ACID–BASE BALANCE OF THE CEREBROSPINAL FLUID IN THE POSTRESUSCITATION PERIOD

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In experiments on dogs whose circulation was stopped for 10 min by electric shock the acid–base balance of the cerebrospinal fluid (CSF) and blood was studied in the postresuscitation period. Although uncompensated systemic acidosis continued for 1 h of the postresuscitation period, acidosis of the CSF was compensated much sooner and was maintained for 6 h at the initial level. Despite the high lactate concentration for 3 h of the postresuscitation period, the bicarbonate concentration during this period remained close to its initial value.

KEY WORDS: *Acid–base balance; lactacidosis; postresuscitation period.*

The study of the pH of the cerebrospinal fluid (CSF) during disturbances of the acid–base balance of the blood has shown it to be relatively independent and stable [3, 7]. Only a few observations relate to the study of acid–base balance of the CSF at a time of complete circulatory arrest, when anoxia leads to the development of acidosis in the organism as a whole, including in the CNS [4, 12].

The object of this investigation was to study the indices of acid–base balance (pH, PCO_2, HCO_3^-, and lactate) of the CSF and to compare it with indices of the acid–base balance of the arterial blood in the postresuscitation period after experimental circulatory arrest for 10 min.

EXPERIMENTAL METHOD

Experiments were carried out on dogs weighing 10-18 kg. Omnopon (6-8 mg/kg) was given for premedication. The blood vessels were dissected under local anesthesia combined with superficial pentobarbital anesthesia (4-5 mg/kg). The circulation was stopped by electric shock for 10 min. The animals were resuscitated by Negovskii's method [5]. Complete restoration of CNS function, to judge from external appearance and behavior, occurred in all ten resuscitated animals.

Indices of the acid–base balance in the blood and CSF were determined with the micro-Astrup apparatus (Radiometer, Danmark). The applicability of this method for CSF investigation has been demonstrated by Lee et al. [8]. For calculation, $pK_1' = 6.13$ and $S = 0.0318$ are taken [6]. Samples of CSF were taken from the cisterna magna. Parameters of the acid–base balance of the CSF and blood were determined in the first case (CSF) immediately after taking the sample, and in the second (blood) in the course of 15 min. Lactate was determined by an enzymic method.

EXPERIMENTAL RESULTS AND DISCUSSION

The results in Table 1 show that in the initial state the ratio between the indices of the acid–base balance of the blood and CSF were rather different from those given by other workers. Indices of the acid–base balance of the CSF obtained in the initial state (premedicated and anesthetized animals) agreed with the figures given by other workers [14]. The CSF is known to have a more acid reaction than the blood. Plum and Siesjo [10], after

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TABLE 1. Indices of Acid–Base Balance of CSF and Blood during Circulatory Arrest and in Postresuscitation Period

Test object	Index studied	Initial data	End of clinical death	Resuscitation period			
				7–10 min	1 h	3 h	6 h
CSF	pH	7,28±0,01	7,04±0,03*	7,24±0,01*	7,33±0,02	7,33±0,02	7,32±0,02
	PCO_2, mm Hg	51,3±3,9	109±8,1*	46,0±3,0	39,5±1,1*	44,6±2,5	36,6±6,2*
	HCO_3^-, meq/liter	22,6±1,6	27,6±1,9*	19,1±1,2	19,1±1,0	21,8±1,6	21,9±1,2
	Lactate, meq/liter	2,17±0,2	—	5,60±0,5	4,90±0,3	2,78±0,2	2,12±0,1
Blood	pH	7,25±0,01	7,18±0,01*	7,10±0,02*	7,24±0,01	7,29±0,01*	7,32±0,01*
	PCO_2, mm Hg	50,2±3,1	62,0±3,9	46,7±4,7	41,9±4,3	39,2±2,0*	35,3±2,1*
	HCO_3^-, meq/liter	20,6±0,9	23,1±0,7	12,4±0,8*	16,5±0,7*	18,4±0,8	17,6±1,1
	Lactate, meq/liter	1,44±0,2	—	8,33±0,6	4,33±0,4	2,09±0,2	1,29±0,2

*Differences from initial data significant ($P < 0.05$).

TABLE 2. Indices of Acid–Base Balance of Arterial Blood with Different Types of Anesthesia

Index	Premedication + anesthesia	Anesthesia without premedication	P
pH	7,25±0,01	7,39±0,01	<0,001
PCO_2, mm Hg	50,20±2,0	41,8±1,6	<0,001
HCO_3^- meq/liter	20,6±0,9	25,2±1,3	>0,05
Base excess (BE), meq/liter	—6,8±1,02	—1,0±0,5	<0,001

analyzing published data and their own observations, showed that the differences between indices for the arterial blood and CSF vary in the case of pH from −0.072 to −0.121, for PCO_2 from +4.8 to +10.7 mm Hg, and for HCO_3^- from −3.2 to +0.6 meq/liter. A somewhat different relationship was obtained in the present experiments on account of the arterial blood values. Wise [15], for instance, gives the following values for unanesthetized dogs with an implanted catheter: pH of the arterial blood 7.453 ± 0.007, PCO_2 35.9 ± 0.7 mm Hg, HCO_3^- 24.5 ± 0.5 meq/liter, and lactate 10.0 ± 0.4 mg %. Similar results for arterial blood were obtained by a number of workers during barbiturate anesthesia [14]. A study of the indices of the acid–base balance in five dogs anesthetized with pentobarbital (25 mg/kg, intramuscularly) was undertaken without preliminary omnopon premedication (Table 2). Omnopon premedication leads to the development of respiratory and metabolic acidosis. Similar results were obtained by Boyarinova and Kuznetsova [1]. No significant differences in the indices of the acid–base balance of the CSF could be found, for the acid–base balance of the CSF during systemic disturbances is distinguished by its stability.

Before the beginning of resuscitation and at the end of clinical death an acidotic shift was observed: It was much more marked in the CSF than in mixed venous blood taken from the right heart (Table 1). The same difference also was observed with respect to CO_2 accumulation. Similar data for blood have also been obtained by other workers [4]. Meanwhile the indices of the acid–base balance of the CSF are very close to those for brain tissue during total brain ischemia [11]. During total ischemia there is evidently an exchange of H^+ ions and removal of CO_2 at the brain–extracellular fluid boundary, whereas at the organ (heart)–blood boundary this process takes place much more slowly. This phenomenon is evidently reflected during the first few minutes after restoration of the circulation: Acid radicals which have accumulated in the tissues gradually begin to find their way into the blood stream and the maximal drop of pH takes place during the first few minutes of the recovery period [2], whereas the pH of the CSF is already starting to rise and is approaching its initial level. The blood bicarbonate concentration at this time is sharply reduced, whereas in the CSF it does not differ significantly from the initial value, although it compensates for the sharply increased lactic acid level.

Toward the end of the first hour of the resuscitation period compensation of acidosis was observed in the arterial blood although the blood lactate concentration was still considerably higher and the bicarbonate concentration and PCO_2 lower than initially. During the period from 3 to 6 h the pH of the arterial blood rose significantly higher than initially on account of considerable loss of CO_2, and the concentration of lactate and bicar-

bonate was close to the initial value. In the course of 6 h of the postresuscitation period the pH of the CSF remained close to its initial level although the lactate concentration did not approach its initial level until after 5 h of the postresuscitation period.

Since CSF acidosis after the first few minutes of resuscitation was compensated and the high lactate concentration was combined with normal pH during 3 h of the postresuscitation period, a very cautious attitude must be adopted toward the assessment of acid–base balance from the lactate level, as some workers have done [9]. The PCO_2 of the CSF in the course of 1-6 h of the postresuscitation period was significantly lower than initially, probably on account not only of the reaction to the increased lactate concentration, but also of diffusion of dissolved CO_2 through the blood–brain barrier into the blood stream, where PCO_2 was considerably reduced at this period.

Compensation of acidosis in the CSF after complete circulatory arrest thus takes place much sooner than in the blood, despite the fact that the buffer capacity of the blood is much higher. Despite the high lactate concentration in the CSF for 3 h of the postresuscitation period, the bicarbonate concentration did not differ significantly from its initial value, evidently because of bicarbonate formation in the choroid plexus [13]. Early compensation of acidosis on account of the removal of bicarbonate may perhaps protect the brain from additional damage.

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