CEREBROSPINAL FLUID PRESSURES DURING HALOTHANE ANAESTHESIA*

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HALOTHANE (Fluothane†) is a valuable agent in the conduct of anaesthesia for neurosurgical procedures because of its physical and pharmacological properties. Its non-flammable and non-explosive nature allows the use of the electrocautery. The bronchodilating effect and the early obtundation of pharyngeal reflexes decreases the incidence of cough, secretions, and reactions to artificial airways. Its rapid action results in prompt awakening. This latter factor is of utmost importance in the differential diagnosis of prolonged or recurrent unconsciousness following intracranial interventions.

In patients with pathologically increased intracranial pressure, the possibility of further elevation due to the effect of drugs or anaesthetic management must be minimized. Intracranial pressure may be evaluated clinically by measuring cerebrospinal fluid pressure. The changes in cerebrospinal fluid pressure induced by different concentrations of halothane were measured in ten elderly patients prior to the onset of prostatic surgery.

METHOD

Eight patients over 60 years of age were medicated with atropine 0.4 mg.; two patients below 60 years of age received meperidine 50 mg. + atropine 0.4 mg. With the patient in the lateral recumbent position, a polythylene catheter (PE 50) was introduced into the intrathecal space between L_3 and L_4, and connected to a water manometer. The patient was then turned supine and placed on a specially constructed foam rubber cushion which prevented obstruction to the transmission of pressure through the catheter. An 18-gauge polyethylene catheter was inserted into an antecubital vein and connected by a three-way stopcock to an infusion of normal saline and to a water manometer. Arterial blood pressure was taken from the contralateral arm by the Riva Rocci method. Tidal volume was measured with a Wright ventilometer. Arterialized capillary blood[1] was drawn intermittently for pH and in three patients also for pCO_2 determinations (method of Astrup). All patients were given topical anaesthesia to pharynx and larynx, and two patients had nasopharyngeal airways inserted while still conscious.

Pressure readings were taken during a control period of 20 minutes or longer before the induction of inhalation anaesthesia. Five patients were anaesthetized

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†Fluothane is the trade name for halothane used by Ayerst Laboratories.

with a nitrous oxide (3 L.) – oxygen (3 L.) – halothane mixture, and five with oxygen (6 L.) – halothane. Respirations were assisted throughout. The agent was volatilized in a Fluotec vaporizer which compensates for both temperature changes and flow variations at flow rates of 4 to 14 litres/minute. Following the intravenous injection of 40 mg. of succinylcholine and ventilation with 100 per cent oxygen for 60 seconds, all patients were intubated with a cuffed endotracheal tube and were then given $\frac{1}{2}$ vol. per cent, 1 vol. per cent, $1\frac{1}{2}$ vol. per cent, and 2 vol. per cent of halothane, using a non-rebreathing system. Each concentration was administered for at least 15 minutes, and the sequence of concentrations was varied. Frequent tidal volume measurements were made in an attempt to keep ventilation constant. Cerebrospinal fluid, venous, and arterial pressures, and pulse rate were monitored at 5-minute intervals. The pH and pCO_2 were determined intermittently.

Results

During the control period, cerebrospinal fluid pressures in the supine position ranged from 200 to 320 mm. H_2O (average 244±32 mm. H_2O), venous pressures from 85 to 150 mm. H_2O (average 126±6 mm. H_2O), and mean arterial pressures from 63 to 117 mm. Hg (average 85±15 mm. Hg). During induction of anaesthesia, progressive rises in cerebrospinal and venous pressure occurred, which were influenced by the degree of excitement and muscular activity displayed by the patient.

During the intubation period, following the fasciculations induced by the intravenous administration of succinylcholine, increases in cerebrospinal fluid pressure ranged from 25 to 95 per cent (average 73±25), and elevations in venous pressure from 25 to 75 per cent (average 51±20) above control values. This was accompanied by a rise in mean arterial pressure in four patients (average +8%), and by a fall in five patients (average −15%). One patient showed no change.

The administration of different concentrations of halothane for periods of 15 to 25 minutes resulted in elevations of cerebrospinal fluid and venous pressures of approximately the same magnitude, and concomitant decreases in mean arterial pressure of greater magnitude (Table I). Figure 1 demonstrates that these changes were proportional to the concentration of halothane. Detailed results

TABLE I

Per Cent Change with Halothane

Concentration of halothane	Cerebrospinal fluid pressure	Venous pressure	Mean arterial pressure
1/2 vol. %	Average +7.1 (±6.1) Range 0 to +17	Average +8.4 (±7.1) Range 0 to +22	Average −22.3 (±14.3)* Range −5 to −36
1 vol. %	Average +10.7 (±6.5) Range 0 to +22	Average +12.4 (±2.3) Range +5 to +23	Average −30.1 (±12.6)† Range −11 to −51
1−1/2 vol. %	Average +18.5 (±11.1)* Range +4 to +35	Average +19.8 (±9.3) Range +8 to +38	Average −42.1 (±14.2)‡ Range −14 to −61
2 vol. %	Average +22.1 (±7.2)† Range +16 to +37	Average +26.3 (±8.8)† Range +16 to +38	Average −48.5 (±9.5)‡ Range −35 to −65

*P = 0.05. †P = 0.01. ‡P = 0.001.

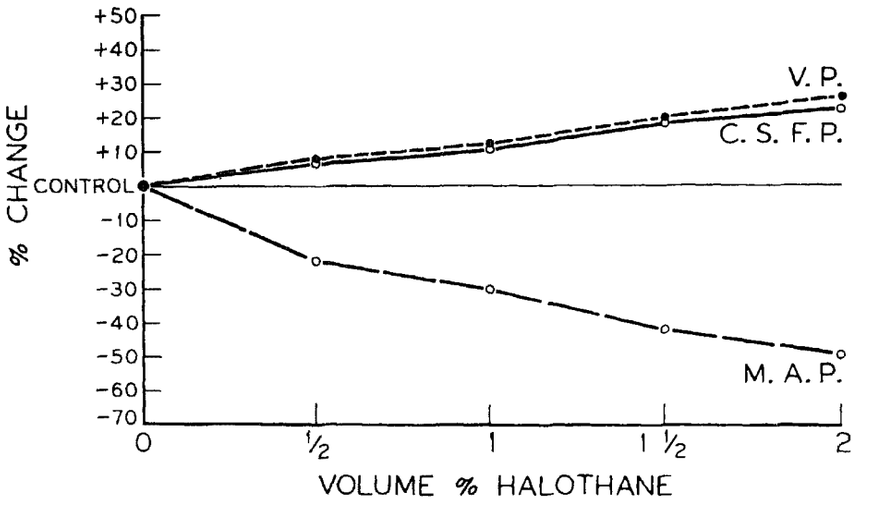


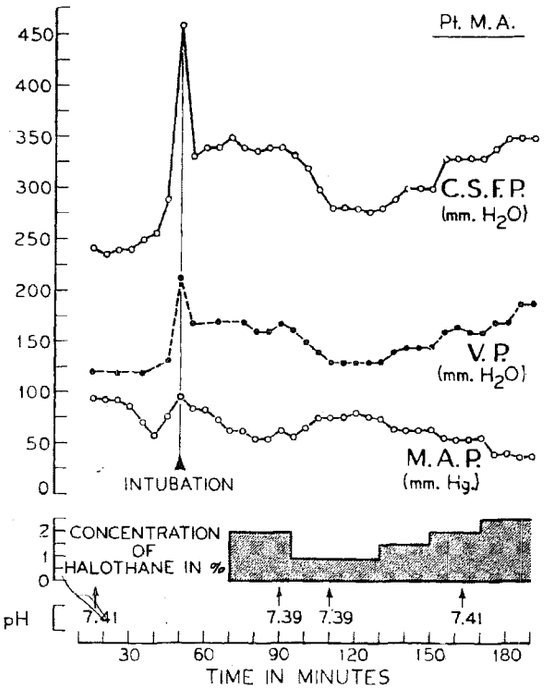
FIGURE 1. Increases in cerebrospinal fluid pressure (CSFP) and venous pressure (VP) and concomitant decreases in mean arterial pressure (MAP) are proportional to the concentrations of halothane anaesthesia (mean of 10 patients)

obtained in two patients are depicted in Figures 2 and 3. They show that the pressure changes are related to the concentration of halothane regardless of the direction in which the concentration is varied.

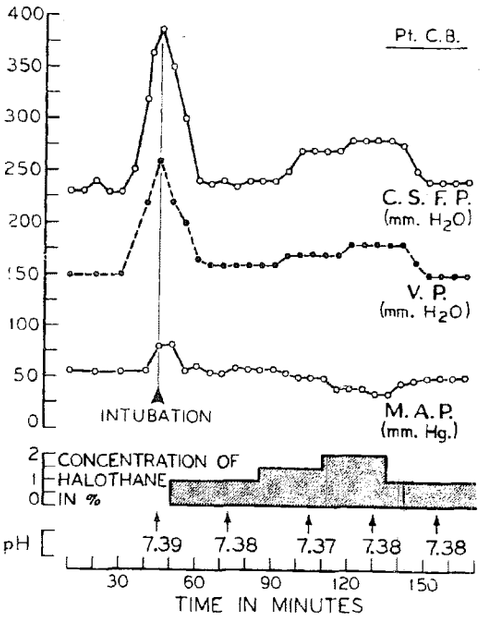
The pH and pCO_2 values remained within normal ranges. pH changes varied from none to 0.03 units, and pCO_2 from 1 to 3 mm. Hg. The calculated buffer base was unchanged at 46.0 mEq./L. in one patient, decreased from 45.5 to to 45.4 mEq. L. in another patient, and increased from 45.3 to 45.4 mEq./L. in a third patient.

DISCUSSION

Cerebrospinal fluid pressure is regulated essentially by the cerebral venous pressure,[2] which, in turn, is dependent upon the systemic venous pressure, the intrathoracic pressure, and the carbon dioxide and oxygen tensions in the blood.[3–5] In the absence of major alterations in intrathoracic pressure or blood gas values, "the systemic venous pressure may be considered a reference level for the craniospinal fluid pressure."[6] During sudden increases in intra-abdominal or intrathoracic pressures, the sharply rising cerebrospinal fluid pressure tends to exceed the venous pressure. This may be due to direct pressure of the dilated vertebral veins on the subarachnoid space[5] or to a direct transmission of the elevated intrathoracic pressure by way of the soft tissues.[7] During hypercapnia, the cerebrospinal fluid pressure increases rapidly and markedly, while the changes in venous and arterial pressures are inconsistent. Upon termination of the hypercapnia, all pressures return promptly to control levels. Cerebral vasodilatation caused by carbon dioxide has been proposed as the probable mechanism. During hypoxia, elevations of cerebrospinal fluid pressure are less than during hypercapnia and are accompanied by marked increases in venous and arterial pressures. Cerebral vasodilatation probably accounts for the changes here, too.[8]



FIGURES 2 (*top*) AND 3 (*bottom*). Charts of two typical patients demonstrating two types of cerebrospinal fluid pressure elevations. During endotracheal intubation, there is a sharp rise in cerebrospinal fluid pressure associated with a less marked increase in venous pressure. During inhalation of different concentrations of halothane, cerebrospinal fluid pressure, venous pressure, and mean arterial pressure changes are related to the concentration.



After the intravenous injection of succinylcholine into hyperventilated anaesthetized patients, cerebrospinal fluid pressures were observed to rise within one minute, whereas blood pressure and pulse rate remained practically unchanged. The rapid increase in cerebrospinal fluid pressure was assumed to be produced by an increase in cerebral blood flow.[9] Another likely explanation is the effect on the venous pressure of the clinical or subclinical fasciculations following the depolarizing action of succinylcholine. Still another possibility is the increase in abdominal pressure during the period of fasciculations.

The resting cerebrospinal fluid pressures obtained in this study fall within the range of normals observed in a previous investigation.[10] The control values for venous and mean arterial pressures are commensurate with the age of the patients (mean age 68±11 years). Two types of rise in cerebrospinal fluid pressure were noted. During endotracheal intubation, cerebrospinal fluid pressure increased sharply and markedly, while venous pressure lagged behind and arterial pressure changed in a variable manner. During the period of undisturbed halothane anaesthesia, elevations of cerebrospinal fluid pressure equalled those of the venous pressure, while arterial pressure declined concomitantly, but to a greater degree. The increases of cerebrospinal fluid and venous pressures and the decreases of arterial pressure were proportional to the concentration of halothane inhaled. The changes observed during endotracheal intubation may be explained by an interplay of three factors: (1) the absence of ventilation, resulting in accumulation of carbon dioxide; (2) the position of head and neck, causing an increase in venous pressure; and (3) the intravenous injection of succinylcholine, producing muscular fasciculations. The alterations seen during the steady state of halothane narcosis, on the other hand, are the result of the cardiovascular effects of the agent. Halothane is known to be a direct myocardial depressant, and to have a peripheral vasodilating and a ganglionic blocking action. One may hypothesize that the rise in venous pressure is an expression of the myocardial depression, whereas the decline in arterial pressure represents the possible summation of all three actions.

Increases in cerebrospinal fluid pressure during inhalation anaesthesia have been reported for ethyl chloride, ether, cyclopropane, halothane, and for nitrous oxide administered in concentrations above 60 per cent.[11–13] When venous pressure was also measured, both pressures were found to change synchronously and with approximately the same amplitude.[11] However, not all of the investigations considered the effects of disturbed respiratory physiology, so that some of the pressure changes may have been due to carbon dioxide accumulation secondary to hypoventilation. Sondergard[12] measured cerebrospinal fluid pressures for periods lasting less than 14 minutes in nine patients; he also determined the CO_2 percentage in inspired and expired air, but did not evaluate venous pressure. He stated that "in all cases, administration of fluothane was observed to produce increases in cerebrospinal fluid pressure. Withdrawal of fluothane resulted in a fall in intracranial pressure provided the CO_2 was kept constant." He concluded that the increased intracranial pressure might be caused by a "dilating effect upon the cerebral vessels with increased cerebral blood flow on account of the sympatholytic effect" of the agent. In our study, successful efforts were made

to prevent hypercarbia and hypoxia. Muscular activity and trauma were avoided, since the investigation was completed prior to the beginning of surgery. Thus our results demonstrate that the increases in cerebrospinal fluid pressure due to halothane *per se* are related solely to increases in systemic venous pressure.

Conclusion

In the course of a neurosurgical anaesthesia, the most acute rise in cerebrospinal fluid pressure is apt to occur during the placement of the endotracheal tube, even in a well-oxygenated patient and in the absence of straining or coughing. This increase, however, is of short duration. Gradual but persistent elevations in cerebrospinal fluid pressure develop during maintenance of anaesthesia and are basically parallel to concomitant rises in systemic venous pressure. Thus, in patients undergoing neurosurgical procedures, halothane, whether used as the sole agent or in conjunction with nitrous oxide, should be administered in the lowest possible concentration that will permit the surgical manipulations.

It may be implied that, in patients who have a markedly raised intracranial pressure, the use of halothane should be preceded by steps to reduce the pressure, since the magnitude of a change in cerebrospinal fluid pressure due to a given change in cerebral blood flow is greater whenever the original pressure is elevated.[3] It may also be advocated that the administration of halothane in large amounts to produce hypotension during neurosurgical procedures should be postponed until after the dura mater has been opened.

Summary

Changes in cerebrospinal fluid pressure, venous pressure, and arterial pressure induced by different concentrations of halothane were measured in ten elderly patients prior to the beginning of surgery. Cerebrospinal fluid and venous pressures were recorded by a water manometer and arterial pressure with a sphygmomanometer. Tidal volume, pH, and pCO_2 were determined intermittently in order to prevent changes in respiratory physiology. The patients were intubated with a cuffed endotracheal tube and given $\frac{1}{2}$ to 2 vol. per cent halothane, for periods of 15 minutes or longer, using a non-rebreathing system and a Fluotec vaporizer.

Two types of increase in cerebrospinal fluid pressure were noted. During endotracheal intubation, cerebrospinal fluid pressure rose sharply while venous pressure lagged behind and arterial pressure changed in a variable manner. During the period of undisturbed halothane anaesthesia, elevations of cerebrospinal fluid pressure corresponded with increases in the venous pressure, while arterial pressure decreased concomitantly. The magnitude of these changes was proportional to the concentration of halothane.

The implications of these findings on the administration of halothane anaesthesia during neurosurgical operations are discussed.

RÉSUMÉ

Chez dix malades âgés, nous avons mesuré avant le début de la chirurgie les changements de la pression du liquide céphalorachidien, du sang veineux et du sang artériel, qui surviennent au cours de l'induction de l'anesthésie au fluothane à diverses concentrations. Les pressions du liquide céphalorachidien et du sang veineux ont été prises au moyen d'un manomètre à l'eau et la pression du sang artériel au moyen d'un sphygmomanomètre. Pour éviter des changements dans la physiologie respiratoire, nous avons calculé, de façon intermittente, l'air courant, le pH et le pCO_2. Tous les malades étaient intubés avec un tube à ballonnet et, durant 15 minutes ou plus, recevaient du fluothane à des concentrations variant de $\frac{1}{2}$ à 2 vol. per cent, en employant la vaporisateur fluotec et un système sans réinspiration.

Nous avons observé deux sortes d'augmentation de pression du liquide céphalorachidien. Au cours de l'intubation endotrachéale, la pression du liquide céphalorachidien s'est élevée en flèche, la pression du sang veineux est demeurée basse et la pression du sang artériel a présenté des variantes. Au cours de la période de distribution de l'agent anesthésique, les augmentations de la pression du liquide céphalorachidien correspondaient aux augmentations de la pression du sang veineux, pendant que la pression du sang artériel diminuait au même moment.

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