

CHANGES IN SOME INDICES OF NATURAL IMMUNITY IN DOGS RESUSCITATED AFTER ELECTROCUTION

S. K. Anan'eva

UDC 617-001.21-036.882-07:612.017.1

Overall bactericidal, complementary, and lysozyme activity of the serum was studied in dogs after resuscitation from cardiac arrest lasting 10 and 12 min. The bactericidal and complementary activities of the serum were reduced immediately after resuscitation, but later they increased. The lysozyme activity was very high during the first hour after resuscitation and remained above its initial level for 5-6 days.

The study of the state of natural immunity in the period after resuscitation is of considerable practical importance because one of the most serious complications at this time is the development of infection. In addition, in the period of cardiac arrest and recovery of vital functions after clinical death it is possible to investigate the role of the nervous system in the mechanisms of natural resistance. This is particularly true if it is remembered that during cardiac arrest and resuscitation the functional state of the central nervous system undergoes regular changes which are characterized by a series of stages determined by the sensitivity of its different levels to hypoxia. The more complex and, as a rule, phylogenetically younger functions are affected earlier and to a greater degree [1, 5].

There is very little information in the literature on changes in the indices of natural immunity in the period after resuscitation [2, 4, 7].

In the investigation described below, the dynamics of the bactericidal, complementary, and lysozyme activities of the blood serum was studied in animals resuscitated after electrocution.

EXPERIMENTAL METHOD

Mongrel dogs of different ages and sexes were used. The animals were electrocuted from the city supply system (127 V, exposure 3-4 sec).

Resuscitation was carried out by a combined method developed in Professor V. A. Negovskii's laboratory: artificial respiration, indirect cardiac massage, and intra-arterial injection of physiological saline (about 50 ml) containing adrenalin (0.5-1 ml) [5, 6].

The overall bactericidal activity of the serum (against *Escherichia coli*) was determined nephelometrically [8]. The complementary activity of the serum was measured by the 50% hemolysis method [10]. Lysozyme activity was estimated by a turbidimetric method based on the degree of clearing of a suspension of acetone powder of *M. lysodeikticus* in phosphate buffer after incubation for 1 h with the test serum at 37°C [3].

Two series of experiments were performed. In Series I resuscitation of the animals began 10 min after lethal electric shock. The initial blood samples were taken (twice or three times during the week before the experiment and on the day of the experiment, immediately before electrocution) and in the recovery period - immediately after recovery of the corneal reflexes, 2 h and 24 h later, and then every

Laboratory of Physiology of Immunity, Institute of Normal and Pathological Physiology, Academy of Medical Sciences of the USSR, Moscow. Laboratory of Experimental Physiology of Resuscitation, Academy of Medical Sciences of the USSR, Moscow. (Presented by Academician of the Academy of Medical Sciences of the USSR N. A. Fedorov.) Translated from *Byulleten' Eksperimental'noi Biologii i Meditsiny*, Vol. 73, No. 2, pp. 80-83, February, 1972. Original article submitted May 31, 1971.

© 1972 Consultants Bureau, a division of Plenum Publishing Corporation, 227 West 17th Street, New York, N. Y. 10011. All rights reserved. This article cannot be reproduced for any purpose whatsoever without permission of the publisher. A copy of this article is available from the publisher for \$15.00.

TABLE 1. Changes in Overall Bactericidal, Complementary, and Lysozyme Activities of Blood Serum of Dogs during Resuscitation after 10-Min Cardiac Arrest

Activity of serum	Initial value	Time of observation				
		in recovery of corneal reflexes	2 h	24 h	6 days	14-15 days
Bactericidal (in percent) P	43,6	-12,8±3,5 <0,01	-3,7±3,3	+12,0±5,2 <0,05	-4,2±8,1	+8,2±7,9
Complementary (in units/ml) P	26,1	-0,7±1,4	+3,9±1,2 <0,01	+4,8±2,6 <0,05 ¹	+5,9±1,8 <0,01	+4,7±1,0 <0,01
Lysozyme (in units) P	14,8	+11,0±1,7 <0,001	+7,9±1,7 <0,001	+4,8±1,2 <0,01	+2,8±1,3 <0,05 ¹	+0,5±1,9

¹Probability of possible error determined by Wilcoxon's criterion.

TABLE 2. Change in Overall Bactericidal, Complementary, and Lysozyme Activities of Blood Serum of Dogs during Resuscitation after 12-Min Cardiac Arrest

Activity of serum	Initial value	Time of observation				
		in restoration of respiration	30 min	24h	5-6 days	9 days
Bactericidal (in percent) P	68,0	-13,5±2,2 <0,01	-9,4±3,8 <0,05	+16,4±3,8 <0,01	+24,6±6,0 <0,01	+13,1±3,4 <0,02
Complementary (in units/ml) P	26,1	-5,4±2,1 <0,05	-4,7±1,1 <0,01	+11,8±5,5 <0,01 ¹	+8,6±3,7 <0,01 ¹	+7,2±2,7
Lysozyme (in units) P	13,9	+4,3±2,1	+6,4±1,5 <0,01	+5,8±1,5 <0,02	+5,2±1,4 <0,02	+3,4±2,3

¹Probability of possible error determined by Wilcoxon's criterion.

2-3 days for 1 month. In Series II resuscitation began 12 min after electrocution. The initial blood samples were taken as in Series I, but in the recovery period blood was taken immediately after restoration of respiration, 30 min and 24 h later, and then every 3-4 days for 1 month.

EXPERIMENTAL RESULTS

In the experiments of Series I nine of the 16 dogs survived, three dogs died on the 1st day, two on the 3rd, and one on the 12th day after electrocution. The dynamics of the indices studied is shown in Table 1.

At the time of recovery of the corneal reflexes a significant decrease in the bactericidal activity of the serum was observed, although 2 h after restoration of respiration it had been slightly increased, close on the average to the initial level. However, 24 h later the bactericidal activity was below its initial level in only two dogs, and in the rest it was significantly higher than initially. Later, individual variations in bactericidal activity of the serum were observed, and they coincided with periods of elevation of the animals' rectal temperature. From the second half of the month a significant increase in bactericidal activity of the sera was again observed and it reached its maximum on the 30th day.

The complementary activity of the serum showed a tendency to diminish at the time of restoration of the corneal reflexes. However, 2 h after restoration of respiration it was considerably increased, and it remained above its initial level throughout the period of observation.

The lysozyme activity of the serum of the experimental animals was also doubled at the time of recovery of the corneal reflexes, after which it fell gradually but still remained significantly higher than its initial level 2 and 24 h after resuscitation. From the second half of the month the serum lysozyme activity showed a tendency to diminish, and at the end of the month it was significantly below its initial value.

In the experiments of series II six of the ten experimental dogs survived, two dogs died on the 1st day, one on the 2nd day, and one on the 6th day. The results of this series of experiments are given in Table 2.

The bactericidal activity of the serum at the time of restoration of respiration and 30 min later was below its initial value in all dogs. After 24 h, a significant increase in bactericidal activity was observed, and in all dogs this persisted for 9-10 days.

The complementary activity at the time of restoration of respiration and 30 min later was significantly below its initial level. After 24 h, all dogs showed an increase in the complement titer, which was still significantly raised on the 6th day.

The lysozyme activity of the serum was higher than initially immediately after restoration of respiration. It was higher still after 30 min and remained significantly higher than initially for 5-6 days.

In the course of the observations regular changes were thus observed in the bactericidal, complementary, and lysozyme activities of the blood serum, indicating the participation of protective mechanisms maintaining the natural immunity of the animal in the pathophysiological process during the period after resuscitation. Tissue hypoxia and a disturbance of nervous regulation, especially in the early stage of the recovery period, must certainly impair these mechanisms. Evidence has also been obtained that in severe forms of shock the serum of animals possesses a certain toxicity [9, 11, 12]. According to one hypothesis, confirmed experimentally, this toxicity is due partly to the penetration of bacterial endotoxin from the intestine [9, 12]. With this in mind, the possibility cannot be ruled out that the decrease in bactericidal and complementary activities in the present experiments during the 30 min after resuscitation may be due to neutralization of toxin entering the blood stream. The increased lysozyme concentration in the blood serum in the initial stage of the recovery period may perhaps be due to liberation of this enzyme from cells, especially leucocytes, which are probably undergoing more rapid destruction at this period. The possibility likewise cannot be ruled out that activation of the lysozyme activity of the blood serum is one of the protective and compensatory responses of the living organism.

LITERATURE CITED

1. A. M. Gurvich, *Electrical Activity of the Dying and Reviving Brain* [in Russian], Leningrad (1966).
2. V. G. Zhdanov, in: *Proceedings of the Second Plenum of the Siberian Branch of the All-Union Society of Pathophysiologists* [in Russian], Chita (1958), p. 70.
3. K. A. Kagramanova and Z. V. Ermol'eva, *Antibiotiki*, No. 10, 917 (1966).
4. M. G. Kolpakov and O. V. Shushponnikova, in: *Proceedings of the Fourth Plenum of Pathophysiologists of Siberia and the Far East* [in Russian], Tomsk (1962), p. 49.
5. V. A. Negovskii, *The Pathophysiology and Treatment of Agony and Clinical Death* [in Russian], Moscow (1954).
6. V. A. Negovskii (editor), *Fundamentals of Resuscitation* [in Russian], Moscow (1966).
7. A. A. Sarkisyan et al., *Abstracts of Proceedings of the 3rd All-Union Conference of Pathophysiologists* [in Russian], Moscow (1960), p. 146.
8. O. V. Smirnova and G. A. Kuz'mina, *Zh. Mikrobiol.*, No. 4, 8 (1966).
9. S. Jacob et al., *Am. J. Physiol.*, 179, 523 (1954).
10. E. A. Kabat and M. M. Mayer, *Experimental Immunochemistry*, Thomas, Springfield, Ill. (1961).
11. S. H. Rutenburg and J. Fine, *Proc. Soc. Exp. Biol. (New York)*, 91, 217 (1956).
12. F. B. Schweinburg et al., *Proc. Soc. Exp. Biol. (New York)*, 95, 646 (1957).