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BLOOD GASES IN CRANIOCEREBRAL HYPOTHERMIA

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Experiments on dogs showed that during craniocerebral cooling from 38 to 28°C the partial pressure of oxygen in the blood increases whereas that of carbon dioxide decreases. In deep hypothermia (24°C) the concentration of the blood gases is lower than at 28°C, but is still higher than initially. These changes are due to the long persistence of adequate pulmonary ventilation in the hypothermic organism.

KEY WORDS: hypothermia; partial pressure of oxygen; partial pressure of carbon dioxide.

An important advantage of craniocerebral hypothermia is its ability to depress the level of oxidative processes considerably. In this connection it is interesting to investigate the intensity of carbon dioxide formation and also the pattern of oxygen transport and utilization in the hypothermic organism. For this purpose the partial pressures of oxygen and carbon dioxide in the blood were studied in dogs during craniocerebral hypothermia.

EXPERIMENTAL METHOD

Experiments were carried out on 25 dogs weighing 10-15 kg. After trimeperidine premedication and intravenous hexobarbital anesthesia the animals were transferred to basal ether-air anesthesia. The animal's head was placed in the Kholod-2F factory-made hypothermic apparatus. Mixed venous blood and blood from the femoral artery were taken before exposure to cold and as the body temperature fell at 38, 36, 34, 30, 28, and 24°C. The blood gases were analyzed by the AZIV-2 apparatus. The partial pressure of oxygen in the arterial (p_aO_2) and venous (p_vO_2) blood was measured by means of the polarographic attachment to the apparatus. The partial pressure of carbon dioxide (p_aCO_2 and p_vCO_2) was determined by means of the Siggaard-Andersen nomogram, with correction for temperature by Rosenthal's method [9].

The depth and frequency of the respiratory movements, the minute volume of respiration (MVR), and the oxygen consumption were measured with the META 1-25 spirograph.

The results were subjected to statistical analysis by the Minsk-32 computer.

EXPERIMENTAL RESULTS AND DISCUSSION

As the body temperature fell from 38 to 28°C the partial pressure of oxygen in the arterial and venous blood increased. At the end of cooling (24°C) the partial pressure of the blood gases was lower than at 28°C, although still higher than initially (Table 1).

The pattern of oxygen transport is closely connected with the processes of entry of the gas into the body. During local brain cooling the depth and frequency of respiration change, so that MVR in the case of superficial or moderate hypothermia (36-32°C) was higher than initially, whereas during moderate or deep hypothermia it

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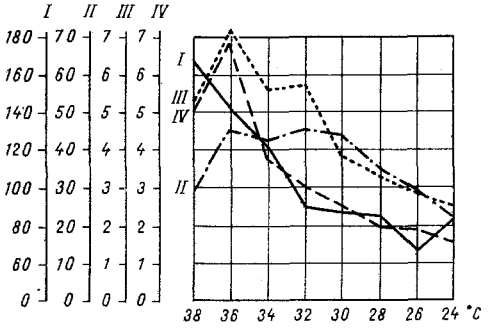

Fig. 1. Changes in depth (in ml, I) and frequency (II) of respiration, in MVR (in liters/min, III), and in oxygen consumption (in ml/min, IV) during craniocerebral hypothermia.

TABLE 1. Partial Pressures of Oxygen and Carbon Dioxide in Blood during Craniocerebral Hypothermia (M ± m)

Body temperature, °C	pO_2, mm Hg			pCO_2, mm Hg	
	arterial blood	mixed venous blood	arteriovenous difference	arterial blood	mixed venous blood
38	78,9±1,52	42,4±2,08	34,6±2,75	42,8±0,65	50,5±0,7
36	78,5±1,78	46,5±2,68	32,7±2,68	40,6±0,56*	50,0±0,91
34	83,5±1,8*	53,9±2,85*	27,6±2,09*	39,6±0,65*	46,6±0,73*
30	88,7±2,53*	65,7±2,6*	22,2±2,16*	38,3±0,41*	44,0±0,56*
28	98,4±2,52*	80,2±2,15*	16,9±1,26*	37,2±0,6*	42,4±0,61*
24	92,6±2,7*	72,8±2,16*	19,8±1,7*	42,1±0,85	47,0±1,0*

*P < 0.01 compared with initial value (at 38°C).

was 50-60% lower than initially (Fig. 1). The increase in the partial pressure of oxygen in the arterial blood observed at temperatures of 36-32°C was evidently due to an increase in MVR and slowing of the blood flow in the pulmonary capillaries [6], increasing the duration of contact between air and blood. With deepening of hypothermia to 30°C or lower, when MVR was reduced, the maintenance of p_aO_2 at above the normal level was due, in the writers' opinion, to a reduction by half of the circulating plasma volume [5] and to a 75% reduction in the rate of the capillary blood flow [6].

As hypothermia deepened, the partial pressure of oxygen in the venous blood rose faster than in arterial blood. The values of p_vO_2 and p_aO_2 became closer and the difference between them was reduced by half, evidence of a corresponding decrease in oxygen utilization by the tissues of the hypothermic organism. This was confirmed by direct determination of the oxygen consumption (Fig. 1).

The results do not agree with data in the literature in which the partial pressure of oxygen in the blood fell as the temperature was lowered [1, 2, 7, 8].

The disagreement between the present results and those described in the literature can be attributed to the use of different methods of cooling. Even if the same method of lowering the temperature was used, a change in the conditions of cooling (the rate of cooling, the depth of anesthesia) would be reflected in the efficiency of ventilation and, consequently, the state of the oxygen metabolism. In the present experiments, during local cooling of the brain, lowering the body temperature from 36 to 28°C led to a decrease in the partial pressure of carbon dioxide in the blood. This is evidence that the pulmonary ventilation was adequate for the needs of the body. That is why the value of pO_2 rose successively within the range of temperatures studied. Deepening the craniocerebral hypothermia to 24°C led to an increase in the partial pressure of CO_2 and a simultaneous decrease in pO_2, but even in this case they did not reach their initial levels: pO_2 remained 17.5% higher and pCO_2 5% lower than the normothermic values (Table 1). Investigation of the blood gases during general hypothermia [1, 2, 8] shows that carbon dioxide begins to accumulate in the blood during cooling of the body below 30°C. In the experiments cited 40% of the animals died when cooled to below 25°C because of respiratory failure, expressed as a high CO_2 concentration and low partial pressure of oxygen. In craniocerebral cooling experiments conducted in the writers' laboratory, spontaneous respiration persisted to a rectal temperature of 23-22°C and a brain temperature of 16.3°C, i.e., to temperatures at which connections with the respiratory center were interrupted.

Respiratory failure appears when the activity of the respiratory center is blocked by cold. This can be explained [3] by inhibition of the reticular structures of the medulla, which are closely connected functionally with the bulbar respiratory zone. When gradual blocking of the various parts of the respiratory center takes place during cooling, a disturbance of the coordinated regulation of the respiratory act develops and pathological forms of respiration appear. The degree of this disturbance of coordination is evidently determined by the method of cooling. In general hypothermia, when the brain is cooled by blood flowing from the internal organs, the depth of cooling and the order of inhibition of the various parts depend on the character of their blood supply. Variation in the time of extinction of functions is more marked than during craniocerebral hypothermia, when the source of cold is applied to the head. In the latter case, the lowering of the brain temperature, due mainly to the thermal conductivity of the brain tissue and its distance from the source of cold, takes place much faster and more uniformly than in the first case [4] and it is this which determines the long preservation of adequate respiration even during deep hypothermia.

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EFFECT OF EXTIRPATION OF AREA SI ON CORTICAL INTERACTION AND INTRAHEMISPHERIC RELATIONS

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Interaction between different cortical areas of the same hemisphere and intrahemispheric relations during application of stimuli of different modalities were studied by the evoked potentials method in acute experiments on unanesthetized cats immobilized with listhenon. In the intact brain influences of the somatosensory areas on visual cortical responses were shown to be mainly facilitatory in character, whereas these effects disappeared as the result of extirpation of area SI. It is concluded that functional reorganization of interhemispheric relations plays a role in the mechanisms of compensation after injury.

KEY WORDS: interhemispheric relations; compensation.

Despite many investigations [1-6, 9-13] the precise mechanisms of interhemispheric relations in the brain have not yet been explained. Nevertheless this problem is interesting from the standpoint of analysis of possible pathways of interaction and of replacement of the functions of individual systems and structures following injury. The study of disturbed brain functions from this aspect can shed light on the neurodynamic mechanisms of relations between symmetrical brain zones and may also make a contribution to the study of

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