CIRCULATORY DISTURBANCES IN THE POSTRESUSCITATION PERIOD OF MYOCARDIAL INFARCTION AND THE ROLE OF BODY FLUID VOLUME CHANGES IN THEIR DEVELOPMENT

G. V. Lisachenko and N. A. Ivanova

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Resuscitation measures in myocardial infarction are not very effective and there is a wide gap between the initial and end results [7, 10-12]. Hence the need for research into the fucntional state of the circulatory system, which largely determines the course and outcome of the resuscitation process [6].

The aim of this investigation was to study the principles governing disturbances of the hemodynamics and to discover the role of the movement of water among the body fluid compartments in their development.

EXPERIMENTAL METHOD

The investigation was conducted on two groups of mongrel dogs (altogether 43) of both sexes under pentobarbital anesthesia (40 mg/kg). In the animals of experimental group 1, a model of clinical death was produced against the background of acute myocardial infarction of coronary genesis, produced under closed chest conditions by the sliding ligature method [8]. Fibrillatory cardiac arrest was induced 1 h after tightening of the ligature by electrical stimulation (30-50 V, 3 sec). In the animals of group 2 (control) clinical death (fibrillation) was induced in animals with an intact myocardium. The dogs were resuscitated 5 min after cardiac arrest by closed cardiac massage, defibrillation, and artificial respiration with moderate hyperventilation. The cardiac output (CO, by the thermodilution method), ECT, systemic BP, central venous pressure (CVP), and pressure in the left ventricle with its first derivative (Mingograf-34, Seimens-Elema), were recorded. The systolic volume (SV) and total peripheral resistance (TPR) and the circulating erythrocyte volume (CEV) were calculated. The water compartments were studied by the one-stage indicator dilution method. The total water content (TW) was studied by the urea dilution method [5], the extracellular fluid volume (EFV) by the thiocyanate method [1], and the circulating plasma volume (CPV) with the aid of albumin-^{137}I. The intracellular water content (ICW), interstitial fluid volume (IFV), and the circulating blood volume (CBV) were calculated.

EXPERIMENTAL RESULTS

Changes in the systemic hemodynamics in the postresuscitation period in the experimental and control groups were similar in character (Table 1). During the first minute of the postresuscitation period the cardiac output increased, but thereafter it progressively decreased. In the presence of myocardial infarction, hyperperfusion was less marked and was shorter in duration, and the subsequent inhibition of the circulation was greater.

Two stages can be distinguished in the development of hypoperfusion. Initially (3-15 min) reduction of cardiac output was due mainly to disturbance of myocardial contractility. This is shown by the sharp fall in the maximal velocity of contraction and relaxation, of the systolic pressure in the left ventricle, and of Veragut's index, accompanied by a more than twofold increase in the end-diastolic pressure (EDP), and all of TPR (Table 1). Depression of myocardial contractility was probably connected with the effect of toxic substances [2], released with the lymph from tissues with hypoxic damage [3, 4].

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TABLE 1. Parameters of Systemic Hemodynamics and Myocardial Contractile Function (M ± m) in Dogs during Postresuscitation Period

Parameters	Experimental conditions	Initial value	1 h of occlusion	Postresuscitation period					
				1 min	3 min	15 min	3 h	9 h	24 h
CO, ml/kg	I	168,0±5,6	141,0±5,3*	199,0±11,9*	158,0±15,4	134,0±7,9*	99,0±5,5*	91,0±9,6*	120,0±8,4*
	II	142,0±5,7	—	180,0±11,1*	155,0±15,0	136,0±7,4	101,0±6,8*	102,8±8,1*	141,0±10,4
SV, ml/kg	I	1,02±0,04	0,87±0,05*	1,34±0,09*	1,04±0,09	0,83±0,06*	0,60±0,04*	0,53±0,03*	0,85±0,06*
	II	0,87±0 05	—	1,48±0,15*	1,05±0,09**	0,76±0,06	0,61±0,05*	0,57±0,04*	0,87±0,06
BP, kPa	I	19,8±0,48	18,5±0,56	19,3±1,43	13,2±1,36*	13,6±0,56*	17,0±0,57**	15,2±0,56*	13,1±0,76*
	II	18,2±0,63	—	19,5±1,22	12,8±0,87*	12,8±0,60*	16,2±0,52**	15,5±0,76*	16,0±0,49*
TPR, kPa/sec·liter^{-1}	I	805,0±64,0	878,0±70,0	662,0±79,0**	532,0±59,0*	708,0±82,0**	1225,0±193,0**	1254,0±137,0*	731,0±80,0**
	II	884,0±124,0	—	729,0±99,0**	630,0±106,0**	679,0±94,0**	1118,0±141,0**	986,0±143,0**	794,0±161,0
$P_{syst.l.v.}$, kPa	I	21,6±0,93	19,7±0.86	22,5±2,13	13,4±1,56*	13,9±0,76*	18,1±0,88*	17,8±0,80*	15,9±0,81*
	II	20,8±1,02	—	23,2±2,26	16,6±2,13**	16,4±1,20*	18,7±0,92**	19,7±1,38	17,6±1,65**
$\Delta t/\Delta tmax$, kPa/sec	I	377,0±22,3	340,0±20,7	488,0±43,7**	196,0±26,3*	205,0±15,7*	272,0±28,0*	263,0±39,7*	230,0±24,8*
	II	377,0±13,4	—	460,0±63,4**	241,0±40,8*	230,0±16,1*	305,0±24,5*	350,0±29,0	301,0±43,0**
$\Delta P/\Delta tmin$, kPa/sec	I	274,0±12,8	246,3±13,0	327,0±30,0**	132,0±20,1*	133,0±18,9*	203,0±22,4*	219,0±14,8*	167,1±21,6*
	II	273,0±12,5	—	331,0±41,8**	134,0±23,3*	172,0±18,0*	208,0±30,0**	244,0±28,9	218,0±28,9**
$\Delta P/\Delta tmax/IP \cdot sec^{-1}$	I	35,9±1,82	33,9±2,24	39,0±2,29**	28,1±1,33*	29,0±1,26*	30,9±2,0	33,5±1,56	29,4±1,60*
	II	38,3±1,32	—	42,9±2,30**	32,3±1,54*	32,8±1,73*	34,4±1,19	35,6±1,62	32,7±2,90**
$EDP_{l.v.}$, kPa	I	0,75±0,085	0,75±0,092	2,19±0,085*	2,20±0,086*	1,53±0,090*	0,69±0 090	0,57±0,126	0,77±0,081
	II	0,61±0,080	—	1,97±0,152*	2,13±0,322*	1,86±0,241*	0,45±0,154	0,50±0,053	0,61±0,077

Legend. Difference significant (*p < 0.05) by Student's test and **p < 0.05) by Wilcoxon's test, when compared with initial data, and (***p < 0.05) between groups by Student's test. I) Acute myocardial infarction (n = 31); II) without cardiac damage (n = 12).

TABLE 2. Water Compartments in Postresuscitation Period (M ± m)

Parameter		Experimental conditions	Initial value	Postresuscitation period		
				2,5 h	9 h	24 h
TW	ml/kg	I	600,0±12,9	576,2±12,9**	563,7±22,9**	544,0±18,6**
		II	635,2±18,9	621,7±18,6**	602,9±11,4**	587,5±10,5**
EFV		I	259,2±9,5	212,8±6,4*	225,7±16,7	269,6±17,7
		II	259,8±10,9	214,3±10,5*	225,2±10,0*	253,3±15,8
ICW		I	340,8±12,4	363,4±10,8**	338,0±18,1	274,4±15,7*,***
		II	375,4±17,7	407,4±16,0**	377,7±13,8	334,2±13,7**
IFV		I	214,2±9,4	177,3±6,4*	188,2±16,3	227,1±16,9**
		II	210,5±10,0	175,2±8,2*	188,2±11,5	195,8±17,2
CPV		I	45,0±1,5	35,5±1,5*	37,5±2,5**	42,5±2,5**,***
		II	49,3±1,8	39,1±3,3*	46,8±3,7**	54,7±2,6**
CEV		I	39,4±2,1	36,6±1,6	34,6±2,6	31,6±2,2*,***
		II	41,6±2,8	40,8±3,9	43,4±3,7	41,6±3,9
CBV		I	84,4±3,3	72,1±2,8*	72,1±4,7**,***	74,1±4,2**,***
		II	90,9±4,1	79,9±6,9*	90,2±6,9	96,3±5,3*
Hematocrit, liter/liter		I	0,46±0,01	0,51±0,08*	0,48±0,01	0,42±0,01*
		II	0,45±0,01	0,51±0,01*	0,48±0,01**	0,43±0,02

Legend. Difference significant (*p < 0.05) bu Student's test and **p < 0.05) by Wilcoxon's test, when compared with initial data, and (***p < 0.05) between groups by Student's test. I) Acute myocardial infarction (n = 24); II) without cardiac damage (n = 12).

During the next 3-9 hypoperfusion progressed under conditions of relative recovery of myocardial contractility, evidence of the important contribution of extracardiac factors. The most important of these is hypovolemia, whose development is linked with movement of fluid between the compartments. On the whole, the dynamics of the movement of fluid among the sectors in the two groups of experiments was qualitatively similar (Table 2). The fall of TW 2.5 h after resuscitation was accompanied by a decrease in the extracellular and an increase in the intracellular fluid volumes. Reduction of the extracellular space was due to reduction of both the interstitial and the intravascular volumes. The cell volume under these circumstances was virtually unchanged. Toward 9 h of the postresuscitation period movement of water in the reverse direction began to take place for the intracellular to the extracellular space. The intensity of this process did not differ significantly in groups 1 and 2.

Improvement of the volume perfusion parameters 24 h after resuscitation took place in spite of worsening of myocardial contractility, and it was due mainly to restoration of the intravascular volume. Movement of water continues in both groups in the direction from the cells to the interstitial tissues and to the blood vessels. In animals with myocardial infarction, cell dehydration was more marked and was coupled with retention of water in the interstitial space. Together with reduction of the cell fraction, this prevented the recovery of CBV.

Myocardial infarction greatly aggravated the course of the postresuscitation period and lowered the survival rate of the animals. Of 31 dogs exposed to clinical death under conditions of myocardial infarction only eight survived, significantly fewer ($p_{\chi^2} < 0.05$) than in the control group (in which eight of 12 dogs survived).

Thus in animals resuscitated from clinical death under conditions of a damaged (infarct) and intact myocardium, changes in the circulation and the water compartments of the body were qualitatively similar. The postresuscitation period of myocardial infarction is characterized by more profound circulatory disorders, connected both with disturbance of the contractile function and with the pattern of water movement between the compartments.

LITERATURE CITED

1. E. B. Berkhin and Yu. I. Ivanov, Methods of Experimental Investigation of the Kidneys and of Water and Mineral Metabolism [in Russian], Barnual (1972).
2. A. Ya. Evtushenko and G. V. Lisachenko, Patol. Fiziol., No. 1, 32 (1975).
3. A. Ya. Evtushenko, A. S. Razumov, and N. A. Ivanova, Anest. Reanimatol., No. 1, 50 (1987).
4. M. M. Minnebaev, V. F. Bakhtiozin, and M. S. Musin, Byull. Éksp. Biol. Med., No. 3, 281 (1981).
5. V. I. Moin, Lab. Delo, No. 2, 28 (1980).
6. V. A. Negovskii, A. M. Gurvich, and E. S. Zolotokrylina, Postresuscitation Sickness [in Russian], Moscow (1987).
7. E. I. Chazov, Vest. Akad. Med. Nauk SSSR, No. 10, 14 (1974).
8. Yu. S. Chechulin, Cardiac Damage [in Russian], Moscow (1975).
9. L. G. Shikunova, Current Problems in Resuscitation [in Russian], Moscow (1980), pp. 127-134.
10. R. J. Myerburg, C. A. Conde, R. J. Sung, et al., Am. J. Med., 68, 568 (1980).
11. G. Pagliarello, W. Demajo, M. J. Girotti, et al., Crit. Care Med., 15, 369 (1987).
12. K. von Seggers, M. Egar, and B. P. Fuhrman, Crit. Care Med., 14, 275 (1986).