Clinical Application of Hyperbaric Oxygenation in the Treatment of Acute Cerebral Damage

Heitaro Mogami, M.D., Toru Hayakawa, M.D., Nobuhiro Kanai, M.D., Ryotaro Kuroda, M.D., Ryohio Yamaa, M.D.,, Takuya Ikeda, M.D., Kikuji Katsurada, M.D., and Tsuyoshi Sugimoto, M.D.

Departments of Neurosurgery and Traumatology, Osaka University Medical School, Osaka, Japan

There have been many reports of the management of cerebral ischemia by hyperbaric oxygenation (OHP), but few evaluations of the effect of OHP on cerebral injury other than several experimental studies in animals. We are reporting the results of OHP treatment in 66 patients suffering from severe acute cerebral damage.

Clinical Material and Methods

Selection of Patients. Most of the patients selected for this study had had head injuries, and all had evidence of severe brain damage (Table 1). In some cases, severe disturbances of consciousness persisted for about 24 hours or longer after the accident; in other cases, surgical procedures revealed severe brain damage.

The remaining patients were suffering from neurological disorders subsequent to the postoperative course of a brain tumor, cerebrovascular disease, or cerebral ischemia. Brain tumor cases were treated with OHP because they showed no signs of returning to consciousness after surgery. Cerebrovascular cases were treated with OHP because of severe carotid thrombosis, and cerebral ischemia cases because of accidental transient cardiac arrests. All of these patients had serious neurological disorders, and most were comatose.

Twenty six patients had respiratory disorders requiring use of a respirator.

Clinical Management. The patient was treated in a hyperbaric chamber designed to operate at high pressure and large enough to accommodate the attendant staff and essential monitoring and therapeutic equipment. The OHP treatment was usually given at a pressure of 2 atmospheres absolute (ATA) for 1 hour, once or twice a day; six of the treatments, however, were given at 3 ATA for 30 minutes. Pure oxygen was continuously administered to the patient by non-rebreathing face mask or tracheal tube. Compression was carried out at 0.1 to 0.2 kg/cm²/min until the desired pressure was reached, and decompression was carried out according to Meijn's new decompression schedule.

Physicians always accompanied the patient in the chamber, checked the vital and neurological signs, and collected arterial blood samples for analysis of oxygen and carbon dioxide (PO₂, PCO₂) and pH. Continuous EEG recordings were examined in 34 treatments of 24 patients. The recording was made on an 8-channel instrument, and qualitative analysis was commonly employed. In 13 cases, changes in cerebrospinal fluid pressure were continuously measured through a catheter inserted intraventricularly. Cerebrospinal fluid (CSF) specimens were collected before, during, and after OHP treatment in 13 cases. CSF lactate was determined by the method of Barker and Summerson and pyruvate by the method of Friedemann and Haugen.

Results

Changes in Neurological Symptoms and Signs. During OHP, 33 patients (50%) showed clinical improvement, 21 of them to a remarkable degree (Table 1), which included restoration of mental as well as neurological function. The most impressive responses were increased awareness and responsiveness. Patients who had been mildly confused became themselves soon after the beginning of OHP, moved extremities, spoke, and responded appropriately to instruction. Other patients who had more se-
Hyperbaric Oxygenation for Cerebral Damage

**TABLE 1**

*Clinical results of hyperbaric oxygen therapy in 66 patients with acute cerebral damage*

<table>
<thead>
<tr>
<th>Cause of Damage</th>
<th>No. of Cases</th>
<th>No. of Treatments</th>
<th>Improvement</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Great</td>
</tr>
<tr>
<td>Head injury</td>
<td>51</td>
<td>97</td>
<td>17</td>
</tr>
<tr>
<td>Brain tumor</td>
<td>10</td>
<td>18</td>
<td>2</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>2</td>
<td>9</td>
<td>0</td>
</tr>
<tr>
<td>Cerebral ischemia</td>
<td>3</td>
<td>19</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>66</td>
<td>143</td>
<td>21</td>
</tr>
</tbody>
</table>

were neurological deficits and were comatose rapidly became responsive to painful stimuli and simple commands. We noted that the improvements were generally remarkable in the cases in which the neurological deficits were relatively mild, and were hardly noticeable in the patients who were in deep coma.

The improvements occurred with the beginning of OHP and persisted during OHP. However, most of these favorable responses were temporary, and regression occurred immediately after decompression. Thus, in most patients the picture of clinical improvement by OHP reverted almost completely to the pretreatment level soon after the treatment ended.

However, there were three patients who became convalescent with the definite help of OHP treatments. Especially in one patient, who had suffered a severe head injury and had been admitted to the hospital comatose in a very poor condition, permanent neurological improvement occurred dramatically under OHP treatments. At the beginning of the first OHP treatment, he woke up rapidly and became responsive to simple instruction. The improvement persisted after the treatment and, furthermore, apparently increased in stepwise fashion whenever the patient was treated with OHP. The patient was given OHP treatments seven times during 5 days and was discharged 20 days after the admission with only a mild residual neurological deficit.

There were four cases which showed clinical improvement during OHP but which became much worse afterward; one of these patients died soon after the end of the treatment. No cases showed significant clinical deterioration during OHP except when carbon dioxide (CO₂)-mixed gas inhalations were tried. But there was one patient whose convulsive seizures caused by head injury slightly increased during OHP.

**EEG Changes.** Most of the EEG recordings from patients with cerebral damage were regarded as abnormal, with slowing of the background and prominent activity in delta ranges. During OHP, the most common EEG changes were an increase of fast components in the background, a decrease and lowering of delta activity, and an increase or appearance of definite alpha activity (Fig. 1). These changes were regarded as a reduction of abnormalities, that is, positive improvement in EEG. Of 24 patients whose EEG's were studied (Table 2), some degree of improvement was observed in seven and remarkable improvement in nine. However, of the nine patients who were in very poor condition and in deep coma, only three showed EEG improvement with OHP. Noticeably, no EEG response to OHP was obtained in four cases in which pretreatment EEG recordings showed almost no activity.

Qualitative analysis of 18 cases of severe head injury showed a decrease of delta activity in 10, an appearance or increase of alpha activity in 15, and an increase of fast components in alpha ranges in three (Fig. 2). In general, increase of alpha activity accompanied increase of beta activity. On the other hand, changes in the theta ranges were variable, but in most cases where there was an increase of theta activity there was a decrease of delta quantity.

Most of the EEG changes were noted only during OHP, and, like the clinical changes, regression occurred soon after decompression. There were two cases where the EEG deteriorated significantly after OHP. The EEG improvements were com-