Direction of Flow in Posterior Communicating Artery on Magnetic Resonance Angiography in Patients With Occipital Lobe Infarcts

Jacqueline C.F. Jongen, MD; Cees L. Franke, PhD; Lino M.P. Ramos, MD; Jan T. Wilmink, PhD; Jan van Gijn, FRCP

Background and Purpose—In some people the blood supply to the posterior cerebral artery occurs partly or even exclusively via the carotid system. This anatomic configuration may influence the risk of occipital lobe infarction. We studied the presence and direction of flow in the posterior communicating artery (PCoA) in patients with an occipital lobe infarct and in healthy controls.

Methods—Forty-seven patients with an occipital lobe infarct were studied by MR angiography, as well as 50 young healthy controls. Special attention was paid to the presence of a PCoA and, if present, to the direction of flow.

Results—Significantly fewer patients than controls had an exclusive blood supply to the posterior cerebral artery via the carotid system, in both the affected (4% versus 17%; 95% CI of difference, 4% to 22%) and unaffected hemispheres (5% versus 17%; 95% CI of difference, 3% to 22%). Patients also less often had a patent PCoA with anteroposterior flow than controls (affected hemisphere, 8% versus 22%; unaffected hemisphere, 12% versus 22%; 95% CI of differences, 3% to 25% and −2% to 23%, respectively). With analysis at the level of individuals, significantly more patients showed no anteroposterior flow through the PCoA in either hemisphere than controls (79% versus 42%; 95% CI of difference, 19% to 55%).

Conclusions—Supply of the posterior cerebral artery by the carotid system occurs less often in patients with an occipital lobe infarct than in healthy controls. The same was true for the unaffected hemisphere of patients, which suggests that the anatomic difference represents a causal factor (fewer collateral pathways after occlusion of the posterior cerebral artery or its branches) rather than a consequence (redistribution of blood flow after occipital infarction). (Stroke. 2004; 35:104-108.)

Key Words: cerebral arteries ■ cerebral ischemia ■ cerebrovascular circulation ■ collateral circulation ■ magnetic resonance angiography

In some subjects the posterior cerebral artery (PCA) is exclusively supplied by the internal carotid artery, via the posterior communicating artery (PCoA), with agenesia or hypoplasia of the P1 segment of the PCA. The proportion of individuals with such a “fetal variant” has been estimated at 15% to 46%, on the basis of anatomic1–4 and angiographic5 studies. If a patient has an occipital lobe infarct, a possible relationship between the infarct and the parent artery of the PCA needs to be considered. For example, in a patient with a fetal variant or even a patent PCoA with anteroposterior flow, a severe carotid stenosis could be responsible, and carotid endarterectomy might be warranted to prevent further ischemic strokes. This also applies to patients with a persistent trigeminal artery, which is much rarer.6,7

It is unknown whether contribution to the occipital lobe from the carotid territory in patients with occipital lobe infarction is more common or less common than in the general population. A double blood supply (patent PCoA with anteroposterior flow) would expose the occipital lobe to a double source of emboli and might predispose to embolic infarction in the occipital lobe. Alternatively, a supply by the posterior or anterior circulation alone might make the occipital lobe relatively vulnerable. To answer this question, it is important to assess the direction of flow through the PCoA, without influencing this direction by invasive tests. Previous studies of the hemodynamics of the circle of Willis have been done,8–10 but none with special attention to occipital lobe infarction.
In a series of patients with an occipital lobe infarct, we prospectively investigated the frequency of a fetal variant of the blood supply to the PCA and of an anteroposterior direction of flow in case of a patent PCoA. A group of young healthy individuals served as controls.

Subjects and Methods

We reviewed the files of 121 consecutive patients with an infarct in the occipital lobe demonstrated by CT or MRI scanning of the brain in the university hospitals of Utrecht and Maastricht and in the Atrium Medical Center in Heerlen in the Netherlands, from January 1997 to March 2002. We excluded patients with insufficient data, migraine as a potential cause of the infarct, incidental finding of occipital lobe infarction, possible border zone infarction, and presence of cerebral metastases. The remaining patients were asked for written informed consent to allow the use of their medical data and to undergo an MRI study including a MR angiogram (MRA). We reviewed the medical history with special attention to diabetes mellitus, hypertension, hypercholesterolemia, cardiac arrhythmias, thrombophilia, peripheral artery disease, migraine, malignant disease, and a history of stroke. We also extracted data about medication, in particular oral anticoagulation or acetylsalicylic acid at the time of occipital lobe infarction, smoking or alcohol use, and the results of investigations (blood chemistry, ECG, chest x-ray, echocardiogram, carotid artery duplex scanning, and CT or MRI of the brain).

Forty-seven patients were included in the study and gave permission for an MRA of the brain. One other patient was excluded at a later stage because in the intervening period he appeared to have developed an asymptomatic carotid artery occlusion on the side of the occipital lobe infarct. The remaining 73 patients were not included for the following reasons: the patient had died (n=16) or was terminally ill (n=1), the patient was not prepared or able to come to the hospital for the MRA or had no means of transport (n=29), or the patient did not respond to our letter and at least 1 phone call (n=24). Three patients could not cooperate because of metal implants in their body. Four of the 47 participating patients had bilateral occipital lobe infarcts, so that the number of affected hemispheres was 51.

Patients and controls underwent MRI with MRA in a 1.5-T system. Standard MRI included a sagittal T1-weighted sequence with repetition time (TR)/echo time (TE) 598/18 ms; 19×5.0/0.5-mm slices; field of view (FOV) 250 mm; matrix 179/256; number of excitations (NEX) 1; and an axial dual-echo proton density T2-weighted sequence with TR/TE/echo train length 2200/9.1 to 100 ms/14; 19×6.0/1.2-mm slices; FOV 230 mm; matrix 224/256; NEX 1. MRA was performed with a 3-dimensional time-of-flight (TOF) sequence10,11 with TR/TE/flip angle 274/6.5 ms/20°; partitions 100×0.8/0.4 mm overcontiguous; FOV 110 mm; matrix 123×256; NEX 2; and two 2-dimensional phase-contrast directional flow sequences (right to left and anterior to posterior) with TR/TE/flip angle 148/9.9 ms/7.5°; slice 13 mm; velocity encoding 40 cm/s; FOV 250 mm; matrix 205×256; NEX 8. The 3-dimensional TOF images were displayed as orthogonal maximum intensity projections and as a set of 12 maximum intensity projection images rotated around the left-right axis. The 2-dimensional phase-contrast acquisitions were displayed as directional flow images, right to left and anterior to posterior. The validity of the 2-dimensional sequences for determining the direction of flow was established in a previous study.12

The interval between the time of the occipital lobe infarct and that of scanning ranged between 1 and 47 months (median, 15 months). The 22 patients originally seen in the hospitals in Heerlen and Maastricht were scanned in Maastricht, and the other 25 patients were scanned in Utrecht. Fifty healthy persons aged between 19 and 30 years served as a control group and underwent the same MRA protocol and assessment as described above; 25 were investigated in Utrecht and 25 in Maastricht.

All 97 MRA studies were evaluated in consensus by 2 experienced neuroradiologists (J.T.W. and L.M.P.R.). They reported on the brain anatomy (any incidental findings or new infarcts) and on the anatomy of the circle of Willis and the direction of flow. Special attention was paid to the presence of the PCoA and, if present, whether the direction of flow was anteroposterior (a→p) or posteroanterior (p→a) (Figures 1 and 2).

Patients were divided into 3 groups, according to the blood supply of the PCA: (1) fetal variant; (2) PCoA, a→p flow (patent PCoA with anteroposterior flow); and (3) no a→p flow (all subjects who showed no PCoA on MRA or who had a patent PCoA with a posteroanterior or undetermined flow).

Figure 1. Large PCoA with a hypoplastic or absent P1 segment on the right (R) (fetal variant, single arrow). The vascular outline in the region of the right PCA represents a superior cerebellar artery. A patent PCoA is shown on the left (L) (double arrow).

Figure 2. Anterior-to-posterior phase-directional flow image reveals anteroposterior flow within both PCoAs. R indicates right; L, left.
The protocol was approved by the institutional review boards of the Atrium Medical Center in Heerlen and of the university hospitals in Utrecht and Maastricht.

Results

The baseline characteristics are listed in Table 1. There were no marked differences, except for malignant disease and use of oral anticoagulants. Three patients had atrial fibrillation or another major source of emboli in the heart. In 13 patients (28%) no ultrasound scanning or CT angiography of the carotid and vertebral arteries was performed; the results of the available data of the remaining 34 patients are listed in Table 2.

Tables 3 and 4 list the MRA results of patients and controls, per hemisphere and per individual. Patients significantly less often had a fetal variant (blood supply to the PCA exclusively via the carotid system and PCoA) than controls (Table 3) in both the affected hemisphere (4% versus 17%; 95% CI of difference, 4% to 22%) and the unaffected hemisphere (5% versus 17%; 95% CI of difference, 3% to 22%). Patients also had a patent PCoA with anteroposterior flow less often than controls (affected hemisphere, 8% versus 22%; 95% CI of difference, 3% to 25%; unaffected hemisphere, 12% versus 22%; 95% CI of difference, -2% to 23%). Together, significantly more hemispheres in patients showed no anteroposterior flow through the PCoA than controls (68% versus 35%; 95% CI of difference, 20% to 46%).

There were no significant differences between the affected and unaffected hemispheres in patients. On analysis at the level of individuals, significantly more patients had no anteroposterior flow through the PCoA than controls (86% versus 61%; 95% CI of difference, 13% to 37%). The direction of flow through the PCoA could not be determined with confidence in 44% of all visible PCoAs [Table 3: (9 + 8 + 26)/(4 + 5 + 22 + 5 + 4 + 14 + 9 + 8 + 26)]. When the analysis was repeated on the assumption that all these patients in fact had some anteroposterior flow, the difference between patients and controls remained significant (68% versus 35%; 95% CI of difference, 20% to 46%).

Seven patients showed additional parenchymal infarcts on the MRI study that had not been evident on the original CT scan of the brain; there were no other incidental findings. In

### Table 1. Baseline Characteristics

<table>
<thead>
<tr>
<th></th>
<th>Total Group n=121</th>
<th>Included Group n=47</th>
<th>Controls n=50</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age range, y</td>
<td>20–92</td>
<td>25–81</td>
<td>19–30</td>
</tr>
<tr>
<td>Median age</td>
<td>72</td>
<td>66</td>
<td>25</td>
</tr>
<tr>
<td>Sex (M:F)</td>
<td>65:56</td>
<td>26:21</td>
<td>21:29</td>
</tr>
<tr>
<td>Interval of MRA after infarction</td>
<td>N/A</td>
<td>Range, 1–47 mo</td>
<td>Median, 15 mo</td>
</tr>
</tbody>
</table>

### Table 2. Results of Available Imaging of the Arteries of the Neck by Duplex Scanning or CT Angiography in 34 Patients

<table>
<thead>
<tr>
<th></th>
<th>Carotid Arteries</th>
<th>Vertebral Arteries</th>
</tr>
</thead>
<tbody>
<tr>
<td>n=34</td>
<td>(100%)</td>
<td></td>
</tr>
<tr>
<td>25</td>
<td>(74%)</td>
<td>No stenosis or &lt;70%</td>
</tr>
<tr>
<td>2</td>
<td>(6%)</td>
<td>Unilateral stenosis &gt;70%</td>
</tr>
<tr>
<td>2</td>
<td>(6%)</td>
<td>Unilateral carotid occlusion</td>
</tr>
<tr>
<td>1</td>
<td>(3%)</td>
<td>No stenosis or &lt;70%</td>
</tr>
<tr>
<td>1</td>
<td>(3%)</td>
<td>No stenosis or &lt;70%</td>
</tr>
<tr>
<td>1</td>
<td>(3%)</td>
<td>No stenosis or &lt;70%</td>
</tr>
</tbody>
</table>

*Numbers do not add up to 100% due to rounding of percentages.*
the control group, 2 subjects showed prominent VirchowRobin spaces; no other abnormalities were found.

### Discussion

The results of this study show a significant difference in the hemodynamics in the circle of Willis between patients with occipital lobe infarction and controls, in that the occipital lobes in both affected and unaffected hemispheres of patients less often received blood entirely or partly via the carotid system than control subjects. The controls were younger than the patients, but an earlier study in the general population found no significant differences according to age with respect to the function of the circle of Willis.13

Our finding that the unaffected hemisphere in patients was also less often supplied via the anterior circulation than in controls argues in favor of an anatomic predisposition rather than redistribution. In an earlier study we showed that there is some degree of interdependence in the shape of the hemicircles of Willis in either hemisphere.5

We did not include the size of the infarct in the analysis since this is determined by many unknown factors other than the presence of anteroposterior flow in the PCoA: the site and duration of occlusion of the PCA, the territories of the major cerebral arteries in a given individual, and the role of collateral pathways on the pial surface.

Our selection criteria did not exclude a dissection of the carotid or vertebral artery as the cause of occipital lobe infarction because emboli thus formed will follow the blood stream in the same way as with atherosclerotic lesions. We excluded patients in whom migrainous infarction was suspected because the pathogenesis of infarction in such cases is probably unrelated to the vascular anatomy of the occipital lobe. Another potential source of bias is the interval between the MRA study and the occipital lobe infarct. In the meantime, the anatomy of the circle of Willis might have changed. For example, occlusion in the anterior circulation might have caused an anteroposterior flow through the PCoA to become posteroanterior (and vice versa, but the incidence of “silent”

### TABLE 3. Relationship of the Internal Carotid and Posterior Cerebral Artery per Hemisphere (Affected or Unaffected)

<table>
<thead>
<tr>
<th>MRA Results</th>
<th>Patients</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Affected (n=51)</td>
<td>Unaffected (n=43)</td>
</tr>
<tr>
<td>Fetal variant</td>
<td>2 (4%, 0%-14%)</td>
<td>2 (5%, 1%-16%)</td>
</tr>
<tr>
<td>Patent PCoA, a→p flow</td>
<td>4 (8%, 2%-19%)</td>
<td>5 (12%, 4%-25%)</td>
</tr>
<tr>
<td>No a→p flow</td>
<td>45 (88%, 76%-96%)</td>
<td>36 (84%, 69%-93%)</td>
</tr>
<tr>
<td>No PCoA</td>
<td>n=31</td>
<td>n=24</td>
</tr>
<tr>
<td>Patent PCoA, p→a flow</td>
<td>n=5</td>
<td>n=4</td>
</tr>
<tr>
<td>Patent PCoA, flow unclear</td>
<td>n=9</td>
<td>n=8</td>
</tr>
</tbody>
</table>

* Four patients had bilateral occipital infarcts.

95% CI indicates 95% confidence interval.

PCoA, a→p flow=patent posterior communicating artery with anteroposterior flow.

No a→p flow=no anteroposterior flow through the PCoA, if existent.

PCoA, p→a flow=patent posterior communicating artery with posteranterior flow.

PCoA, flow unclear=patent posterior communicating artery, direction of flow could not be determined.

### TABLE 4. Relationship of the Internal Carotid and Posterior Cerebral Artery per Individual; Variations of the Posterior Communicating Artery

<table>
<thead>
<tr>
<th>MRA Results</th>
<th>Patients</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n=47 (% , 95% CI)</td>
<td>n=50 (% , 95% CI)</td>
</tr>
<tr>
<td>Fetal variant, both sides</td>
<td>1 (2%, 0%-11%)</td>
<td>2 (4%, 0%-14%)</td>
</tr>
<tr>
<td>Fetal variant one side, a→p flow other side</td>
<td>0 (0%, 0%-8%)</td>
<td>3 (6%, 1%-17%)</td>
</tr>
<tr>
<td>Fetal variant one side, no a→p flow other side</td>
<td>2 (4%, 1%-15%)</td>
<td>10 (20%, 10%-34%)</td>
</tr>
<tr>
<td>a→p flow, both sides</td>
<td>2 (4%, 1%-15%)</td>
<td>5 (10%, 3%-22%)</td>
</tr>
<tr>
<td>a→p flow one side, no a→p flow other side</td>
<td>5 (11%, 4%-23%)</td>
<td>9 (18%, 9%-31%)</td>
</tr>
<tr>
<td>No a→p flow, both sides</td>
<td>37 (79%, 64%-89%)</td>
<td>21 (42%, 28%-57%)</td>
</tr>
</tbody>
</table>
carotid occlusion is much higher than that of vertebral artery occlusion\(^\text{15}\)). It is precisely for this reason that we excluded 1 patient in whom carotid occlusion had recently developed. Unfortunately, we did not have sufficient data on ultrasound scanning of the patients’ carotid and vertebral arteries because the clinicians were not interested in these data as it would not have any consequence for surgery. To change this perception is exactly why we performed this study (among others).

Use of MRA to assess blood flow over the circle of Willis is not associated with transient increases in arterial blood pressure due to contrast injection, as is the case in conventional angiography. Although injection-related flow changes can usually be recognized by careful study of early washout of the contrast medium after injection pressure has dropped, MRA avoids any possible ambiguity in this respect. Accordingly, the proportion of control subjects in this study in whom the PCA derived its blood supply exclusively from the carotid artery (17%) is consistent with the proportion of 11% we found in a previous study of angiograms.\(^\text{5}\)

In the present study the neuroradiologists could not determine direction of flow through the PCoA with confidence in 44% of all visible PCoAs. Two aspects in the scan protocol may have contributed to this problem. First, the directional flow images were scanned at a fixed, predetermined angle in all subjects, whereas on sagittal images some subjects showed a relatively vertical course of the PCoA. Second, flow velocity encoding was set on 40 cm/s, which was sufficiently accurate in most cases but not in others, resulting in insufficient flow imaging. This problem might have been reduced by scanning the directional flow images in an individually variable angle determined by the sagittal image and by adjusting the flow velocity encoding settings when no flow could be determined in an otherwise visible artery. Our uniform protocol may have caused a slight underestimation of the number of subjects (in both groups) with anteroposterior flow, since in healthy controls this direction of flow was more often identified than posteranterior flow (Table 3; 22 versus 14 subjects). Since this applies to patients as well as controls, we do not think this technical factor can have greatly influenced the results. A third reason for failure to determine flow direction in the PCoA may be that in some subjects the flow may have been to and fro. In that case there is no misclassification because the anterior and posterior circulations are separate in a physiological sense.

In summary, we found that a contribution from the carotid system to the occipital lobe was less frequent in patients with occipital lobe infarction than in healthy controls. That we found the same for the unaffected hemispheres in the patient group strongly suggests that the difference represents a causal factor and not a consequence. Whichever explanation is correct, patients with occipital infarction in whom the occipital lobe is entirely or mostly supplied by the carotid system, although relatively rare, should in our view be investigated for the presence of ipsilateral carotid stenosis, with a view to surgery.

Acknowledgments

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