Interhemispheric steal

An experimental study

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In 21 cats the pressure in the occluded middle cerebral artery (MCA) was recorded by way of a catheter, introduced in the most proximal portion of this artery by way of the transorbital approach. The effect of temporary occlusion of the ipsilateral and contralateral common carotid artery on the pressure in the occluded MCA was studied. The results seem to prove the existence of the so-called “interhemispheric steal” syndrome.

KEY WORDS - carotid arteries - cerebral arteries - collateral circulation - interhemispheric steal - brain infarction

In some patients with an occlusion or severe stenosis of one internal carotid artery (ICA), infarction develops in the contralateral cerebral hemisphere. This probably occurs when small-vessel disease (occlusion or stenosis of one or more of the branches of the middle cerebral artery (MCA) or anterior cerebral artery (ACA)) was already present on that side, but the blood supply to that hemisphere was sufficient to prevent infarction. Occlusion or stenosis of the opposite ICA produces a redistribution of the blood supply to the brain from the patent ICA and the basilar artery via the circle of Willis, preventing infarction of the cerebral hemisphere ipsilateral to the compromised ICA and producing infarction in the contralateral hemisphere, where small-vessel disease was already present. This phenomenon is called by some authors “interhemispheric steal.” We will present some experimental evidence for the existence of this “interhemispheric steal” syndrome.

Materials and Methods

General anesthesia was induced in 21 cats with pentobarbital sodium (25 mg/kg body weight) injected intraperitoneally. The anesthesia was maintained by additional intravenous injections of a solution of 1 ml pentobarbital sodium diluted in 10 ml saline.

A catheter was placed into one femoral artery and pushed upward until the tip was in the aortic arch. The systemic arterial blood pressure (SABP) was continuously recorded by way of this catheter. Another catheter was introduced into the femoral vein for intravenous injections and infusions. Tracheostomy was performed and a tracheal cannula was introduced, which was connected to a Harvard animal respirator.* Each common carotid artery was exposed, and a ligature for temporary occlusion was placed around it. The animal was put in the prone position with the head fixed in a stereotaxic head holder.

By an orbital approach, the right MCA was exposed at its origin from the ICA with the aid of the operating microscope, according to the method of Hudgins and Garcia.2

*Respirator manufactured by Harvard Apparatus, Millis, Massachusetts.
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Two ligatures were put around the MCA near its origin. The proximal ligature was tied, occluding the artery. A small opening was made with microscissors just distal to the occlusion, and backflow bleeding was prevented by light traction on the distal ligature. A polyethylene tubing,† with inner diameter 0.011 in. and outer diameter 0.024 in., was introduced with its tip directed distally and a ligature tied firmly around it. As the tip of this PE10 catheter was usually too thick, its diameter was slightly reduced by stretching the catheter. The tip of the catheter was bent through 90° since it is otherwise impossible to introduce the catheter into the MCA via the transorbital approach. The catheter was filled with a heparin and saline solution (500 units of heparin/10 ml saline) and connected to a Statham strain gauge,‡ the output of which was recorded by a polygraph.§ The pressure in the occluded MCA was continuously recorded via this catheter.

In five of the 21 cats the left MCA was also catheterized 2 to 3 hours after catheterization of the right MCA, and the MCA pressure was recorded on both sides. The end tidal CO₂ (ETCO₂) was continuously recorded with a capnograph.§ In three animals the orbita was occluded in a watertight manner with dental cement, and the intracranial pressure (ICP) was continuously recorded by way of a Millar catheter || introduced via a burr hole into the subdural space. In the remaining animals the orbita was left open. The right, left, and both common carotid arteries simultaneously were temporarily occluded for periods of 2 to 5 minutes, sometimes longer, and the changes in MCA pressure and SABP were studied.

†Intramedic PE10 catheter manufactured by Clay-Adams, Inc., 141 East 25th Street, New York, New York.
‡Strain gauge manufactured by Statham Laboratories, Oxnard, California.
§Capnograph manufactured by Litton-Mijnhardt, 45 Singel, Odijk, The Netherlands.
|| Catheter manufactured by Millar Instruments, Inc., 6001 Gulf Freeway, Houston, Texas.
Results

Occlusion of the common carotid artery ipsilateral to the occluded MCA produced no, or in some cases only a minimal and transient, decrease in MCA pressure (Fig. 1 b). Occlusion of the contralateral common carotid artery produced in every animal a marked decrease in MCA pressure, that lasted as long as the common carotid artery remained occluded (Figs 1 a and 2). The results of occlusion of the contralateral common carotid artery are summarized in Table I.

Occlusion of both carotid arteries produced in most animals the same drop in MCA pressure as occlusion of only the contralateral common carotid artery. In some cases the decrease in MCA pressure was slightly more than when only the contralateral common carotid artery was occluded, but in a few minutes the pressure increased until that level was reached. In five cats, where the MCA pressure was recorded on both sides (which means that both MCA's were occluded), occlusion of one common carotid artery produced the same slight decrease (2 to 3 mm Hg) of MCA pressure on both sides (Fig. 3).

The ICP was ± 0 in 18 animals in which the orbita was left open. In the three cats in which the orbita was closed, the ICP was very low (0 to 3 mm Hg) during these experiments.

Discussion

Occlusion of the contralateral common carotid artery produces in cats a very consistent and significant decrease of the pressure in the occluded MCA, which is recorded by way of a catheter in the proximal portion of this artery, just distal from the occlusion. The
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Fig. 3. Occlusion of left common carotid artery in a cat. The pressure is recorded in the occluded right and left middle cerebral artery (MCAP). SABP = systemic arterial blood pressure.

### TABLE 1

Occlusion of common carotid artery contralateral to the occluded MCA in 16 cats*

<table>
<thead>
<tr>
<th>Time</th>
<th>ETCO₂ (%)</th>
<th>mSABP (mm Hg)</th>
<th>MCAP (mm Hg)</th>
<th>MCAP × 100% mSABP (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>before occlusion</td>
<td>3.3 ± 0.5</td>
<td>112.2 ± 25.6</td>
<td>19.8 ± 6.4</td>
<td>17.9 ± 4.1</td>
</tr>
<tr>
<td>after occlusion</td>
<td>3.3 ± 0.5</td>
<td>118.4 ± 25.9</td>
<td>13.6 ± 5.1</td>
<td>11.4 ± 3.9</td>
</tr>
<tr>
<td>NS</td>
<td></td>
<td></td>
<td></td>
<td>p &lt; 0.005</td>
</tr>
<tr>
<td>p &lt; 0.0005</td>
<td></td>
<td></td>
<td></td>
<td>p &lt; 0.0005</td>
</tr>
</tbody>
</table>

*ETCO₂ = end tidal CO₂; mSABP = mean systemic arterial blood pressure; MCAP = middle cerebral artery pressure; NS = not significant.
pressure fall occurs despite an increase in SABP. The ratio MCAP: mean SABP, which is a measure for the effectiveness of the leptomeningeal collateral circulation to the occluded MCA, therefore decreases markedly (Table 1). The same phenomenon was noted in a previous study in eight monkeys.

The decrease of collateral flow to the occluded MCA is probably an expression of the so-called “interhemispheric steal” syndrome. Occlusion of the common carotid artery produces a fall in pressure in its branches (MCA and ACA), which is immediately followed by a redistribution of the blood supply to the brain from the opposite carotid artery and the basilar artery via the circle of Willis, in favor of the hemisphere ipsilateral to the occlusion and at the cost of the collateral flow to the occluded MCA in the opposite hemisphere.

When the common carotid artery ipsilateral to the occluded MCA is occluded, no or only a very slight and transient decrease of MCA pressure occurs. Since the major branch of this carotid artery, the MCA, is occluded already, the intracranial hemodynamics are scarcely changed and the collateral flow to the occluded MCA is therefore not or only minimally influenced.

When both MCAs are occluded, occlusion of one common carotid artery produces exactly the same slight decrease of pressure in both MCAs. Obviously, when one carotid artery is occluded, the opposite one supplies an equal amount of blood via both ACA's (in the monkey via the single ACA) to the occluded MCA's, through the leptomeningeal collaterals between the ACA and MCA.

The most remarkable finding of this experimental study, the very consistent and significant decrease in collateral flow to the occluded MCA when the contralateral common carotid artery is occluded, has, probably, a clinical implication. In patients suffering from ischemic insults in one cerebral hemisphere and occlusion or severe stenosis of the contralateral ICA, the same type of interhemispheric steal may exist. Endarterectomy of the diseased carotid artery or an extra-intracranial bypass procedure contralateral to the ischemic cerebral hemisphere seems to be indicated in those cases.

References

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