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Study of the relationship between hemostasis and vascular endothelium in hypoxic lesions of the nervous system in newborns

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Resume. The study of the statement of the hemostatic system and vascular endothelium was made in newborns with hypoxic lesions of the nervous system. Some indicators of hemostasis (PTT, APTT, TT and fibrinogen) were determined in cord blood, as well as endothelial dysfunction marker Endothelin-1. In newborns who underwent a chronic hypoxia, a statistically significant increase of the fibrinogen level and endothelin-1 were revealed in the umbilical cord blood. During hypoxic lesions of the nervous system in newborns the vascular endothelium primarily reacts, causing activation of hemostasis and cerebral blood flow disorders.

Key words: cerebral hypoxia, hemostasis, vascular endothelium, endothelin-1, asphyxia, fibrinogen.

The purpose of the study. Study of the state of hemostasis system and vascular endothelium in hypoxic damage of the nervous system in newborns.

Materials and methods.
37 newborns with hypoxic damage of the nervous system were observed. A blood sample of 5.0 ml was taken from the umbilical cord immediately after birth to study the indicators.
Group I consisted of 12 healthy newborns born to healthy mothers aged 21 to 33 years, who did not have a difficult obstetric anamnesis with a physiological course of pregnancy and childbirth. 8 of them were born at term and 4 were born "conditionally healthy" prematurely. "Conditionally healthy" preterm group includes children with a gestational age of 35 to 37 weeks and a body weight of 1500 to 2500 grams.
The second group consisted of 12 babies born to healthy mothers who had acute asphyxia during childbirth.
The third group consisted of 13 newborns with chronic intrauterine hypoxia.
Causes of chronic fetal hypoxia:
1) severe anemia (hemoglobin-70g/l and below) in 6 mothers;
2) 4 babies born to mothers suffering from chronic pyelonephritis, severe preeclampsia.
3) severe anemia, high blood pressure and edema in one of the mothers.
4) In 2 mothers, the pregnancy continued against the background of vomiting of pregnant women and the threat of termination of pregnancy.
Laboratory tests:
1. Coagulogram prothrombin time (PTT), prothrombin index (PTI), international normalized ratio (INR), activated partial thromboplastin time (APTT), fibrinogen and thrombin time (TV) were determined on the device "Human clot junior" (2000).
2. Endothelin-1, a marker of endothelial dysfunction, was detected in the blood using the Mindray MR-96A apparatus using the immune enzyme method.
Statistical processing of the received data was carried out using Statistica 10.0 and Microsoft Excel 2017 special programs.

Results and discussion. When hemostasis parameters PTT, INR, APTT and TV were examined in acute and chronic hypoxia of the newborn, their changes did not have a statistically significant difference. Thus, PTT in acute asphyxia was 13.11 ± 0.74 sec, in chronic hypoxia 12.75 ± 0.82 sec, prothrombin index was 105.50 ± 5.85 sec in acute hypoxia and 110.15 ± 1 in chronic hypoxia, respectively. A significant decrease in INR and APTT indicators was observed in newborn sick babies compared to healthy babies. However, a significant decrease in TV was observed only in acute hypoxia and averaged 35.60±3.92 seconds. It should be noted that there was no statistically significant difference between PTT, TT, PTI, INR and APTT in sick and healthy

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newborns. However, among hemostasis indicators, only fibrinogen level between chronic intrauterine hypoxia and healthy newborns was 3.96 ± 0.58 g / l had a statistically significant difference (p1<0.01), but in acute asphyxia and chronic hypoxia there was no statistically significant difference p2>0.2.

Blood coagulation system and vascular endothelium indicators in newborns on the 1st day of life (M ± m)

Table 1

№	Indicators	Healthy babies born group	A group of infants born with acute hypoxia	Group of babies born with chronic hypoxia
		M±m	M±m	M±m
1	PTV(sek)	14,14±1,02	13,11±0,74 p>0.5	12,75±0,82 p1>0.2; p2>0.5
2	PTI(%)	93,43±6,91	105,50±5,85 p>0.2	110,15±6,03 p1>0.1; p2>0.5
3	INR	1,35±0,16	1,09±0,06 p>0.1	1,06±0,08 p1>0.1; p2>0.5
4	ARTT(sek)	39,01±4,80	33,83±2,39 p>0.5	33,34±1,38 p1>0.2;p2>0.5
5	TV(sek)	46,43±8,52	35,60±3,92 p>0.2	48,15±6,59 p1>0.5;p2>0.1
6	Fib(g/l)	2,11±0,42	2,99±0,65 p>0.2	3,96±0,58 p1<0.01;p2>0.2
7	Endothelin -1 (pg/ml)	0,04±0,001	1,24±0,36p<0.001	1,06±0,24 p1<0.001;p2<0.5

Note: p - is the reliability of the difference in scores between healthy newborns and newborns born with acute asphyxia.

P1 - the reliability of the differences in indicators of healthy newborns and babies born with chronic intrauterine hypoxia.

P2 - the reliability of differences in indicators of babies born with acute and chronic intrauterine hypoxia.

When examining the state of vascular endothelium, a statistically significant (p<0.001) increase in endothelin-1, a marker of endothelial dysfunction, was observed up to 1.24 ± 0.36 pg/ml in acute hypoxia and 1.06 ± 0.24 pg/ml in chronic hypoxia.

Summary. Thus, as a result of the research, it was found that in the case of hypoxic damage of the nervous system in newborns, vascular endothelium is primarily affected, leading to activation of hemostasis and hemodynamic changes in cerebral blood vessels.

Conclusion: When the state of the hemostasis system and vascular endothelium was studied, a statistically significant increase in the level of endothelin-1 and fibrinogen in the umbilical cord blood of newborns exposed to chronic hypoxia was found. In the case of hypoxic damage to the nervous system of newborns, the vascular endothelium is primarily affected, which leads to the activation of hemostasis. and causes hemodynamic changes in cerebral blood vessels.

Enter. 60-80% of central nervous system (CNS) diseases in children are associated with perinatal hypoxia [8]. According to the information obtained from the literature, among children recognized as disabled for the first time, about half are patients with cerebral palsy, the origin of this disease is connected with perinatal hypoxia [6].

More than half of all cases of CNS damage in children are not manifested by acute hypoxia of the fetus and newborn, but by chronic hypoxia [2]. Cerebrovascular pathology takes the leading place in perinatal damage of the nervous system in babies [3,4,5]. Thus, acute asphyxia mainly causes focal damage in the form of

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thrombosis, which leads to the development of limited necrosis, and chronic hypoxia causes diffuse changes in nervous tissue [1,9].

ET-1 secretion is activated under the influence of hypoxia [6,7,10]. The main mechanism of action of ET is the activation of calcium release, which leads to:

1. Enhance platelet adhesion and aggregation and secondary hemostasis;
2. Contraction and growth of vascular smooth muscles leads to thickening of the vascular wall and vasoconstriction [5,6,7,10].

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