PATHOLOGICAL PHYSIOLOGY AND GENERAL PATHOLOGY

CHANGE IN THE REACTION OF THE ORGANISM TO CERTAIN SHOCK STIMULI IN THE PERIOD OF RESTORATION OF VITAL FUNCTIONS AFTER CLINICAL DEATH

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The complex technique of revivification of the organism after clinical death consists in combination of artificial (mechanical) respiration and arterial urging of the blood with glucose and adrenalin in the direction of the heart [4]. After the heartbeat resumes some blood is introduced intravenously. Independent respiration is quickly restored. It is fully understandable that the rise in arterial pressure and restoration of respiration are very important, but all the same they are only the first link in revivifying the organism. For stable revivification, strengthening of the results achieved on the one hand, and, on the other, successful treatment of a multitude of possible complications are necessary.

The investigations we conducted [2, 3] showed that the development of shock or collapse as a result of loss of blood sharply changes the reactivity of the organism and changes in sensitivity are observed not only in the period of shock or collapse but after their apparent disappearance, i. e. after seeming recovery. The introduction in this case of shock doses of peptone, foreign blood, morphine (experiments on dogs), histamine (experiments on cats) and other stimuli does not produce the usual shock reaction, arterial pressure showing no appreciable fall. The findings of the experiments convinced us that the resistance to shock stimuli after emergence from a state of collapse or shock is a result of the parabiotic process localized chiefly in the region of the center of the medulla oblongata, which is in agreement with the data on the parabiotic nature of traumatic shock [1, 6, 7]. The onset of a state of clinical death in essence is the maximum expression of the suppression of the functions of the organism. One might have expected that disturbance of the excitatory processes in the central nervous system and, in connection with this, change in its reactivity after restoration of the vital functions of the organism subject to clinical death, would be no less marked than after collapse or shock. Determination of such changes in reactivity, testifying to the traced parabiotic processes in the reviviled animals, it must be assumed, is of crucial importance in therapy of terminal states and determination of the outcome of revivification. In order to clarify these changes special investigations were undertaken.

EXPERIMENTAL METHODS

The experiments were conducted on cats (first series) and on dogs (second series).

In the first series of experiments, in the animal in a state of chloral narcosis (1-2 ml of 1% solution per 1 kg body weight, intraperitoneally), the femoral artery and vein, the carotid and trachea were dissected. In the trachea was placed a glass T-piece, connected by one arm with the bellows for artificial respiration; in the carotid a cannula was inserted for recording arterial pressure by means of a mercury manometer on the smoked drum of a kymograph. The respiration was recorded by a pneumogram collar. Lethal hemogrape was produced

from the femoral artery. The moment of death was established according to the last agonal breath. Three to five minutes after death revivification was undertaken according to the complex technique of V. A. Negovsky [5].

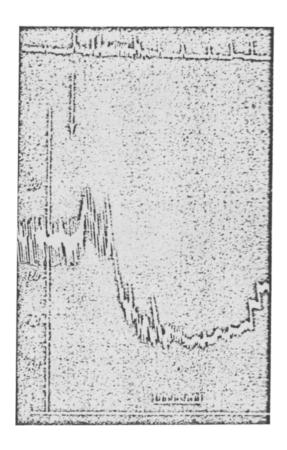
Ten to fifteen minutes after restoration of respiration and steady arterial pressure a shock dose of histamine was introduced under the skin of the fore paw: 0.2-0.25 ml 1% solution per kg body weight.

In the control experiments histamine in the same dose was introduced in cats in a state of chloral narcosis.

In the second series of experiments, 30-40 minutes before the experiment, morphine (0.5-1% solution, subcutaneously) was introduced in the dog. After lethal hemorrhage the restoration of the vital functions (method of V. A. Negovsky) occurred within 3-5 minutes. In 10-15 minutes 30 per cent solution of peptone at the rate of 1 ml per kg body weight was introduced into the femoral artery. For the control, peptone was introduced in the dogs previously given a preparatory injection of morphine only.

EXPERIMENTAL RESULTS

In the 10 experiments of the first series subcutaneous injection in the cats of histamine in the period of restoration of the vital functions after revivification, as a rule, did not produce a fall in arterial pressure, typical of histamine. In the control animals histamine caused at first a slight increase and then an exceptionally swift fall in arterial pressure. The stage of increase corresponded to a distinct intensification in respiratory movements.



In the cats undergoing clinical death, introduction of histamine did not produce the slightest incfease in arterial pressure. The fall in arterial pressure, when it was observed, occurred only 0.5-1 minute after introduction of histamine, i. e., it was delayed, steady and developed gradually without exceeding 20-30 mm Hg. Even greater discrepancies in the reactions of the organism were observed as regards respiration; in the experimental cats, unlike the controls, introduction of histamine did not produce a distinct change in respiration.

In the second series a change in the reactions of the revivified animals to intra-arterial introduction of a solution of peptone was observed. The dosage of peptone which we administered to the dogs in the femoral artery (control), as a rule, caused a sharp temporary dysphea and a slight increase in arterial pressure, quickly replaced by a fall to 50-70 mm Hg. In the course of several minutes a gradual levelling of arterial pressure occurred (Fig. 1). At the same time, upon introduction of peptone 10-15 minutes following revivification there was neither a sharp excitation of respiration nor the typical reaction of arterial pressure which only slightly and gradually fell (Fig. 2).

DISCUSSION OF RESULTS

As is clear from the data presented neither peptone in the dogs nor histamine in the cats produced after revivification those reactions which are observed in the usual experimental conditions. The absence of excitation of respiration, of the usual increase in arterial pressure, replaced by stages of acute and persistent reduction, indicates that neither peptone nor histamine produced a picture of shock in the experimental animals, i. e., the animals in a state of restoration of the vital functions after clinical death were relatively resistant to shock stimulation. The change in the sensitivity to peptone or histamine after clinical death, of course, is not specific; it is determined by the general change in reactivity, and by analogy with the mechanism of resistance, discovered by us after shock or collapse [2, 3], the resistance of the organism to traumatic shock in the experiments conducted may be considered as a consequence of the parabiotic process in the nerve centers.

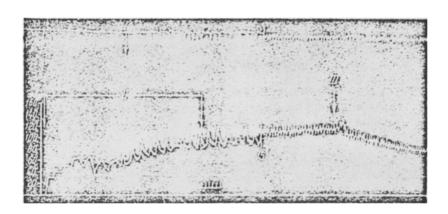


Fig. 2. Change in respiration and arterial pressure after introduction of peptone in dog, subjected to clinical death. Significance of tracings as in Fig. 1. $I - \frac{1}{2}$ artificial respiration; $II - \frac{1}{2}$ arterio-venous pressure of blood after 3 minute clinical death resulting from hemorrhage; $III - \frac{1}{2}$ introduction of 12 ml 1% peptone solution in right femoral artery.

Confirmation of the presence of a parabiotic state of the centers after revivification is provided by the study of spinal areflexia conducted by I.R. Petrov [6]; however, any conclusion on the parabiotic nature of change in reactivity of the revivified animals requires more direct evidence. Nevertheless, there is no doubt that in the period of restoration of the vital functions after clinical death a protective inhibition appears, which reduces sensitivity to traumatic shock.

From this point of view the fact described of the resistance of the organism to peptone or histamine should be considered only as a particular manifestation of general change in the reactivity of the revivified animals.

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