
TRANSTENTORIAL DIASCHISIS: REDUCTION OF CEREBELLAR BLOOD FLOW CAUSED BY SUPRATENTORIAL LOCAL CEREBRAL ISCHEMIA IN THE GERBIL

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SUMMARY To assess the effect of supratentorial cerebral ischemia on infratentorial brain function, changes in regional cerebellar blood flow (rCBF), after right carotid occlusion for 4 hours, were studied in 30 mongolian gerbils. The regional cerebral blood flow (rCBF) in the occluded cerebral hemisphere and rCBF in both cerebellar hemispheres were measured simultaneously by hydrogen clearance methods. Before carotid occlusion, rCBF was 0.44 ± 0.07 ml/g brain/min, and rCeBF in the left and right cerebellar hemispheres was 0.37 ± 0.09 and 0.40 ± 0.09 ml/g brain/min, respectively. After carotid occlusion, rCBF decreased in all animals showing levels of above 0.20 ml/g brain/min in 14 (group A), between 0.10 and 0.19 ml/g brain/min in 7 (group B) and below 0.10 ml/g brain/min in 9 (group C). rCeBF exhibited no changes in group A and a mild reduction in group B after carotid occlusion. In group C, rCeBF was significantly reduced 30 min after carotid occlusion in the left cerebellar hemisphere followed subsequently by bilateral reduction. In groups B and C, supratentorial brain edema was observed 4 hours after occlusion, but the degree of edema was moderate. The results of the present study suggest that depression of infratentorial brain function may occur after supratentorial local cerebral ischemia, presumably due to diaschisis.

Stroke, Vol 14, No 2, 1983

IT HAS BEEN REPEATEDLY SHOWN during the last several decades that in acute unilateral cerebral infarction, cerebral blood flow and metabolism may be reduced in both cerebral hemispheres. As first described by Von Monakow who used the term “diaschisis,” local cerebral ischemia may cause depression of the function in remote areas of brain presumably by a transneural mechanism. However, little is yet known concerning whether such a remote effect also influences the infratentorial brain reducing the blood flow and metabolism of the brain stem as well as the cerebellum.

In the present study, the regional cerebellar blood flow (rCeBF) was repeatedly measured before and after unilateral carotid occlusion in mongolian gerbils. The purpose of the study was to clarify whether supratentorial cerebral ischemia provides remote depressive effects on the infratentorial brain function.

METHODS

OPERATIVE PROCEDURE

Thirty-six adult gerbils weighing 55 to 90 g were lightly anesthetized by intraperitoneal injection of pentobarbital (40 mg/kg). The animals were allowed to breathe spontaneously throughout the experiments. PE-10 polyethylene catheters were introduced into the
femoral arteries, and the arterial blood pressure was monitored continuously on a polygraph (Nihon Kohden RM-6000). The scalp was removed, and 3 small skull holes were cut in the parietal regions, one on the left and the other 2 on the right. Three platinum electrodes for measurement of cerebral and cerebellar blood flow were inserted through the skull holes, one into the right parietal cortex and the other 2 into the left and right cerebellar hemispheres, respectively. These electrodes were 200 μ in diameter and were insulated except for their tips, 1 mm in length, which were platinized. Placement of the electrodes into the cerebellar hemispheres was made in a crossing manner from one parietal skull hole penetrating the cerebrum into the contralateral cerebellar hemisphere. In a preliminary study, this procedure was found to lead the electrodes successfully into the cerebellar hemisphere. All these electrodes were attached tightly to the skull with dental cement. The location of the electrodes and any damage to the brain, which might be caused by electrode implantation, were later checked at the end of experiments. A neck incision was then made at the midline to expose the common carotid arteries, and the right common carotid artery was carefully separated from the adjacent veins and sympathetic nerves.

Measurement of Cerebral and Cerebellar Blood Flow

The regional cerebral blood flow (rCBF) in the right cerebral hemisphere as well as rCeBF in both cerebellar hemispheres were measured simultaneously by hydrogen clearance methods. Hydrogen clearance curves were obtained by the inhalation of hydrogen gas, and rCBF as well as rCeBF values were calculated from the initial 5 min of the wash-out curves. In the calculation of the flow values, the tissue-blood partition coefficient for hydrogen gas was taken as 1.0 both for the cerebral and cerebellar hemispheres. rCBF and rCeBF were measured twice in a steady state to test the reproducibility of the methods and to obtain control values. The coefficient of variation between the two flow values measured in a steady state gave 5.9 ± 4.6% for rCBF and 6.7 ± 4.1% for rCeBF indicating that the present methods were fairly reproducible. Subsequently, the right carotid artery was permanently ligated, and rCBF as well as rCeBF values were measured at 30 min, 2 hours and 4 hours after the occlusion, respectively. One hundred μl of arterial blood was sampled before the occlusion, at 30 min and 4 hours after the occlusion, and the arterial gas was estimated with IL Micro-13 gas analyzer.

Measurement of Cerebral Water Content

After 4 hours of occlusion, the animals were sacrificed, and the location of the electrodes in the parietal cortex and cerebellar hemispheres was confirmed. Experiments with inaccurate electrode placements were excluded from data analysis. The whole brain was removed from the skull, and any damage to the brain due to the electrode implantation was investigated visually. If damage occurred, that experiment was excluded from data analysis. The left and right cerebral hemispheres were separated from the rest of the brain, and the wet weight of the respective cerebral hemispheres was estimated. The cerebral hemispheres were then placed in a heated oven at a temperature of 100.0 ± 2.0°C for 24 hours. After measurement of the dry weight, the cerebral water content in each cerebral hemisphere was calculated as follows:

\[
\text{Cerebral Water Content} = \frac{\text{Wet Weight} - \text{Dry Weight}}{\text{Wet Weight}} \times 100\%
\]

Normal control values for the cerebral water content were obtained from 20 other gerbils which received no carotid occlusion.

In this paper, all values obtained from one group of animals are presented as the mean ± standard deviation, and changes in the parameters after carotid occlusion were analyzed by the paired t-test.

Results

Successful experimental results were obtained in 30 out of the 36 animals. In the other 6, the results obtained were unreliable: 2 of these showed suppression of respiration after carotid occlusion, 3 had incorrect localization of electrodes and one suffered a small cerebral hemorrhage due to the electrode implantation. These 6 animals were excluded from the following analysis.

1. Reduction of rCBF in the Occluded Cerebral Hemisphere

The rCBF in the right cerebral hemisphere measured during the steady state was 0.44 ± 0.07 ml/g brain/min for the group as a whole. Thirty min after the occlusion, rCBF was decreased to various extents in all animals. In 6 animals, the rCBF reduction was extremely severe, and inhalation of hydrogen gas did not provide satisfactory clearance curves. The rCBF in such cases was estimated to be zero in the present study. On the other hand, the rCBF reduction was very moderate in some animals, where a tendency towards rCBF recovery was observed with a passage of time. In order to compare the changes in cerebellar blood flow among those with completely different degrees of rCBF reduction, all the animals were divided into three groups according to the extent of rCBF reduction observed at 30 min after the occlusion as follows: group A with rCBF values of above 0.20 ml/g brain/min (n = 14), group B with rCBF values of between 0.10 and 0.19 ml/g brain/min (n = 7) and group C with rCBF values of below 0.10 ml/g brain/min (n = 9). The rCBF values measured before the occlusion were the same in all three groups as shown in table 1. The extent of rCBF reduction seen at 30 min after the occlusion did not alter thereafter in most animals (table 1). In group A, some animals showed a tendency towards rCBF recovery, although none revealed a decrease such as below 0.20 ml/g brain/min. In group B, one showed rCBF recovery exceeding the level of 0.20 ml/g brain/min at 4 hours after the occlusion, and another exhibited a transient reduction below 0.10 ml/g brain/min at 2 hours after the occlusion. Otherwise, rCBF
was maintained between 0.10 and 0.19 ml/g brain/min in this group. In group C, all animals constantly revealed rCBF values below 0.10 ml/g brain/min.

2. Changes in Arterial Blood Pressure and PaCO2 after the Occlusion

The changes in mean arterial blood pressure (MABP) and PaCO2 after carotid occlusion are summarized in tables 2 and 3. The MABP tended to increase in the majority of animals at 30 min after the occlusion, and the increase was significant in groups A and C (p < 0.02, p < 0.05, respectively). Thereafter, MABP returned towards the control level in all groups. The PaCO2 level was unaltered at 30 min after carotid occlusion in all groups, but at 4 hours of the occlusion it was decreased compared to the control level by 3.4, 4.4 and 4.5 mmHg in groups A, B and C, respectively. These decreases in PaCO2 were all significant (p < 0.005; p < 0.005; p < 0.01, respectively). The PaO2 level was maintained at between 70.0 and 115.0 mmHg in all animals throughout the experiments.

3. Changes in rCeBF after Carotid Occlusion

The rCeBF in the left and right cerebellar hemispheres measured during the steady state was 0.38 ± 0.09 and 0.41 ± 0.10 ml/g brain/min in group A; 0.36 ± 0.09 and 0.40 ± 0.10 ml/g brain/min in group B and 0.38 ± 0.10 and 0.37 ± 0.07 ml/g brain/min in group C, respectively. Thus, rCeBF values showed relatively wide standard deviation in all groups, yet the mean values as well as standard deviations in each respective group were essentially the same for both the left and right cerebellar hemispheres.

The changes in rCeBF after carotid occlusion were quite different among three groups as shown in figures 1, 2 and 3. In these figures, the rCeBF changes are expressed as percentage increases or decreases compared to the control values. In group A (fig. 1), rCeBF was slightly increased at 30 min after the occlusion both in the left (+6.8 ± 16.6%) and right (+3.7 ± 12.8%) cerebellar hemispheres, although these changes were not significant. Two hours after the occlusion, rCeBF returned towards the control level in both cerebellar hemispheres (-1.5 ± 8.6% in the left, +2.6 ± 7.7% in the right). In this group, rCeBF remained unchanged also at 4 hours after the occlusion both in the left (-3.7 ± 12.8%) and right (-3.5 ± 14.2%) cerebellar hemispheres.

In group B (fig. 2), the rCeBF measured at 30 min after carotid occlusion was slightly but not significantly reduced in the left (-6.3 ± 12.8%) and right (-7.0 ± 12.1%) cerebellar hemispheres. Two hours after the occlusion, rCeBF showed further decrease in both cerebellar hemispheres (-9.9 ± 16.3% in the left, -9.9 ± 17.0% in the right), however these decreases were not significant. Four hours after the occlusion, rCeBF became significantly reduced in the left (-16.6 ± 14.2%, p < 0.05) as well as the right (-16.3 ± 16.3%, p < 0.05) cerebellar hemispheres. The extent of rCeBF reduction in both hemispheres was thus the same.

The most remarkable rCeBF changes were observed in group C (fig. 3). In this group, there was a significant rCeBF reduction in the left cerebellar hemisphere (-16.1 ± 12.4%, p < 0.05) and a similar but not significant rCeBF reduction in the right cerebellar hemisphere (-11.8 ± 19.4%) at 30 min after carotid occlusion. Two hours after the occlusion, the same grade of rCeBF reduction continued in the left cerebellar hemisphere (-14.6 ± 15.3%, p < 0.05), and the rCeBF reduction also became significant in the right cerebellar hemisphere (-16.1 ± 17.4%, p < 0.05). Four hours after the occlusion, the rCeBF reduction became more remarkable in both the left (-25.9 ± 14.0%, p < 0.005) and right (-21.4 ± 14.4%, p < 0.005) cerebellar hemispheres, and these reductions were significantly greater than those observed at 30 min after the occlusion (p < 0.02, p < 0.05). In this group, the extent of rCeBF reduction in the left cerebellar hemisphere tended to be greater compared to that in the right cerebellar hemisphere, however, the difference between the two hemispheres was not significant at any stage, even if assessed by the paired t-test.

4. Cerebral Water Content in the Occluded and Healthy Cerebral Hemisphere

The normal cerebral water content obtained from 20 control animals was 77.6 ± 0.6%. Compared to these controls, there was no increase in cerebral water content in group A both for the occluded and healthy cerebral hemispheres (77.8 ± 0.6%, 77.5 ± 0.4%, respectively). In group B, the cerebral water content in

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**Table 1 Changes in rCBF after Carotid Occlusion**

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of animals</th>
<th>Control</th>
<th>30 min</th>
<th>2 hours</th>
<th>4 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>14</td>
<td>0.45 ± 0.07</td>
<td>0.29 ± 0.06</td>
<td>0.32 ± 0.05</td>
<td>0.30 ± 0.06</td>
</tr>
<tr>
<td>B</td>
<td>7</td>
<td>0.43 ± 0.07</td>
<td>0.15 ± 0.04</td>
<td>0.14 ± 0.04</td>
<td>0.14 ± 0.04</td>
</tr>
<tr>
<td>C</td>
<td>9</td>
<td>0.42 ± 0.06</td>
<td>0.02 ± 0.03</td>
<td>0.02 ± 0.03</td>
<td>0.02 ± 0.02</td>
</tr>
</tbody>
</table>

rCBF values are in ml/g brain/min.

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**Table 2 Changes in Mean Arterial Blood Pressure after Carotid Occlusion**

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of animals</th>
<th>Control</th>
<th>30 min</th>
<th>2 hours</th>
<th>4 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>14</td>
<td>81 ± 12</td>
<td>86 ± 12†</td>
<td>82 ± 12</td>
<td>79 ± 13</td>
</tr>
<tr>
<td>B</td>
<td>7</td>
<td>82 ± 12</td>
<td>84 ± 10</td>
<td>80 ± 11</td>
<td>80 ± 11</td>
</tr>
<tr>
<td>C</td>
<td>9</td>
<td>81 ± 10</td>
<td>86 ± 14*</td>
<td>82 ± 16</td>
<td>80 ± 16</td>
</tr>
</tbody>
</table>

MABP values are in mm Hg.

* *p < 0.05 significant increase compared to control.
† †p < 0.02 significant increase compared to control.
TABLE 3  Changes in PaCO₂ after Carotid Occlusion

<table>
<thead>
<tr>
<th>Group</th>
<th>No. of animals</th>
<th>Control</th>
<th>30 min</th>
<th>4 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>14</td>
<td>36.5±3.3</td>
<td>36.1±4.1</td>
<td>33.1±4.6†</td>
</tr>
<tr>
<td>B</td>
<td>7</td>
<td>37.2±2.8</td>
<td>35.4±3.6</td>
<td>32.8±3.6†</td>
</tr>
<tr>
<td>C</td>
<td>9</td>
<td>37.9±2.8</td>
<td>36.6±3.2</td>
<td>33.4±4.3*</td>
</tr>
</tbody>
</table>

PaCO₂ values are in mm Hg.
* p < 0.01 significant decrease compared to control.
† p < 0.005 significant decrease compared to control.

Discussion

In the present study, two out of three groups of gerbils showed a significant reduction of rCeBF after unilateral carotid occlusion. Though no sham-operated control experiments were performed in this study, these rCeBF changes do not seem to be derived from the operative manipulation of neck structures or other experimental procedures because there was no rCeBF change in one group, which received the same procedures as the others. Gerbils are known to often reveal seizures after carotid occlusion, which might cause some changes in the cerebellar circulation. However in the present study, experiments were all performed under pentobarbital anesthesia, and probably for this reason, none of the animals showed seizures. The influence of seizures, therefore, can be completely excluded from possible causes of rCeBF reduction. After carotid occlusion, the arterial blood pressure never decreased significantly, and PaCO₂ showed only moderate reduction at the end of experiments in all three groups. This moderate PaCO₂ change did not significantly alter rCeBF values in one group. Consequently, the same degree of PaCO₂ changes is unlikely to cause a large extent of rCeBF reduction in the other two groups. Thus, the changes in arterial blood pressure as well as PaCO₂ are not regarded as substantial contributing factors of rCeBF reduction.

It is clear that the depression of rCeBF seen in this study is closely related with the extent of rCBF reduction in the occluded cerebral hemisphere. In group A, rCBF of which was maintained above 0.20 ml/g brain/min, no rCeBF change occurred throughout the experiments. While in groups B and C, rCBF of which was reduced below 0.20 ml/g brain/min and below 0.10 ml/g brain/min, respectively, rCeBF more or less decreased significantly, and grade of decrease was greater in group C. Experimental workers investigating a relationship between the extent of cerebral blood flow
reduction and the occurrence of cerebral ischemia have indicated that the critical blood flow level for the development of cerebral ischemia lays somewhere between 0.15 and 0.20 ml/g brain/min. There seems to be, therefore, little doubt that the depression of rCeBF indicates that the critical blood flow level for the development of cerebral ischemia is connected with the development of cerebral ischemia in the occluded cerebral hemisphere.

Regarding the mechanism of rCeBF reduction, three possibilities can be considered: (1) a steal phenomenon, the blood flow to the cerebellum being diverted to the occluded cerebral hemisphere; (2) effects of supratentorial brain swelling produced by development of ischemic cerebral edema and (3) diaschisis. Van der Drift and other clinical workers have documented that the critical blood flow level for the development of cerebral ischemia widely involves the cerebral hemisphere. In gerbils, the size of infarction produced by unilateral carotid occlusion is huge involving almost the entire cerebral hemisphere, and hence massive brain swelling could occur after the development of ischemic cerebral edema. The development of ischemic cerebral edema is, however, usually gradual in manner. As far as the first several hours of cerebral ischemia is concerned, the extent of brain swelling appears to be small generally. In the present study, the increase of cerebral water content estimated at 4 hours after the onset of cerebral ischemia was less than 3.0% of brain weight. Clinicopathological studies have shown that in patients with supratentorial cerebral infarction, a secondary damage to the infratentorial structures occurred only if the extent of brain swelling exceeded 10.0% in terms of brain weight. Though such a data obtained in human patients may not be directly comparable with experimental results, it seems that the extent of brain edema seen in this study is too moderate to provide the suppressive effect on the infratentorial brain function.

Finally the third possibility, diaschisis, is considered to be most likely for the explanation of the present results. This peculiar phenomenon, in which the local cerebral damage causes the functional depression in remote areas of the central nervous system presumably by a transneural mechanism, was first described by Von Monakow as a clinical condition and was later more clearly demonstrated by Kempinsky in experimental animals. Though a detailed mechanism of this phenomenon has not yet been fully understood to date, the nerve pathways connecting two remote areas seem to play an important role in the functional depression of the secondary regions. Recent experimental studies have shown that after focal cerebral ischemia of the unilateral cerebral hemisphere, neurotransmitters may be reduced in both cerebral hemispheres. Such widespread changes in neurotransmitters may also participate with the exertion of diaschisis.

In acute unilateral cerebral infarction, cerebral blood flow and metabolism is often reduced in both cerebral hemispheres, and these observations are usually explained on the basis of diaschisis. The decrease of CBF in the healthy cerebral hemisphere was found to occur even at 30 min after the onset of unilateral cerebral ischemia in experimental animals and was shown to become progressively remarkable during the first several days of the ictus in patients with cerebral infarction. In gerbils, however, such a steal phenomenon is unlikely to occur because of anatomical reasons. In this particular animal species, the posterior communication of the circle of Willis is known to be incomplete, and there seem to be only insignificant anastomoses between the carotid and the vertebrobasilar arterial systems. In such a circumstance, little blood flow may be redistributed from the vertebrobasilar arterial system to the occluded cerebral hemisphere.

The second possibility, that mass effect of supratentorial brain edema secondarily depresses the infratentorial brain function, may always exist when the ischemia widely involves the cerebral hemisphere. In gerbils, the size of infarction produced by unilateral carotid occlusion is huge involving almost the entire cerebral hemisphere, and hence massive brain swelling could occur after the development of ischemic cerebral edema. The development of ischemic cerebral edema is, however, usually gradual in manner. As far as the first several hours of cerebral ischemia is concerned, the extent of brain swelling appears to be small generally. In the present study, the increase of cerebral water content estimated at 4 hours after the onset of cerebral ischemia was less than 3.0% of brain weight. Clinicopathological studies have shown that in patients with supratentorial cerebral infarction, a secondary damage to the infratentorial structures occurred only if the extent of brain swelling exceeded 10.0% in terms of brain weight. Though such a data obtained in human patients may not be directly comparable with experimental results, it seems that the extent of brain edema seen in this study is too moderate to provide the suppressive effect on the infratentorial brain function.

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diaschisis was recently reported by Baron et al. in man. They performed a positron emission tomography study using $^{15}$O inhalation in patients with supratentorial cerebral infarction and found a reduction of oxygen consumption as well as blood flow in the cerebellar hemisphere contralateral to the side of infarct. They assumed that these changes probably result from diaschisis. In human subjects, most of the nerve fibers connecting the cerebral hemispheres and the cerebellar hemispheres appear to cross from one to the other. Such a crossing nerve connection may play a role in the depression of the function in the contralateral cerebellar hemispheres, as suggested by Kempinsky. The results obtained in the present study agree essentially with those reported by Baron et al. However, in the present study, rCeBF reduction was seen in both cerebellar hemispheres with slight dominance in the contralateral cerebellar hemispheres. In this respect, the present results show slight difference from those of Baron et al. It should be noted here that the present results represent rCeBF changes encountered in early stage of cerebral ischemia, while those of Baron et al. were mainly dealing with rCeBF changes observed days after the onset of cerebral ischemia. Such a difference in the time of the observation may be one reason for the disagreement of results. Alternatively, the discrepancy of the results may be explained by the species difference between the man and the gerbil. The neuroanatomy of the gerbil has, unfortunately, not yet been established in detail, and the mode of neural connection between the cerebral hemispheres and the cerebellar hemispheres is unknown. It is considered likely that in the gerbil, one cerebral hemisphere is equally connected with both cerebellar hemispheres. In such circumstances, bilateral rCeBF reduction could be brought about even after the unilateral cerebral ischemia. Yet, this interpretation is only speculative, and further investigations are needed to clarify the detailed reasons underlying bilateral rCeBF reduction in gerbils.

The present study has shown that after supratentorial cerebral ischemia, a rapid and progressive reduction of rCeBF may occur in gerbils. Such a rCeBF reduction is considered to derive mainly from transtentorial diaschisis, although the effect of supratentorial brain edema or hypocarbia may be partly involved. The present author considers that a more detailed investigation of intratentorial brain function including the measurement of brain stem blood flow and metabolism is necessary to understand further pathophysiology of acute supratentorial stroke.

Acknowledgment

The author gratefully acknowledges the advice of Dr. Fumio Gotoh, Professor of Department of Neurology, Keio University School of Medicine, in preparing this work.

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*Stroke*. 1983;14:213-218
doi: 10.1161/01.STR.14.2.213

*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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