The Effect of Cerebral Infarction on the Regional Cerebral Blood Flow of the Contralateral Hemisphere

BY SYLVAN LAVY, M.D., ELDAD MELAMED, M.D., AND ZIPPORA PORTNOY, M.SC.

Abstract: Regional cerebral blood flow (rCBF) measurements were performed over the contralateral hemisphere by the 133Xe intracarotid injection method in 20 patients with acute cerebral infarction in the territory of the internal carotid artery. The rCBF was found to be reduced, sometimes remarkably, in all of the patients. The mean reduction was 30% to 36% from the lowest normal value for the mean age of these patients. In the younger age group (40 to 59) the reduction was greater, 40% to 47% from the lowest normal value for this age.

The rCBF depression was not related to cerebral dominance, previous hypertension or arterial Pco2 levels. It occurred in both patients who were fully alert and those with disturbances of consciousness, although it tended to be more diminished in the latter.

It can be assumed that the flow reduction in the nonaffected hemisphere is part of a general phenomenon affecting the entire brain and caused by globally reduced cerebral metabolism.

Additional Key Words: hemodynamic changes cerebral metabolism ischemia 133Xenon intracarotid injection method

Introduction

It is well known that in some patients with cerebral infarction there is a reduction of the cerebral blood flow not only in the limited region of ischemia but also globally depressed over the whole affected hemisphere. Less is known of the hemodynamic changes occurring in the contralateral hemisphere of stroke patients with unilateral cerebral infarction. Several authors observed a reduction in the cerebral blood flow of the nonaffected hemisphere in a small number of stroke patients and in some experimental animals with occlusion of the middle cerebral artery. Such results were not obtained by others, either in human subjects or in experimental animals. A more detailed and systematic study using a larger number of cases therefore seemed warranted.

The aim of this study is to present the results of rCBF measurements over the contralateral hemisphere determined by the 133Xe intracarotid injection method and performed during the acute stage of cerebral infarction in 20 consecutive patients.

Methods

Twenty consecutive patients, hospitalized with acute cerebrovascular accident due to ischemia in the territory of the internal carotid arterial system, were studied. Their ages ranged from 48 to 85 years, with a mean age of 65 years. Twelve were males and eight females. In each patient, the diagnosis of cerebral infarction in the distribution of the internal carotid artery was established on the basis of the history, careful neurological examination, lumbar punctures, serial EEG recordings and 99m Tc brain scans. Cerebral angiograms, ipsilateral to the hemispheric lesion, were performed in all cases. Occlusion of a major artery, either the internal carotid or the middle cerebral artery, was observed in eight patients. In an additional case, occlusion of the left internal carotid artery with massive hemispheric infarction was demonstrated at autopsy. All of the patients were, as a rule, severely affected and had hemiparesis or hemiplegia with either hemianesthesia, homonymous hemianopia or aphasia in varying degrees of severity. At the time of the study, none were stuporous or in coma. Ten patients were fully alert and cooperative, while the other ten had disturbances of consciousness such as drowsiness, confusion or disorientation. These were rendered alert before and during the study by vocal or painful stimuli.

Patients with evidence of pre-existing dementia and/or an old stroke in the contralateral hemisphere were not included in this study. Patients diagnosed as having capsular infarction were likewise excluded.

Patients were considered as hypertensive prior to stroke if a reliable history of diastolic hypertension of 100 mm Hg and above combined with at least two recordings of arterial pressure of 160/95 mm Hg and above taken seven days or more after the acute event on follow-up examinations were obtained. Thus, 12 patients were regarded as hypertensive and eight normotensive prior to the stroke.

rCBF was determined over the contralateral hemisphere...
EFFECT OF CEREBRAL INFARCTION ON rCBF

TABLE 1

rCBF Studies in Hemisphere Contralateral to Cerebral Infarction

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age</th>
<th>Sex</th>
<th>State of consciousness</th>
<th>Hemisphere studied</th>
<th>MABP</th>
<th>Pco2</th>
<th>Mean rCBF2 (ml/100 gm/min)</th>
<th>Mean rCBF10 (ml/100 gm/min)</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>56</td>
<td>M</td>
<td>N</td>
<td>Right</td>
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<td>28</td>
<td>27</td>
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<tr>
<td>2</td>
<td>78</td>
<td>F</td>
<td>Drowsy</td>
<td>Left</td>
<td>116</td>
<td>32</td>
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<td>13</td>
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<td>3</td>
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<td>Mildly confused and disoriented</td>
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<td>130</td>
<td>34</td>
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<td>9</td>
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<tr>
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<td>Left</td>
<td>120</td>
<td>40</td>
<td>19</td>
<td>15</td>
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<tr>
<td>6</td>
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<td>N</td>
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<td>30</td>
<td>27</td>
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<td>66</td>
<td>M</td>
<td>N</td>
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<td>26</td>
<td>18</td>
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<tr>
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<td>32</td>
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<td>Left</td>
<td>106</td>
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<tr>
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<td>M</td>
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<td>Drowsy</td>
<td>Left</td>
<td>103</td>
<td>34</td>
<td>8</td>
<td>11</td>
</tr>
</tbody>
</table>

Results

The pertinent clinical data and the results obtained are presented in table 1.

The rCBF measured over the contralateral hemisphere was found to be reduced in all of the patients studied. As this reduction was uniform over the entire hemisphere, and with no significant difference among the regions studied, the results are presented as mean rCBF values. For all age groups, the mean rCBF2 was 23 and the mean rCBF10 was 25 ml/100 gm per minute. This is a reduction of 36% and 30% accordingly from the lowest normal value obtained in our laboratory for the mean age of 65, which is 36 ml/100 gm per minute. For the 40 to 59 age group, the mean rCBF2 was 24 and the mean rCBF10 was 27 ml/100 gm per minute. This is a reduction of 47% and 40% accordingly from our lowest normal value of 45 ml/100 gm per minute for this age group. For the age group above 60 with mean rCBF2 of 21 and rCBF10 of 23 ml/100 gm per minute, this is a reduction of 41% and 36% accordingly from our lowest normal value recorded for this age group. In six patients (Case Nos. 2, 4, 5, 8, 17, and 20) the rCBF of the contralateral hemisphere was remarkably diminished. The mean rCBF values were approximately the same for all age groups, except for the range of 70 to 79 years where lower levels were noted (table 2).

The rCBF reduction was the same for patients with lesions in the dominant and non-dominant hemispheres (table 3). It was the same for patients who were either hypertensive or normotensive prior to the stroke (table 3). In ten patients, the arterial Pco2 at the time of the study was lower than 34 mm Hg, which is the lowest normal level in our laboratory. However,
TABLE 3

Mean rCBF of Contralateral Hemisphere According to Side, State of Consciousness, Previous Hypertension and Arterial Pco2

<table>
<thead>
<tr>
<th>Contralateral hemisphere studied</th>
<th>State of consciousness</th>
<th>Previous hypertension</th>
<th>Arterial Pco2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Right (10)</td>
<td>Left (10)</td>
<td></td>
</tr>
<tr>
<td>Mean rCBF2 (ml/100 gm/min)</td>
<td>24</td>
<td>23</td>
<td>26</td>
</tr>
<tr>
<td>Mean rCBF10 (ml/100 gm/min)</td>
<td>24</td>
<td>25</td>
<td>27</td>
</tr>
</tbody>
</table>

This did not seem to play an important role in the degree of rCBF reduction, since contrary to the expected, the patients with lower arterial Pco2 had somewhat higher values (table 3).

None of the patients was stuporous or in coma. However, the rCBF was lower in those with disturbances of consciousness such as drowsiness or confusion, although they were rendered alert at the time of the study by vocal or painful stimuli (table 3).

Bilateral rCBF measurements were performed in four patients (table 4). The hemisphere contralateral to the lesion was studied first in each patient. The rCBF of the ipsilateral hemisphere was determined after an interval of one week. In three cases, the mean rCBF values measured over the affected hemisphere were the same or somewhat lower than those of the opposite one, and in one case they were higher.

Discussion

The results of this study show that in this series of patients with acute cerebral infarction in the territory of the internal carotid arterial system, the rCBF is reduced, sometimes remarkably, in the hemisphere contralateral to the lesion. This is in accordance with the findings of Hedt-Rasmussen and Skinhøj, Fieschi et al., and McHenry. It appears from the present study and from the results of other investigators that even though the rCBF is sometimes more reduced in the infarcted hemisphere its depression in the contralateral hemisphere is part of a general, global phenomenon affecting the entire cerebrum. There is recent evidence that not only the cerebral blood flow but also the autoregulation may be abnormal in the opposite hemisphere.

This phenomenon is clearly related to the acute stroke as it is highly unlikely that a reduction of CBF, which was sometimes remarkable, would not have produced focal or diffuse neurological signs before the onset of the infarction.

Factors such as pre-existing cerebral arteriosclerotic changes, increased intracranial pressure in some patients with cerebral infarction, and cerebrovascular spasm in some cases with hemorrhagic stroke may contribute to the overall depression of cerebral blood flow but do not provide the main explanation for this phenomenon. The reduction of rCBF was found to be greater in patients with disturbed consciousness but occurred also in the absence of such abnormalities. It did not seem to be related to cerebral dominance, previous hypertension, or the level of arterial Pco2.

In some stroke patients, there is an elevation of pyruvate and lactate with lowering of the pH in the cerebrospinal fluid, reflecting the presence of brain tissue acidosis due to the infarction. However, were the acidosis to spread and implicate the opposite hemisphere, it would lead to hyperemia rather than to a depression of the CBF.

It seems more likely that the global depression of cerebral blood flow in the affected and “non-diseased” hemispheres is induced by the overall reduction of cerebral metabolism occurring in stroke patients. It was suggested by Hedt-Rasmussen and Skinhøj that neurogenic mechanisms play a role in this process, and they used the term “transneural depression.” Thus, the injured neurons in the infarcted region lead to a decrease in function of neurons in other, remote parts of the brain bilaterally, as their...
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ability to activate the intrahemispheric and interhemispheric connections is lost or reduced. This results in depressed metabolism, not only in the focus of infarction, but in other regions of the same hemisphere and the contralateral one with the ensuing global reduction of the cerebral blood flow. Support for this theory is lent by the observation that global depression of cerebral metabolism and blood flow may be caused by brain-stem lesions affecting the reticular formation. It also may be possible that the reduced mental activity in stroke patients leads to the depression of cerebral metabolism and blood flow, as occurs in patients with dementia. Each theory has yet to be proved.

Acknowledgment
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References
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