mediator. More recently, however, the possibility of an atypical action of acetylcholine has been reported. Pappano [12], who observed accelerating effects of acetylcholine on the chick embryonic hearts, postulates an increase in sodium permeability by this mediator. Other workers [11], on the basis of experiments with radioactive potassium, concluded that in low concentrations both endogenous and exogenous acetylcholine can reduce potassium permeability. The increase in the rate of SDD during parasympathetic acceleration may perhaps be due to a similar "atypical" effect of acetylcholine on the permeability of the cell membrane, i.e., to its ability to increase sodium and reduce potassium permeability. This problem is a matter for future research.

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RESTORATION OF VITAL FUNCTIONS OF ANIMALS REVIVED WITH A DONOR CIRCULATION AFTER PROLONGED CIRCULATORY ARREST

V. I. Soboleva, I. E. Trubina, and E. A. Mutuskina

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Anesthetized dogs were revived by means of an artificial donor circulation after circulatory arrest lasting 15-20 min. In group 1 the donor's blood was injected toward the heart of the resuscitated dog, whereas in group 2 it was injected toward the heart and brain. In donor-aided resuscitation (especially in group 2) the vital functions were restored more quickly and the number of surviving animals was greater than when other methods of resuscitation were used. Despite the outwardly full recovery of the animals after prolonged circulatory arrest, various degrees of injury took place to their brains, depending both on the duration of clinical death and on the methods of resuscitation.

KEY WORDS: *resuscitation; donor circulation.*

The question of the longest possible period of anoxia tolerated by the brain and the possibility of resuscitation after long periods of clinical death is not a new one [2, 4-9,

Laboratory of Experimental Physiology of Resuscitation, Academy of Medical Sciences of the USSR, Moscow.

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12-14]. However, it is difficult to compare the results which have been obtained because of the use of different methods of resuscitation, different ways of causing death, and different ways of preparing the animals for the experiments. Insufficient attention has been paid to an assessment of the degree of recovery or of brain damage.

The investigation described below was carried out in an attempt to shed light on this problem.

EXPERIMENTAL METHOD

Experiments were carried out on 30 dogs weighing 5-9.5 kg. Before the experiment the animals received an injection of 2% pantopon solution (4-8 mg/kg) and 1% pentobarbital solution (8-16 mg/kg). The circulation was arrested for 15-20 min by electrical shock. Resuscitation was carried out with the aid of a donor dog, the weight of which was four to five times greater than that of the resuscitated animals [1]. The donor's carotid artery was connected to the carotid artery of the resuscitated animals. In group 1 (19 dogs) blood from the donor was injected toward the heart of the resuscitated dogs. In group 2 (11 dogs) blood from the donor was injected toward the heart and brain of the resuscitated animals. Blood from the recipient's jugular vein was simultaneously pumped into the donor's jugular vein. To maintain a constant balance of blood the resuscitated dogs were kept on scales. The cardiac activity was restored by pulsed discharge from a defibrillator. During perfusion the donor breathed oxygen. Artificial ventilation of the lungs was carried out only on the animals of group 2. During the experiments the blood pressure (BP), pneumogram, ECG, EMG, and electrocorticogram (ECoG) were recorded. Observations were made on recovery of the neurological status and the acid–base balance was studied.

EXPERIMENTAL RESULTS

Circulatory arrest lasted 15 min in 14 dogs of group 1 and 20 min in five dogs; in the dogs of group 2 it lasted 15 min.

After the beginning of perfusion BP in the resuscitated animals of both groups averaged 46-50 mm Hg before restoration of cardiac activity. After 3-6 min of resuscitation the amplitude of the defibrillator oscillations in the ECG increased, and cardiac activity was restored after the capacitor discharges.

In group 1, when circulatory arrest lasted 15 min, cardiac activity was restored after 5.42 ± 0.60 min; if it lasted 20 min, it was restored after 6.48 ± 1.12 min. In 10 of the 19 dogs of group 1 cardiac activity was not restored until after the second or third discharge. Respiration reappeared after 7.35 ± 0.68 min in animals with circulatory arrest for 15 min. The structure of the respiratory act was fully restored to normal after 40.6 min. Corneal reflexes were restored after 24.9 ± 1.7 min. With an increase in the duration of circulatory arrest to 20 min all the functions investigated were restored later (Table 1). The first signs of electrical activity of the brain appeared on average 30 min after the beginning of resuscitation after circulatory arrest for 15 min, and continuous electrical activity was restored after 60 min.

In group 2, in which the donor's blood was injected simultaneously toward the heart and brain, the restoration of cardiac activity, respiration, and the corneal reflexes took place sooner in the dogs than in group 1, and their cardiac activity was restored after a single defibrillation. In the dogs of both groups, the acid–base balance of the blood at the beginning of resuscitation (after perfusion for 5-10 min) was characterized by moderate acidosis: The concentration of organic acids was 16.4 ± 1.0 meq/liter, the base deficit 12.6 ± 0.8 meq/liter, and pH 7.17 ± 0.01. During the period of assisted circulation (30-40 min) the concentration of organic acids fell to 14.9 ± 0.8 meq/liter and pH returned to its initial level. The metabolic disturbances were much less marked than during resuscitation by the artificial circulation apparatus or by direct cardiac massage [11].

Because of the absence of artificial ventilation of the lungs in the animals of group 1, before spontaneous breathing was restored the arterial blood was undersaturated with oxygen (82% HbO_2); in some dogs the degree of oxygen saturation of the hemoglobin was reduced to 64-70%. Possibly this may have acted unfavorably on the dynamics of recovery of certain neurological functions. In the experiments of group 2, as a result of the artificial ventila-

TABLE 1. Dynamics of Restoration of Functions in Dogs Resuscitated with the Aid of a Donor

Conditions of resusciation	Number of animals	Duration of circulatory arrest, min	Time of recovery, min, of			Result of resuscitation	
			cardiac activity	respiration	corneal reflex	survived	died
Injection of blood toward the heart	5	20	6,48±1,22	10,61±2,14	38,1±4,1	3	2
	14	15	5,42±0,60	7,35±0,68	24,9±0,1	10	4
P			<0,02	<0,05	<0,001		
Injection of blood toward the brain and heart	11	15	3,35±0,25	5,59±0,54	18,1±1,2	10	1

tion of the lungs the O_2 concentration and degree of O_2 saturation of hemoglobin in the arterial blood remained at the normal level (98-100%) throughout the period of observation. From 2 to 3 h after resuscitation the O_2 concentration and percentage of HbO_2 in the mixed venous blood fell in the animals of both groups to 52-59%. The arteriovenous O_2 difference rose to 8.9-9.2%. This fact was evidently associated with a decrease in the minute volume of the heart in the posthypoxic period in the animals surviving a long period of clinical death.

Outwardly complete recovery of the vital functions by the 2nd-10th day occurred in 10 of the 14 dogs of group 1 after circulatory arrest for 15 min; four dogs died soon after the experiment. Of the five dogs in which circulatory arrest lasted 20 min, three survived.

Despite the apparent normalization of the neurological status in the animals surviving after circulatory arrest for 15 min, the mean amplitude of the high-frequency component of the ECoG of all the dogs was lowered (it fell gradually during 4 weeks of observation) by more than 50% below its initial value. This indicated considerable damage to the cerebral and cerebellar cortical cells, as morphological investigations have shown [3].

Of the 11 dogs of group 2, outwardly complete recovery of function took place in 10. However, the mean amplitude of the high-frequency components of the ECoG was restored to normal in only two of the six animals studied. Counting the Purkinje cells of the cerebellum showed a lower degree of brain damage in group 2 than in group 1.

Comparison of the results of resuscitation by the various methods showed that apparently complete recovery of vital functions after fibrillation for 15 min took place in only 14.3% of animals revived by means of external cardiac massage combined with artificial ventilation of the lungs. After resuscitation by an artificial circulation apparatus survival of the animals after the same periods of circulatory arrest depended on the time of defibrillation of the heart [2, 10]. In the case of late defibrillation (10-12 min of resuscitation) only 14.2% of the animals survived, compared with 66.6% after early defibrillation (2-6 min of resuscitation). Meanwhile, 71.4% of dogs in the experiments of group 1 and 90.8% in those of group 2 survived after donor-assisted resuscitation.

After resuscitation with the aid of a donor, unlike by other methods (cardiac massage, artificial circulation apparatus), the vital functions of the body are restored more rapidly and more animals survived. It must be emphasized, however, that despite the outwardly complete recovery of the animals after circulatory arrest for 15-20 min under normothermic conditions, a varied degree of damage to the higher levels of the brain was present in these animals. The degree of damage is determined not only by the duration of circulatory arrest, but also by the resuscitation method used.

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STATE OF THE MICROCIRCULATION AFTER MASSIVE TRANSFUSION OF DOGS WITH HOMOLOGOUS DONORS' BLOOD

V. P. Matvienko

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Normovolemic exchange transfusion with homologous donors' blood causes moderate slowing of the blood flow in the microcirculation of the recipient dogs together with mild or moderately severe intravascular aggregation of erythrocytes. In 25% of cases acute phenomena of retention of blood in the mesenteric microvessels develop, accompanied by a fall in the level of perfusion in the microvessels of the bulbar conjunctiva. Acute disturbances of the microcirculation are transient in character.

KEY WORDS: *blood transfusion; complications of blood transfusion; microcirculation.*

Transfusion with massive doses of homologous donors' blood leads to the development of a series of disturbances in the recipient collectively described as the homologous blood syndrome [1, 8, 15-17]. Disturbances of the hemodynamics, gas exchange [12], hematopoiesis, the peripheral blood [4, 6], liver function, and the system of phagocytic monocytes [7] have been investigated experimentally in this condition. However, few histopathological investigations have been made of the microcirculatory disorders, which play an important role in the pathogenesis of this syndrome [1, 8].

This paper describes the results of a biomicroscopic study of the microcirculation in the mesentery and the bulbar conjunctiva of dogs during normovolemic exchange transfusion of homologous donors' blood using an arteriovenous shunt [16].

Laboratory of Pathological Physiology, Central Institute of Hematology and Blood Transfusion, Moscow. (Presented by Academician of the Academy of Medical Sciences of the USSR N. A. Fedorov.) Translated from Byulleten' Éksperimental'noi Biologii i Meditsiny, Vol. 84, No. 9, pp. 273-276, September, 1977. Original article submitted March 21, 1977.