THE BLOOD LACTIC ACID AND PROGNOSIS FOR RESUSCITATION AFTER EXPERIMENTAL CIRCULATORY ARREST

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Circulatory arrest lasting 10 or 15 min was induced by electric shock in experiments on dogs and the concentrations of lactate and pyruvate, the ratio between them, the lactate dehydrogenase activity, and the pH of the arterial blood and the lactate concentration in the CSF were studied in the postresuscitation period (for 9 h). The maximal concentrations of lactate and pyruvate in the arterial blood at the beginning of the recovery period (after 5 min of resuscitation) were the same in both groups of experiments. Differences between experiments with circulatory arrest of different duration began to appear only 6 h after the beginning of resuscitation. The most characteristic index of the severity of the hypoxia is the lactate level in the CSF.

It is considered by some authorities that the chance of survival after shock of varied etiology is directly dependent upon the production of lactate, the end product of anaerobic metabolism [6, 10, 11].

The dynamics of the concentrations of lactate and pyruvate in the arterial blood and the lactate dehydrogenase (LDH) activity was studied in the recovery period after saturatory arrest of two different durations in order to examine their value as indices of the severity of the hypoxia and the irreversibility of the changes which arise.

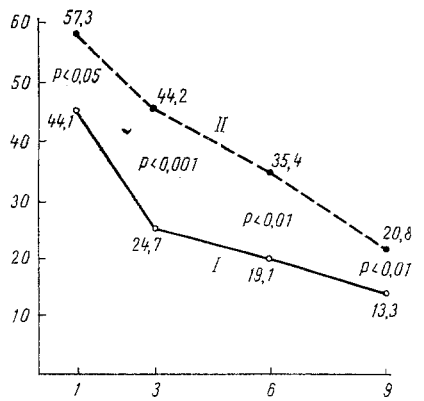

Fig. 1. Dynamics of lactate concentration in CSF in postresuscitation period after clinical death lasting 10 (I) and 15 (II) min. Abscissa, hours of postresuscitation period; ordinate, lactate concentration (in mg/100 ml).

EXPERIMENTAL METHOD

Sixteen experiments were carried out on dogs with circulatory arrest induced by electric shock. Resuscitation was carried out by the method of Nevogskii et al. [1]. In group 1 (circulatory arrest for 10 min) complete restoration of the functions of the CNS, to judge from the external appearance and behavior of the dogs, occurred in 9 of the 10 animals, while in group 2 (circulatory arrest for 15-17 min) it occurred in 2 of the 6 animals; 3 dogs died during the 5 days after the experiment, and in one animal the functions of the higher levels of the brain were not restored.

The following parameters were studied: pH of the arterial blood, lactate concentration in the arterial blood, and pyruvate concentration in the arterial blood (by enzymic methods), and the blood LDH activity [12]; the lactate/pyruvate ratio (L/P) was calculated.

EXPERIMENTAL RESULTS AND DISCUSSION

As Table 1 shows, the maximal increase in the concentrations of lactate and pyruvate in the arterial blood was observed with the

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TABLE 1. Changes in Concentrations of Lactate (L) and Pyruvate (P), L/P Ratio, and LDH Activity in Arterial Blood in Postresuscitation Period (M ± m)

Index studied	Before circulatory arrest		Postresuscitation period									
			5 min		1 min		3 min		6 min		9 min	
	group 1	group 2	group 1	group 2	group 1	group 2	group 1	group 2	group 1	group 2	group 1	group 2
Lactate (in mg %)	13,0±1,9	15,6±1,8	75,0±8,1*	72,1±9,0*	39,0±5,9*	41,8±4,7*	18,8±2,7*	18,7±1,8*	11,5±2,3	16,5±1,7	8,1±1,8*	12,0±1,9*
Pyruvate (in mg %)	0,99±0,17	1,09±0,20	1,53±0,32*	1,70±0,37*	1,59±0,27*	1,59±0,30*	0,93±0,13	1,01±0,11	0,63±0,12	0,64±0,13	0,47±0,11	0,49±0,10*
L/P ratio	13,6±1,8	15,1±2,0	49,7±5,3*	41,6±5,8*	24,5±3,4*	26,3±3,6*	20,2±2,3	18,5±3,0	18,2±2,4	25,7±2,9*	17,2±1,9	24,4±2,2
LDH activity (in iu/ml)	56±6,3		158±12,1*		195±16,8*		187±20,1*		165±13,3*		98±12,5*	

* Indices differ significantly from initial values ($P \leq 0.05$).

resumption of the spontaneous circulation accompanied by a decrease in pH below 7.0. Lactate production rose sharply over pyruvate production, as shown by an increase in the L/P ratio.

Subsequently, all the parameters fell in both groups of experiments. However, whereas the pyruvate level returned to its initial value towards the end of the third hour of resuscitation (simultaneously with restoration of the normal pH), the normal lactate level was restored only after 6 h. The concentrations of lactate and pyruvate 9 h after the beginning of resuscitation were below their initial level ($P < 0.01$) although the L/P ratio remained increased, reflecting a disturbance of oxidation in the tissues.

The maximal increase in LDH activity was somewhat delayed compared with the maximal increase in substrate concentration: it reached its highest values by the end of the first hour of resuscitation and then remained for 6 h at a level 3-4 times higher than initially. Even after 9 h, LDH activity was still 1.5-2 times above its initial level.

It is stated in the literature [2, 4, 7, 8] that the maximal lactate concentration or the maximal L/P ratio and "lactate excess" can be used as prognostic signs in shock of varied etiology. In the present experiments no difference was found between the maximal values of these parameters after circulatory arrest for different periods. In both groups of experiments the maximal lactate concentration in the arterial blood was close to the value regarded as an indication of irreversibility of the disturbances [3]. Correlation between the degree of anaerobiosis and the chance of survival evidently occurs in hemorrhagic or other types of shock in which, despite the oxygen lack, the supply of oxidation substrates to the tissues is not interrupted. Under these conditions the maximal increase in lactate concentrations may reflect the depth of the disturbance of carbohydrate metabolism. After circulatory arrest, the carbohydrate reserves are evidently completely exhausted by the 10th minute.

Differences between the first and second groups of experiments began to appear only 6 h after the beginning of resuscitation: the lactate level after circulatory arrest for 10 min was significantly lower at this time than after circulatory arrest for 15 min ($P < 0.05$). It is thus clear that it was not the maximal lactate concentration, but its dynamics which is of prognostic value in resuscitation after circulatory arrest. No significant differences were found between the other parameters investigated during hypoxia of different degrees of severity, in agreement with the observations of Weil and Affi [13].

The composition of the CSF reflects the state of brain metabolism [5, 9], and the lactate concentration in the CSF is a sensitive indicator of cerebral hypoxia. It will be clear from Fig. 1 that the general direction of the changes in the lactate concentration in the CSF coincides with those in the arterial blood, but the lactate level in the CSF was higher than in the blood starting from the first hour of resuscitation. The more severe hypoxia in the experiments of group 2 led to maintenance of a higher lactate concentration in the CSF. This difference remained sigficant during 9 h of the resuscitation period.

The severity of the hypoxia following circulatory arrest can thus be judged from the lactate concentration in the arterial blood in the late period of resuscitation, but the lactate level in the CSF is the most characteristic index.

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