ELECTROENCEPHALOGRAPHIC CHANGES DURING GENERAL HYPOTHERMIA AND REGIONAL COOLING OF THE HEAD

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Two series of experiments were carried out on dogs. In series I (80 experiments) the animals were subjected to deep general hypothermia by covering the whole body with finely crushed ice. In series II (29 experiments) isolated deep hypothermia was produced in the head alone by means of a femoral-carotid arterial by-pass and cooling the blood in a heat exchanger. In both series the brain temperature of the animals was reduced to 18-22°C. Comparison of the electroencephalographic changes in the two series showed that cooling the brain leads to a reduction in its electrical activity evenly distributed among all frequencies in the EEG. Predominance of slow waves in the series with generalized hypothermia was not due to the effect of the low brain temperature, as other workers have previously but erroneously maintained, but to the reduction in the cerebral blood flow resulting from the decrease in the minute volume of the circulation accompanying general hypothermia, as other experiments carried out by the writers confirmed.

KEY WORDS: immersion hypothermia; perfusion hypothermia; EEG frequencies; EEG reduction; minute volume of the circulation.

Artificial hypothermia in various forms is nowadays quite widely used in clinical medicine. The chief indication for hypothermia in surgery is protection of the body as a whole or of a single organ from hypoxia during total or regional exclusion of the circulation in order to perform an operation under bloodless conditions. Operations on the dry organs are most frequently performed in cardiac surgery, but their use is also gaining ground in neurosurgery and in other fields. This has brought with it the need for a study of the pathophysiology of hypothermia. For example, it is very important to differentiate between the effects of depth of anesthesia, lowering of the temperature, elements of hypoxia, and other factors during operation for all can give rise to essentially similar changes in the EEG.

Many papers have been published on the character of changes in the amplitude and frequency parameters of the EEG at lowered temperatures. However, some aspects of the problem still remain highly debatable. For instance, Gurvich [1], on the basis of his experiments on dogs in Negovskii's laboratory, considers that hypothermia of a depth described in clinical practice as "moderate" (28-30°C), is characterized by a relative increase in the contribution of Δ-activity on the EEG. On the other hand, according to the observation of Fukukei et al. [6], also on dogs, cooling to 30°C does not significantly alter the frequency composition of the EEG. There are also disagreements in the evaluation of EEG changes during hypothermia in the experiments of Scott et al. [5] on monkeys and of Lipp [3] on cats.

Since the ultimate purpose of the present investigations was to establish criteria for assessing the state of the body as a whole during different forms of hypothermia used in clinical medicine, attempts were made to discover changes in the EEG that are due to hypothermia itself and not to the accompanying factors mentioned above.

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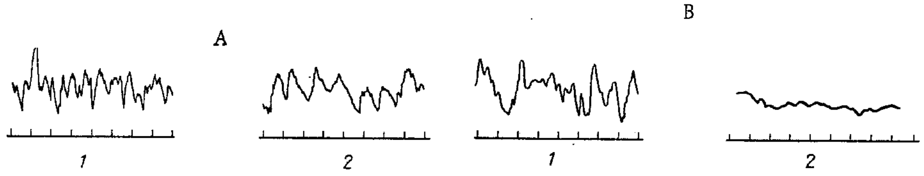

Fig. 1. EEG changes in deep general hypothermia (A) and deep isolated hypothermia of the head (B). In A: 1) initial EEG at 35.4°C; 2) EEG at maximal depth of hypothermia, 21.5°C. In B: 1) initial EEG at 36.9°C; 2) EEG at maximal depth of hypothermia, brain temperature 23.6°C.

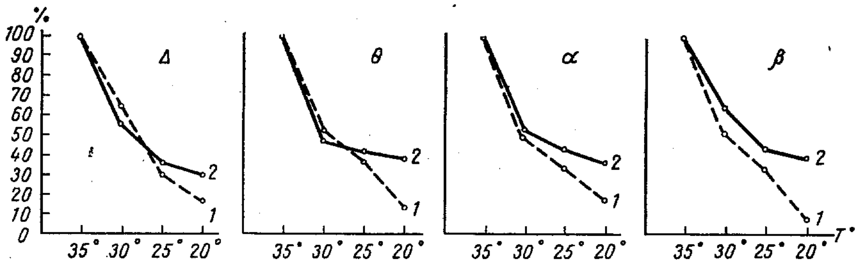

Fig. 2. Comparison of changes in brain electrical activity for EEG waves of different frequencies during deep general hypothermia and regional cooling of the head in experiments on dogs: 1) general hypothermia; 2) cooling of the head. Changes given as percentages of corresponding normothermic values.

EXPERIMENTAL METHOD

Two series of experiments were carried out on dogs of both sexes and of different body weights. In series I (80 experiments) the animals were subjected to deep general hypothermia produced by external cooling through the application of bags containing finely crushed ice to the whole body. In series II (29 experiments) deep perfusion hypothermia of the head was carried out by means of a femoral-carotid arterial by-pass with cooling of the blood in a heat exchanger. The pump of the AIK RP-64 artificial circulation apparatus and a Soviet heat exchanger were used in the experiments. During cranial hypothermia the rest of the body was warmed by means of a metal tubular heat exchanger introduced into the inferior vena cava [2]. In all other respects the experimental conditions were identical: premedication with morphine (0.5 mg/kg), intravenous hexobarbital (20 mg/kg) anesthesia to a depth of surgical stage II-III, monitored by clinical features and the EEG. The frequency composition of the EEG was as follows: On the average, 22% of the total electrical activity of the EEG was accounted for by α waves, 17% by β, 28% by θ, and 33% by Δ waves; the EEG was recorded on a Hungarian MB-5202 instrument.

The EEG was recorded with bipolar frontal parietal leads symmetrically from both hemispheres using needle electrodes. The frequency—amplitude characteristics of brain electrical activity were determined from the recordings by the analyzer of the electroencephalograph used and by subsequent integration of the frequency bands; the readings of the integrator for the various bands were aggregated and the resulting sum was taken as 100% (at the beginning of the experiment, normothermia) and the various terms (also expressed as percentages) showed the proportion of the total electrical activity contributed by the various frequency bands.

All temperature measurements were made with thermistor sensors of the electrothermometer of the RM-150 polygraph (Japan). Measurements were made at the middle third of the esophagus, in the rectum at a depth of 15 cm, and in the parietal cortex. The blood temperature also was measured in the arch of the aorta. The arterial and venous pressure, values of the acid—base balance, the minute volume of the circulation (MV; measured by Fegler's [3] thermodilution method), etc., also were recorded.

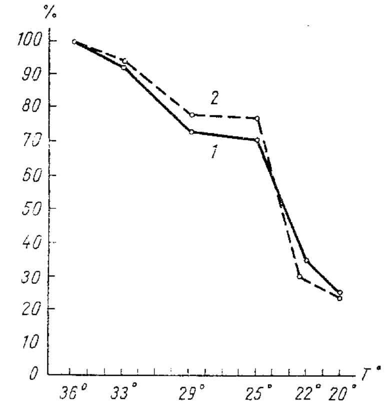

Fig. 3. Changes in heart rate per minute and MV in dogs during deep general immersion hypothermia: 1) heart rate, beats/min; 2) MV (mean values in experiments).

In both series of experiments the depth of cerebral hypothermia in the animals was 18-22°C. In the experiments of series I the general body temperature (in the esophagus and rectum of the animals) was the same, but in series II it was 35-36°C. In the experiments with general hypothermia the circulation was stopped in the whole body by clamping both venae cavae and the aorta, whereas in series II the cerebral circulation was stopped by clamping the vertebral and common carotid arteries when the maximal depth of hypothermia was achieved.

EXPERIMENTAL RESULTS

In the experiments with general hypothermia, at the stage of moderate hypothermia (30°C) the global brain electrical activity was almost halved: The α activity was 51.4%, β 52.7%, θ 52%, and Δ 61% of their initial level (mean values for the whole series). In the experiments with regional cooling of the head at the same level of hypothermia the global brain electrical activity was reduced by about the same degree, but the fast-wave component of the EEG, notably the β waves, remained at a higher level. The mean frequency values in these experiments were: α–56.8%, β 66.8%, θ 53.8%, and Δ 57.1% of the corresponding initial values.

In both series of experiments a further reduction in the animals' brain temperatures led to continued depression of the EEG, but it was more marked in the experiments of series I (Fig. 1) when the following changes were observed for the separate frequencies: α 17%, β 8.8%, θ 13.1%, and Δ 16.6% of the corresponding initial levels. In the experiments of series II, with the same depth of cerebral hypothermia (20-22°C), the decrease in brain electrical activity was more uniformly distributed among the frequencies (Fig. 2), so that the α activity was 37.8%, β 39.7%, θ 38.4%, and Δ activity 30.3% of their initial values.

At the stage of occlusion of the above-mentioned vessels in all the experiments for 15-25 sec after stopping the cerebral circulation the EEG waves disappeared completely. After resumption of the cerebral blood flow and restoration of normothermia, the initial brain electrical activity recovered completely.

The observations showed that in general hypothermia at the level of 30°C the α activity was reduced more than during regional hypothermia. As the depth of hypothermia increased, the difference became greater and reached a maximum in deep levels of hypothermia. On the whole, the changes in β activity followed the same pattern.

The dynamics was different in the slow-wave range. At the level of moderate hypothermia and down to 25°C the changes were more uniform and gradual in character. At deeper levels of hypothermia, however, the changes observed followed the same patterns as the α and β activities. It can be concluded from these results that, for the same brain temperature, differences in the intensity of depression of brain electrical activity are found in general and regional hypothermia. In an attempt to explain this phenomenon other parameters of the state of the body were compared and differences were found only in the minute volume of the circulation (MV), one of the principal hemodynamic indices, which fell rapidly in the experiments of series I as hypothermia deepened. At the lowest depths of hypothermia MV was only 40% of its initial value. The decrease in MV with a fall in temperature to 25-26°C took place mainly as a result of the development of bradycardia, a characteristic feature of general hypothermia. In the experiments of series II no bradycardia was observed in the animals, for the general body temperature was maintained at the normothermic level. Even at the greatest depth of cerebral hypothermia in the experiments with regional cooling MV on the average did not fall below 70% of its initial value, i.e., it fell by about the same amount as the demand of the brain on its arterial blood supply fell (Fig. 3).

It can be concluded that in general hypothermia, once the temperature reaches 28-30° but, more especially, at lower temperatures, there is a decrease in MV which is accompanied by a decrease in the cerebral blood flow; meanwhile, according to the findings of Rosomoff and Holaday [5], and of others, at the stage of moderate hypothermia the oxygen demand of the brain tissue is not significantly reduced (it falls on the average by 6.7 ± 1.3% for each degree of falling temperature). In general hypothermia the predominance of slow-wave activity in the EEG is thus largely determined by the mismatching between the cerebral blood flow and the body's requirements. In the form of local brain hypothermia used in the experiments of series II this mismatching did not take place, for the volume blood flow of the femoral-carotid arterial by-pass remained unchanged throughout the period of cerebral hypothermia (about 7 ml/kg/min). In the writers' view this explains the contradictory data in the literature on the character of the EEG changes during general hypothermia.

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