THE CEREBRAL CIRCULATION IN POSTOPERATIVE INTRACRANIAL HYPOTENSION*

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The syndrome of postoperative intracranial hypotension has been frequently described as a persistent semicomatose or lethargic state following intracranial surgery associated with a low cerebrospinal fluid pressure. It is most often seen after evacuation of a chronic subdural hematoma and following the relief of an obstruction causing a chronic internal hydrocephalus. It has been assumed that the decrease in cerebrospinal fluid pressure was causally related to the protracted disturbance of consciousness. Intrathecal instillation of fluid to restore cerebrospinal fluid pressure has been suggested frequently as treatment for this syndrome.

Chorobski\(^1\) has recently reviewed the theories of the pathogenesis of postoperative intracranial hypotension and agreed with the suggestions that this syndrome was caused by cessation or insufficient formation of cerebrospinal fluid.

Leriche\(^4\) first suggested that the clinical picture was the result of cessation of production of cerebrospinal fluid caused by cerebral vasoconstriction and ischemia of the choroid plexus. Wolff\(^5\) agreed that a reduced formation of cerebrospinal fluid is at fault and ascribed this to a disturbed venous circulation of the choroid plexus.

Chorobski\(^3\) postulated that as a result of the operative procedure there occurs a decrease in cerebral blood flow causing an ischemia of the diencephalon. He further assumed that a center exists in the latter structure controlling production of cerebrospinal fluid from the choroid plexus together with a center for consciousness. The disturbance of these centers by the ischemia was suggested as the origin of the syndrome of postoperative intracranial hypotension. It thus appears that it would be of interest to report on the state of the cerebral circulation in this syndrome now that a reasonably accurate method for its determination in man is available.

MATERIAL AND METHOD

Three patients were studied within 3 to 5 days after evacuation of typical chronic subdural hematomas. All 3 patients failed to respond in the usual manner following surgery, remaining semicomatose or comatose, and a postoperative lumbar puncture in each instance revealed a profound cerebral hypotension. The first 2 patients, both of whom remained comatose following evacuation of bilateral subdural hematomas, were treated with

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normal saline instillation into the lumbar subarachnoid sac. The first patient, aged 45, survived, requiring 4 weeks for consciousness to be fully regained. The second patient, aged 65, died 1 week after operation and 3 days after the study reported in this article had been performed. The third patient made a prompt recovery after institution of therapy consisting of carbon dioxide inhalations and intravenous fluids. At no time was the condition of this last patient as serious as the others.

The nitrous oxide technique as originally described was used. Mean arterial blood pressure was recorded directly from the femoral artery by means of a damped mercury manometer. Cerebrospinal fluid pressure in the lumbar subarachnoid sac was recorded continuously upon a water manometer with the patient lying supine upon a mattress originally designed for use with patients receiving continuous spinal analgesia. It has been observed that the spinal fluid pressure is consistently at least 50 to 75 mm. of H₂O higher in the supine position than in the lateral recumbent position.

RESULTS

Reference to Tables 1 and 2 shows that in all patients studied a diminished cerebral blood flow was associated with the reduced cerebrospinal

TABLE 1

Blood gas constituents in patients with intracranial hypotension

<table>
<thead>
<tr>
<th>Pt.</th>
<th>Age</th>
<th>Sex</th>
<th>C.S.F.P.</th>
<th>M.A.B.P.</th>
<th>Arterial</th>
<th>Internal Jugular</th>
<th>pH</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>mm. H₂O</td>
<td>mm. Hg</td>
<td>I II I II</td>
<td>I II I II</td>
<td>I II I II</td>
</tr>
<tr>
<td>1</td>
<td>46</td>
<td>M</td>
<td>90</td>
<td>174</td>
<td>94 44</td>
<td>11.7 11.8</td>
<td>52.5 51.1</td>
</tr>
<tr>
<td>2</td>
<td>65</td>
<td>M</td>
<td>92</td>
<td>190</td>
<td>78 34</td>
<td>15.4 15.0</td>
<td>45.9 44.6</td>
</tr>
<tr>
<td>3</td>
<td>38</td>
<td>M</td>
<td>88</td>
<td>106</td>
<td>81 85</td>
<td>19.4 19.9</td>
<td>40.8 45.6</td>
</tr>
</tbody>
</table>

C.S.F.P. Cerebrospinal fluid pressure.
M.A.B.P. Mean Arterial Blood Pressure.
II Study following intrathecal instillation of normal saline in patients 1 and 2, and 5% CO₂ inhalations in patient 3.

fluid pressure. The reduction in cerebral blood flow ranged from 30 to 46 per cent below the average normal as measured in healthy young males. The cerebrovascular resistance in each patient was clearly elevated and correlated well with the reduction of cerebral blood flow. The mean arterial blood pressure was within normal limits in each patient. The oxygen consumption was generally reduced and correlated well with reduced state of consciousness observed at the time of the study.

In the first two patients following the initial observations, normal saline solution was inserted into the spinal subarachnoid space sufficient to raise the cerebrospinal fluid pressure to normal. This resulted in some, but not a