Prolonged Disturbances of Regional Cerebral Blood Flow in Transient Ischemic Attacks

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SUMMARY Regional cerebral blood flow (rCBF) was measured over both hemispheres in 20 patients with unilateral transient ischemic attacks (TIA) of the territory of the internal carotid artery on the day of the TIA. rCBF was estimated with the nontraumatic Xenon 133-inhalation technique using the initial slope index. 13 patients experienced their first TIA, 7 had several attacks. In 14 patients the first rCBF-measurement was performed during the presentation of clinical symptoms. The 2nd rCBF-measurement was done on day 2, the last one on day 7. Scans of the 15 patients studied with CT were normal.

On day 1 mean rCBF of the TIA-side was significantly lower than that of the contralateral hemispheres. 22% of all areas showed a significant reduction of flow compared to mean rCBF. Mean rCBF of both the TIA- and the contralateral side was significantly reduced compared to the bi-hemispheric mean rCBF of a control group with no history of TIA or completed strokes but at least 2 risk factors for cerebrovascular disease.

Whereas mean rCBF did not change in the contralateral side it increased significantly (+ 6.9%) in the TIA-side from day 1 to day 2 but not from there to day 7. This is reflected by the increase of the total number of ROI with normal flow from day 1 to day 2. Considering the actual flow and the flow course of that tissue which was believed to be responsible for the clinical symptoms the following regional patterns were observed: normal rCBF in 6 patients; early return to normal concomitant to the clinical course (n = 4); delayed return to normal with ischemia on day 2 but not on day 7 (n = 3); early hyperperfusion with perifocal ischemia on day 1 (n = 2); delayed hyperperfusion on day 2 with ischemia on day 1 (n = 2); persistent ischemia (n = 3).

The data indicate that transient ischemic attacks are accompanied by varying patterns of focal CBF with negligible participation of the contralateral hemisphere. 60% of all cases present with flow abnormalities on the day of the TIA and 40% on the day following the clinical symptoms. The method seems to be highly sensitive in indicating pathophysiological abnormalities in patients with transient ischemia of the brain.

THE INTRODUCTION OF AN ATRAUMATIC METHOD of measuring regional cerebral blood flow (rCBF) in humans \(^{1,2}\) made it possible to study rCBF repeatedly in acute ischemia. As studies of rCBF in transient ischemic attacks (TIA) have been limited and repeated measurements have not been done \(^{3-9,23}\) we became interested in the course and degree of rCBF changes in such cases. The functional integrity of the brain depends on adequate cellular metabolism. If metabolism is reduced because of insufficient blood flow, functional failure will occur. With recovery of rCBF the tissue may regain its functional activity unless tissue necrosis has resulted in permanent damage. The arbitrary classification of ischemic stroke defines those ischemic attacks with neurological symptoms of less than 24 hours duration as TIAs. Following the observations of Perrone et al \(^{10}\) and Ladurner et al \(^{11}\) it has been suggested that TIAs might be accompanied by parenchymal damage as judged by computerized tomography. However, sequential changes with time have not been reported. Therefore it is still unclear whether TIAs might be associated with prolonged alteration of cerebral tissue appearance as judged by computerized tomography or by early and consistent changes in the regional cerebral blood flow.

Between 1979 and 1983 we studied rCBF in 20 patients with TIA during the attack or within 6 hours of clinical recovery. In all cases rCBF was studied three times within seven days.

Patients Twenty patients (14 males, 6 females, mean age 57.4 ± 9.4) with a final diagnosis of TIA in the territory of the right (n = 9) or the left (n = 11) internal carotid artery completed the protocol. All patients complained of one or more of the following symptoms: hemiparesis with brachial predominance (n = 14), hemihypesthesia (n = 11), motor aphasia (n = 4, all right handed), homonymous hemianopia (n = 4). None of the patients complained of impairment of consciousness. All patients with symptoms of pure vertebrobasilar insufficiency were excluded.

In two of the four patients with transient homonymous hemianopia the symptoms were attributed to reduction of blood flow in the territory dependent on the internal carotid artery. In the other two we did not find any other symptoms or sign which could be attributed to vertebro-basilar insufficiency. According to the history of each patient 13 patients experienced their first attack which led to admission: in 10 of these cases neurologic symptoms were still present during the first rCBF-measurement but had disappeared within 24 hours of onset. The other three patients entered the hospital within 1–6 hours after the end of the attack. Of the 20 cases seven had a history of several similar attacks (maximum 7) of which the latest one was still present at the time of the first rCBF-measurement in four cases. The other three cases with multiple attacks stated that symptoms had vanished not longer than six hours before the CBF measurement. In all 20 cases
symptoms lasted no longer than 24 hours, in the majority less than four hours. In two cases one additional TIA was observed between the second and the third rCBF measurement.

Fifteen in-patients were evaluated with Doppler sonography and computerized tomography, ten of them by cerebral angiography. Five patients refused admission, but agreed to ambulatory evaluation including rCBF measurement, Doppler sonography and computerized tomography (n = 4). Computerized tomography was performed between the second and the fifth day after the TIA and was reported as being normal in all cases studied.

The final explanations for the TIA were as follows: Cardiac arrhythmia (n = 4), hypertensive crisis (n = 2), arteriosclerosis with narrowing (n = 3) or occlusion (n = 2) of the internal carotid artery. In nine cases no explanation of the TIA could be given. Migraine accompagnée, focal seizures, inflammatory diseases of the brain and former completed strokes or PRIND were excluded.

Data were compared to those of 20 patients with at least two risk factors for cerebrovascular disease, but no history of acute cerebral ischemic attacks (control group). These data were collected over a period of three years, independently of the TIA-protocol. In these cases the second CBF measurement (CBF 2) was made within three days and the third (CBF 3) within 14 days of the first measurement.

Method and Protocol

rCBF was measured using the atraumatic design as proposed by Veall and Mallett, further developed by Obrist and slightly modified by ourselves. The examinations took place in a quiet and darkened room. The patient inhaled a mixture of Xenon-133 and air (concentration 3-4 mCi/l) from an eight litre airbag via a mouth tube which completely prevented any leakage of gas. The nose was closed by a clamp. After 60 sec the valve systems were switched to room air and for the next ten minutes up to 32 raw curves were recorded by externally placed detectors, containing a NaI, crystal 0.75 inches in diameter. The collimation was 20 mm. The window was set to 81 keV with an opening of ±20%. The detectors close to the vertex were arranged slightly angled to the inter-ear axis. The detectors covered the hemisphere with the exception of the most anterior and the most posterior areas of the brain. The cerebellum was not covered. The expired air passed another detector for calculation of the end expiratory Xenon-133 activity, which is an analog of the arterial concentration, thus allowing calculation of recirculation. The CO2-volume percentage of the end expiratory air was recorded continuously and used for calculation of PaCO2. Blood pressure was measured repeatedly by auscultation. If the peak count of each curve was less than 1000 c/0.5 sec the individual detector was excluded from the calculation. This occurred in 56 out of 1638 focal clearance curves. Since the initial slope index (ISI) of Risberg is considered to be stable in test-retest procedures and does not require lambda (the blood-brain tissue coefficient), it was used for expressing the rCBF. Changes of ISI from measurement to measurement of at least 10% were considered to be of statistical significance. The normal ISI in our laboratory for a sex matched group of patients aged 49–54 years, with no signs and symptoms of cerebral disease, is 50.4 ± 5.3 for the right and 49.4 ± 4.1 (sec−1) for the left hemisphere. There is no statistically significant difference between the right and left hemispheres.

The first rCBF measurement (CBF 1) was undertaken within 12 hours of the onset of neurologic symptoms. In 14 cases it was done during the attack. The second measurement (CBF 2) was performed on day 2, when clinical signs had vanished in all patients, and the third procedure (CBF 3) between days 6 and 8 after the TIA.

Since the PaCO2 did not differ by more than 2 mm Hg from measurement to measurement in any of the cases, correction for PaCO2 was not done. At CBF 1 PaCO2 was 39.2 ± 2.8 mm Hg in the TIA group and 40.6 ± 1.7 mm Hg in the control group. We did not attempt to orient individual detectors to particular arteries.

Results

A) Mean Regional Cerebral Blood Flow

During the first CBF measurements (CBF 1) mean rCBF (mrCBF) on both the side of the TIA and in the contralateral hemisphere, was normal in 13 cases (table 1). Compared to the mean bi-hemisphere flow value of the “control-group” mrCBF was significantly lower (more than two standard deviations) over the affected side in seven cases and over the contralateral side in two cases. In none of the patients was the initial mrCBF over the side of the TIA more than 15% higher than that of the contralateral side. mrCBF on the side of the TIA for the total group was significantly lower than that of the contralateral hemisphere (p < 0.005).

The mrCBF of both the TIA side and the contralateral side calculated for the total group was significantly lower than that of the control group (p < 0.005 and p < 0.0005, table 1).

In the “control” group there was no change of mean flow between all three CBF measurements (table 2). There was no change of mean rCBF in the contralateral side of the TIA patients between CBF 1, CBF 2 and CBF 3 (table 2 and fig. 1). mrCBF of the contralateral side changed significantly from CBF 2 to CBF 3 in only two individual cases (fig. 1). This indicates that flow in the hemisphere opposite to the side of the TIA represents a stable flow more influenced by the pre-existing stable metabolic state than by the acute ischemic attack. In ten cases mrCBF on the side of the TIA increased significantly from CBF 1 to CBF 2 and in three cases from CBF 2 to CBF 3. The mean rCBF of the group on the side of the TIA was statistically higher for CBF 2 compared to CBF 1 (p < 0.0025). There was no statistical change in this side from CBF 2 to CBF 3 (table 2, fig. 1).

These data indicate that:
### TABLE 1  Mean Regional Cerebral Blood Flow (ISI) and Standard Deviation on Day 1 in Patients with TIA's and in Control Patients

<table>
<thead>
<tr>
<th>TIA-Patients</th>
<th>Control patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>TIA-Side</td>
<td>Contralateral</td>
</tr>
<tr>
<td>1</td>
<td>46.1 ± 5.1</td>
</tr>
<tr>
<td>2</td>
<td>44.7 ± 5.0</td>
</tr>
<tr>
<td>3</td>
<td>45.3 ± 6.7</td>
</tr>
<tr>
<td>4</td>
<td>47.1 ± 5.8</td>
</tr>
<tr>
<td>5</td>
<td>44.7 ± 3.7</td>
</tr>
<tr>
<td>6</td>
<td>43.9 ± 7.1*</td>
</tr>
<tr>
<td>7</td>
<td>34.1 ± 6.0†</td>
</tr>
<tr>
<td>8</td>
<td>39.0 ± 5.1†</td>
</tr>
<tr>
<td>9</td>
<td>38.2 ± 3.2†</td>
</tr>
<tr>
<td>10</td>
<td>52.1 ± 4.9</td>
</tr>
<tr>
<td>11</td>
<td>49.1 ± 5.4</td>
</tr>
<tr>
<td>12</td>
<td>44.8 ± 2.3</td>
</tr>
<tr>
<td>13</td>
<td>37.6 ± 6.1†</td>
</tr>
<tr>
<td>14</td>
<td>40.9 ± 4.1</td>
</tr>
<tr>
<td>15</td>
<td>44.1 ± 2.2</td>
</tr>
<tr>
<td>16</td>
<td>42.5 ± 3.3</td>
</tr>
<tr>
<td>17</td>
<td>35.1 ± 3.1†</td>
</tr>
<tr>
<td>18</td>
<td>36.1 ± 4.0†</td>
</tr>
<tr>
<td>19</td>
<td>37.9 ± 3.6†</td>
</tr>
<tr>
<td>20</td>
<td>40.5 ± 2.1</td>
</tr>
<tr>
<td>(X)</td>
<td>42.2</td>
</tr>
<tr>
<td>SD</td>
<td>4.8</td>
</tr>
<tr>
<td>(p &lt;)</td>
<td>0.005</td>
</tr>
</tbody>
</table>

*Significant difference between TIA-side and contralateral side. Paired t-test for comparison between hemispheres in the TIA-group and control group. Initial slope index (ISI) as \((sec^{-1})\).

†Value differs by more than 2 SD from mean rCBF of the control group.

1) During CBF 1 blood flow is significantly reduced in the affected hemisphere compared to the contralateral side and to a control group with a similar risk profile.

2) By the day after the TIA there is a significant improvement of cerebral blood flow only in the hemisphere suffering from ischemia.

### B) Relative Flow Distribution

Since mean rCBF is able to mask regional flow abnormalities, regional CBF variations were calculated. In normals, regional deviation from mrCBF does not exceed 0.6 ± 9.8%. Therefore deviations from the individual patient's mrCBF of more than 20% will be considered as being relatively reduced (hypoemic) or increased (hyperemic), even though the absolute flow value may be normal. The rCBF data which are within 20% of the mean regional value are called relatively normal, even though their absolute flow may be above or below the value in normal volunteers.

In the involved side 277 regions of interest (ROI) and on the contralateral side 269 ROI were used for calculation. Sixty-one ROI on the involved side were relatively hypoemic during CBF 1, 17 ROI showed relative hyperemia and in 199 the flow was relatively normal. Already during CBF 2, 228 ROI and at CBF 3 252 ROI were relatively normal. This indicates a trend towards a slow return to normal of the rCBF map. Since the rCBF was not measured after the seventh day in the majority of the cases it cannot be decided whether CBF 3 represents the final stable flow distribution. However, since the distribution of relative flow in the contralateral hemisphere includes 13 increased, 10 reduced and 264 normal ROI during CBF 1, with only minor changes until CBF 3, we assume that the similar flow-patterns over both sides at CBF 3 reflects a stable pattern of flow. This distribution is still different from that in normal subjects. Since CBF in the contralateral side is stable in consecutive measurements, the return to normal of the rCBF over the side of the TIA can be recognized by considering the percentage difference of mrCBF between the two sides. A percentage difference between two corresponding regional areas of more than 12% is statistically significant (mean value and standard deviation).
of right-left difference for normal volunteers = −0.6 ± 5.6%).

If one considers only the areas on the side of the TIA with relatively decreased and increased flow, and compares them to the corresponding areas in the opposite hemisphere the following results were obtained: At CBF 1 all areas of reduced flow deviate from the findings on the opposite side by −25.1 ± 11.7% (fig. 2). At CBF 2 this difference is reduced to a mean value of −14.9 ± 10.2%. At CBF 3 the difference is −12.4 ± 10.8%. The areas of relatively increased flow on the side of the TIA differ from the contralateral areas at CBF 1 by 22.9 ± 11.2%, at CBF 2 by 20.0 ± 12.3% and at CBF 3 by 6.0 ± 5.5%.

These data indicate that the tissue with a relatively reduced flow shows a maximum change in flow (as judged by the percentage difference from the corresponding tissue) from CBF 1 to CBF 2. However, since the flow of the relatively hyperemic tissue shows a significant difference from the corresponding areas at CBF 2 but not at CBF 3, it can be suggested that the flow map still changes its appearance from CBF 2 to CBF 3.

C) Regional Flow Data

To identify the regional flow changes in the territory of the ischemic attack, all detectors were classified as follows: Those two detectors which were believed to be localized over the assumed ischemic area during CBF 1 were called the "focal detectors". Since computer tomography was normal in all studied cases and electroencephalography showed focal abnormalities only in a few instances the identification of the focal detectors was purely done on clinical grounds. Except for the groups with "early hyperperfusion" and "normal flow" all other 12 patients showed in these focal detectors a flow which was more than 20% below mean rCBF during CBF 1. The 3–6 detectors which directly adjoined all "focal detectors" represent the "perifocal" detectors and the rest of 14–16 detectors over the hemisphere were called the "distal detectors". All areas were compared to the corresponding contralateral region. Since there was no uniform pattern of rCBF alteration from patient to patient all cases were classified according to their actual "focal" flow at CBF 1 and the "focal" flow changes from CBF 1 to CBF 2.

Test-retest measurements in the controls under identical conditions have shown a mean deviation between consecutive measurements of 2.4 ± 3.6%. Therefore a regional change of more than 10% in the TIA-group can be considered as being statistically significant. Table 3 lists the mean change in the subgroups. The only change of more than 10% from measurement to measurement was observed in the patients with a "delayed return to normal" of "focal" flow from CBF 2 to CBF 3 and in the patients with "delayed hyperperfusion" of "focal" flow from CBF 1 to CBF 2 and CBF 3. Consideration of regional flow changes revealed significant changes in all classes of detectors which were partially neutralized by insignificant changes in other areas if the mean flow was calculated. The following description refers to the regional flow changes in the groups as named in table 3.

1. **Normal flow (n = 6)**

   In six patients rCBF of the involved and the contralateral side was normal and did not change during the time of observation (not included in fig. 3).

2. **Early return to normal (n = 4)**

   The rCBF increased from CBF 1 to CBF 2 by more than 10% and remained stable till CBF 3. There was no pathological flow observed in the perifocal or distal areas (fig. 3).

3. **Delayed return to normal (n = 3)**

   In these three cases rCBF initially was reduced because of focal ischemia. The next day (CBF 2) mrCBF was improved but was still significantly below that of CBF 3. Fig. 3 indicates that this delayed recovery was due only to ischemia in the "focus" without ischemia in the other detectors.

4. **Early hyperperfusion (n = 2)**

   During CBF 1 rCBF was higher than during CBF 2 and CBF 3. The regional map indicated in both cases...
that hyperperfusion paralleled reduced flow in the neighboring detectors. Both cases suffered from transient brachiofacial hemiparesis, which corresponded to the initial increased flow in the lower detectors over the central fissure with reduced flow over the parietal tissue. Frontal and occipital flow were normal.

5. Delayed hyperperfusion with early ischemia \((n = 2)\)

The rCBF during CBF 1 was below normal because of ischemia in the focal, perifocal and distal detectors. During CBF 2, rCBF in the focus was 17.1% above the flow of CBF 3. This means that CBF decreases from CBF 2 to CBF 3 (which is the "final" normal valve). Hyperemia in the focal detectors was accompanied by normal rCBF in all perifocal and more remote tissue. However, at the conclusion of the tests the mrCBF of the involved side was still below that of the contralateral hemisphere, which was true for all detectors.

6. Persistent ischemia \((n = 3)\)

Compared to the contralateral side and to the control group mrCBF was within normal limits. However, the focal areas of the TIA-side presented with ischemia for the total time of observation. There was no change of both mrCBF and the focally reduced flow from CBF 1 to CBF 3. The perifocal tissue of the involved side increased its blood flow slightly from CBF 2 to CBF 3.

**Discussion**

TIA is a rather arbitrary description of neurologic symptoms of limited duration. It does not define the length and cause of the ischemia itself, since it solely defines the duration of clinical presentation. It is not definitely proven that permanent tissue damage occurs as a result of TIA. Not all studies which have presented visualisation of brain tissue by computerized tomography (CT) have reported repeat observations. Therefore, the observed tissue alteration might represent only the results of previous ischemic attacks. None of the CT-scans which were performed in our patients indicated tissue damage which could be correlated to the most recent ischemic insult. In contrast, we have observed abnormal flow patterns with alteration of CBF with time in about 60% of all cases.

However, if one compares both techniques the reliability of the CBF-method should be considered. Our own studies measuring rCBF by the intracarotid Xenon 133 injection technique immediately followed by the inhalation technique during the same session indicated a high correlation for the initial slope index \((\text{ISI}_\text{in} = 0.95 \cdot \text{ISI}_\text{inh} + 6.5)^{10} \). Crosstalk (measuring radioactivity over the contralateral hemisphere originating from the homolateral side) was below 15% if all detectors close to the base and to the superior sagittal sinus were omitted or at least directed away from these points by slightly tilting their axis toward a focussing center behind the contralateral side. Test-retest procedures indicated that rCBF changes vary little from measurement to measurement in the absence of any disease. Our calculations revealed that rCBF changes of more than 10% represent true flow alterations.

However, since 2-dimensional techniques measure flow predominantly in the superficial grey matter, regional ischemic foci of deeper location might be overlooked or at least underestimated. Still it can be assumed that crosstalk or look through phenomena do not play a decisive role in patients of this kind since none of our patients presented with a "mirror focus" or with flow changes over the contralateral hemisphere which were identical to that of the homolateral (TIA) side. Although the peak-valley map (relative distribution of flow compared to the absolute mean regional value) in the TIA-side changed over the period of the tests, the relative variation of rCBF over the contralateral side was not altered. The stable flow map of the hemisphere which was not involved in the TIA is related to its preexisting state.

If measured flow and its pattern changes are considered as reflecting true flow values, what then causes the individual pattern? There are only a few observations on rCBF in TIA in the literature, as summarized in table 4.
With a few individual exceptions CBF-measurements of all patients were performed in the sub-acute and chronic state of TIA (days to weeks after the attack). Later serial measurements were not a part of these investigations. Taking into account all the literature which is known to us there are no reports about early and repeated measurements of rCBF in TIA.

Our studies indicate that even in the very early phase of the attack mean rCBF was normal in most of the patients. However, different types of focal disturbance can be detected on the day of the attack. These include reduction of rCBF in 22% of all areas, but hyperemia in only 6%. This distribution changes rather rapidly by the next day, but more than a tenth of all areas still presented with relative ischemia one day after the ischemic attack (fig. 3). In contrast to blood flow in complete infarction, diaschisis \(^6\) \(^7\) was not observed. Separating all patients who still had symptoms during the first CBF-measurement from those who had become normal when CBF 1 was done, there was no significant difference between the actual flow, the distribution of relative decreased or increased flow and the flow changes over the next seven days. A 3-dimensional method of measuring CBF \(^5\) might detect differ-

**Figure 3.** The course of rCBF in all patients, grouped according to the actual focal flow and the flow change in the focus. The areas of the hemispheres are classified in focal, perifocal and distal areas. Black points plus vertical bars indicate the flow and the standard deviation in the involved side, open rhombs the flow in the corresponding areas of the contralateral hemisphere. Flow expressed as initial slope index (sec \(^{-1}\)). Early normalization (Group II): rCBF improves significantly from CBF 1 to CBF 2. Delayed normalization (Group III): rCBF improves significantly from CBF 2 to CBF 3. Early hyperperfusion (Group IV): rCBF in the "focal" detectors of the TIA-side is significantly higher than in the corresponding contralateral areas at CBF 1. Delayed hyperperfusion (Group V): rCBF in the focus increases significantly from CBF 1 to CBF 2 and decreases from CBF 2 to CBF 3. Persistent focus (Group VI): In the focus there is no change of rCBF which is significantly lower than in the corresponding areas of the contralateral side.
ent rCBF-patterns, but there is no report about the use of these techniques in the very acute phase of TIA. In addition it is possible that the similar behavior of these subgroups (patients with symptoms during CBF 1 and patients who have become normal before the first CBF estimation) based on the fact that all CBF-levels were found above 25 (sec$^{-1}$). This is still above the level where electrical function is affected.18

Regarding the individual patterns of rCBF changes (table 3, fig. 3) it is not possible to explain all of the differences. In ten patients (group I and II) CBF was normal on day 2, which corresponds to the clinical course. In group III rCBF still increased until day 7, which might indicate a further improvement of flow and recovery of blood flow control at a level where clinical symptoms do not appear. This is in agreement with the observations of others who have reported disturbances of autoregulation4 and blood gas reactivity3,9 a long time after the attack. In two patients (group IV) early elevated flow was measured thus indicating relative luxury perfusion.19 This relative hyperemia was accompanied by transient ischemia in the neighbour-hood, which might represent a steal phenomenon.

Two other patients (group V) developed focal hyperemia on day 2 accompanied by normal flow in the perifocal detectors (parietal areas in both hemiparetic patients with upper limb predominance). In group VI (persistent focus) there was no significant change of flow from CBF 1 to CBF 3 except for the perifocal areas which showed an increased flow from day 2 to day 7. The stable flow pattern in the focal detectors might represent the acute ischemia of the TIA with loss of recovery, but on the other hand it could represent the stable flow which was already present before the attack. One of our three patients suffered from complete occlusion of one ICA (and could have suffered from a transient hemodynamic crisis). One case had arterio-sclerotic disease of the neck vessel (which raises the question whether he suffered from TIA as a result of embolic episodes). In the third case no cause for the TIA could be found.

All the patients in our series had a normal hematocrit and a normal hemoglobin. This excludes a transient disturbance of tissue perfusion due to alteration of blood characteristics, apart from an increased level of fibrinogen, which however was not estimated in all of the cases. Lacunar infarcts cannot be excluded as a cause of the attacks20 particularly among those patients where initial blood flow was normal, since ischemic lesions in deeper structures might remain undetected by 2-dimensional CBF-measurements.

The pattern of blood flow after an ischemic event does not depend only on possible reperfusion. Damage to metabolic processes through prolonged uncoupling, tissue acidosis and hyperoxidation21 do influence tissue function in spite of a normal CBF. Lenzi et al,22 using positron emission tomography in patients with TIA some time after the attack, observed pathological values for both blood flow and oxygen metabolism with greater regional defects for the former. Our studies indicate that blood flow might be abnormal even 24 hours after the ischemic attack. This is an agreement

<table>
<thead>
<tr>
<th>Author et al (Ref.)</th>
<th>Method</th>
<th>Time from TIA to CBF-measurement</th>
<th>Focal abnormalities</th>
<th>Repeated measurements</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rees (3)</td>
<td>i.a. Xe 133</td>
<td>11 8-90 days</td>
<td>7 cases with hypoemia and hyperemia</td>
<td>no</td>
</tr>
<tr>
<td>Skinhoj (4)</td>
<td>i.a. Xe 133</td>
<td>12 40 min-4 years</td>
<td>focal hyperemia in 1 patient 30 min after TIA. Focal ischemia in 1 patient 1 day after TIA. No focal abnormalities in 9 cases. 1 patient with disturbed autoregulation</td>
<td>no</td>
</tr>
<tr>
<td>Wong (5)</td>
<td>i.a. Xe 133</td>
<td>22 1-90 days</td>
<td>in about 80% of all cases focal abnormalities plus clinical symptoms. In many areas focal abnormalities without clinical symptoms</td>
<td>no</td>
</tr>
<tr>
<td>Ackerman (6)</td>
<td>inhal. Xe 133</td>
<td>22 not mentioned</td>
<td>not mentioned</td>
<td>no</td>
</tr>
<tr>
<td>Naritomi (7)</td>
<td>inhal. Xe 133</td>
<td>15 not mentioned</td>
<td>not mentioned. Patients presented with normal autoregulation</td>
<td>no</td>
</tr>
<tr>
<td>Yonekura (8)</td>
<td>inhal. Xe 133</td>
<td>87 not mentioned</td>
<td>occipital, frontal, parietal, but not temporal. Some cases presented with bilateral flow reduction</td>
<td>no</td>
</tr>
<tr>
<td>Tsuda (9)</td>
<td>i.a. Xe 133 detectors placed over the vertex</td>
<td>18 4 days to 3 months</td>
<td>CO$_2$-responsiveness normal in TIA-patients with internal carotid artery stenosis but abnormal in patients with internal carotid artery occlusion</td>
<td>no</td>
</tr>
<tr>
<td>Vorstrup (15)</td>
<td>inhal. Xe 133 (SPECT)</td>
<td>14 days to weeks</td>
<td>in 9 cases focal abnormalities. No change of rCBF in some cases after extracranial-intracranial bypass-surgery</td>
<td>yes</td>
</tr>
<tr>
<td>Lenzi (22)</td>
<td>positron emission tomography</td>
<td>11 not mentioned</td>
<td>blood flow disturbance was more prominent than metabolic alteration</td>
<td>no</td>
</tr>
<tr>
<td>Meyer (23)</td>
<td>inhal. Xe 133</td>
<td>19 3-about 75 days</td>
<td>focal abnormalities in the first 3 weeks after TIA</td>
<td>no</td>
</tr>
</tbody>
</table>
with the observation of Meyer et al., who reported reduction of flow over the involved and the contralateral hemisphere up to 3 weeks after the ischemic insult. However, repeat studies were not done.

Studies of blood flow in the brain do not differentiate attacks caused by embolic events from those due to hemodynamic crises. This study indicates that blood flow might not have returned to normal by the day after the attack, so that any additional attack occurs at a stage when the ischemia could be less well tolerated.

References
Prolonged disturbances of regional cerebral blood flow in transient ischemic attacks.

A Hartmann

Stroke. 1985;16:932-939
doi: 10.1161/01.STR.16.6.932

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