PATHOLOGICAL PHYSIOLOGY AND GENERAL PATHOLOGY

RESUSCITATION OF THE ORGANISM AFTER CLINICAL DEATH IN DOGS WITH DENERVATED CAROTID SINUS

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In resuscitation of the organism through arterial transfusion of blood and artificial respiration a positive result depends first and foremost on timely restoration of the circulation. With considerable pressure in the blood-pumping system (180-200 mm mercury) the blood pressure in the arterial vessels (femoral artery) of the resuscitated animal did not exceed 40 mm mercury up to the moment when the heart activity was resumed, at which time it rose abruptly and the blood circulation was restored. Most authors believe that the "awakening" of heart activity is a result both of the restoration of nutrition of the myocardium and of reflex stimulation emanating from the receptors of the coronary vessels and other parts of the vascular system [1]. There is no doubt that the pressure created in the arteries with centripetal infusion of blood stimulates the baroceptors of the arterial vessels and contributes to the restoration of reflex regulation of circulation.

The importance of stimulation of the receptors of the blood vessels in resuscitation by the Andreev-Negovsky method is demonstrated by the experiments of E. M. Smirenskaya [3], who showed that preliminary stimulation of the baroceptors of the peripheral arteries accelerates the restoration of heart activity. E. M. Smirenskaya and V. I. Prokhorov also observed resumption of heart activity with transfusion into the peripheral end of an artery not combined with centripetal introduction of blood.

Restoration to life after clinical death is achieved in a complex method through restoration of the nutrition of the central, and evolutionarily older, pressor reflex regulation of the circulation, without which a good result cannot be achieved in resuscitation.

The role of the phylogenetically younger depressor regulation of the circulation has not been made entirely clear. Nor has the role of the carotid depressor apparatus been studied. On the basis of his own investigations and the work of his assistants, I. R. Petrov concludes that with clinical death all the conditions are created for the appearance of phase reactions when the receptors of the carotid sinus are stimulated. Therefore, during the period of resuscitation the depressor carotid reflex is converted into a pressor reflex and contributes to the restoration of the circulation. These opinions of I. R. Petrov are based on data obtained in study of the carotid depressor reaction in shock conditions, when it frequently is paradoxal.

In V. A. Negovsky's opinion, the ultraparadoxal reaction, although possible, does not appear regularly, and therefore it cannot be assigned the place of decisive importance in all cases of resuscitation of the organism.

In a previous communication from our laboratory (1953) we expressed the opinion that I. R. Petrov's point of view [2] required experimental confirmation, since it was not based on facts established in resuscitation of the organism after clinical death.

We set up experiments in resuscitation of dogs with denervated carotid sinuses. We proceeded on the premise that if I. R. Petrov's opinion was correct resuscitation of animals with denervated carotid sinuses should

be more difficult and should be successfully achieved in a smaller percentage of cases because of the absence of the above-mentioned paradoxal (pressor) reaction.

EXPERIMENTAL METHODS

Our experiments were conducted on two groups of dogs – experimental and control (12 animals in each group).

The experimental dogs were subjected to a bilateral denervation of the carotid sinus 10 days before clinical death and resuscitation. Each dog of the control group was also subjected to an operation at the same time; all the elements of operative trauma were reproduced, with the exception of denervation of the carotid sinus and the preparation of the adventitia.

For resuscitation of the organism after clinical death we employed a complex method which is used in the laboratory directed by V. A. Negovsky [1].

Before the beginning of the experiments in resuscitation the animal received an injection of pantopon – 8 mg per 1 kg of body weight, and the femoral vessels were prepared with anesthesia by a 0.5% solution of novocaine. Heparin was used as an anticoagulant in the amount of 0.10 ml/kg (Richter).

The duration of clinical death was 3½ minutes. We transfused the animal's own blood without adding glucose and oxygen.

Artificial respiration was effected with a two-chamber bellows through intubation.

EXPERIMENTAL RESULTS

There was no observable reflection of the denervation of the carotid sinus in the general state of the animals. The post-operative period proceeded normally in the experimental and control animals, although elevated blood pressure was noted in most of the experimental dogs (above 160 mm mercury). Undoubtedly, this hypertension was the result of the denervation. However, it was not substantially reflected in the success of the resuscitation experiments (Fig. 1).

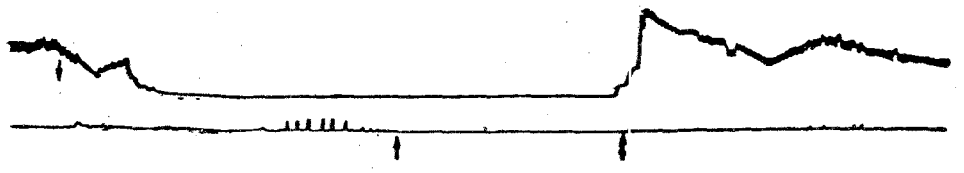

Fig. 1. Kymogram of experiment No. 17. Bleeding and resuscitation of dog with denervated carotid sinuses. Meaning of the curves (top to bottom): blood pressure, respiration. ↓ Beginning of bleeding. ↑ Beginning of clinical death. ↕ Beginning of resuscitation.

After clinical death lasting 3½ minutes we succeeded in reviving 18 animals; restoration of functions was not achieved in 3 experimental and 3 control dogs (see table).

If resuscitation is viewed as the basic indicator of the effect of denervation of the carotid sinus in our experiments, then it must be noted that the ratio of the number of dogs resuscitated to the number not resuscitated was identical in the experimental and the control groups.

However, it must be noted that some difference was observed in the animals of the two groups with respect both to dying and to resuscitation.

The length of the dying period, as was established in the laboratory directed by V. A. Negovsky, is a very important factor in the successful resuscitation of the organism after clinical death. It will be seen from the table that clinical death occurred more quickly in the experimental animals than in the control animals. For example,

Results of the Experiments in Resuscitation of Dogs After Clinical Death

Experimental dogs			Control dogs		
Animal No.	Duration of dying period	Results	Animal No.	Duration of dying period	Results
2	13 min	Not revived	7	28 min	Not revived
5	8 min	" "	9	10 min 30 sec	" "
12	11 min	" "	14	21 min	" "
19	5 min 30 sec	Lived 72 hours	4	17 min 30 sec	Lived 25 min
20	12 min	Lived 7 days	11	17 min 30 sec	Lived 48 hours
22	9 min	Lived 7 days	21	12 min 30 sec	Lived 13 days
1	12 min	Alive	6	4 min	Alive
3	6 min 30 sec	Alive	8	13 min 30 sec	Alive
10	11 min	Alive	15	8 min	Alive
13	9 min 30 sec	Alive	18	10 min 30 sec	Alive
16	9 min	Alive	23	10 min 30 sec	Alive
17	6 min	Alive	24	9 min	Alive

the dying period lasted more than 10 minutes in only 4 of the experimental dogs (Nos. 2, 10, 12, and 20), whereas a longer dying period was observed in 9 dogs of the control group (Nos. 4, 7, 8, 9, 11, 14, 18, 21, and 23). This distinction was especially noticeable in the animals which were not revived or which died at varying lengths of time after resuscitation.

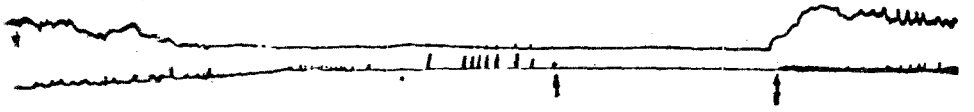

Fig. 2. Kymogram of Experiment No. 6. Bleeding and resuscitation of control dog. Curves: same as in Fig. 1.

We observed two types of reactions with restoration of the circulation: 1) quick and considerable heightening of the blood pressure and 2) slow and less marked heightening. In the reaction of the first type the blood pressure increased within the first few seconds after restoration of heart action, reaching 180-220 mm. This reaction was more characteristic for animals with denervated carotid sinus (it was observed in 6 out of the 9 resuscitated experimental dogs: (Nos. 10, 16, 17, 19, 20 and 22).

We also observed a hypertonic reaction in 4 of the 9 resuscitated control animals (Nos. 8, 15, 18, and 24), but their blood pressure immediately after the restoration of heart activity was considerable lower than in the experimental dogs.

In 7 of the 9 resuscitated control dogs heart action was restored within 40 seconds from the beginning of the resuscitation procedure, and in the other two (Nos. 4 and 21) after more than 40 seconds. In the experimental dogs heart action was restored within 40 seconds in only 4 cases. Thus, in denervated animals restoration of heart action took somewhat longer than in the control animals.

We also observed some difference in the restoration of breathing. Spontaneous breathing commenced more quickly in the experimental animals than in those of the control group: it had appeared by the third minute in 5 of the 9 resuscitated experimental animals and in only 2 of the 9 control animals.

From the above data it will be seen that there is an inverse relationship between the time of restoration of heart activity and of respiration in "denervated" and in "undenervated" dogs: heart activity revived more slowly in the experimental dogs and spontaneous respiration more slowly in the control dogs.

The corneal reflex appeared between the 7th (No. 17) and the 23rd minutes (No. 11). We were unable to establish any difference in the time taken for this reflex to appear in the experimental and the control animals.

After restoration of heart action, respiration, and the corneal reflex, the animals of both groups were in a state of sleep. After the sleep, and in some of the animals even immediately after resuscitation, rigidity of the muscles set in. In most of the revived dogs the rigidity was inconsiderable and was observed only in the fore limbs during the first few hours after resuscitation; on the following day it disappeared, and coordinated movements appeared in connection with eating; later the animals also began to walk freely.

The animals which died soon after resuscitation (Nos. 11, 19, 20, and 21) showed symptoms of pronounced rigidity in their muscles at the time of death, or sometimes the rigidity appeared in them after a period when they were in a comparatively good condition.

The most severe complications after resuscitation were observed in Dog No. 1. Symptoms of considerable damage to the cerebral cortex continued in this animal up to the time of its death 3 months after resuscitation.

Generalizing the data obtained, we may reach the following conclusions. Denervation of the carotid sinus causes certain peculiarities in the reaction of dying and resuscitation which are not observed in the control animals. For example, dogs with denervated sinus die more quickly with acute loss of blood, and they endure prolonged dying with more difficulty. During the period of resuscitation the restoration of heart action took somewhat longer in them than in the control dogs. However, after the circulation had been restored a preponderantly hypertonic reaction was noted in them. Respiration was restored sooner in the experimental dogs than in the control dogs. Despite these differences, however, the number of denervated dogs resuscitated was identical with the number of control group dogs resuscitated.

We link the fact that the experimental animals died more quickly with the presence of hypertonia, which contributes to a greater loss of blood in a shorter period of time. It is not clear why heart action was restored more slowly in the denervated dogs. The possibility that the depressor nerve conducts impulses "awakening" heart activity cannot be excluded. It is difficult to say whether these impulses are an expression of the paradoxal reaction accelerating heart action, referred to by I. R. Petrov. However, it is evident from our material that immediately after the renewal of heart activity there is an adequate (depressor) but not paradoxal (pressor) reaction. This is evident from the fact that the experimental dogs react with a considerably more pronounced hypertonic reaction because of the absence of the sinus nerves. This reaction was weaker in the control animals.

The speedier restoration of respiration in the denervated animals is apparently connected with the absence of the depressor action of the depressor apparatus on the respiratory center which appears when the blood pressure in the carotid sinus rises above the normal level.

Despite the differences noted in the restoration of life after clinical death in dogs with denervated carotid sinus and in the control dogs, our investigations show that the role of the carotid sinus in the restoration of life is not so great as is suggested by some authors (I. R. Petrov). The best indicator of this is the identical numbers of experimental and control animals revived.

LITERATURE CITED

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[3] E. M. Smirenskaya, Theses of the Reports of the Conference on the Problem of the Pathophysiology and Therapy of Terminal Conditions in the Clinic and in First Aid Practice* (Moscow, 1952), pp. 6-8.

*In Russian.