Effect of Small Deep Hemispheric Infarction on the Ipsilateral Cortical Blood Flow in Man

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SUMMARY The effect of small, deep ischemic lesions on the ipsilateral cortical circulation was investigated in 10 patients with persistent mild or moderate neurological deficits due to infarcts in the internal capsule. rCBF studies by the $^{133}$Xe intracarotid injection method were performed 14–180 days after the onset of the infarction. The rCBF functional image was made up from the data of $^{133}$Xe dynamic images measured by an Anger-type gamma camera and the rCBF values were calculated by the initial slope method. The average value of mean rCBFs (mCBF) in 10 patients was 44.9 ± 7.1 ml/100g/min (average PaCO$_2$: 39.9 ± 4.3 mm Hg). In the rCBF functional images, a focal hypoperfusion area was observed in all cases and localized around the central sulcus, especially in the precentral and central areas. Significant decreases of mCBF and the tendency to decrease of the rCBFs in the hypoperfusion focus were noted in the patients with the larger infarcts in comparison with those with the smaller ones. These results suggest that a small, deep ischemic lesion such as a capsular infarct may have remote effects on the ipsilateral cortical circulation, due probably to the damage of a number of fibers passing through the lesion.

The purpose of this study is to investigate the influence of a small, capsular infarct on the ipsilateral cortical blood flow.

Subjects and Methods

Subjects

Ten patients aged 39–78 years old, with a mean ± SD of 56.4 ± 13.5, who suffered from mild or moderate hemiparesis after cerebral infarct were studied. The diagnosis of capsular infarction was made on the basis of clinical symptoms, neurological findings, cerebral angiogram and CT-scan of the brain. Informed consent was obtained from each subject prior to the initiation of the study in accordance with the Helsinki Declaration of 1975. The clinical summary of the subjects studied is shown in table 1. All patients had a clinical history of hypertension. Four patients showed pure motor hemiparesis and 6 manifested both motor and sensory disturbances. Five cases had a mild, and the remainder a moderate severity of neurological deficit. The severity (mild or moderate) of neurological deficit in each case was determined by the activities of daily living. All patients were successfully investigated 14–180 days after the ictus. In each case, CT-scans were carried out 2–3 times before and after the rCBF measurement, and the existence of a small low density area at the internal parts distant from the lesion, in the human brain. 9 Sev-
capsule was confirmed. There was no low density area in other regions of the brain. The size of the low density area was determined by measuring the diameter (mm) in the printed-out numeric image of the CT-scan.

No occlusive lesions were detected in the intra- and extracranial arteries in serial cerebral angiograms.

### rCBF Measurement

The rCBF measurement was performed as follows: Following an insertion of a small catheter (19 G, 90 mm) into an internal carotid artery, an Anger-type gamma camera (Picker Co. Ltd., USA) was placed over the lateral (9 cases) or vertical (1 case) aspect of a patient’s head. Arterial blood was obtained anaerobically for the blood gas analysis, and after this approximately 4 mCi of $^{133}$Xe in saline was injected rapidly through the catheter. The radioactivity of $^{133}$Xe was measured at the sampling time of one second for 130 seconds, and serial images were stored on the magnetic tape in a digital form of a 64 x 64 matrix of 4 x 4 mm$^2$ elements. Arterial blood pressures were measured periodically before and during the rCBF measurement and arterial blood gases were analyzed using an automatic gas analyzer (Corning, Model 175). Thereafter, angiography was carried out through the same catheter. All measurements were done in a resting state.

### rCBF Functional Image

The rCBF functional image was made up from the collected data according to the method reported previously. The region of interest (ROI) was defined as the area over 70% of the maximum count in the accumulated image of the initial 50 frames after the injection. One region consisted of the neighbouring 4 elements (8 x 8 mm$^2$). The rCBF values were calculated by the partial fraction method and the value of 0.87 was used as the partition coefficient of $^{133}$Xe. The calculated rCBF values were rearranged to the corresponding regions in the 32 x 32 matrix, and interpolated in the 64 x 64 matrix, then displayed on the cathode ray tube in a gray map with brightness modulation of the intensity as a functional image. Typewriter printings of rCBF values in 2 or 3 digital decimal numbers in a map format of the 32 x 32 matrix were also carried out as a numeric display. The mean CBF (mCBF) was obtained as the mean of rCBFs in all regions.

### Determination of Hypo- or Hyperperfusion Areas

Hypo- or hyperperfusion areas were defined in the following way. The variation of the regional blood flow in rCBF functional image of the normal patient, expressed as a standard deviation (SD), was 7% in our institute. Therefore, areas with relative flows less than -15% (< 2SD) were defined as hypoperfusion areas and those with relative flows more than 15% (> 2SD) as hyperperfusion areas. To identify the anatomical regions where abnormal flows were detected, the

### Table 1 Clinical Summary of 10 Patients

<table>
<thead>
<tr>
<th>Case</th>
<th>Name</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Neurological deficits at the time of the rCBF study</th>
<th>Severity</th>
<th>Interval* (days)</th>
<th>CT-scan†</th>
<th>Size (mm$^2$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>TM</td>
<td>70</td>
<td>M</td>
<td>weakness &amp; numbness of lt. upper &amp; lower limbs, dysarthria</td>
<td>mild</td>
<td>14</td>
<td>rt. i.c. (P)</td>
<td>9 x 6</td>
</tr>
<tr>
<td>2</td>
<td>YN</td>
<td>39</td>
<td>M</td>
<td>weakness &amp; numbness of lt. upper &amp; lower limbs, dysarthria</td>
<td>mild</td>
<td>18</td>
<td>rt. i.c. (A)</td>
<td>16 x 12</td>
</tr>
<tr>
<td>3</td>
<td>FY</td>
<td>58</td>
<td>M</td>
<td>lt. hemiparesis, dysarthria, lt. facial palsy, lt. hemihypesthesia</td>
<td>moderate</td>
<td>18</td>
<td>rt. i.c. (K)</td>
<td>6 x 6</td>
</tr>
<tr>
<td>4</td>
<td>SH</td>
<td>44</td>
<td>M</td>
<td>weakness of rt. upper limb, dysarthria, normal sensation</td>
<td>mild</td>
<td>28</td>
<td>lt. g.p.</td>
<td>20 x 12</td>
</tr>
<tr>
<td>5</td>
<td>KI</td>
<td>53</td>
<td>F</td>
<td>rt. hemiparesis, rt. facial palsy, dysarthria, normal sensation</td>
<td>moderate</td>
<td>45</td>
<td>lt. g.p.</td>
<td>10 x 7</td>
</tr>
<tr>
<td>6</td>
<td>FU</td>
<td>72</td>
<td>M</td>
<td>rt. hemiparesis, rt. facial palsy, dysarthria, normal sensation</td>
<td>moderate</td>
<td>51</td>
<td>lt. i.c. (K)</td>
<td>27 x 15</td>
</tr>
<tr>
<td>7</td>
<td>YN</td>
<td>57</td>
<td>M</td>
<td>lt. hemiplegia, lt. facial palsy, dysarthria, lt. hemihypesthesia</td>
<td>moderate</td>
<td>64</td>
<td>rt. g.p.</td>
<td>29 x 15</td>
</tr>
<tr>
<td>8</td>
<td>ST</td>
<td>53</td>
<td>M</td>
<td>weakness of lt. upper limb, dysarthria, normal sensation</td>
<td>mild</td>
<td>120</td>
<td>rt. i.c. (A)</td>
<td>12 x 8</td>
</tr>
<tr>
<td>9</td>
<td>IM</td>
<td>78</td>
<td>F</td>
<td>lt. hemiparesis, lt. facial palsy, lt. hemihypesthesia</td>
<td>moderate</td>
<td>120</td>
<td>rt. i.c. (K)</td>
<td>10 x 9</td>
</tr>
<tr>
<td>10</td>
<td>KK</td>
<td>40</td>
<td>M</td>
<td>weakness of rt. upper &amp; lower limbs, rt. hemihypesthesia</td>
<td>mild</td>
<td>180</td>
<td>lt. i.c. (K)</td>
<td>10 x 8</td>
</tr>
</tbody>
</table>

Mean ± SD 56.4 13.5

*Interval from the onset to the rCBF measurement.
†Location of low density area on CT-scan.

Abbreviations: CT = computed tomography; M = male; F = female; lt. = left; rt. = right; i.c. = internal capsule; g.p. = globus pallidus; A = anterior limb; P = posterior limb; K = knee portion.
rCBF functional image was magnified to be of the same size as the corresponding angiogram and the
angiogram was superimposed on it.

Results
Mean arterial pressure, PaCO₂, mCBF, mean value of rCBFs and relative flow in focal hypoperfusion area
in each case are summarized in table 2, and the findings on the rCBF functional image in table 3. The
rCBF study was performed in an almost normocapnic condition (PaCO₂ = 39.9 ± 4.3 mm Hg), though
significant increase of mean arterial pressure was observed during the examination. In our institute, the
mean value of mCBFs in age-matched normal controls was 57.5 ± 4.0 ml/100g/min in normocapnic condi-
tion, and according to the unpaired t-test a significant decrease of the mean value of mCBFs was noted
in the stroke patients studied in the present investigation (mCBF = 44.9 ± 7.1 ml/100g/min). In the rCBF
functional image, no patients showed any hyperperfusion foci and as presented in table 3, a focal hypoperfu-
sion area was observed in every patient. The mean value of the regional blood flows in such a focus was
35.0 ± 7.6 ml/100g/min and significantly different from the mean of mCBFs. The relative flow in a focal
hypoperfusion area ranged from −39% to −15% (−22.6 ± 7.4%). Both the location and extent of the
focal hypoperfusion areas detected in each case are presented in table 3, with the schematic drawings of
the rCBF functional images. A hypoperfusion focus was identified around the central sulcus, especially
in the precentral and central areas. The extent of the hypoperfusion areas ranged from 4 to 14 regions (2.56 to
8.96 cm²).

For reference, one of the typical cases is presented in detail: case 5 was a 53-year-old female patient.
Early in the morning, she awoke to note right hemiparesis and dysarthria, and was admitted to our hospital. A
diagnosis of cerebral infarction in the left internal capsule was made from the neurological findings, CT-
scans, though no low density areas were observed on the first CT-scan. The rCBF measurement and angiography were performed simultaneously 45 days after

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**TABLE 2** Overall Results of rCBF Measurement in 10 Patients

<table>
<thead>
<tr>
<th>Case</th>
<th>Mean AP Before (mm Hg)</th>
<th>During (mm Hg)</th>
<th>PaCO₂ (mm Hg)</th>
<th>mCBF (ml/100g/min)</th>
<th>rCBF in focal hypoperfusion area (ml/100g/min)</th>
<th>Relative flow (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>87</td>
<td>119</td>
<td>41</td>
<td>49</td>
<td>41</td>
<td>−16</td>
</tr>
<tr>
<td>2</td>
<td>87</td>
<td>97</td>
<td>43</td>
<td>46</td>
<td>39</td>
<td>−15</td>
</tr>
<tr>
<td>3</td>
<td>105</td>
<td>107</td>
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<td>31</td>
<td>−26</td>
</tr>
<tr>
<td>4</td>
<td>109</td>
<td>118</td>
<td>34</td>
<td>36</td>
<td>22</td>
<td>−39</td>
</tr>
<tr>
<td>5</td>
<td>101</td>
<td>105</td>
<td>43</td>
<td>51</td>
<td>37</td>
<td>−27</td>
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<tr>
<td>6</td>
<td>95</td>
<td>121</td>
<td>40</td>
<td>37</td>
<td>29</td>
<td>−22</td>
</tr>
<tr>
<td>7</td>
<td>97</td>
<td>137</td>
<td>36</td>
<td>41</td>
<td>34</td>
<td>−17</td>
</tr>
<tr>
<td>8</td>
<td>103</td>
<td>116</td>
<td>41</td>
<td>39</td>
<td>29</td>
<td>−26</td>
</tr>
<tr>
<td>9</td>
<td>95</td>
<td>149</td>
<td>38</td>
<td>58</td>
<td>49</td>
<td>−16</td>
</tr>
<tr>
<td>10</td>
<td>99</td>
<td>116</td>
<td>48</td>
<td>50</td>
<td>39</td>
<td>−22</td>
</tr>
<tr>
<td>Mean</td>
<td>97.8 ± SD 7.2</td>
<td>116.8*</td>
<td>39.9</td>
<td>44.9†</td>
<td>35.0‡</td>
<td>−22.6</td>
</tr>
</tbody>
</table>

Abbreviations: Mean AP = mean arterial pressure.
*p < 0.01 (paired t-test): Significantly different from the mean of mean arterial pressure before the rCBF measurement.
†p < 0.005 (unpaired t-test): Significantly different from the mean of mCBF in the normal control (57.5 ± 4.0 ml/100g/min).
‡p < 0.001 (paired t-test): Significantly different from the mean of mCBF.

**TABLE 3** Localization of Focal Hypoperfusion Area on rCBF Functional Image

<table>
<thead>
<tr>
<th>No. of regions</th>
<th>rCBF functional image</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case</td>
<td>Precentral</td>
</tr>
<tr>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>3</td>
<td>7</td>
</tr>
<tr>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>5</td>
<td>3</td>
</tr>
<tr>
<td>6</td>
<td>4</td>
</tr>
<tr>
<td>7</td>
<td>3</td>
</tr>
<tr>
<td>8‡</td>
<td>7</td>
</tr>
<tr>
<td>9</td>
<td>4</td>
</tr>
<tr>
<td>10</td>
<td>4</td>
</tr>
</tbody>
</table>

*Hypoperfusion regions localized in the precentral, central and postcentral areas.
†In case 8, the approximate localization of the focal hypoperfusion area was presented, though it was difficult to identify the precise localization.
FIGURE 1a. CT-scan of case 5, 30 days after the onset of stroke. A small low density area (arrow) was localized at the left globus pallidus and the knee portion of the internal capsule.

FIGURE 1b. rCBF functional image of case 5 (left lateral view) 45 days after the onset. Functional image is shown in a gray map with gray scales (right side), representing 70, 60, . . . , 20 ml/100g/min. Black coloured area represents focal hypoperfusion regions in which rCBF's are reduced less than -15% of mCBF.

FIGURE 1c. Left carotid angiogram of case 5. When the angiogram was superimposed on the magnified rCBF functional image, the hypoperfusion area, denoted simply by an open circle, was identified around the central sulcus (curved line), higher than the Sylvian triangle (broken line).

The interrelationships between the neurological deficits (the existence of sensory disturbances, severity of the deficit), the interval from the ictus to the rCBF measurement, CT findings (location and size of the infarction), mCBF, rCBF or relative flow in the focal hypoperfusion area, and the findings on the rCBF functional image (location or extent of the hypoperfusion focus) were also investigated. Part of the results are presented in table 4. There were no clear relationships between the mCBF values and the severity of neurological deficit or the interval from the ictus to the rCBF measurement. On the contrary, significant decrease of mCBF was noted in the patients with the larger infarct in comparison with those with the smaller one. The rCBF in the focal hypoperfusion areas also tended to decrease in the patients with large infarcts on the CT-scan, though significant differences could not

<table>
<thead>
<tr>
<th>Severity of neurological deficits</th>
<th>mCBF (ml/100g/min)</th>
<th>rCBF in focal hypoperfusion area (ml/100g/min)</th>
<th>Relative flow (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild (n = 5)</td>
<td>44.0 ± 6.2</td>
<td>34.0 ± 8.2</td>
<td>-23.6 ± 9.7</td>
</tr>
<tr>
<td>Moderate (n = 5)</td>
<td>45.8 ± 8.5</td>
<td>36.0 ± 7.9</td>
<td>-21.6 ± 5.0</td>
</tr>
</tbody>
</table>

Size of low density area on CT-scan*<br>
<table>
<thead>
<tr>
<th>Size of low density area on CT-scan*</th>
<th>mCBF (ml/100g/min)</th>
<th>rCBF in focal hypoperfusion area (ml/100g/min)</th>
<th>Relative flow (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 10 mm (n = 5)</td>
<td>50.0 ± 5.7</td>
<td>39.4 ± 6.5</td>
<td>-21.4 ± 5.3</td>
</tr>
<tr>
<td>10 mm ≤ (n = 5)</td>
<td>39.8 ± 4.0†</td>
<td>30.6 ± 6.3</td>
<td>-23.8 ± 9.5</td>
</tr>
</tbody>
</table>

*The patients were divided into two groups, according to the largest diameter of the low density area presented in table 1.<br>
†p < 0.05 (unpaired t-test): Significantly different from the mean of mCBF in the patients with the smaller infarct (< 10 mm).
be obtained between the mean values in those with the large and small infarcts (0.05 < p < 0.1). There were no significant differences in relative flows between the patients with mild and moderate neurological deficits, and between those with small and large infarctions. With respect to the findings on the rCBF functional image, a focal hypoperfusion area was identified mostly in the precentral and central areas, whether or not the motor disturbances were accompanied by sensory abnormalities, and no significant correlations were observed between the location or extent of the hypoperfusion focus and several other parameters mentioned above.

Discussion
The brain is a complex organ having many structural and functional components which not only operate separately but also cooperate with each other through neuronal pathways to maintain the integrated function of the central nervous system. Therefore, if any damage occurs in one part of the brain, some circulatory and metabolic disturbances may be induced in distant parts of the brain. Many studies have been reported from this point of view. Høedt-Rasmussen et al. first described a reduction of hemispheric blood flow in the healthy side as well as in the infarcted hemisphere in patients with unilateral cerebral infarction, and this phenomenon was thought to be caused by some dysfunction of the interhemispheric neuronal connection via the corpus callosum. Other investigators reported that an ischemic lesion in one area of the brain induced remote effects on distant areas. In these studies, however, the infarcted lesions were too large or variable to make a specific analysis of the circulatory changes in the distant related areas. In the present study, we assessed the influence of a small, deep ischemic lesion on the ipsilateral cortical circulation by measuring regional cerebral blood flows in patients with persistent mild or moderate neurological deficits due to an infarct in the internal capsule.

A significant decrease of mCBF (table 2) was noted in these patients and the rCBF functional image showed a focal hypoperfusion area around the central sulcus in all the patients studied (table 3). These results strongly suggest that the small infarct in the internal capsule has remote effects on the overlying cerebral cortex. Although it has been shown that two-dimensional recording of CBF with $^{133}$Xe is subject to serious errors when applied to densely ischemic brain tissue, this "look-through" artifact can hardly be invoked to explain our findings, considering both the small size and the deep situation of the infarcted area. In another study, presented in a preliminary report, we showed a significant increase of rCBF in such a hypoperfusion focus during hypercapnia, and this excludes the possibility of an unrecognized infarction of such cortical areas. It is difficult to explain the mechanism of the deactivation phenomenon noted in our study, but the irreversible ischemic damage of the connecting fibers that pass through the internal capsule may induce neuronal loss or antegrade and retrograde degeneration in the related cortical areas, where focal hypoperfusion was detected.

From the clinical application of positron emission tomography, Baron et al. reported that both the blood flow and oxygen metabolism were reduced in the cerebellar hemisphere contralateral to the supratentorial infarcts, in deep capsular or large supratentorial infarcts, and this phenomenon of "crossed cerebellar diaschisis" was demonstrated particularly in the patients with recent (less than 2 months) infarction and significantly related to the presence of hemiparesis. In another study, they also reported that in comparison with the opposite side, there was an 11% reduction of the regional cerebral blood flow in the ipsilateral temporoparietal cortical area adjacent to the capsular infarction. Using the same method, Celesia et al. have reported recently that the decreased flow was noted in the visual cortex distal to the optic radiation lesion and in the frontal cortex ipsilateral to the infarct. These results also demonstrated clearly the presence of remote effects in patients with a deep, capsular infarction, though only a few such cases were studied and the precise localization and size of the hypoperfusion area were not reported.

In the present study, we showed the precise localization and size of the hypoperfusion focus on the rCBF functional image in 10 patients with a small, deep capsular infarction, and a close relationship was detected between the neurological deficits (hemiparesis or hemiplegia) and the location of focal hypoperfusion area (largely detected in precentral and central regions) (table 3). As presented in table 4, in studies on the interrelationship between several parameters, we noted a significant decrease of mCBF in the patients with a large infarct and the tendency to decrease of the rCBF in the hypoperfusion focus though with no statistical significance. These results suggest that the relatively larger capsular infarcts have rather large remote effects on the corresponding cortical areas, due probably to the damage of a large number of fibers passing through the internal capsule. In conclusion, we propose that cerebral infarction may induce circulatory disorders in the brain, distant from the lesion. The remote effect, a type of "intrahemispheric diaschisis", may be present in the human brain resulting from an affect on the afferent and efferent fibers by a small, deep capsular infarct.

Acknowledgments
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Hemispheric Blood Flow in the Rat After Unilateral Common Carotid Occlusion: Evolution With Time

Gaspard De Ley, V.M.D., Jean-Berchmans Nshimyumuremyi, M.D., and Isidoor Leusen, M.D.

SUMMARY Acute occlusion of one common carotid artery in the anesthetized normocapnic rat results in a moderate cerebral blood flow (CBF) decrease in both cerebral hemispheres. No asymmetrical perfusion is observed when the overall flow in each hemisphere is considered. The increase in blood flow which normally occurs in hypercapnia is strongly impaired in the cerebral hemisphere on the occluded side resulting in an important asymmetrical hemispheric perfusion.

The days (1, 5, 15, 30) following unilateral carotid occlusion normal control CBF values are found in both hemispheres in normocapnic conditions. Hemispheric perfusion asymmetry in hypercapnia also becomes progressively less pronounced with time but a slight asymmetry still persists one month after unilateral carotid occlusion.

RAPID ADJUSTMENT of the collateral circulation after acute occlusion of one common carotid artery is able to keep cerebral blood flow (CBF) above the critical level for appropriate metabolic supply in many animal species although blood flow may be reduced in cerebral regions dependent on the occluded vessel.1–4

In the anesthetized rat acute unilateral carotid occlusion reduces the hemispheric blood flow on the side of the occlusion.5–7 Circulatory reserve is also depressed in these conditions as indicated by the inadequate adaptation of CBF to hypoxia, to hypercapnia and to hypertension.5–7 Restoration of such a circulatory reserve in the days following the occlusion is an indication that collateral circulation and CBF adaptation progressively develop in the post-occlusion period. In order to obtain more information on this eventuality, blood flow in both brain hemispheres was studied in rats at different time intervals after unilateral carotid occlusion.

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