LEAD TISSUE CONCENTRATION IN NEW BORN RATS DURING HYPOXIC LESSION

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Hypoxic-ischemic lesionsis one of importantproblems of neonatology, which is determined by theirplacein the structure ofmorbidity, perinatalmortality anda valuein the disordersformation. Microelements provide course of important biologicalreactions and are catalysts of manyof them.Micronutrient disbalanceisone of themechanisms ofdamage ofmembranes. The roleof microelements andespeciallyultramicroelementsin metabolicadaptation ofnewbornson the backgroundof hypoxiais staying unknown. Providing vital organs, such as cerebrum, heart, liver andkidneys, with microelements and ultramicroelements in the caseof hypoxia is uninvestigated too. Herewith,the roleof toxicmicroelements,namelylead, is not determined.

The present objective was to research toxiclead dynamics in vital organstissues (brain, heart, liver, kidneys) of newborn rats in the case of experimentalhypoxiaof various severity degrees.

All rats were randomly divided in two groups. The first group (12 rats) was control. Hipobaric model of hypoxia was used in second experimental group (48 rats). Moderate hypoxia was achieved by the putting rates into hermetic chamber for 2 hours, where the air pressure of 525 millimeters of mercury column was created, which is corresponding to the partial pressure of oxygen of 110 millimeters of mercury column. The absorption ofcarbon dioxidein the chamber was exercised by soda lime. Hypoxia of the severe degree was achieved by the keeping animals for 2 hours in the chamber, where the air pressure was 380 millimeters of mercury column, which is corresponding to the partial pressure of oxygen of 80 millimeters of mercury column.

The animals were taken out of the experiment after 12 hours and on the 7^{th} day. Brain, heart, liver and kidneys were removed aseptically and weighed. Organs were dried for 36 h and digested with a mixed solution of hydrochloric and nitric acids (1:3, vol/vol) and slowly heated to 100^0 C until the digestion was complete and added by 10ml by distilleted watter.Brain, heart, liver and kidneys were removed and measure of Pb in this organs.

The content oftoxicleadis the largestin brainand almosttwiceincreases in liver, heart andkidneys.The levelof leadis stableduring the firstweek of lifein liverandheart, butits content inkidneysincreasesin three times as much as decrease in brain tissues of rats. Effect ofmoderatehypoxialeadsto increase ofaccumulationof leadin heartand kidneys– in 8 times, in liver– in 3 timesandin brain(28.2%, $p < 0.05$). In case of severe hypoxia we can see a decreaseof lead contentin liver, but in other organsits content ismuchhigher thanresults of control groupandanimals,affected bymoderatehypoxia. Accumulationof leadis accompanied byformationof correlations of medium strengthin kidneysandliver($r = 0,43$)under conditionsof moderatehypoxiaandthe strongconnection- in case ofseverehypoxicinjury($r = 0,76$).A significantpowerrelationshipis formedabout theelementcontentin heartand kidneysbothin the caseof light($r = 0,92$),andsevere($r = 0,81$)hypoxia. Two-factor analysis of variance of influence of controlled factors on the content of microelements shows significant influence of age and hypoxia. The age factor has predominant influence in brain tissues, heart and kidneys. In liver lead content is equal dependent on age and degree of hypoxic damage.