The concentration of cerebrospinal fluid potassium during systemic disturbances of acid-base metabolism

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SYNOPSIS  A possible relationship between blood acid-base state and the concentration of cerebrospinal fluid potassium has been examined in patients with systemic disturbances of acid-base metabolism. Over the range of values studied it was not possible to demonstrate any significant correlation between these parameters.

A decrease in the concentration of cerebrospinal fluid (CSF) potassium has been demonstrated in patients after subarachnoid haemorrhage and a stroke (Sambrook, Hutchinson, and Aber, 1973). Although these patients also developed a systemic alkalosis no significant correlation could be demonstrated between changes in CSF potassium concentration and arterial blood acid-base values. In order to investigate further the possible relationship between arterial blood pH and CSF potassium a study has been made in patients with systemic disturbance of acid-base metabolism.

Patients Studied

Reference values for blood and CSF acid-base parameters and potassium concentration were obtained from 40 control patients who had a lumbar puncture performed during the course of routine neurological investigations but in whom no significant central nervous system pathology was subsequently demonstrated. Samples of CSF were rejected if the white cell count was greater than 4/mm³ or the protein concentration in excess of 40 mg/100 ml.

Ten patients with systemic acid-base disorders were studied (table 1). Four patients had chronic renal failure and were observed during acute and subacute disturbance of acid base. Patients with primary hyperaldosteronism, Cushing's syndrome, and liver cirrhosis had stable blood acid-base values for at least seven days before obtaining simultaneous samples of blood and CSF whereas in the remaining three patients the abnormalities of acid base had developed acutely within 24 hours. The purpose of the investigation was explained to patients or their relatives before permission for CSF examination was requested.

Methodology

Simultaneous samples of lumbar CSF and femoral arterial and antecubital venous blood were obtained for measurement of CSF and blood acid-base values and potassium concentration. Samples of CSF were withdrawn anaerobically into a glass syringe and sealed with a metal cap, the dead space of the syringe having first been filled with the patient's CSF before taking the full sample.

Measurements of arterial blood and lumbar CSF pH were made with a G.9027/2 microelectrode and PCO₂ with a direct reading E.5036 Severinghaus electrode using PHM 27 meter and gas monitor PHA 927b (Radiometer Company, Copenhagen). The G.9027/2 microelectrode was calibrated with BDH precision buffers (pH 6-850, 7-380, and 7-420) and the PCO₂ electrode with special gas preparations of 4 and 8% CO₂. Analyses were performed within 20 minutes of taking samples. The bicarbonate concentration of arterial blood was derived from the Siggard-Anderson nomogram and for CSF it was calculated from the Henderson-Hasselbalch equation using a solubility coefficient for CO₂ in CSF of 0-0312 mmol/1 and a pK value of 6-13 (the mean of values calculated by Alexander, Gelfand, and Lamberts, 1961; Cowie, Lambie, and Robson, 1962; Mitchell, Carman, Severinghaus, Richardson, Singer, and Shnider, 1965; Alroy and Flenley, 1967).

The concentration of potassium in CSF and plasma was estimated by flame photometry using an EEL 150 clinical flame photometer.

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Results

Reference values for blood and CSF acid-base measurements and potassium concentration established in 40 control patients are given in table I and agree well with the results of previous workers (Bradbury, Stubbs, Hughes, and Parker, 1963; Huang and Lyons, 1966). It is noteworthy that the mean values and range for CSF potassium were much smaller than those for plasma.

The biochemical findings in patients with systemic disturbances of acid-base are detailed in table II. The range of values for arterial blood pH was 7.15-7.51, actual blood [HCO₃⁻] 10.38 mmol/l and PaCO₂ 2000-6800 Nm⁻² (15-51 mm Hg) and for CSF pH 7.30-7.38, CSF [HCO₃⁻] 10.9-27.2 mmol/l and Pcsf CO₂ 3066-7600 Nm⁻² (23-57 mm Hg). Except in three patients values for CSF potassium concentration remained within the range established in control subjects and were not influenced by

![Graph](http://jcp.bmj.com/)

**Figs 1 and 2** Simultaneous values for CSF potassium concentration and (1) blood pH and (2) PaCO₂ in 10 patients with systemic disturbance of acid-base metabolism (31 estimations)

### Table I

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Cerebrospinal Fluid</th>
<th>Blood/Plasma</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.33±0.02 (7.30-7.36)</td>
<td>7.41±0.03 (7.37-7.45)</td>
</tr>
<tr>
<td>[HCO₃⁻] (mmol/l)</td>
<td>22.2±1.3 (19.8-24.8)</td>
<td>25.8±2.3 (23.0-29.4)</td>
</tr>
<tr>
<td>PCO₂(Nm⁻¹)</td>
<td>6240±440 (5600-7200)</td>
<td>5426±333 (4800-6400)</td>
</tr>
<tr>
<td>Potassium (mmol/l)</td>
<td>2.88±0.13 (2.6-3.1)</td>
<td>3.95±0.38 (3.4-4.7)</td>
</tr>
</tbody>
</table>

**Table I** Values for CSF and plasma acid-base parameters and potassium concentration in 40 control patients

### Table II

<table>
<thead>
<tr>
<th>Patient</th>
<th>Diagnosis</th>
<th>pH</th>
<th>Actual [HCO₃⁻] (mmol/l)</th>
<th>PCO₂(Nm⁻¹)</th>
<th>Potassium (mmol/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 G. P.</td>
<td>Chronic renal failure*</td>
<td>7.315</td>
<td>7.190</td>
<td>16.0</td>
<td>12.0</td>
</tr>
<tr>
<td>2 A. B.</td>
<td>Chronic renal failure*</td>
<td>7.375</td>
<td>7.150</td>
<td>18.0</td>
<td>11.0</td>
</tr>
<tr>
<td>3 M. S.</td>
<td>Chronic renal failure*</td>
<td>7.325</td>
<td>7.470</td>
<td>26.0</td>
<td>35.0</td>
</tr>
<tr>
<td>4 R. T.</td>
<td>Chronic renal failure*</td>
<td>7.340</td>
<td>7.280</td>
<td>21.2</td>
<td>16.5</td>
</tr>
<tr>
<td>5 A. B.</td>
<td>Primary hyperaldosteronism*</td>
<td>7.335</td>
<td>7.480</td>
<td>27.2</td>
<td>38.0</td>
</tr>
<tr>
<td>6 J. B.</td>
<td>Cushing's syndrome</td>
<td>7.380</td>
<td>7.510</td>
<td>26.0</td>
<td>34.0</td>
</tr>
<tr>
<td>7 J. E.</td>
<td>Mandrax overdose*</td>
<td>7.300</td>
<td>7.300</td>
<td>23.4</td>
<td>24.6</td>
</tr>
<tr>
<td>8 E. W.</td>
<td>Salicylate overdose</td>
<td>7.330</td>
<td>7.440</td>
<td>10.9</td>
<td>10.0</td>
</tr>
<tr>
<td>9 J. K.</td>
<td>Liver cirrhosis</td>
<td>7.380</td>
<td>7.420</td>
<td>19.1</td>
<td>19.1</td>
</tr>
<tr>
<td>10 A. S.</td>
<td>Gastroenteritis</td>
<td>7.355</td>
<td>7.330</td>
<td>22.0</td>
<td>20.8</td>
</tr>
</tbody>
</table>

**Table II** Diagnosis and values for CSF and plasma acid-base parameters and potassium concentration in 10 patients with systemic disturbance of acid-base metabolism

*Several measurements were made in six patients* during their illness and the tabulated values are those recorded during the period of maximum acid-base disturbance.
Changes in either blood or CSF, pH, P\textsubscript{CO\textsubscript{2}}, or [\text{HCO}_3^-] (figs 1 and 2). The mean concentration of CSF potassium in patients with values for blood pH below 7.40 was 2.93 ± 0.13 mmol/l and for pH 7.40 and above was 2.88 ± 0.25 mmol/l (not significantly different from values in controls). Values for plasma potassium ranged from 1.89 to 5.7 mmol/l.

**Discussion**

Changes in the concentration of plasma and extracellular fluid potassium occur with systemic disturbances of acid-base metabolism and are due to both renal and extrarenal mechanisms (Berliner, Kennedy, and Orloff, 1951; Keating, Weichselbaum, Alanis, Margraf, and Elman, 1953; Black, 1967). Since the concentrations of ions in cerebral extracellular fluid and CSF are similar (Fencel, Miller, and Pappenheimer, 1966) the possible influence of disturbances of systemic acid-base metabolism upon CSF potassium concentration has been studied. Measurements were made in patients with respiratory and metabolic alkalosis and acidosis and where possible several observations were recorded during the patient’s illness. The results failed to show any consistent pattern of change in the concentration of CSF potassium over the range of blood and CSF acid-base parameters studied.

The absence of any demonstrable influence of blood pH upon CSF potassium may be due to two factors. First, an electrical potential difference exists between CSF and plasma which is sensitive to changes in blood pH, and second changes in blood pH are often accompanied by relatively small alterations in that of cerebrospinal fluid.

Tschirgi and Taylor (1958) and Held, Fencel, and Pappenheimer (1964) demonstrated that CSF was 4.5 m volts positive in relation to plasma and this potential difference increased during acidosis and decreased and became negative during alkalosis. An increase in CSF-plasma potential difference would oppose the movement of cations into the cerebral extracellular compartment whilst a decrease would oppose their exit. Thus in systemic acidosis an increased movement of potassium ions from intra-to extracellular fluid would be opposed by an increase in electrical potential difference. The reverse situation would occur in systemic alkalosis.

Changes in blood pH are associated with smaller changes in pH of CSF (Bradley and Semple, 1962; Pauli, Vorburger, and Reubi, 1962; Mitchell et al, 1965; Posner, Swanson, and Plum, 1965), and it is possible that the flux of potassium ions between cerebral extracellular fluid and cerebral parenchyma is influenced by changes in blood pH to a much smaller degree than in other tissues. In the patients studied only three had values for CSF pH outside the range established in controls but even in these the concentration of CSF potassium was normal.

Carbon dioxide rapidly diffuses between blood and CSF and the possibility that the concentration of CSF potassium could be related to changes in Pa\textsubscript{CO\textsubscript{2}} was examined. Although the concentration of CSF potassium was lowest (2.5 and 2.7 mmol/l) in the two patients with lowest values for Pa\textsubscript{CO\textsubscript{2}} (2000 and 4000 Nm\textsuperscript{-2}) (15 and 30 mm Hg) there was no significant overall correlation between them.

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**References**


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