The Effect of Halothane on Intracranial Pressure in Cerebral Tumors*

Report of Two Cases

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Halothane in anesthetic concentrations causes a marked increase in cerebrospinal fluid (C.S.F.) pressure as reported by Sondergard, Marx, Hunter, and McDowall, et al. The observations in our own study were made on patients with normal C.S.F. pathways who were anesthetized with nitrous oxide-oxygen during controlled ventilation to produce normocapnia. The addition of 0.5% Halothane to the anesthetic gases increased lumbar C.S.F. pressure in every case (Fig. 1), the mean increase being 68.2 mm H$_2$O (S.D. ±11.2 mm H$_2$O; statistical significance $P<0.001$). It was suggested that this rise in C.S.F. pressure might prove dangerous to patients in whom the pressure was already high due to an intracranial space-occupying lesion. Our report concerns the effect of Halothane on the intracranial pressure in two such patients.

Case Reports

Case 1. A 48-year-old man with headache of 2 months' duration was admitted to the hospital after noticing visual hallucinations on the left side for 3 weeks. On examination he had left homonymous hemianopia, mild left hemiparesis, and early papilledema; there was nystagmus and left-sided cerebellar ataxia. Carotid angiography suggested hydrocephalus, and air ventriculography showed a cerebellar tumor. At operation on the same day as the ventriculogram, a metastatic tumor was removed from the cerebellum; it was thought likely that there was at least one other metastasis in the right cerebral hemisphere to account for his visual signs.

Case 2. A 36-year-old woman was admitted to the hospital after 5 weeks of headache and vomiting. She had had a major epileptic fit during the puerperium 4 years earlier, and for 2 years had been slowing down mentally. She had marked papilledema; the only neurological sign was facial weakness. Angiography revealed a large frontal tumor. At operation this proved to be an extensive infiltrating glioma, reported histologically to be an anaplastic astrocytoma.

Method

Each case was premedicated with 0.6 mg atropine. Anesthesia was induced with a deep dose of thiopentone and, after the intravenous injection of suxamethonium chloride, auffed endotracheal tube was

* Supported by grants from the Medical Research Council and the Scottish Hospital Endowments Research Trust.

FIG. 1. Effect of Halothane on cerebrospinal fluid pressure in patients with normal C.S.F. pathways. (Reproduced from Anaesthesia by courtesy of the editor.)
Halothane Effects on Intracranial Pressure

passed. Controlled ventilation with nitrous oxide-oxygen was instituted, and tubocurare was administered at the earliest indication of returning muscle tone. A period of about 30 minutes was then allowed for stabilization during which the head was draped for craniotomy and a frontal burr hole was made. A metal brain cannula was inserted into the lateral ventricle through a burr hole and connected to a Statham pressure transducer, amplifier, and ultraviolet recorder. Stability of the intracranial pressure was assessed for 10 minutes, and Halothane (0.5% in Case 1 and 1.0% in Case 2) was then introduced. No further surgery was undertaken until all measurements of intracranial pressure were completed. Blood pressure was measured intermittently by upper arm sphygmomanometry, the systolic pressure being determined by palpation. The minute volume was measured with a Dräger Volumeter on the expiratory limb of the non-return anesthetic circuit. Arterial pH and PaCO₂ were measured on capillary blood before, during, and after the Halothane administration by the micro-Astrup technique (Andersen, et al.1). Esophageal temperature was monitored to allow correction of the arterial PaCO₂ (PaCO₂) for temperature, using Rosenthal's factor.

Results

Case 1. The intracranial pressure rose from 180/160 mms H₂O to 500/440 mms H₂O after 13 minutes of administration, at which time the Halothane was discontinued. In the following minute the intracranial pressure rose further to 520/460 mms H₂O, but thereafter fell rapidly towards the control value (Fig. 2). Throughout the period of measurement the PaCO₂ was 38 mm Hg, and the mean esophageal temperature was 36.3°C.

Case 2. After 7 minutes of 1% Halothane, intracranial pressure rose from 155/130 mm H₂O to 580/470 mm H₂O and the Halothane was then discontinued. During the next minute the intracranial pressure continued to rise precipitously, reaching a peak value of 800/620 mm H₂O, after which it returned rapidly to the control value (Fig. 3). During these measurements, PaCO₂ was 34 mm Hg; the mean esophageal temperature was 35.8°C; and the systolic blood pressure was

![Fig. 2. Effect of Halothane on cerebrospinal fluid pressure in Case 1.](image1)

![Fig. 3. Effect of Halothane on cerebrospinal fluid pressure in Case 2.](image2)