EFFECT OF THE METHOD OF RESUSCITATION ON THE OXYGEN BUDGET AND ACID-BASE BALANCE OF THE BLOOD OF ANIMALS AFTER PROLONGED CIRCULATORY ARREST

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Dogs were anesthetized, circulatory arrest was produced for 15 min by electric shock, and the animals were resuscitated by means of the artificial circulation apparatus (ACA) or by external cardiac massage. A comparative study was made of the acid-base balance and gas composition of the blood, the oxygen demand, and the cardiac output of the two groups of dogs during the period of 3 h after resuscitation. A higher rate of survival and less severe disturbance of the acid-base balance were found after resuscitation with the ACA. Irrespective of the method used, 2-3 h after resuscitation the minute volume of the heart was considerably lower than in the early recovery period or in the initial state.

Various types of extracorporeal circulation have been used sussessfully for experimental resuscitation [1, 2, 7, 9, 10].

To compare the effectiveness of this method of resuscitation the acid-base balance and gas composition of the blood, certain indices of the gas exchange and hemodynamics were studied in animals resuscitated by means of the "heart-lung" apparatus or by external cardiac massage.

EXPERIMENTAL METHOD

Sixteen dogs were anesthetized (4-8 mg/kg pantopon, 10 mg/kg pentobarbital). Circulatory arrest for 15 min was induced by electric shock leading to fibrillation of the heart. The animals were revived by means of the AIK-RP-64 artificial circulation apparatus, filled with fresh donor's blood (group 1) or by external cardiac massage [5] in conjunction with artificial ventilation of the lungs (group 2). The duration of the artificial circulation in group 1 was 27.6 ± 2.0 min. Cardiac activity was restored by means of the pulse discharge of a defibrillator: in group 1 after resuscitation for 7.67 ± 0.83 min, in group 2 after resuscitation for 4.42 ± 0.36 min. The pH, O_2 saturation, O_2 and CO_2 concentrations by Van Slike's method, were determined in the arterial and mixed venous blood, while the total organic acids were determined in arterial blood and the values of pCO_2, actual bicarbonate, and base deficit were calculated. The expired air was analyzed on a gas chromatograph, and the O_2 consumption and CO_2 excretion in the animal's lungs were determined [6]. The minute volume of the heart was calculated by Fick's equation.

EXPERIMENTAL RESULTS AND DISCUSSION

Complete recovery of the vital functions on external inspection occurred in five of the nine animals of group 1 but only in one of the seven dogs of group 2. The time taken for recovery of respiration and the corneal reflexes was the same in the dogs of both groups.

At the beginning of the recovery period a severe and uncompensated metabolic acidosis was observed in all the animals, but it was more severe in group 2 throughout the period of investigation. Results showing

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TABLE 1. Indices (M ± m) of Acid-Base Balance and Gas Composition of Blood, Oxygen Demand, and Cardiac Output in Animals Resuscitated with the Artificial Circulation Apparatus (1) or by External Cardiac Massage (2)

Index	Initial state (anesthetized)		Recovery period after resuscitation							
			5–10 min		40–60 min		120 min		180 min	
	1	2	1	2	1	2	1	2	1	2
Organic acids (in meq/liter) . . .	11,0±1,0	12,0±1,2	21,2±1,5*	35,4±2,1*	19,7±1,7*	29,3±2,6*	16,4±1,5*	24,6±2,4*	15,6±1,6*	20,0±1,1*
P	>0,1		<0,001		<0,001		<0,01		<0,05	
PCO_2 (in mm Hg), arterial	41,5±2,3	43,0±2,3	53,2±4,6*	43,5±4,5	30,3±2,1*	31,2±2,5*	28,9±1,1*	29,4±2,2*	22,8±2,0*	30,0±2,1*
P	>0,1		>0,1		>0,1		>0,1		0,02	
pH, arterial	7,26±0,02	7,20±0,01	7,08±0,03*	6,94±0,02*	7,23±0,02	7,18±0,02	7,29±0,01*	7,25±0,01*	7,36±0,02*	7,25±0,02*
P	<0,02		<0,002		≥0,05		<0,02		<0,002	
$Hb O_2$ (in %), venous	70±2,4	73±1,7	—	72±5,0	76±1,9*	66±4,8	53±1,7*	60±4,1*	45±1,2*	49±5,2*
P	>0,1				≥0,05		>0,1		>0,1	
O_2 demand (in ml/kg/min)	6,5±0,4	5,4±0,4	—	—	6,3±0,7	6,4±0,4	6,5±0,9	6,6±0,8	7,0±0,9	6,3±0,6
P	>0,1				>0,1		>0,1		>0,1	
A-VO_2 (in vols.%) .	4,6±0,2	4,8±0,2	—	5,5±0,8	3,5±0,4*	5,7±0,6	9,4±0,2*	7,2±0,6*	11,9±0,5*	8,7±0,9*
P	>0,1				<0,01		<0,05		<0,01	
Minute volume of heart (in ml/kg/min) . . .	154,4±20,6	108,5±7,1	—	—	168,1±22,8	121,0±15,0	69,0±11,2*	93,6±11,8	59,9±11,5*	75,5±3,4*
P	>0,05				=0,1		>0,1		>0,1	

*Indices differing significantly from original ($P < 0.05$).

the increase in the concentration of organic acids and the pH shift toward acidosis are given in Table 1. The base deficit at the beginning of resuscitation was increased in group 1 to -16.6 ± 0.9 meq/liter and in group 2 to -26.2 ± 0.7 meq/liter, while the bicarbonate concentration was reduced to 15.5 ± 0.6 and 9.1 ± 0.6 mmoles/liter, respectively ($P < 0.001$).

During the next few hours of the recovery period the metabolic components of the acid-base balance showed a tendency to return to normal, but in both groups they were significantly above their initial values after 3 h.

The much smaller metabolic changes, especially in the early stages of resuscitation, after application of the ACA are evidently the result of the partial blood replacement taking place through the use of fresh donor's blood for resuscitation. The higher volume velocity of the blood flow in the body created by the ACA within a few minutes of the beginning of resuscitation than during external cardiac massage also contributed to the better supply of oxygenated blood to the tissues and the effective removal of toxic metabolic products, thus leading to quicker recovery of oxidative processes.

Investigation of the respiratory function of the blood showed that during the artificial circulation (group 1), just as during application of artificial respiration (group 2), the elimination of CO_2 was adequate and the degree of O_2 saturation of the arterial blood ($92 \pm 0.9\%$) was satisfactory, and this state of affairs continued during the period of spontaneous cardiac activity and respiration. The dynamics of the CO_2 concentration and pCO_2 in the blood of the animals of both groups reflects the gradual development of gaseous alkalosis in the recovery periods, to a more severe degree in group 1.

The percentage of oxyhemoglobin in the venous blood was considerably reduced 2-3 h after resuscitation in all the animals, and the arterio–venous oxygen difference was increased (Table 1). The reason was evidently not an increase in the oxygen demand of the tissues, but a circulatory effect: slowing of the volume velocity of the blood flow. An increase in the cardiac output was found at the beginning of the recovery period, and it decreased progressively after the first hour of resuscitation regardless of its outcome. The arterial pressure level did not change significantly, and it differed only slightly from initially, indicating an increase in the total peripheral vascular resistance. The decrease in the minute volume of the heart in the period following resuscitation after clinical death from blood loss has been reported previously [4, 8]. Its principal cause is evidently hypoxic damage to the myocardium, inhibition of its functions by the action of toxic products, and the state of the circulation in the vascular system after hypoxia [3, 11, 12].

The role of complications associated with the particular methods of resuscitation used likewise cannot be ruled out.

The use of the "heart-lung" apparatus for resuscitation thus partly abolished or neutralized the metabolic disturbances in the early stages of resuscitation and improved its final results. Irrespective of the method of resuscitation, a marked decrease in the cardiac output was observed 2-3 h after resuscitation.

LITERATURE CITED

1. A. A. Bozh'ev, Use of the "Heart-lung" Apparatus for Resuscitation after Sudden Death Caused by Ventricular Fibrillation, Author's Abstract of Candidate's Dissertation, Moscow (1970).
2. S. S. Bryukhonenko, The Artificial Circulation [in Russian], Moscow (1964).
3. A. C. Guyton, Circulatory Physiology: Cardiac Output and Its Regulation, Philadelphia and London (1963).
4. A. Ya. Evtushenko and S. Ya. Evtushenko, Pat. Fiziol., No. 3, 65 (1971).
5. V. A. Negovskii, Indirect Cardiac Massage and Expiratory Artificial Respiration [in Russian], Moscow (1966).
6. É. M. Nikolaenko, in: Current Problems in the Biochemistry of Respiration and Its Clinical Features [in Russian], Ivanovo (1970), p. 413.
7. V. I. Soboleva, S. V. Tolova, N. L. Gurvich, et al., Pat. Fiziol., No. 5, 24 (1970).
8. I. E. Trubina, Pat. Fiziol., No. 3, 57 (1971).
9. V. D. Yankovskii, A. P. Kovtun, and A. P. Morozov, in: Proceedings of a Symposium on the Artificial Circulation [in Russian], Leningrad (1964), p. 101.
10. V. A. Chernyak, The Study of the Effectiveness of the Artificial Circulation for Resuscitation after Prolonged Clinical Death, Author's Abstract of Candidate's Dissertation, Moscow (1968).
11. G. H. A. Clowes, Jr., G. A. Sabga, A. Konitaxis, et al., Ann. Surg., 154, 524 (1961).
12. T. M. Glenn, A. M. Lefer, I. B. Martin, et al., Am. Heart J., 82, 78 (1971).