TREATMENT OF EXPERIMENTAL TERMINAL STATES CAUSED BY BLOOD LOSS BY MEANS OF AN ARTIFICIAL CIRCULATION

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In acute anemia, hypotension, and cerebral hypoxia develop and within a few hours irreversible changes take place in the brain. Resuscitation measures carried out subsequently are thus ineffective [3, 4, 9].

Because of the great sensitivity of the brain to hypoxia, a number of authors have suggested injecting blood into the peripheral end of the carotid artery [10-12]. However, in Negovskii's opinion [4], such perfusion can be used only if cardiac activity is present, however weak. Injection of blood into the coronary artery restores the coronary circulation and creates conditions for strengthening the contractile activity of the heart [1-6].

In the present investigation, a comparative study was made of the results of resuscitation of dogs after acute blood loss by means of various types of artificial circulation.

EXPERIMENTAL METHOD

To resuscitate dogs after acute blood loss, Yankovskii [8] obtained good results with Bryukhonenko's autojector. Skorik, Ballyuzek, and Entina [7], in experiments on dogs, used a portable artificial circulation apparatus for resuscitation. The present authors used the ISL-2 apparatus, based on Ballyuzek's system and intended for artificial circulation during open-heart operations, for the treatment of terminal states.

Two series of experiments were carried out to resuscitate adult dogs of both sexes weighing from 14 to 20 kg. In series I, blood was injected into the femoral artery, and in series II into the carotid artery.

In most experiments the perfused blood had been used once or twice previously.

In 9 dogs anesthetized with morphine and hexobarbital massive bleeding was carried out of the femoral artery, and in 12 dogs from the carotid artery, in a volume of between 800 and 1500 ml, depending on the animal's body weight, equivalent to 62-75% of the total circulating blood volume. Before the bleeding the dogs were given an injection of heparin (1.0-1.5 mg/kg). Heparinized blood was given in a volume of between 2 and 3 liters, taking compatibility into account. To neutralize the clotting power of the blood, heparin was used in a dose of 50-60 mg/liter blood.

During the experiment the arterial pressure was recorded in the femoral artery with a mercury manometer, the venous pressure with a Waldman's phlebotonometer, the ECG was recorded in the standard leads, the EEG (symmetrical fronto-occipital leads) was recorded with needle electrodes introduced subperiosteally into the scalped skull, and respiration was recorded with a Marey's capsule.

EXPERIMENTAL RESULTS

After bleeding severe posthemorrhagic shock developed.

The duration of the shock before the onset of clinical death was 8-12 min, counting from the time when the arterial pressure fell after bleeding to 50/10 mm (in 6 animals between 8 min and 8 min 30 sec, in 8 from 9-10 min, and in 7 from 11 to 12 min). In both series of experiments the duration of the shock

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and of clinical death before the beginning of resuscitation with the artificial circulation was the same. The period of clinical death for the animals of both series of experiments, apart from 2 dogs in series I (for which it was 5-6 min) was 3-4 min after total disappearance of the cerebral potentials coupled with disappearance of all other signs of life in the animal. Only in 5 experiments were sporadic ventricular complexes recorded on the ECG.

In all the experiments cannulas were introduced into the vessels before bleeding. With the main venous trunk closed, blood was injected in a volume close to the volume of the blood loss, in order to restore the circulating blood volume.

In the experiments of series I (9 dogs) the apparatus was connected to cannulas in the femoral vessels. The vital functions were restored in the opposite order to that in which they were lost after the blood loss. The normal cardiac activity was restored after perfusion for 5 min, and regular cortical potentials reappeared after perfusion for 10-15 min (after 10-12 min in 3 dogs and 13-15 min in 4 dogs).

In the case of the two animals whose cortical electrical activity ceased completely for 5 and 7 min, respectively, perfusion via the femoral vessels was ineffective. Their cardiac contractions and respiration were restored, but not their cortical activity. Despite prolonged perfusion, the dogs died from secondary cardiac and respiratory arrest.

In the experiments of series II the method of connecting the ISL-2 apparatus was changed. The arterial trunk was connected by two cannulas through a T-tube to the central and peripheral ends of the divided carotid artery. In this way oxygenated blood could be injected during perfusion toward the heart and the brain simultaneously. With this method the cortical activity was restored more rapidly than in the experiments of series I. Perfusion into the carotid artery continued for 2-3 min until the cortical and cardiac activities were restored. Later, the apparatus was switched over to perfusion via the femoral vessels until the arterial pressure and the ECG and EEG were stabilized. This required the use of the artificial circulation for 5-15 min (5-7 min for 3 dogs, 8-10 min for 4, and 11-15 min for 3 dogs). Altogether this method was used for 12 experiments.

Comparison of these resuscitation methods showed that better results were obtained when blood was injected simultaneously into the carotid artery toward the heart and the brain. It was therefore concluded that carotid perfusion results in the more rapid restoration of the lost vital functions. The cortical and cardiac activities were restored far more quickly than with perfusion via the femoral arteries. However, it must be remembered that the rapid and massive injection of blood into the carotid artery causes overfilling of the heart, and against the background of a myocardium weakened by hypoxia, this could cause sudden cardiac arrest or fibrillation. Such a reaction to perfusion was observed in two experiments. For this reason, the volume velocity of perfusion in most experiments did not exceed 50-70 ml/kg/min.

These investigations thus showed that perfusion via the carotid artery may be used very effectively for the treatment of clinical death.

Of the 21 experimental animals, 17 recovered, while 4 died during the first day with disturbances of respiration and cardiac activity. The postoperative period for all the surviving dogs was uncomplicated. They began to walk and take food at the end of the first day. Three dogs in series II began to walk 10 min after the end of resuscitation. In three experiments of series I, transient ataxia was observed, and in two experiments severe motor excitation occurred for 30 min. Comparison of the postoperative period for the five dogs which preserved sporadic ventricular complexes with that for the animals whose cardiac electrical activity disappeared completely showed no difference.

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