROS-generation and AOS enzymes activity is observed: increase of activity of processes the ROS-generation and decrease of AOS activity in comparison with healthy newborn. Apparently from the Table 2, at children who have been given birth in acute anoxia, MDA level not so is expressed it is increased, rather than at children who have transferred chronic hypoxia, however SOD activity at these patients is sharply lowered.

In umbilical blood of the patients who have transferred chronic intra-uterine hypoxia owing to an anemia, appreciable downturn of SOD activity and increase of catalase activity is revealed. Chronic hypoxia at FPI it was accompanied by oppression of activity as SOD, and catalase. This interesting fact of different changes of activity of ROS-scavenger enzymes in group of patients with chronic intra-uterine hypoxia (FPI and the anemia) specifies that response of antiradical system at hypoxia conditions is stereotyped whereas response antiperoxidative systems of protection depends on duration of hypoxia.

At short-term hypoxia, caused by acute anoxia, in umbilical blood there is an oppression of SOD activity at normal values catalase, whereas in blood of mother downturn of catalase activity takes place at normal values SOD.
One more fact has drawn our attention: despite of the common tendency in changes of parameters of system the ROS generation/AOS inside groups of the surveyed patients were available different shifts.

So, at a part of children who have been given birth from mothers with an anemia, catalase activity in umbilical blood was lowered, whereas as a whole in this group the level of activity of this enzyme was higher than norm. Lines of mothers from group of risk on chronic hypoxia of fetus had normal MDA level in blood, whereas SOD and catalase' activities was lowered. Not in all cases of change in blood of mother were same to changes in umbilical blood.

**TABLE 3. THE MODIFIED SCALE OF A SUB-OPTIMALITY OF THE PRENATAL RISK FACTORS**

<table>
<thead>
<tr>
<th>Groups of newborns</th>
<th>Groups of Mothers</th>
<th>Apgar scale</th>
<th>Biochemical Criteria</th>
<th>The neurologic criteria</th>
<th>Neurosonographic criteria</th>
<th>Changes in the child development at first year of life</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low prenatal risk of HIE</td>
<td>Women 18-25 y.o. with gynecologic and somatic diseases, but it is rather favorable current pregnancy</td>
<td>6-7/8</td>
<td>Activity of the SOD and catalase in norm at mother. Slight increase (less 15%) of catalase activity, decreasing of the SOD activity less than 15% at norm, the MDA level corresponds to norm in umbilical blood.</td>
<td>Transitory neurologic frustration: muscular distonia, a syndrome of hyper -excitability; Presence of clinical symptoms no more than 7 days.</td>
<td>Transitory brain ischemia and transitory increase of speed of bloodstream in the basic vessels of the brain.</td>
<td>Psychiologic and physical development corresponds to age norm</td>
</tr>
<tr>
<td>Increased prenatal risk of HIE</td>
<td>Women at age senior 25 y.o. with expressed somatic, obstetric and gynecologic pathology; a combination more 2x adverse factors during pregnancy; pathological pregnancy and delivery</td>
<td>6-7</td>
<td>Increasing the catalase activity less than 15%, decreasing the SOD activity to 15-30%, the MDA level - less 2 times higher than norm at mothers blood. The high MDA level (2 times at norm), decreasing the SOD activity to 15-30% at norm, increasing catalase activity less than 15% in the blood of umbilical cord.</td>
<td>Compensative increasing of the bloodstream in the basic arteries of a brain within the first 7 days.</td>
<td>Physiological reflexes by duration from 7 about 20 days</td>
<td>Retarding on psycho-motor development The minimal brain dysfunctions</td>
</tr>
<tr>
<td>High prenatal risk of HIE</td>
<td>Pathological pregnancy, a combination more than 3 pathological factors; Numerous threat of interruption of pregnancy; early gestosis at pregnancy.</td>
<td>2-4</td>
<td>Increasing the catalase activity more than 15% at norm, opposition the SOD activity more than 50%, MDA level – more than 2 times at norm in the mother’s blood. The high MDA level (more than 3-4 times at norm), very low SOD activity (oppression more, than 75% at norm), reduction of catalase activity more than 15% at norm in the blood of umbilical cord.</td>
<td>Rough neurologic symptoms in neonatal period, expressed syndrome of oppression; the convulsive syndrome, the expressed muscular hypertension or hypotonia. Presence of pathological symptoms more than 20 days.</td>
<td>Decrease (reduction) of bloodstream speed, ventricular dilatation, perihemorrhages.</td>
<td>Psychiologic-neurological frustration: children’s cerebral paralysis.</td>
</tr>
</tbody>
</table>
In this connection, we have found expedient to analyze values of biochemical parameters of adaptable systems antiradical and antiperoxidative protection and a level of ROS generation in the blood of the mother and umbilical blood, having compared them with clinical symptoms dynamics in the early neonatal period, with the neurologic status of children during the first year of life and with the degree of a cerebral ischemia. Such approach corresponds to recommendations IDK-10, where it is taken into account not only duration of hypoxia, but its degree. So, at easy acute anoxia the I degree of a cerebral ischemia is observed, the cerebral ischemia of II degree is observed at intra-uterine chronic hypoxia, and also at average degree of acute anoxia. The cerebral ischemia of III degree is diagnosed at heavy acute anoxia and at intra-uterine hypoxia.

Depending on activity of ROS-scavenger enzymes such as SOD and catalase the observed pairs (mother/child) were divided into four groups. At the 1-st group (both at mother, and in umbilical blood) it is established high enough (in comparison with other groups) activity of the SOD and catalase enzymes and normal level for their age of the MDA. The high SOD and catalase activity corresponded to good adaptation of an organism of the child in neonatal period, the cerebral ischemia did not develop in this patients. In the patients of the 2-nd and 3-rd groups were different changes of ROS-scavenger enzymes activity at high MDA level. In 2-nd group on a background of sufficient catalase activity, the SOD activity was markedly reduced. In the 3-rd group of patients on a background of normal catalase activity rather low SOD activity was determined. At newborn of the 2-nd and 3-rd groups, the processes, occurring at a cellular level and which were established with the help of definition of oxidative stress intensity, were shown by a few clinical symptoms. So, at all children it was marked tightened current (about 8-10 days) of transitory conditions. Besides, at the majority newborn from 2-nd and 3-rd groups, in the first 7-10 days of life there were noted a CNS defeat with syndromes of hyper excitability or oppression; that characterizes change of phases of cerebral dysfunction and is treated as an attribute of a cerebral ischemia of II degree. Pairs of the 4 groups had a significant depression of the SOD and catalase activities, and strongly increased the MDA level. In this group progressing loss of cerebral activity was observed, cerebral hypertensia and failures of disease were registered.

According to results of clinical observation and data of neurosonography for newborns with attributes of a cerebral ischemia both studying clinical and biochemical parameters in blood at their mothers and in the umbilical blood the scale of a sub-optimality of the
prenatal risk for adverse factors is made. In it degrees of risk of development HIE (Table 3) are offered.

Conclusion

There are an increase of catalase activity and reduction of SOD activity both in the blood of mother and in umbilical cord’s blood are revealed in conditions of chronic hypoxia caused by an anemia of mothers.

There is an oppression of SOD activity and normal parameters of catalase at acute anoxia in the umbilical cord blood, that probably due to the short duration of hypoxia and the partial compensation of the oxygen radical absorbance capacity, which are observed at short action of the oxygen deficiency and a short phase of reoxygenation.

Expressiveness of oxidizing stress and generation of ROS at women with an anemia and FPI before sorts and in the umbilical cord blood has an identical set.

It is possible to predict risk of HIE according to data of the SOD and catalase activities, MDA level both in the umbilical cord blood and in the mothers blood.

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