damage to the blood-brain barrier during epileptic seizures induced by electric shock could be abolished by high cervical cord section which prevented blood pressure rise. The damage to the blood-brain barrier was examined after injection of horseradish peroxidase. Increased pinocytosis was shown to be the cause of the edema, since the endothelial tight junctions remained intact. Paulson and Hertz (Copenhagen) have shown that intactness of the blood-brain barrier (BBB) may be shown in man using the indicator dilution technique since water transport across the BBB is influenced by injection of hyperosmolar solutions. The transport of metabolic substrates such as glucose and lactate across the BBB can similarly be measured. These measurements require combined carotid and jugular vein puncture as well as multiple injections of 133Xe and labeled indicator substances.

Sanguineous Cerebrospinal Fluid in Recanalized Cerebral Infarction

TADAYOSHI IRINO, M.D., MAMORU TANEDA, M.D., AND TAKAO MINAMI, M.D.

SUMMARY To clarify the causal relationship between spontaneous recanalization of the occluded cerebral artery and development of hemorrhagic infarction, 15 patients with internal carotid or middle cerebral arterial axis occlusion were submitted to consecutive lumbar punctures and follow-up cerebral angiography. Consequently, six of seven recanalized patients had sanguineous cerebrospinal fluid (CSF) on the second or third day after ictus, while only one of eight non-recanalized patients had bloody CSF.

Atrial fibrillation was present on the ECG in six patients (table 1). Fifteen patients who had no previous stroke had sudden onset of severe hemiplegia and disturbance of consciousness. Fibrinolytic agents were not used in the treatment of these patients.

Angiography was first performed within 24 hours after the onset, using 7 ml of 60% Amidotrizoate. Angiography also was performed on the second or third day in order to study the occluded artery. When recanalization was not demonstrated, additional angiography was performed between the fifth and seventh day.

CSF was obtained by lumbar puncture on admission and then at one to three-day intervals in the first week; the appearance and pressure of the CSF were noted. When the CSF was bloody, it was placed in three test tubes and the red cell counts were compared with each other to eliminate a traumatic tap. Sanguineous CSF always contained more than 1,500 red blood cells per cubic millimeter. The appearance of the CSF was classified as clear, xanthenochromic or sanguineous. The results were compared in two groups: recanalized and non-recanalized patients.

Methods

One hundred fifty-six consecutive stroke patients, who were admitted to the Division of Cerebrovascular Disease of Hanwa Hospital between May, 1974, and February, 1975, had cerebral angiography performed within 24 hours after the onset of stroke. Eighty-six patients were diagnosed as having cerebral infarction on the basis of physical and angiographical findings. Of these 86 patients, all (except one who died 32 hours after the stroke) who had completed stroke due to occlusion of the internal carotid artery or the middle cerebral arterial axis were selected for the present study (15 patients); there were eight patients with internal carotid arterial occlusion and seven patients with middle cerebral arterial axis occlusion.

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SANGUINEOUS CSF IN RECANALIZED CEREBRAL INFARCTION/Irino et al.

Table 1  Case Summary

<table>
<thead>
<tr>
<th>Case no./age/sex</th>
<th>First</th>
<th>Second</th>
<th>Third</th>
<th>CSF Xanthochromic or sanguineous</th>
<th>Outcome within a month</th>
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<tbody>
<tr>
<td>1/47/F*</td>
<td>R ICA</td>
<td>Recanalized</td>
<td>—</td>
<td>yes</td>
<td>Alive</td>
</tr>
<tr>
<td>2/62/M</td>
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<td>—</td>
<td>yes</td>
<td>Alive</td>
</tr>
<tr>
<td>3/62/M*</td>
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<td>L MCA</td>
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</tr>
<tr>
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<td>L MCA</td>
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<tr>
<td>5/44/F</td>
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<td>—</td>
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<td>Dead (Day 3)</td>
</tr>
<tr>
<td>6/63/F*</td>
<td>L MCA</td>
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<td>—</td>
<td>yes</td>
<td>Dead (Day 7)</td>
</tr>
<tr>
<td>7/73/F*</td>
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<td>—</td>
<td>no</td>
<td>Dead (Day 7)</td>
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<tr>
<td>8/70/M*</td>
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<td>—</td>
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<td>Alive</td>
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<tr>
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<tr>
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<tr>
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<tr>
<td>13/72/M</td>
<td>L ICA</td>
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<td>L ICA</td>
<td>no</td>
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<tr>
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<td>Alive</td>
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<tr>
<td>15/72/F</td>
<td>R MCA</td>
<td>R MCA</td>
<td>R MCA</td>
<td>no</td>
<td>Alive</td>
</tr>
</tbody>
</table>

*Patients with atrial fibrillation. ICA = internal carotid artery, MCA = middle cerebral artery, L = left, R = right, M = male, F = female.

Table 1: Case Summary

The remaining patient was not recanalized. Five patients (Cases 3, 4, 5, 6 and 7) of the recanalized group died within a month and were autopsied. The angiograms of Case 1 are demonstrated in Figure 1. Among the fatal cases, sanguineous or xanthochromic CSF occurred between the second and fourth day in four patients (Cases 3, 4, 5 and 6); in the remaining patient (Case 7), the CSF was constantly clear (Fig. 2). These five cases had infarction pathologically.

None of the patients with non-recanalized vessels died within one month of onset.

Five (three with bloody CSF) of six patients with atrial fibrillation showed recanalization angiographically. Three (all with bloody or xanthochromic CSF) of nine patients without atrial fibrillation had recanalization.

The CSF pressure reached the highest level between the second and fourth day in the non-recanalized group. In the recanalized group, the CSF pressure continued to increase until death occurred in three patients (Cases 3, 4 and 5), while the CSF pressure remained low in two patients (Cases 6 and 7) who died of heart failure accompanying atrial fibrillation.

![Figure 1](http://stroke.ahajournals.org/Downloaded from http://stroke.ahajournals.org/ by guest on April 13, 2017)
Discussion

Although it is well recognized that spontaneous recanalization of the occluded arteries frequently occurs in cerebral infarction, as first reported by Lehrer in 1958, the clinical course of such patients has not been thoroughly investigated. In the many cases reported as recanalized infarction, the restoration of circulation did not produce significant clinical improvement. Some of the cases, as described in the papers reported by Dalai, had hemorrhagic infarction at autopsy examination. In our present study, six of seven patients with recanalization showed sanguineous or xanthochromic CSF and five of them had hemorrhagic infarction at autopsy, while the CSF of the non-recanalized patients became sanguineous less frequently, i.e., only one of seven non-recanalized patients. The development of hemorrhagic infarction correlated well with the occurrence of spontaneous recanalization.

Fisher et al. reported that hemorrhagic cerebral infarction was often observed at autopsy while occluded arteries were rarely found. They suggested that hemorrhagic infarction was caused by the restoration of blood flow through the recanalized arteries. The patients did not have consecutive cerebral angiograms during life, and it was not certain whether the affected arteries recanalized or not. The present observations support the hypothesis suggested by Fisher et al. that the absence of an occluded artery in hemorrhagic infarction is probably the result of spontaneous recanalization.

Harvey and Rasmussen reported that experimental hemorrhagic infarction was commonly associated with transient occlusion of a cerebral artery, but not associated with permanent occlusion. Others produced hemorrhagic infarction by utilizing transient occlusion of cerebral arteries.

However, some differences have been noted. Similar problems have been encountered after thrombo-embolectomy in stroke patients with acute major cerebral arterial occlusion; this therapeutic procedure has been discarded by some neurosurgeons.

A definite conclusion concerning CSF pressure is not certain because there was no special direction of change in the recanalized and non-recanalized groups.

We suspect that the frequency of development of abnormal CSF appearance might depend on whether the patients had cerebral thrombosis or embolism, since embolism is frequently accompanied by hemorrhagic infarction. Although atrial fibrillation is considered to be diagnostic for embolism, the definite differentiation between embolism and thrombosis is difficult clinically. However, there may be few exceptions. We considered that the cerebral arterial occlusions with sudden onset in the present study were all embolism, whether accompanied by atrial fibrillation or not.

We presume that since sanguineous CSF appeared especially in patients with recanalized occlusion that spontaneous recanalization of the occluded artery had an important relationship to the occurrence of hemorrhagic infarction and caused a change in CSF appearance. Spontaneous recanalization was associated with a poor prognosis in those patients with cerebral infarction.

References

Sanguineous cerebrospinal fluid in recanalized cerebral infarction.
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