Cerebrovascular Effects of Cerebrospinal Fluid Removal

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SUMMARY

The effects on cerebral blood flow of withdrawal of graded volumes of cerebrospinal fluid (CSF) from the cisterna magna were studied in 12 barbiturate-anaesthetized baboons. The results show that withdrawal of CSF was accompanied by a decreased CSF pressure, an increased CSF PCO2, a decreased CSF PO2 and an increased cerebral blood flow. The possible clinical significance of these findings is pointed out.


Withdrawal of cerebrospinal fluid (CSF) is accompanied by a decrease in CSF pressure in proportion to the volume of fluid aspirated. This decrease in CSF pressure effectively increases the cerebrovascular perfusion pressure, since this perfusion pressure = mean arterial blood pressure — (cerebral venous pressure + CSF pressure). Davson maintains that such a procedure does not affect cerebral blood flow. This seems reasonable, since the perfusion pressure changes would fall well within the limits of cerebrovascular 'autoregulation'. There are several studies which support this hypothesis. While most show no significant alteration of blood flow, a recent study indicated that withdrawal of CSF is accompanied by a slight, non-significant increase in blood flow, which was possibly due to alteration of cerebral metabolism. Other recent studies have also demonstrated that cerebral blood flow may be decreased in patients with Alzheimer's disease after removal of CSF, while in patients with normal pressure hydrocephalus, this procedure results in an increased cerebral blood flow. Taking the above into account and seeing that it is important to know whether CSF removal during spinal puncture or other procedures is associated with an increase or decrease in cerebral blood flow, we have investigated flow changes with graded withdrawal of CSF volume in normal baboons, and have attempted to correlate changes in cerebral blood flow with CSF PCO2, PO2 and pH.

MATERIALS AND METHODS

Cerebral blood flow was measured in 12 barbiturate-anaesthetized adult baboons of both sexes, by the intracarotid xenon-133 injection method, using the methods described previously. The 133Xe was injected into the internal carotid artery via a catheter retrograde in the lingual artery. The cerebral uptake and clearance of the bolus of xenon were monitored with a highly collimated 50-mm diameter sodium iodide detector placed over the parietal region. Blood flow was measured from each clearance curve by measuring the slope of the initial 2 minutes of this curve and calculating flow according to the methods of Kanno and Vemura.

The animals were intubated and ventilated with a positive pressure respirator in order to maintain arterial blood PO2, PCO2 and pH within normal range for this altitude. These blood gases were measured before each blood flow determination using an IL 313 blood gas analyser, and end-tidal expired air carbon dioxide content was continuously monitored by a Goddard capnograph. Arterial blood pressure was recorded via a catheter placed in a femoral artery and a Statham transducer. CSF pressure was monitored by cisternal puncture with a Statham pressure transducer and the needle also served to withdraw CSF. The experimental procedure was as follows: two baseline cerebral blood flow measurements were made and then an initial 2 ml of CSF was rapidly withdrawn. The sample was used to determine baseline CSF PO2, PCO2 and pH. Immediately after withdrawal of this CSF sample, blood flow was determined again. Thereafter, further CSF samples of increasing volumes were withdrawn, and blood flow was determined after each withdrawal. A 45-minute recovery period was allowed between each withdrawal and the samples were used to determine the CSF PO2, PCO2 and pH relating to the respective previous cerebral blood flow determinations (see Discussion).

RESULTS

Fig. 1 shows the alterations in CSF pressure, CSF PO2, CSF PCO2 and cerebral blood flow after removal of graded volume of CSF. It should be noted that CSF PO2, PCO2 and pH values could only be determined after a CSF sample had been withdrawn. Therefore, the control values were obtained from the first 2 ml withdrawn. The effects of withdrawal of that 2 ml on CSF gas values were then assessed after the next sample was obtained and so on (see Discussion).

Baseline values for cerebral blood flow were 36,62 ± 1.65 ml/100 g/min, for CSF pressure 14,00 ± 1,11 mmHg, for CSF PCO2 31,34 ± 4,10 mmHg, for CSF PO2 84,91 ± 5,17 mmHg and for CSF pH 7,36 ± 0,06 (means ± SE). Fig. 1 shows that with graded CSF withdrawal there was a progressive reduction in CSF pressure, a decreased CSF PO2, increased CSF PCO2 and an increased cerebral blood flow (when 3 ml or more of CSF was removed). There was no change in CSF pH, the value remaining constant at 7,36 ± 0,06. The increase in blood flow after removal of CSF was correlated with the CSF pressure, the CSF PO2 and the CSF PCO2, with statistically significant correlation coefficients of −0,93, −0,81 and 0,67 respectively.

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In a further series of six experiments we investigated the time required for cerebral blood flow to return to normal after 4 ml of CSF had been withdrawn. It was found that a period of 20-30 minutes was necessary for a return to baseline values. This coincided with a slow return to normal of CSF pressure, although this did not reach the prewithdrawal values at the end of the 30-minute period.

There was no significant alteration in blood pressure, blood Po, blood Pco, or pH during the experiments reported above.

**DISCUSSION**

Cerebral blood flow is primarily dependent on the arterial inflow pressure, the venous pressure, the CSF pressure and the cerebrovascular resistance.

Elevation of arterial pressure from the normal value of about 100 mmHg to 140 mmHg does not significantly alter cerebral blood flow. This represents an elevation of perfusion pressure by 40 mmHg if cerebral venous pressure and CSF pressure remain constant, which is compensated for by an increase in cerebrovascular resistance in order to maintain cerebral blood flow. In the present experiments the maximal drop in CSF pressure was 10.5 mmHg and this, therefore, represents an increase in perfusion pressure which is much less than the 40 mmHg needed to overcome normal cerebrovascular 'autoregulation'. Despite this we have recorded a significantly increased cerebral blood flow with decreased CSF pressure and have also shown that these two variables are significantly correlated. Thus the blood flow did not 'autoregulate' in response to this small increase in perfusion pressure. The present increments in blood flow have also been correlated with the accompanying increase in CSF Pco, and decrease in CSF Po, due to the fact that the effects of CSF withdrawal on these two variables could only be assessed after a period of 45 minutes, it may be that the Po, and Pco, changes in CSF directly after sample withdrawal were of much greater magnitude and better correlated with the changes in cerebral blood flow. Indwelling sensors, lodged in the cisterna magna, which could record Po, Pco, and pH, would indicate the immediate changes in these variables after CSF removal. These were, however, not available to us.

Miyakawa et al. have shown a similar trend to increased cerebral blood flow with CSF withdrawal, although the increase was not significant. A possible explanation for the difference between their results and ours may be that they only withdrew a single sample of 5-6 ml. In our experiments we withdrew graded volumes of CSF and the total amount of fluid withdrawn was greater than 6 ml. It is possible that this manoeuvre may alter cerebral metabolism, seeing that CSF withdrawal in normal animals is known to be associated with dilation of pial veins and venules and constriction of pial arteries. The decrease in CSF Po, and increase in Pco, which are directly influenced by the brain Po, and Pco, are thus possibly a consequence of altered cerebral metabolism and this altered metabolism causes the change in cerebral blood flow. Alternatively, removal of CSF is known to result in increased CSF production. This newly formed fluid may have a high Pco, which would result in an increased cerebral blood flow.

These observations may have clinical implications; if CSF is withdrawn during spinal, cisternal or ventricular puncture, it cannot be assumed that cerebral blood flow will not change because autoregulation will take care of the perfusion pressure. The present study has shown that there may be changes in cerebral blood flow which are accompanied by changes in CSF composition.

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**REFERENCES**