Regional cerebral blood flow, sensory evoked potentials, and intracranial pressure in dogs with MCA occlusion by embolization or trapping

YOSHIKAZU OKADA, M.D., TAKESHI SHIMA, M.D., MITSUO YAMAMOTO, M.D., AND TOHRU UOZUMI, M.D.

Department of Neurosurgery, School of Medicine, Hiroshima University, Hiroshima, Japan

Regional cerebral blood flow (rCBF), sensory evoked potentials (SEP), and intracranial pressure (ICP) were investigated in dogs with focal cerebral ischemia produced by a silicone cylinder embolus in the middle cerebral artery (MCA) trunk as compared to that produced by trapping the same vessel. These variables were measured at intervals of 1 hour for a period of 6 hours after MCA occlusion.

In the embolized animals, rCBF decreased most extensively at the basal ganglia, from a control level of 53.9 ± 3.9 (mean ± SE) to 21.5 ± 2.7 ml/100 gm/min at the 6th hour. Sensory evoked potentials decreased progressively from the resting level of 100% to 53.0% ± 7.2% at the 3rd hour. Intracranial pressure, measured by epidural pressure on the occluded side, increased rapidly during the first 3 hours, from 10.6 ± 0.3 to about 30 cm H2O.

In the animals with trapping, the decreases in rCBF and declines of SEP were significantly less than those in the embolized animals, and no evident brain swelling was observed. This study demonstrates that MCA trunk occlusion by silicone cylinder embolization produces a more marked decrease in deep CBF, with diminution of SEP and increase in ICP, than that produced by trapping.

KEY WORDS cerebral blood flow □9 intracranial pressure □9 evoked potentials □9 cerebral infarction □9 embolization

The pathophysiology of acute cerebrovascular occlusive disease has been studied in experimental middle cerebral artery (MCA) occlusion by many investigators. In normotensive animals, MCA occlusion produces a varying degree of ischemic brain damage which depends on the collateral flow potential of experimental animals and the involvement of the perforating arteries. Many studies have been performed to scrutinize the changes in regional cerebral blood flow (rCBF), cerebrovascular responses, cerebral function, and intracranial pressure (ICP) following direct surgical MCA occlusion. However, there are few reports describing the pathophysiology of deep cerebral infarction involving the internal capsule and basal ganglia. This may be due to the difficulties in consistently producing an experimental deep cerebral infarction caused by the involvement of the perforating arteries originating from the MCA trunk.

Recently, Molinari reported that deep cerebral infarction can be produced consistently by placing a silicone cylinder embolus in the proximal MCA in dogs. In recent experiments in our laboratory, we have observed distinct clinicopathological differences between silicone cylinder embolization of the MCA trunk and trapping of the same vessel. The former shows deep cerebral infarction with serious neurological deficits, while the latter displays no macroscopic lesions. These disparities must depend on the degree of ischemia in the territory of the perforating arteries.

Therefore, in this study, comparative analyses of rCBF, cerebral function, and ICP associated with occlusion of the MCA trunk produced by the two methods, silicone cylinder embolization and trapping, were performed to investigate the critical points for producing deep cerebral infarction.

Materials and Methods

Sixty adult mongrel dogs, each weighing 7 to 15 kg, were intubated under sodium pentobarbital (25 to 30
Effect of MCA occlusion on rCBF, SEP, and ICP

mg/kg) anesthesia, and respiration was controlled mechanically to ensure normocapnia and normoxia. Systemic arterial blood pressure was recorded from a catheter cannulated into the femoral artery, and arterial blood pO₂, pCO₂, and pH were measured with a blood gas analyzer.*

Production of Occlusion

A cylinder, 1.1 mm in diameter and 8 mm in length, made by molding silicone rubber† was injected into the right cervical internal carotid artery, producing segmental occlusion of the MCA trunk. Although the cylinders lodged in the various parts of the cerebral arteries, in this study we used data only from those animals in which the cylinder lodged in the lateral segment the MCA trunk, with the tip of the cylinder reaching the trifurcation of the MCA (Fig. 1A). Thirty-five out of 45 animals were embolized successfully in this study.

In a separate "trapped" group, right temporal craniectomy was performed, and the MCA trunk was exposed through the subtemporal approach. Then, Scoville microclips under microscopic control were applied to the proximal and distal segments of the MCA trunk, about 8 mm apart, with the distal clip at the trifurcation of the MCA (Fig. 1B and C). This group consisted of 15 animals.

Regional Cerebral Blood Flow Measurement

After surgical preparation for MCA trunk occlusion with embolization or trapping, the animal's head was fixed in a stereotaxic apparatus.‡ In the embolized group, a small right temporal craniectomy was performed to introduce hydrogen electrodes. The rCBF was measured at the cortex of the anterior Sylvian gyrus (ASG) and posterior Sylvian gyrus (PSG) within approximately 1 cm from the trifurcation of the MCA and the basal ganglia on the occluded side. Hydrogen electrodes of platinum wire (300 μm in diameter, insulated down to their tips with Teflon so as to leave only about 1 mm exposed) were then placed at three locations using micromanipulators. The electrode at the basal ganglia was introduced into the putamen stereotaxically according to the stereotaxic atlas of Lim, et al. Large Ag/AgCl electrodes were placed subcutaneously in the scalp to serve as reference electrodes. All electrodes were allowed to stabilize for at least 30 minutes after placement in the tissue, and their satisfactory functioning was verified by two or three control clearances with amplifiers following 10% hydrogen gas inspiration for 1 to 2 minutes.§ Blood flow was calculated using the initial-slope method to analyze the clearance curve for 2 to 3 minutes, excluding the first 40 seconds after discontinuation of hydrogen gas inspiration.16

Sensory Evoked Potential Recording

To estimate cerebral function, sensory evoked potentials (SEP) were recorded at the sensory cortex on the occluded side following contralateral median nerve stimulation. A bipolar stimulus electrode was placed on the left median nerve, and the stimulus (2 to 3 V, 1 msec, 1 Hz) was applied to the nerve via an electric stimulator.¶ The cortical electrode was then positioned on the dura in the sensory cortex area with the reference electrode on the ear. Signals from the cortical electrode were amplified and averaged using a computer on-line with 100 responses produced by stimulation. The averaged peak-to-peak amplitudes of the primary responses were expressed as percentages of the control amplitude recorded before MCA occlusion.

Intracranial Pressure Measurement

The ICP was measured in terms of epidural and intracisternal pressure before and after embolization. The animals were placed in a headholder in the prone

* IL 214 blood gas analyzer manufactured by Instrumentation Laboratories, Inc., 133 Hartwell Avenue, Lexington, Massachusetts.
† Silicone rubber, Xythantopren, Bayern, or medical elastomer, No. 832, manufactured by Dow Corning Corp., Medical Products Division, Midland, Michigan.
‡ Narishige SN-1-M stereotaxic apparatus manufactured by Natsume Seiskusho, Tokyo, Japan.
§ PHG-201 amplifiers manufactured by Unique Medical Co., Ltd., 1326 Izumi, Komae-shi, Tokyo, Japan.
¶ SEN 1101 electric stimulator manufactured by Nihon Kohden Kogyo Co., Inc., Tokyo, Japan.
Y. Okada, T. Shima, M. Yamamoto and T. Uozumi

TABLE 1
Findings before and at various times after MCA occlusion by embolization or trapping in dogs *

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Control</th>
<th>1 Hour</th>
<th>2 Hours</th>
<th>3 Hours</th>
<th>4 Hours</th>
<th>5 Hours</th>
<th>6 Hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>embolization</td>
<td>rCBF (ml/100 gm/min)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>basal ganglia</td>
<td>53.9 ± 3.9</td>
<td>24.6 ± 2.3</td>
<td>23.4 ± 2.5</td>
<td>24.4 ± 3.1</td>
<td>22.4 ± 2.8</td>
<td>22.1 ± 2.8</td>
<td>21.5 ± 2.7</td>
</tr>
<tr>
<td>ASG</td>
<td>60.9 ± 3.0</td>
<td>36.7 ± 2.4</td>
<td>35.2 ± 2.5</td>
<td>34.8 ± 3.1</td>
<td>33.9 ± 3.1</td>
<td>33.4 ± 3.3</td>
<td>32.5 ± 3.3</td>
</tr>
<tr>
<td>PSG</td>
<td>53.0 ± 2.9</td>
<td>39.3 ± 3.8</td>
<td>37.3 ± 3.2</td>
<td>34.6 ± 2.9</td>
<td>36.3 ± 3.2</td>
<td>33.1 ± 4.2</td>
<td>37.2 ± 4.8</td>
</tr>
<tr>
<td>MABP (mm Hg)</td>
<td>99 ± 4.5</td>
<td>100 ± 4.6</td>
<td>103 ± 3.4</td>
<td>105 ± 4.6</td>
<td>110 ± 4.1</td>
<td>109 ± 3.1</td>
<td>109 ± 3.8</td>
</tr>
<tr>
<td>gas analysis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pH</td>
<td>7.39 ± 0.02</td>
<td>7.38 ± 0.01</td>
<td>7.37 ± 0.02</td>
<td>7.37 ± 0.02</td>
<td>7.36 ± 0.02</td>
<td>7.38 ± 0.01</td>
<td>7.38 ± 0.01</td>
</tr>
<tr>
<td>pCO₂ (mm Hg)</td>
<td>35.3 ± 1.37</td>
<td>36.1 ± 1.07</td>
<td>34.3 ± 1.01</td>
<td>35.2 ± 1.13</td>
<td>34.5 ± 1.05</td>
<td>35.7 ± 1.05</td>
<td>36.6 ± 1.11</td>
</tr>
<tr>
<td>pO₂ (mm Hg)</td>
<td>95.6 ± 3.7</td>
<td>96.8 ± 3.1</td>
<td>95.2 ± 3.0</td>
<td>97.9 ± 4.3</td>
<td>100.5 ± 4.6</td>
<td>102.4 ± 4.7</td>
<td>99.1 ± 2.9</td>
</tr>
</tbody>
</table>

| trapping | rCBF (ml/100 gm/min) |   |   |   |   |   |   |
| basal ganglia | 53.2 ± 2.8 | 31.2 ± 2.6 | 30.9 ± 2.8 | 33.3 ± 2.9 | 34.5 ± 2.5 | 35.0 ± 2.3 | 35.9 ± 2.0 |
| ASG | 58.4 ± 2.5 | 39.6 ± 3.9 | 38.7 ± 3.6 | 39.8 ± 3.1 | 40.2 ± 3.1 | 40.3 ± 3.7 | 43.1 ± 4.5 |
| PSG | 48.8 ± 3.0 | 49.4 ± 4.4 | 46.0 ± 2.1 | 45.3 ± 2.7 | 45.0 ± 2.9 | 45.7 ± 4.0 | 44.9 ± 2.9 |
| MABP (mm Hg) | 99 ± 7.3 | 106 ± 7.4 | 106 ± 7.8 | 112 ± 8.6 | 110 ± 7.9 | 109 ± 8.5 | 111 ± 7.3 |
| gas analysis |   |   |   |   |   |   |   |
| pH | 7.36 ± 0.01 | 7.37 ± 0.01 | 7.35 ± 0.01 | 7.35 ± 0.01 | 7.37 ± 0.02 | 7.35 ± 0.01 | 7.36 ± 0.01 |
| pCO₂ (mm Hg) | 36.1 ± 2.50 | 34.7 ± 1.37 | 37.2 ± 1.78 | 37.5 ± 0.87 | 37.7 ± 1.30 | 36.0 ± 1.16 | 36.0 ± 1.71 |
| pO₂ (mm Hg) | 90.6 ± 3.55 | 95.4 ± 5.2 | 94.2 ± 5.1 | 99.7 ± 2.8 | 98.4 ± 2.4 | 96.1 ± 3.3 | 94.1 ± 2.8 |

* Regional cerebral blood flow (rCBF) at the basal ganglia, and the cortex of the anterior Sylvian gyrus (ASG) and the posterior Sylvian gyrus (PSG), mean arterial blood pressure (MABP), and arterial blood pH, pCO₂, and pO₂ during the 6 hours after middle cerebral artery (MCA) occlusion. These values are expressed as mean ± standard error.

position after surgical preparation for embolization, and burr holes of 1.5 x 0.5 cm were made at bilateral parietal regions. The dura was detached from the cranium around these holes, and the epidural pressure devices* were inserted into the epidural spaces in both hemispheres to measure epidural pressure. To measure intracisternal pressure, the cisterna magna was punctured by a No. 23 needle and connected to a pressure transducer† by a non-distensible catheter. Bilateral epidural and intracisternal pressures were recorded with arterial blood pressure on rectigraphs and a polygraph, respectively.‡

After a period of at least 1 hour, during which control measurements of rCBF, SEP, and ICP were made, the MCA trunk was occluded by either the embolization or the trapping method. Measurements of these variables were performed every hour for a period of 6 hours.

The 35 embolized and 15 trapped animals were all prepared for measuring rCBF, SEP, and ICP. The rCBF at the basal ganglia and ASG was measured simultaneously in 10 embolized and five trapped animals. In another eight embolized and seven trapped animals, the rCBF was measured at the PSG. The SEP were measured simultaneously in these eight embolized animals and in five out of the seven trapped animals. In all, the SEP were successfully measured in 11 embolized and five trapped animals. The ICP was measured in 14 embolized animals, and the brain was observed macroscopically in three trapped animals.

Statistical significance of the results was determined using Student's t-test, and p < 0.05 was considered significant.

Results

Regional Cerebral Blood Flow

The mean values of rCBF prior to MCA occlusion at the three regions were 50 to 65 ml/100 gm/min in both groups (Table 1). Changes in rCBF following MCA occlusion were different at the three regions (basal ganglia, ASG, and PSG) in both groups. In the embolized group, rCBF decreased significantly to 24.6 ± 2.3 (mean ± SE), 36.7 ± 2.4, and 39.3 ± 3.8 ml/100 gm/min at the basal ganglia, ASG, and PSG, respectively. The reduction in rCBF at the basal ganglia was significantly greater than that at the ASG and PSG during the 6 hours, and reached 21.5 ± 2.7 ml/100 gm/min. In the trapped group, rCBF decreased significantly to 33.2 ± 5.7 ml/100 gm/min at the basal ganglia, and to 39.6 ± 3.9 ml/100 gm/min at the ASG 1 hour after trapping, but recovery of rCBF was observed within a few hours.

Figure 2 expresses the relative changes in rCBF...
Effect of MCA occlusion on rCBF, SEP, and ICP

Following MCA occlusion at the three regions as a percentage of the preocclusion values at each region in both groups. The relative changes in rCBF demonstrated that the degree of reduction in the embolized group was greater than that in the trapped group at all regions during the 6 hours. Thus, 1 hour after MCA occlusion, rCBF in the embolized group decreased to 45.4 ± 2.5%, 61.1 ± 3.8%, and 75.8 ± 7.7% at the basal ganglia, ASG, and PSG, respectively. In the trapped group, rCBF decreased to 59.3 ± 5.5%, 67.6 ± 4.0%, and 97.0 ± 6.7% at the basal ganglia, ASG, and PSG, respectively. Further, the most remarkable changes in rCBF were shown at the basal ganglia in both groups during the 6-hour period.

Sensory Evoked Potentials

Typical changes in SEP in the embolized group are shown in Fig. 3A, which exhibits evident decline of SEP with time. On the other hand, Fig. 3B presents an example of recorded SEP in the trapped group, which displays no particular decline of SEP.

Figure 4 shows the relative changes in SEP in both groups and their mean values. In the embolized group, the degree of changes in SEP differed from animal to animal (Fig. 4A). In two of 11 embolized animals, SEP disappeared almost completely within 1 hour after embolization. After an initial fall, recovery of SEP was seen in two other animals. On the other hand, changes in SEP in five trapped animals were all within 10% to 20% of the control levels (Fig. 4B). The mean SEP in 11 embolized animals decreased to 72.3 ± 8.6% (± SE) at the 1st hour (Fig. 4C), and this reduction continued, reaching 53.0 ± 7.2% by the 3rd hour, a value that differed significantly from that at the 1st hour. However, after the 3rd hour the mean SEP remained at approximately 50%, without evident changes. In the trapped group, changes in the mean SEP were insignificant.

The difference in the mean SEP between the embolized and trapped groups was 18.5% at the 1st hour after MCA occlusion, and became more remarkable with time, reaching about 50% at the 5th hour (Fig. 4C). These results suggest that the changes in SEP between the embolized and trapped groups would be detectable within a few hours after occlusion.
FIG. 4. Changes in sensory evoked potentials (SEP) in 11 embolized (A) and five trapped animals (B) and the mean SEP of both groups (C) are shown. The decline of the mean SEP in the embolized group shown in C is significantly greater than that in the trapped group (p < 0.05 at 1 hour, p < 0.01 at 2 to 6 hours after occlusion). EP = evoked potentials; MCA = middle cerebral artery.

Relationship Between rCBF and SEP

We assumed that the mean SEP would vary in proportion to the logarithmic values of rCBF at the three regions measured. In Fig. 5, the mean values of rCBF at the three regions are plotted on the abscissa on semilogarithmic graph paper against the percentage of mean evoked potentials on the ordinate. Regression lines are drawn between the mean SEP and the corresponding logarithmic values of the mean rCBF at each region. These regression equations were

Y = 141.3 logX - 131.4 at the basal ganglia (r = 0.867, p < 0.01); Y = 197.3 logX - 238.9 at the ASG (r = 0.797, p < 0.01); and Y = 318.9 logX - 439.8 at the PSG (r = 0.947, p < 0.01). These results suggest that the decline of SEP is closely related to the decrease in rCBF due to MCA trunk occlusion.

Intracranial Pressure

The ICP was measured in 14 embolized animals as bilateral epidural pressure and intracisternal pressure. Epidural pressure in both hemispheres and cisternal pressure prior to embolization were 10.6 ± 0.3 (± SE), 10.9 ± 0.4, and 11.4 ± 0.5 cm H₂O, respectively. Figure 6 shows an example of epidural pressure recording after middle cerebral artery (MCA) trunk embolization.

FIG. 5. Relationship between decline of sensory evoked potentials and decrease in regional cerebral blood flow at the basal ganglia (1), cortex of the anterior Sylvian gyrus (Ant. Sylv. G.) (2), and cortex of the posterior Sylvian gyrus (Post. Sylv. G.) (3).

FIG. 6. Example of recording of epidural pressure following middle cerebral artery (MCA) trunk embolization.
Effect of MCA occlusion on rCBF, SEP, and ICP

FIG. 7. Left: Changes in epidural pressure (EDP) on the occluded side in 14 embolized animals. Right: Mean EDP on both sides and cisternal pressure rose progressively within the first 3 hours after middle cerebral artery (MCA) trunk embolization, without significant pressure difference.

cording which increases progressively within the first 2 to 3 hours after embolization. The degree of change in ICP varied from animal to animal. The changes in epidural pressure on the occluded side of the 14 embolized animals are summarized in Fig. 7 left. The epidural pressure increased above 20 cm H2O at the 1st hour after embolization in 12 of the 14 animals and above 30 cm H2O at the 2nd hour in six; after the 3rd hour there was little variation. The mean epidural pressures on both sides and the cisternal pressure are shown in Fig. 7 right. The mean epidural pressure on the occluded side increased significantly to 24.1 ± 1.7 cm H2O in the 1st hour, 28.9 ± 2.2 cm H2O in the 2nd hour, and 31.9 ± 2.7 cm H2O in the 3rd hour after embolization. The epidural pressure on the non-occluded side increased at the same degree as that on the occluded side, and no significant pressure difference could be detected. The cisternal pressure was slightly higher than the bilateral epidural pressures, but the changes in cisternal pressure were similar to those of the epidural pressures. In other words, no significant pressure differences could be seen between cisternal and epidural pressures.

In the trapped group, brain swelling could not be observed macroscopically.

The mean arterial blood pressure (MABP) measured in the 14 embolized animals increased from 101.4 ± 3.7 (± SE) to 114.6 ± 4.1 mm Hg during the 6 hours. Consequently, the perfusion pressure (MABP - epidural pressure on the occluded side) varied slightly, ranging from 93.8 ± 3.7 to 88.4 ± 5.1 mm Hg.

Arterial Blood Pressure and Gas Analysis

Measurements of MABP and gas analyses were performed during all experimental procedures. These results in measurement of rCBF in 18 embolized and 12 trapped animals are presented in Table 1. The MABP, which was approximately 100 mm Hg prior to MCA occlusion, increased by 10% within 6 hours, but there was no significant difference between the embolized and the trapped group. Arterial gas analyses showed that arterial pO2, pCO2, and pH were maintained within the ranges of 90 to 110 mm Hg, 30 to 40 mm Hg, and 7.30 to 7.40, respectively. The data in animals with SEP and ICP recording were similar to those with rCBF measurement.

Discussion

We found that the reduction in rCBF in the embolized group was significantly greater than that in the trapped group at all times and locations within 6 hours after MCA occlusion. A gradation of reduction in rCBF's could be observed in the three regions, being greatest at the basal ganglia and smallest at the posterior Sylvian gyrus (PSG) in both groups. The rCBF at the basal ganglia decreased to about 40% to 50% of the control level in the embolized group and to about 60% in the trapped group. These rCBF's were relatively high despite MCA occlusion, in contrast with remarkable reductions in similar conditions in baboons6,19 or squirrel monkeys.6,18 Symon, et al.,19 have reported that rCBF at the basal ganglia declined to about 20% of basal levels, and in the most densely

J. Neurosurg. / Volume 58 / April, 1983
Y. Okada, T. Shima, M. Yamamoto and T. Uozumi

ischemic cortical zone reached 25% following MCA clipping in baboons. In the squirrel monkey, the rCBF measured by a krypton-85 washout technique decreased to 20% to 50% of the preocclusional level following MCA clipping. These differences in rCBF following MCA occlusion in various species may be attributable to the fact that precapillary arterial anastomoses among meningocerebral branches of the middle, posterior, and anterior cerebral arteries in dogs are more abundant than those in baboons or squirrel monkeys.

In comparative studies on MCA trunk occlusion by silicone cylinder embolization and trapping, we have observed remarkable differences between the two with regard to clinocopathological features. In the embolized group, the perforating arteries were occluded at their orifices directly and produced distinct deep cerebral infarction with serious neurological deficits. On the other hand, trapping did not produce evident cerebral infarction in dogs insofar as the systemic blood pressure was maintained within the normal range and the collateral evoked potential was preserved. Consequently, the rCBF of 21.5 ± 2.7 ml/100 gm/min at the basal ganglia in the embolized group indicates a critical value which produces irreversible brain damage, while the rCBF of 30.9 ± 2.8 ml/100 gm/min in the trapped group shows a level that produces only reversible ischemic damage.

The effect on cerebral function of reduced rCBF caused by acute cerebral arterial occlusion has been estimated from a variety of standpoints. We have selected the SEP elicited by contralateral median nerve stimulation as a quantitative index of cerebral function. The SEP is considered to closely relate with the motor function, which may be affected frequently by MCA occlusion. In fact, our study shows that, in the embolized group, SEP disappears in a few animals by MCA occlusion. In fact, our study shows that, in the embolized group, SEP disappears in a few animals by MCA occlusion.

From this relationship, when SEP was 0%, rCBF was calculated to be 8.5, 16.2, and 23.0 ml/100 gm/min at the basal ganglia, ASG, and PSG, respectively. These values cannot be applied directly as the critical level of rCBF in focal ischemia, but they may suggest the degree of ischemia that produces serious brain damage. The critical dependence of SEP on rCBF has been investigated by simultaneously recorded SEP and rCBF at the same site in the parietal cortex in baboons. Results showed that a decrease in rCBF to 16 ml/100 gm/min caused a decrease of the amplitude of SEP, and at an rCBF below 12 ml/100 gm/min, SEP disappeared completely. Boysen has also demonstrated in man that when the rCBF was reduced to the level of 16 to 22 ml/100 gm/min by a 2-minute carotid occlusion, the electroencephalogram (EEG) was slow, and when the rCBF was reduced to 11 to 19 ml/100 gm/min, the EEG became flat. The critical rCBF's obtained from our study agreed with these clinical and experimental data.

Next after changes in rCBF and cerebral function, brain swelling is the most important problem in acute cerebrovascular occlusion. Brain swelling has been estimated as increases in ICP. In experimental studies on brain swelling due to acute cerebrovascular occlusion, models which closely resemble the human condition have been required. During procedures of experimental cerebrovascular occlusion, an open cranium, the drainage of the cerebrospinal fluid, and direct brain manipulation might influence subsequent changes in ICP. Therefore, the embolization method is one of the most valuable models to study changes in ICP due to intracranial cerebral artery occlusion.

We have confirmed that, in embolized animals, ICP increases rapidly within the first 3 hours after MCA trunk occlusion. Clinical and experimental studies have shown that if the patients or the experimental animals survived the acute stage, cerebral edema subsequent to an acute stroke reached a maximum level within a few days and eventually subsided in about 1 to 3 weeks. Little has demonstrated morphologically the primary and secondary phases in the evolution of ischemic cerebral edema following surgical clipping of the MCA in squirrel monkeys. The primary phase, beginning shortly after arterial occlusion, was characterized by mild swelling of the gray and white matter with gradual increase in severity for 3 to 6 hours. Then the secondary phase, characterized by massive swelling, began and reached its peak at 24 hours or later. Hence the early increase in ICP observed in our embolized animals probably corresponds to the primary phase.

Consequently, our study suggests that the abrupt decrease in rCBF below the critical level at the basal ganglia due to segmental MCA trunk occlusion produces progressive deterioration of deep cerebral energy metabolism resulting in decline of SEP and increase in ICP within a few hours. The evolution of successive massive brain swelling, which severely affects mortality and morbidity due to MCA trunk occlusion, depends on the degree of the early changes in rCBF and ICP.

References


Manuscript received January 27, 1982.
Accepted in final form October 12, 1982.
Address reprint requests to: Yoshikazu Okada, M.D., Department of Neurosurgery, School of Medicine, Hiroshima University, 1-2-3 Kasumi-cho, Minami-ku, Hiroshima, 734 Japan.