Experimental Cerebral Venous Oxygen Tension during Raised Intracranial Pressure

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Cerebral circulation under raised intracranial pressure has recently attracted considerable attention.2,5,8,14 The results have been somewhat varied. Greenfield and Tindall2 found a clear-cut reduction in blood flow in the internal carotid artery in man at a cerebrospinal fluid pressure of 1.8 times the original pressure; Huber et al.5 found no definite pressure threshold at which internal carotid or vertebral blood flow decreased, and in some cases they even found an increase in blood flow when the intracranial pressure was moderately increased (monkeys, dogs); Langfitt et al.8 found an inverted linear relationship between the blood flow and the intracranial pressure, at least when the cardiovascular response had been abolished by section of the spinal cord (monkeys).

Some of these differences are no doubt due to variations in method. Greenfield and Tindall2 used hydrostatic pressures, whereas Huber et al.5 and Langfitt et al.8 used expanding intracranial balloons to raise the pressure.

The cerebral venous oxygen tension (PcvO2) is a function both of the cerebral blood flow and of the utilization of oxygen by the brain.4,9 The experiments reported here investigate changes in the PcvO2 caused by an artificially induced rise in intracranial pressure.

Methods

Rabbits were used in all experiments. Tracheotomy was performed under intravenous urethane supplemented with local anaesthesia. A catheter was threaded up into the aorta through the femoral artery for recording the blood pressure; another catheter was inserted into a femoral vein. The rabbit was then turned prone and a catheter was inserted into the spinal subarachnoid space through a lumbar laminotomy.

The bone over the confluens sinuum was removed with an electric drill (see Fig. 1 for the cerebral venous vasculature of the rabbit). A short piece of nylon tubing was glued to the surface of the confluens sinuum with Eastman 910 adhesive, and kept anchored by filling the defect in the bone with cold-curing acrylate.

The blood pressure was recorded with a Statham P23AA pressure transducer, fed by a UNA STV 12/1000 D.C. source, and the signal from the transducer was amplified in a Norma 709 D.C. amplifier. The oxygen tension was recorded with a Beckman oxygen microelectrode; this electrode was polarized by a Beckman 160 Physiological Gas Analyzer, which also served to amplify the signal from the electrode. The electrode was calibrated at the rectal temperature of the rabbit. A Norma direct-writing recorder was used.

When the surgical procedure had been completed, 5000 international units of heparin were given intravenously and a Riley needle was inserted into the confluens sinuum (Fig. 1) through the nylon tubing. It was then ascertained that venous blood could be withdrawn through the needle, and that the fluid level in the upper end of the needle pulsated with respiration. The microelectrode was then introduced into the Riley needle.

The intracranial pressure was raised by the application of hydrostatic pressure with physiological saline through the catheter in the spinal subarachnoid space; before hydrostatic pressure was applied, we always checked that fluid could flow freely through the catheter.

Results

Useful data were obtained from 22 animals. In 12 of the 13 animals that were breathing spontaneously, minute changes in PcvO2 were...
seen on the application of hydrostatic pressures up to 50 mmHg. A pressure of above 70 mmHg usually caused a definite drop in Pcvo₂. In the 13th animal, the surgical procedures had taken an unusually long time, and no rise in blood pressure was seen on the application of 50 mmHg hydrostatic pressure. There was a clear-cut reduction in Pcvo₂.

In the 4 animals that were maintained in a respirator, a drop in Pcvo₂ was at first seen in all (Fig. 2) when a hydrostatic pressure of 50 mmHg or over was applied. However, in these 4 animals the Pcvo₂ tended to return to more or less the original level when the cardiovascular response to intracranial hypertension appeared. Whenever the cardiovascular response was insufficient, the Pcvo₂ remained lower than before, until intracranial hypertension was released (Fig. 4) or until a vasopressor agent was administered (Fig. 3). In one animal, a hydrostatic pressure of 50 mmHg did not affect the Pcvo₂ very much; when the pressure was raised to 70 mmHg there was a very marked drop in Pcvo₂ (Fig. 4).

In 5 animals we measured the arterial oxygen tension (Pao₂) during intracranial hypertension and found, as one might expect, that Pao₂ rose during the hyperventilation induced by intracranial hypertension. Our measurements of Pcvo₂ in spontaneously breathing animals suggest that this rise in Pao₂ may be important for the maintenance of the usual Pcvo₂ during intracranial hypertension.

**Discussion**

Several questions concerning our methods should be answered. First, how did we know that the hydrostatic pressure applied through a catheter in the lumbar subarachnoid space would affect the brain? Before the experiments recorded in this paper, we did some experiments in which we removed the calvaria of the skull of the rabbit and applied pressure through a catheter in the spinal subarachnoid space. We found that the brain of the rabbit was always pushed out through the defect in the skull to some extent. Furthermore, the rabbits mostly showed the typical cardiovascular response to intracranial hypertension; in those few that did not, the Pcvo₂ went down.

Secondly, how did we know that there was good blood flow in the confluens sinuum? We always tested the response of the animal and the electrode by having the rabbit breathe nitrogen or re-breathe expired air or by stopping the respirator (see Fig. 2); in all the animals in this study, the response was adequate, i.e., the Pcvo₂ went down after a while, and rose to the previous level when the rabbit was adequately ventilated again.

![Fig. 2. Respirator rate 60/min. Record shows blood pressure (mmHg) and Pcvo₂(Po₂ mmHg). The respirator was stopped for about 2 minutes to ascertain the response of the electrode in the confluens sinuum. A hydrostatic pressure of 100 mmHg was then applied through a catheter in the lumbar subarachnoid space. At “hyperventilation” the rate of the respirator was raised to 120/min.](image)