BLOOD SUPPLY OF THE BRAIN IN CRANIOCEREBRAL HYPOTHERMIA

V. P. Vindyuk and G. G. Shchegol'kova

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The blood supply of the brain was studied in craniocerebral hypothermia. At the beginning of cooling the blood supply is unchanged or slightly reduced. At a rectal temperature of 33-31° (brain temperature 30.1-27.1°) the blood supply is reduced by 40-50%. In deep hypothermia the blood supply is reduced by 65-70% below its initial level.

Reheating restores the normal blood supply to the brain.

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The brain temperature modifies its blood supply, but data on this matter are conflicting [2, 5].

The introduction of craniocerebral hypothermia into clinical practice has necessitated a more complete study of the dynamics of changes in the blood supply to the brain during this method of cooling.

EXPERIMENTAL METHOD

Experiments were carried out on 14 mongrel dogs weighing 5-13 kg. Anesthesia was produced by intravenous injection of a 5-7.5% solution of hexobarbital and maintained with ether and oxygen. Craniocerebral hypothermia was produced in the animals [3, 4] to a rectal temperature of 28-27° (brain temperature 22.4-20.9°), and reheating produced by reflectors to a temperature of 33° (brain temperature 30.1°). Further recovery of the body temperature took place without active heating of the animal. The blood supply was studied by rheography. The rheoencephalogram (REG) was recorded by means of needle electrodes fixed into the frontal and occipital bones.

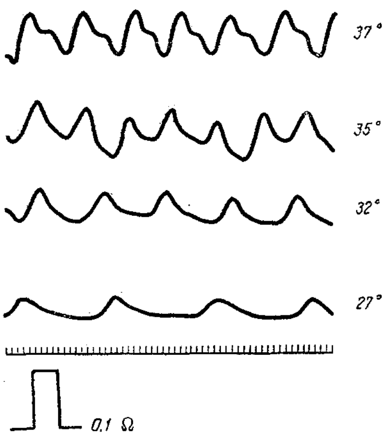

Fig. 1. REG in different stages of craniocerebral hypothermia.

EXPERIMENTAL RESULTS

In normothermia (rectal temperature 38-37°, brain temperature 38-36.5°) the REG shows the following characteristics: the anacrotic part rises steeply, the apex as a rule is pointed, the catocrotic part is sloping. The incisura usually lies in the middle part of the catacrotic phase of the REG. The circulation time of the dogs under these conditions averaged 4.8 ± 0.3 sec.

The beginning of cooling in most experiments was accompanied by a slight decrease in amplitude of the REG. When the temperature was lowered by 1-2° (brain temperature 36.7-33.5°) it was reduced by 10-15% of the initial amplitude. Often the amplitude at the beginning of cooling remained the same as in normothermia. The REG showed no major changes. The circulation time was only slightly altered, and its mean value was 5.3-0.4 sec. Further deepening of the hypothermia to 35-34° (brain temperature 33.5-31.9°) as a rule did not change the amplitude or reduced it only slightly.

Department of Physiology of Man and Animals, P. I. Lebedev-Polyanskii Vladimir Pedagogic Institute.
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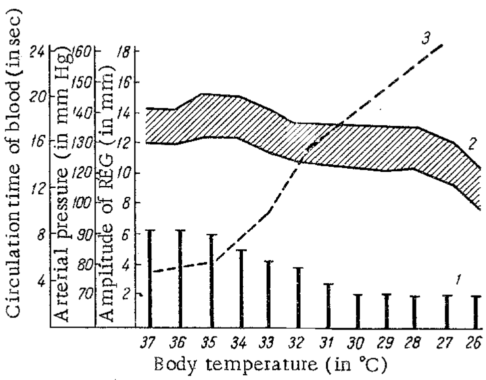

Fig. 2. Dynamics of changes in amplitude of REG, arterial pressure, and circulation time during hypothermia (experiment No. 655, June 14, 1968). 1) Amplitude of REG; 2) arterial pressure; 3) circulation time of blood.

Cooling the animals to 33-31° (brain temperature 30.1-27.1°) was accompanied by a marked decrease in amplitude of the rheographic wave—to 40-50% of its initial value. This change in amplitude of the REG indicates a decrease in blood supply to the brain. The circulation time was 10 ± 0.8 sec, i.e., the velocity of the blood flow was only half its initial value. The apex of the REG became more rounded, the catacrotic and anacrotic parts were more sloping, the major changes affecting the catacrotic phase, while the incisura in most experiments had disappeared.

The most significant changes in the REG were observed during cooling to 30-27° (brain temperature 25.6-20.9°). At this level of hypothermia the amplitude of the REG was reduced by 65-70% compared with initially. Starting with a body temperature of 30° (brain temperature 25.6°), the amplitude of the REG became stabilized and was unchanged during further cooling, i.e., the blood supply to the brain remained at a steady level. However, the velocity of the blood flow still continued to fall until the end of cooling, when the circulation time was 23.8 ± 3.3 sec. The incisura of the REG at this level of hypothermia became obliterated, and the apex became more rounded, although no sign of a plateau appeared (Fig. 1). Once that it had been established that the blood supply and the velocity of blood flow in the vessels of the brain decreased regularly during deepening of hypothermia, an attempt was made to establish the cause of this phenomenon. During the period of elevation of the arterial pressure, which took place in the initial period of cooling, the blood supply to the brain diminished (Fig. 2). At other levels of hypothermia no direct relationship could be found between the arterial pressure, the volume of blood in the brain, and the velocity of the blood flow. At the end of cooling, when the arterial pressure was lowered and the velocity of blood flow decreased, the blood supply to the brain remained at a steady level.

Comparison of the changes in the volume of blood in the cerebral vessels and the velocity of its circulation with changes in arterial pressure revealed no direct relationship between these hemodynamic parameters, although at times they follow a parallel course. Beyond a certain depth of hypothermia, it appears that the predominant factor reducing the blood supply to the brain and the velocity of the blood flow in its vessels is a change in vascular tone.

The beginning of heating causes a sharp increase in amplitude of the REG. This increase is of short duration and is followed by a decrease. Under these conditions, the amplitude of the REG remains the same as at the end of hypothermia until the temperature has risen by 3-4° (rectal temperature 30-31°, brain temperature 25.6-27.1°). At the end of heating the amplitude rises, but as a rule it still remains below the initial level. Heating restores the normal circulation velocity. The technique of energetic heating to 33° (brain temperature 30.1°) used in this investigation results in a comparatively rapid increase in the circulation velocity of the blood. However, its value is always lower during heating than at the same temperature during cooling.

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