Oxygen Tension of the Brain and its Modification with Hypothermia*

An Experimental Study

By

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With 8 Figures in the Text

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Although considerable attention has been directed towards the elucidation of the patho-physiologic factors observed when the body temperature is lowered, there has been a relative paucity of investigation in the area of tissue oxygen tension. The lack of such information is understandable, in view of the difficulties imposed by a direct approach, and the inherent inaccuracies of an indirect approach.

Previous investigations in our laboratory on animals and humans have indicated that continuous quantitative oxygen tension measurements of the cisternal or ventricular cerebrospinal fluid could be easily accomplished using a platinum microelectrode. Furthermore, it was observed that the oxygen tension of the cerebrospinal fluid quickly reflected changes in arterial $pO_2$, cerebral blood flow, and cerebral metabolism. Since the cerebrospinal fluid appeared to be in dynamic equilibrium with the brain and its circulation, it was thought that some advance in the knowledge of the oxygen tension changes of the central nervous system, during hypothermia, might accrue from the application of this technique.

Methods

Experiments were performed on 31 mongrel dogs, weighing an average of 13.8 kg. A total perfusion pump oxygenator technique was used to minimize the variable of cardiac output so that temperatures down to 10°C could be more easily explored. To decrease the effects of anesthesia on cerebral metabolism and normal vasomotor activity, the animals were intubated under light thiamylal sodium (Surital) anesthesia. During the period of preparation, the animals were immobilized with intramuscular succinyl choline and mechanically respirated.

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Oxygen tension of the brain in hypothermia

Using local anesthesia, the right thorax was opened through the fourth interspace and a 40 plastic catheter was inserted into the right atrium. The main pulmonary artery was encircled with a ligature to institute complete cardiopulmonary by-pass. The left atrium was decompressed with a catheter, which was drained by gravity into the venous reservoir. The Clowes membrane oxygenator was used for the majority of these experiments and the temperature of the blood was controlled with a Brown-Emmons heat exchanger interposed in the arterial line.

The sagittal sinus, internal carotid and femoral arteries were cannulated. The following parameters were measured continuously: quantitative cerebrospinal fluid oxygen tension via cisternal puncture with a Beckman platinum micro-electrode, blood pressure, central venous pressure, esophageal temperature, brain temperature, EKG, and EEG. Arterial and sagittal sinus PO2's were for the most part obtained by sampling. The sample oxygen tension determinations were made in an electrode-cuvette system which was thermostated to the animal's temperature within 0.2 of a degree centigrade. The oxygen electrodes were calibrated at the appropriate temperatures. Total cerebral blood flow determinations were made utilizing the Stewart principal. Indoeyanine green dye, 0.5 mgs, was injected into the internal carotid artery, and sagittal sinus concentration curves were obtained with a Water's densitometer by withdrawing blood at a constant rate. Observations were made on the effects of perfusion rates and total circulatory arrest at temperatures ranging from 37°C to 10°C. Sufficient time was allowed at each temperature level so that no temperature gradient existed between brain and esophagus.

Results

The relationship of parameters studied to decreasing temperatures (Fig. 1): The data from 82 observations, when expressed as percent change from control value, revealed that all parameters, except the cerebral metabolic rate of oxygen consumption (CMRO2) and the arterial oxygen content, shift with decreasing temperature in such a direction that one might anticipate a lowering of the ambient partial pressure of oxygen in the brain. The arterial oxygen content remained essentially unchanged from 37°C to 10°C and averaged 18.6 Vol-%. The blood pressure fell to 68% of the control value. The sagittal sinus oxygen tension fell from 37.7 mm Hg to 17 mm Hg, or to 46% of its initial value. A fall from 4.38 cm3/min to 0.472 cm3/min at 10°C was seen in the total cerebral oxygen consumption (CMRO2), or to approximately 10% of its 37°C control value.

Relationship of sagittal sinus oxygen content (SSO2) and sagittal sinus PO2 (SSpO2) to decreasing temperature (Fig. 2): The average of 111 observations of the SSO2 content rose progressively—as the temperature decreased—from 8.6 Vol-% at 37°C to 18.5 Vol-% at 10°C, an increase of 186%. Concomitantly the cerebral venous oxygen tension fell from 37.7 mm Hg to 17 mm Hg, a fall of 54%. The PO2 values at each temperature agree rather closely with the shift to the left of the oxygen dissociation curve of dogs' blood reported by Brown and Hill.

Effect of hypothermia on cerebral blood flow (CBF) regulatory mechanisms (Fig. 3): Sixteen observations were made on the effect of