DYNAMICS OF PHAGOCYTIC ACTIVITY OF THE PERIPHERAL BLOOD NEUTROPHILS IN THE RECOVERY PERIOD AFTER CLINICAL DEATH CAUSED BY ELECTRIC SHOCK

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The state of phagocytic activity of the peripheral blood neutrophils was studied in dogs in the recovery period after clinical death due to electric shock. An increase was found in the digestive ability of the neutrophils 2 h after resuscitation, and this persisted for 5-9 days. The ingestive ability of the neutrophils showed a tendency to increase during the first 30 min after resuscitation. Later the changes in this parameter were not consistent.

Despite the comprehensive character of investigations conducted in the field of resuscitation, the state and importance of immunological reactions in recovery of the organism after clinical death has still been inadequately studied.

Kolpakov and Shushpannikova [3] found a decrease in the phagocytic index in the early period of recovery after clinical death due to blood loss. One hour after restoration of respiration the ingestive power of the neutrophils not only was restored, but was actually rather higher than initially. In the early stage of agony caused by exsanguination, Sarkisyan et al. [6] found a temporary increase in the phagocytic activity of the blood neutrophils; during resuscitation the ingestive power of the neutrophils was restored to normal within 10-30 min.

The object of the present investigation was to study the ingestive and also the digestive power of the peripheral blood neutrophils during recovery after clinical death.

EXPERIMENTAL METHOD

Experiments were carried out on dogs of different ages and sexes. One hour before the experiment the animals received an injection of pantopon (40 mg/kg body weight). Circulatory arrest was induced by electric shock from the town supply (127 V, 3-4 sec).

Resuscitation began 10 (series I) and 12 (series II) min after lethal electric shock. It was done by a combined method developed by Negovskii and collaborators [including artificial respiration, indirect cardiac massage, and intra-arterial infusion of physiological saline (mean dose 50 ml) with adrenalin (0.5-1 ml)].

The method of Berman and Slavskaya [2] was used to determine the phagocytic activity of the neutrophils. The ingestive power of the neutrophils was assessed from the phagocytic number, and their digestive power by the index of completion of phagocytosis.

The experiments of series III (6 dogs), in which the same experimental conditions were reproduced, including injection of pantopon, fixation of the animal to a frame, and dissection and ligation of the femoral vessels, acted as the control. In this series 4 blood samples were taken during immobilization of the animals for 2 h and later.

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TABLE 1. Dynamics of Index of Completion of Phagocytosis in Recovery Period after Cardiac Arrest Lasting 10 Min ($M \pm m$)

Initial	Recovery period							
	8-15 min	2 h	1-st	3-rd	6-7-th	8-th		
value	(respiration of corneal reflexes)		day					
47,5 P	-2,0±2,0 -	+8,7±2,4 <0,01	+16,7±3,3 <0,001	+20,0±5,7 <0,02	+13,3±5,2 <0,05	+3,2±4,4 -		

TABLE 2. Dynamics of Index of Completion of Phagocytosis in Recovery Period after Cardiac Arrest for 12 min $(M \pm m)$

	Recovery period						
Initial	3-5 min	30 min	1-st	5-th	9 ~ th		
value	(restoration)	ov min	day				
44,5 P	-0,7±2,0 -	-3,6±3,4 	+16,9±4,9 <0,02	+26,0±6,9 <0,02	+22,0±2,6 <0,01		

TABLE 3. Dynamics of Index of Completion of Phagocytosis in Intact Animals (control) $(M \pm m)$

Initial value	Time of observation							
	30 min	1S t	2-nd	l=st	5-th	9-th		
		h		day				
34,4 P	-0,5±0,7	—1,0±1,5 —	+0,2±1,2 -	-1,1±4,3 -	$+16.8\pm10.2$ < 0.2	+27,3±2,3 <0,001		

Control tests also were carried out to exclude any possible effect of electric shock on the phagocytic activity of the neutrophils (series IV). In this case the animals (8 dogs) were subjected to electric shock followed by rapid defibrillation of the heart, so that the period of fibrillation did not exceed 10 sec.

EXPERIMENTAL RESULTS

In series I 8 of the 14 dogs and in series II 4 of the 8 dogs survived. The remaining dogs died at different periods after resuscitation during the first 6 days.

The results of series I (Table 1) and II (Table 2), as regards changes in the index of completion of phagocytosis, were similar. During the first 30 min after resuscitation this index in most animals was the same as initially, but in four dogs it was considerably reduced. After 2 h there was a small but statistically significant increase in the index of completion of phagocytosis. During the next 3-5 days it increased still more. On the 8th day in the experiments of series I the index of completion of phagocytosis returned to its initial value, but in series II it fell, although it still remained significantly higher than initially.

The dynamics of the phagocytic number showed no significant changes in the experimental series. In most dogs (in all animals with an initial phagocytic number of not more than 75% in series I), however, there was a marked increase in this index during the first 2 h of the recovery period.

In the control series of experiments during the first 2 h of observation no significant changes were found in the phagocytic activity of the neutrophils. By the end of the first day a significant decrease in the phagocytic number was found in series III (Table 3) and in the index of completion of phagocytosis in series IV. Starting with the 5th day, an increase in phagocytic activity of the neutrophils was detected.

These investigations thus revealed a regular increase in the digestive power of the neutrophils 2 h after resuscitation, and subsequently for 5-9 days. The ingestive power of the neutrophils, by contrast with their bactericidal power, showed a tendency to increase only immediately after resuscitation for a period of 2 h.

It is very difficult at present to express any definite opinion regarding the mechanisms of this phenomenon. The responsible agents in this case could be either factors connected with a disturbance of the neuro-humoral regulation of phagocytosis [4, 5] or the bacteriemia and toxemia accompanying the state of clinical death could be responsible [7].

Whatever the case, the fact that the digestive power of the leukocytes is increased can be interpreted as evidence of mobilization of the protective responses of the organism in the postresuscitation period. This conclusion is in full agreement with earlier observations when the writer showed that during this same period there is increased activity of other anti-infectious protective mechanisms, such as the bactericidal and complementary activity of the serum [1].

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