EFFECT OF ELECTRICAL STIMULATION OF THE MEDULLA AND SPINAL CORD ON RESTORATION OF FUNCTION OF THE RESPIRATORY CENTER AFTER CLINICAL DEATH

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Electrical stimulation of the spinal cord and medulla in the initial period of resuscitation after clinical death lasting 6 min led to a more rapid recovery of spontaneous respiration and increased the number of surviving experimental dogs compared with controls.

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Earlier laboratory investigations [1] showed that the sooner the introduction of air into the lungs for resuscitation purposes begins the more rapidly spontaneous respiration is restored after clinical death. Earlier recovery of function of the respiratory center in turn leads to a more rapid and complete recovery of the functions of other parts of the central nervous system, including the cerebral cortex, thus ensuring a favorable prognosis for resuscitation.

In 1947, L. Ya. Shostak [2] showed that Leduc currents (short pulses of direct current) can be used to excite the central nervous system when in a state of profound inhibition during general anesthesia.

Even in clinical death some nervous reflexes between the internal organs still persist. This is shown, in particular, by the disturbance of the rhythm of bioelectrical potentials of the arrested heart on introduction of an incubation tube into the trachea for carrying out artificial respiration.

In this investigation the possibility of quickening the recovery of function of the respiratory centers after clinical death by electrical stimulation of the medulla and spinal cord was studied.

EXPERIMENTAL METHOD

Experiments were carried out on 11 dogs in which clinical death was produced by acute blood loss and lasted about 6 min. Subsequent resuscitation was carried out by centripetal injection of blood with adrenalin and glucose intraarterially and by artificial respiration using a breathing apparatus pumping air into the lungs under positive pressure. Electrical stimulation was applied to the animal 30-50 sec after the heart had started to work independently, using an interrupted direct current (100 pulses/sec, off-duty factor 1:10). Needle electrodes were inserted beneath the skin: the positive in the region of the occiput and the negative in the dorsal region at the level of the lower thoracic vertebrae. The current was applied for 1 sec and this was repeated at intervals of 30 sec. The total number of stimuli varied from 3 to 10 depending on the rate of recovery of spontaneous respiration by the experimental animals. The strength of stimulation was determined from the appearance of contractions of the neck and tongue muscles (usually 12-15 V).

Besides the 11 experimental dogs, observations were made on the rate of recovery of respiration and survival after clinical death in 20 other dogs which served as controls.

EXPERIMENTAL RESULTS AND DISCUSSION

The period from the beginning of resuscitation until the appearance of the first inspiration in the dogs of the control groups varied from 3 to 9 min after blood loss and death lasting 5-17 min (the bottom row in

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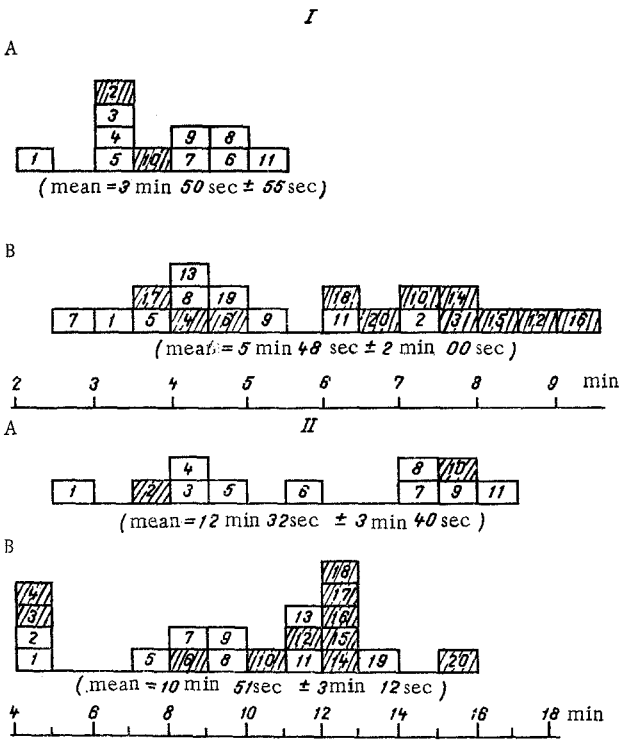

Fig. 1. Duration of periods before recovery of respiration (I) and of clinical death (II) in experimental (a) and control (b) dogs. Shaded squares represent dying animals. Serial numbers of dogs are in order of relative duration of period of recovery of respiration. Horizontal axis, time (in min).

Fig. 1, I and II). No strict relationship between the time of appearance of respiration and the duration of clinical death was observed. In some dogs (Nos. 2 and 3), after a short period of death, respiration was not restored until late, while in others (Nos. 13, 17, 19), after a long period of clinical death, it was restored relatively early.

In the dogs of the experimental group, with approximately the same duration of blood loss and clinical death (6-17 min) certain significant differences from the control animals were observed as regards both the times of appearance of respiration and their pattern. These times were shorter (2-5 min) and did not show such large deviations from the mean value as in the dogs of the control group. A more distinct dependence of the time of appearance of respiration on the duration of clinical death was also apparent, as can be seen by the fact that the position of the individual experimental dogs on the diagrams in Fig. 1 (I and II) coincides with their numerical order.

In the control group of dogs in which the relationship between the time of appearance of respiration and the duration of clinical death was ill-defined, the individual arrangement of the dogs on the diagram (compare I and II) is nearly chaotic in character.

Of the 20 control dogs, 11 (the shaded squares on the diagram) died in the first two days after the experiment. Death of the animals bore some relationship to the duration of clinical death and time taken for respiration to recover. Only 2 of the 9 experimental dogs died ($P < 0.01$). Since the duration of blood loss and clinical death in the dogs of the two groups were the same, the differences observed can be attributed entirely to electrical stimulation in the early stage of resuscitation of the experimental dogs.

In the analysis of the possible mechanisms by which electrical stimulation influences the rate of recovery of functions of the respiratory center, an important fact is that, as well as the more rapid appearance of respiration in the experimental animals, this period also bore a more precise relationship to the duration of blood loss and its deviations from the mean values were smaller. This may be explained as follows.

The time for recovery of respiration after clinical death is determined mainly by the degree of anoxia, because the longer the blood loss the greater the time taken to abolish the anoxia. However, an explanation must be found for the considerable variability frequently observed in the time of appearance of respiration after blood losses of equal duration. It may be postulated that the removal of anoxia by itself does not cause the recovery of respiration but merely provides suitable conditions for recovery to take place.

Triggering of the respiratory center after clinical death evidently requires additional influences and it is thus apparently accidental in character. Electrical stimulation in this case facilitates earlier triggering, and by abolishing the role of chance in this process reveals its relationship to the degree of anoxia (i.e., to the duration of blood loss and clinical death) more clearly.

While electrical stimulation undoubtedly influences the rate of recovery of respiration, the appearance of the first breaths nevertheless did not coincide with the moment of stimulation. The absence of any direct relationship between them suggests that the role of electrical stimulation is not one of direct excitation of activity of the respiratory center, but one of clearing the paths and de-inhibiting the block to conduction. When this has been done, the hitherto latent activity of the respiratory center starts to be manifested in its autonomous rhythm, independent of the rhythm of electrical stimulation, as was observed in the experiments described above.

LITERATURE CITED

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