CORRELATION BETWEEN THE CENTRAL HEMODYNAMICS OF DOGS IN THE EARLY POSTRESUSCITATION PERIOD AND THE OUTCOME OF RESUSCITATION

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Changes in the central hemodynamics were compared with the outcome of resuscitation in 18 dogs after circulatory arrest lasting 12 min caused by ventricular fibrillation. In nine animals resuscitated with evidently complete neurological recovery moderate hypertension was observed in the first 10 min after the beginning of the resuscitation measures: The mean arterial pressure (MAP) was 175.0 ± 8.9 mm Hg. In most of the dogs which died subsequently MAP in this period was not higher than initially, but two animals showed severe hypertension (MAP about 200 mm Hg). In this same period differences also were found in other parameters of the central hemodynamics. Moderate hypertension in the first 10 min of the postresuscitation period evidently leads to rapid recovery of an adequate level of the peripheral blood flow in tissues and organs and, as a result, it aids the survival of animals after a long period of circulatory arrest.

KEY WORDS: fibrillation; postresuscitation period; central hemodynamics; outcome of resuscitation.

Circulatory disturbances play an important role in the genesis of postresuscitation complications leading to death or to irreversible changes in the organs and tissues [1-3]. However, information on the nature of these disturbances and the stages of the recovery period at which they may influence the outcome of resuscitation is contradictory. Considerable importance is attached to the level of the arterial pressure (AP). For example, an increase in AP produced by catecholamines after resuscitation can have a favorable effect on restoration of neurological functions and on survival [4, 7, 10]. The size of the cardiac output also has a definite influence on the course of the postresuscitation period [5]. To study the role of the state of the central hemodynamics for the outcome of resuscitation in the present investigation basic indices of the hemodynamics were compared in the early postresuscitation period in dogs surviving with evidently full neurological recovery after cirulatory arrest lasting 12 min and also in dogs dying during the 1st or 2nd days after resuscitation.

METHODS

Experiments were carried out on 18 anesthetized (2% Omnopon, 6 ml/kg, 1% pentobarbital, 5 ml/kg) dogs weighing 9-21 kg, after circulatory arrest lasting 12 min as a result of ventricular fibrillation induced by electric shock. Resuscitation was carried out by external cardiac massage, intra-arterial injection of 10-20 ml physiological saline with adrenalin (0.1 ml/kg of a 1 : 1000 solution), electrical defibillation of the heart, and artificial ventilation of the lungs with 60% oxygen, with a tidal volume of 30 ml/kg and respiration rate of 16 breaths per minute for 30-40 min. The cardiac output was measured by the thermodilution method. AP was recorded in the aorta and pulmonary artery. The cardiac and stroke indices, the left ventricular stroke work, and the total peripheral resistance were calculated. All indices were measured before electric shock and in the course of 3 h after resuscitation. The surviving dogs were kept under observation for 2 weeks. The results were subjected to statistical analysis by Student's t-test [6].

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TABLE 1. Indices of Central Hemodynamics (M ± m) in Resuscitated Dogs Surviving for a Long Time (group 1) and Dying on the 1st or 2nd Days (group 2) after Circulatory Arrest for 12 min

Time after resuscitation, min	Mean arterial pressure, mm Hg		Pressure in pulmonary artery, mm Hg		Cardiac index, ml/min·kg		Cardiac frequency, beats/min	
	group of animals							
	1	2	1	2	1	2	1	2
Original state	117,9 ±5,7	108,9 ±5,5	12,8 ±1,2	11,4 ±1,2	105,7 ±14,4	102,2 ±10,0	79,2 ±11,7	80,2 ±12,2
10	175,0* ±8,9	106,5* ±8,5	20,3 ±2,0	20,3 ±2,3	126,6 ±39,6	90,6 ±17,0	162,5 ±14,9	190,1 ±11,9
15	120,2 ±11,9	133,5 ±11,4	19,9 ±1,0	19,2 ±1,5	98,0 ±10,8	97,1 ±9,3	164,7 ±13,9	172,8 ±15,7
30	113,9 ±7,6	114,0 ±7,8	16,9 ±1,7	16,0 ±2,2	82,8 ±17,4	108,1 ±11,5	175,6 ±5,4	191,7 ±8,9
45	120,5 ±6,0	116,4 ±6,8	14,0 ±2,8	12,4 ±1,3	84,2† ±10,8	119,8† ±12,0	173,1 ±5,1	187,8 ±8,1
60	110,1 ±3,1	109,0 ±6,1	12,4 ±2,2	11,0 ±0,8	88,8 ±12,1	93,4 ±9,1	168,3 ±4,2	191,8 ±10,1
180	120,4 ±8,9	124,3 ±5,6	8,6 ±1,6	11,6 ±1,1	62,4 ±12,9	58,8 ±8,7	144,6 ±10,8	156,0 ±12,5

*P < 0.001 for comparison of indices in two groups.
†P < 0.05.

RESULTS AND DISCUSSION

Depending on the outcome of the experiments, the animals as a whole were divided into two groups: group 1) nine dogs which survived with evidently full neurological recovery; group 2) nine dogs which died on the 1st or 2nd days after the experiment. The resuscitation measures applied were the same for both groups. The mean AP level (MAP) after external cardiac massage was 77.1 ± 6.7 mm Hg in the dogs of group 1 and 93.4 ± 8.8 mm Hg in the dogs of group 2 (the difference is not statistically significant). The cardiac activity was restored in the dogs of group 1 after 4.4 ± 0.6 min and in the dogs of group 2 after 3.1 ± 0.3 min. Respiration also was restored almost simultaneously in the dogs which survived (3.2 ± 0.2 min) and in those which died (3.8 ± 0.3 min). The corneal reflexes were restored a little sooner in the dogs of group 1 (13.7 ± 0.9 min) than in those of group 2 (after 18.1 ± 2.2 min) but the difference was not statistically significant.

Changes in the basic indices of the central hemodynamics are given in Table 1. Significant differences in the first 10 min of the postresuscitation period were observed in the MAP level. In animals which survived after recovery of their cardiac activity MAP reached 175.0 ± 8.9 mm Hg, whereas in those which subsequently died MAP was 106.5 ± 8.5 mm Hg, i.e., there was no rise of pressure above the initial values. Only in two animals (the results are not given in Table 1) did MAP rise in 196 and 198 mm Hg respectively. After 15 min the values of MAP became equal and throughout the remaining period they were the same as initially in all the dogs. The cardiac index of the dogs of the two groups differed at the beginning of the resuscitation period: In those which survived it was 126.6 ± 39.6 ml/min · kg, whereas in those which subsequently died the output was a little lower, namely 90.6 ± 17.0 ml/min · kg. The cardiac output at this time was maintained at a sufficiently high level in all the animals despite a considerably reduced stroke volume because of a sharp increase in the cardiac frequency; the tachycardia was more marked in the dogs of group 2. Significant differences in the cardiac output between the two groups were found at the 45th minute, i.e., immediately after disconnection of the artificial respiration apparatus. With the ending of artificial ventilation of the lungs the dogs of group 2 responded by an increase in cardiac output, whereas in the dogs of group 1 the output remained practically unchanged compared with its level during artificial respiration (Table 1). By 3 h the cardiac output was reduced to almost half of the initial value of all dogs. The total peripheral resistance and the left ventricular stroke work differed only during the first minutes of the postresuscitation period: The total peripheral resistance was 10% higher in the dogs of group 2 and the work of the left ventricle in the dogs of group 1 was twice that in group 2. Differences in the values of pulmonary arterial pressure were not observed throughout the period of the experiment.

Differences in the central hemodynamic indices in the dogs of the two groups were thus observed immediately after recovery of cardiac activity: In the dogs which survived, the cardiac output and stroke volume and the left ventricular stroke work were rather higher, but the cardiac frequency and the total peripheral vascular resistance were lower. The main differences at this time were concerned with the AP level. In the dogs which survived, moderate hypertension was present, whereas in the animals which died the blood pressure was normal, or in two cases, excessive hypertension was present. A moderately high pressure (about 170 mm Hg) during the first minutes of the postresuscitation period evidently contributes to the rapid attainment of an adequate level of the peripheral circulation in the tissues and organs, including in the brain and, as a result, to more rapid neurological recovery. This suggestion is confirmed by the results of investigations by Hossmann et al. [8, 9], who studied the cerebral blood flow and electrophysiological recovery after compressional cerebral ischemia in rats. An excessively raised pressure may lead to the development of cerebral edema or shunting of the blood flow, resulting in local hypoperfusion of the brain. In the present experiments dogs which had moderate hypertension in the first 10 min of the postresuscitation period survived without any additional treatment after prolonged circulatory arrest. Since in these experiments the dose of catecholamines administered during resuscitation was the same for all animals, it can be tentatively suggested that disturbances of neurohumoral regulation of vascular tone in the first minutes after resuscitation were much more marked in the dogs which subsequently died than in those which survived. Accordingly, in the early stages of the postresuscitation period additional therapeutic measures must be taken with the object of creating an adequate peripheral circulation.

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