Body Fluid Oxygen Tension and Prognosis in Patients With Ruptured Aneurysm

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SUMMARY  Body fluid gas pressure and electrolytes of patients with ruptured aneurysm were continuously analyzed. Intracranial pressure (ICP) was regulated at the level of 120–100 mm H2O by cerebral ventricular drainage. There was no significant change in the pH, PCO2, HCO3, Na+, K+, Ca++ in the cerebrospinal fluid (CSF) of patients with slight or moderate disturbance of consciousness (lethargic-drowsy state). The PcorO2 of the patients with marked disturbances of consciousness (semicoma-coma) was significantly low. PcorO2 of the patients with cerebral vasospasm was significantly lower than for those without vasospasms. PcorO2/Pao2 was 0.27 ± 0.01 in the patients with vasospasm and 0.50 ± 0.01 in those with vasospasm. PcorO2 tended to decrease in patients with markedly bloody CSF. When the bloody CSF was cleared by ventricular drainage, PcorO2 increased. PcorO2 did not return to a normal value in the patients with marked disturbances of consciousness despite sufficient arterial oxygen tension. This suggests that PcorO2 and PcorO2/Pao2 should provide a convenient index for the prognosis of patients with ruptured aneurysm.

DISRUPTION OF consciousness is an important clinical symptom in the acute phase of a ruptured aneurysm. It may be due to an acute increase of intracranial pressure and/or cerebral vasospasm.

Oxygen tension P02 in cerebrospinal fluid (CSF) is one index of oxygen availability to brain tissue, but a decreased P02 value may not always reflect brain hypoxia.

Vasospasm occurs at various intervals after subarachnoid hemorrhage leading to ischemic hypoxia. Some studies suggest a chemical factor for the origin of vasospasms.¹

The present study was designed to evaluate the independent actions of intracranial pressure controlled by ventricular drainage, and to find more convenient indexes of prognosis for ruptured aneurysm.

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Methods

Eleven patients with ruptured aneurysm were examined and the clinical findings are shown in table 1. Patients 5 and 7 had multiple aneurysms. All patients were on continuous ventricular drainage and intracranial pressure (ICP) was regulated at a level of 120–100 mm H2O. Cerebral vasospasm was diagnosed by the criteria of Wilkins et al.² Clinical symptoms were graded by Hunt’s classification at admission as: 8 patients grade III, 2 grade IV, and 1 grade V. The operation (neck clipping of the aneurysm) was performed in 9 patients. Four patients died as a result of cerebral vasospasm and pulmonary disease.

In both CSF and femoral arterial blood pH, PCO2, HCO3 and Base Excess (BE) were examined simultaneously. Na+, K+, Cl−, Ca++ and total protein in both CSF and peripheral venous blood were also examined at the same time. The study was continued for a maximum of 70 days consisting of 8 patients with hypertensive intracerebral hemorrhage, 6 patients with brain tumor and 7 patients with miscellaneous neurological diseases were examined using the same procedures.
Table 1  Case Summary

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Location of aneurysm</th>
<th>Grade* at admission</th>
<th>Consciousness level at admission</th>
<th>Progress course</th>
<th>Vasospasm</th>
<th>Operation (clipping)</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>64</td>
<td>f</td>
<td>A com A</td>
<td>IV</td>
<td>semicoma</td>
<td>(downhill)</td>
<td>+</td>
<td>-</td>
<td>die</td>
</tr>
<tr>
<td>2</td>
<td>42</td>
<td>f</td>
<td>MCA</td>
<td>III</td>
<td>lethargic</td>
<td>(uphill)</td>
<td>-</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>61</td>
<td>f</td>
<td>MCA</td>
<td>III</td>
<td>lethargic</td>
<td>(downhill)</td>
<td>+</td>
<td>-</td>
<td>die</td>
</tr>
<tr>
<td>4</td>
<td>63</td>
<td>f</td>
<td>A com A</td>
<td>III</td>
<td>drowsy</td>
<td>(uphill)</td>
<td>-</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>50</td>
<td>m</td>
<td>MCA, PCA</td>
<td>III</td>
<td>drowsy</td>
<td>(downhill)</td>
<td>+</td>
<td>+</td>
<td>(with coating)</td>
</tr>
<tr>
<td>6</td>
<td>69</td>
<td>f</td>
<td>A com A</td>
<td>III</td>
<td>drowsy</td>
<td>(uphill)</td>
<td>+</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>59</td>
<td>f</td>
<td>MCA, IC-PC</td>
<td>III</td>
<td>drowsy</td>
<td>(uphill)</td>
<td>-</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>62</td>
<td>f</td>
<td>IC-PC</td>
<td>III</td>
<td>drowsy</td>
<td>(stationary)</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>72</td>
<td>f</td>
<td>MCA</td>
<td>III</td>
<td>drowsy</td>
<td>(up &amp; down)</td>
<td>+</td>
<td>+</td>
<td>die</td>
</tr>
<tr>
<td>10</td>
<td>45</td>
<td>m</td>
<td>A com A</td>
<td>V</td>
<td>semicoma</td>
<td>(downhill)</td>
<td>+</td>
<td>+</td>
<td>die</td>
</tr>
<tr>
<td>11</td>
<td>62</td>
<td>m</td>
<td>IC-PC</td>
<td>IV</td>
<td>semicoma</td>
<td>(downhill)</td>
<td>+</td>
<td>+</td>
<td>die</td>
</tr>
</tbody>
</table>

*Hunt’s classification.

Results

1. Relations Between Level of Consciousness, CSF Gas Pressure and Electrolytes

The CSF oxygen tension ($P_{C8fO2}$) of the patients who were semicomatose or in coma was significantly lower than that found in patients without such disturbance. The pH, $P_{CO2}$, $HCO_3^-$, $Na^+$, $K^+$, $Cl^-$, and $Ca^{++}$ in the CSF of patients with ruptured aneurysm were not significantly different from normals. These results are shown in table 2.

2. Cerebral Vasospasm and Metabolic Parameters

The $P_{C8fO2}$ in patients with ruptured aneurysm and vasospasm was significantly lower than in control groups of patients without vasospasm. The $P_{C8fO2}/Pao_2$ in the group with vasospasm was markedly lower than in the group without vasospasm or the control group. The $pH_{C8f}$, $P_{C8fO2}$, $[Na^+/K^+]_{C8f}$, $Ca^{++}_{C8f}$ did not show the significant changes in either the group with or without vasospasm. These results are shown in table 3.

Figure 1 shows the course of a patient and her $P_{C8fO2}$ (patient 4 shown in table 1). This patient did not have vasospasm during the month following onset but she was drowsy for a few days after SAH. She became alert immediately after ventricular drainage. $P_{C8fO2}$ and $P_{C8fO2}/Pao_2$ remained within normal limits but transient increased values were observed for a few days after ventricular drainage and craniotomy (neck clipping of aneurysm).

Figure 2 shows patient 3 in table 1. This patient did not have vasospasm on admission, but vasospasm developed 13 days after the first SAH and the day after re-rupture as shown. This patient displayed diffuse vasospasm of the main intracranial arteries on angiographic study. $P_{C8fO2}$ and $P_{C8fO2}/Pao_2$ were markedly low after the onset of vasospasm. $Pao_2$ was maintained within normal limits or slightly higher by oxygen via an intratracheal tube. The patient died 15 days after the re-rupture of her aneurysm.

All patients who died, as shown in table 1, had vasospasm. The $Pao_2$ level was maintained within normal limits or slightly higher in all patients but the $P_{C8fO2}$ value did not increase.

Discussion

Body fluid gas pressure and electrolytes of patients with ruptured aneurysm were continuously analyzed

Table 2  CSF Metabolic Parameters and Electrolytes of Patients with Ruptured Aneurysms  mean = SEM

<table>
<thead>
<tr>
<th>Consciousness levels</th>
<th>$pH$</th>
<th>$PCO_2$ (mm Hg)</th>
<th>$PO_2$ (mm Hg)</th>
<th>$HCO_3^-$ (mEq/L)</th>
<th>$Na^+$ (mEq/L)</th>
<th>$K^+$ (mEq/L)</th>
<th>$Cl^-$ (mEq/L)</th>
<th>$Ca^{++}$ (mEq/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>alert (n = 13)</td>
<td>7.27 ± 0.01</td>
<td>50.4 ± 0.8</td>
<td>44.1 ± 2.7</td>
<td>22.6 ± 0.7</td>
<td>141.3 ± 1.2</td>
<td>2.45 ± 0.07</td>
<td>120.0 ± 1.3</td>
<td>2.53 ± 0.14</td>
</tr>
<tr>
<td>lethargic (n = 16)</td>
<td>7.29 ± 0.007</td>
<td>51.0 ± 0.8</td>
<td>43.8 ± 0.7</td>
<td>24.3 ± 0.7</td>
<td>146.4 ± 1.5</td>
<td>2.50 ± 0.06</td>
<td>122.8 ± 0.9</td>
<td>2.44 ± 0.04</td>
</tr>
<tr>
<td>drowsy (n = 16)</td>
<td>7.29 ± 0.006</td>
<td>51.3 ± 0.8</td>
<td>35.6 ± 1.7</td>
<td>25.0 ± 1.0</td>
<td>144.8 ± 1.4</td>
<td>2.70 ± 0.07</td>
<td>122.2 ± 1.6</td>
<td>2.60 ± 0.06</td>
</tr>
<tr>
<td>semicoma (n = 16)</td>
<td>7.26 ± 0.01</td>
<td>51.5 ± 1.2</td>
<td>33.0 ± 1.7*</td>
<td>23.2 ± 1.0</td>
<td>144.5 ± 0.3</td>
<td>2.53 ± 0.17</td>
<td>118.0 ± 2.0</td>
<td>2.50 ± 0.03</td>
</tr>
<tr>
<td>coma (n = 9)</td>
<td>7.22 ± 0.02</td>
<td>55.0 ± 2.0</td>
<td>32.5 ± 1.1*</td>
<td>20.9 ± 1.5</td>
<td>145.0 ± 1.5</td>
<td>2.70 ± 0.09</td>
<td>125.0 ± 2.2</td>
<td>2.65 ± 0.05</td>
</tr>
</tbody>
</table>

*Intracranial pressure (ICP) was regulated at the level of 120-100 mm Hg.

* < 0.05 (in comparison with alert group), n: sample numbers.
### Table 3  Metabolic Parameters and Electrolytes of Patients with Ruptured Aneurysm Accompanied by Cerebral Vasospasm

<table>
<thead>
<tr>
<th>Cases</th>
<th>pH</th>
<th>PaCO₂ (mm Hg)</th>
<th>PaO₂ (mm Hg)</th>
<th>BE  (meq/L)</th>
<th>PaO₂/PaCO₂</th>
<th>[Na⁺/K⁺]csf</th>
<th>Ca²⁺csf (mEq/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control group (n = 21)</td>
<td>7.28 ± 0.01</td>
<td>49.7 ± 1.0</td>
<td>43.4 ± 1.1</td>
<td>-2.5</td>
<td>0.50 ± 0.01</td>
<td>57.8 ± 0.7</td>
<td>2.4 ± 0.03</td>
</tr>
<tr>
<td>Groups of ruptured aneurysm</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>with vasospasm (n = 12)</td>
<td>7.31 ± 0.01</td>
<td>51.7 ± 1.1</td>
<td>30.6 ± 1.0*</td>
<td>+2</td>
<td>0.27 ± 0.01*</td>
<td>51.2 ± 3.1</td>
<td>2.6 ± 0.1*</td>
</tr>
<tr>
<td>without vasospasm (n = 14)</td>
<td>7.26 ± 0.01</td>
<td>49.9 ± 0.7</td>
<td>43.0 ± 1.1</td>
<td>-3.4</td>
<td>0.50 ± 0.01</td>
<td>58.6 ± 1.7</td>
<td>2.6 ± 0.01</td>
</tr>
</tbody>
</table>

Intracranial pressure (ICP) was regulated at the level of 120-100 mm Hg. *p < 0.05.

In this study, it has been demonstrated that Ca²⁺ has an effect on vascular diameters if K⁺ and H⁺ are normal. An increase of Ca²⁺ leads to a dose-dependent constriction of vessels, and reduced or no Ca²⁺ in a perfusate leads to dilatation. The action of CO₂ on cerebral vessels is exerted via changes in the extracellular fluid pH and molecular CO₂; bicarbonate ions do not have independent vasoactivity. Our clinical study, unlike animal experiments, showed only a change of oxygen tension in relation to cerebral vasospasm.

Increased ICP may provoke the various metabolic or vasculo-circulatory changes in brain, like reduced cerebral blood flow, accumulation of CO₂ in the tissue, tissue acidosis, decrease of cerebrovascular tone, increase of cerebral vascular bed, brain edema, and swelling, in vicious cycle. SAH induces the increased ICP. Therefore, lateral ventricular drainage was performed to regulate the ICP and also to exclude the bloody CSF in this study.

Some reports indicate a correlation between vasospasm secondary to rupture of an intracranial aneurysm and the prognosis, but, despite all efforts, etiology and pathogenesis of spasm have not been established nor has any form of therapy proven successful in the relief of cerebral vasospasm in humans.

R.H. Wilkins et al. reported an incidence of cerebral spasm in about 35 to 40 percent of all patients with subarachnoid hemorrhage secondary to ruptured aneurysm. Spasm does not usually appear until several days after hemorrhage, usually on about the fifth or sixth day, after hemorrhage. Spasm has not been reported during the first 24 hours. Vasospasm may persist for several weeks and its presence does not always correlate well with the clinical status of the patient. However, in this study, patients with vasospasm of diffuse type had a mortality of 67%.

K. Katsurada et al. reported low PaO₂, high internal jugular venous O₂ content, and high CSF lactate content in patients with acute severe head injury. The internal jugular venous O₂ content of the patients with marked disturbances of consciousness following SAH tended to increase in this study, but this change was not consistent and could not be considered characteristic. T. Sugi et al. reported that an increase in CSF lactate with a concomitant rise in CSF lactate/pyruvate ratio is a useful indicator of brain tissue hypoxia, even when the CSF is hemorrhagic. It is well known that brain hypoxia induces the lactic acidosis and accelerating glycolysis.

The question of the meaning of a low PaO₂ value, in spite of the sufficient arterial oxygen tension, is unanswered but it may indicate a low oxygen availability to the brain tissue. This, however, should cause some alteration of the blood-brain barrier.

The PaO₂ did not tend to return to its normal value in the patients with marked disturbances of con-
consciousness in spite of sufficient arterial oxygen tension which may indicate irreversible brain damage.

The $P_{\text{a}}O_2$ and $P_{\text{a}}O_2/P_{\text{a}}O_2$ may provide a helpful index for the prognosis of patients with ruptured aneurysm and subarachnoid hemorrhage.

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Body fluid oxygen tension and prognosis in patients with ruptured aneurysm.
H Kishikawa, K Iwatsuki, S Fujimoto and A Umeda

Stroke. 1979;10:560-563
doi: 10.1161/01.STR.10.5.560

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0039-2499. Online ISSN: 1524-4628

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