Background and Purpose—Knowledge of the extent and distribution of infarcts of the posterior cerebral artery (PCA) may give insight into the limits of the arterial territory and infarct mechanism. We describe the creation of a digital atlas of PCA infarcts associated with PCA branch and trunk occlusion by magnetic resonance imaging techniques.

Methods—Infarcts were manually segmented on T2-weighted magnetic resonance images obtained ≥24 hours after stroke onset. The images were linearly registered into a common stereotaxic coordinate space. The segmented images were averaged to yield the probability of involvement by infarction at each voxel. Comparisons were made with existing maps of the PCA territory.

Results—Thirty patients with a median age of 61 years (range, 22 to 86 years) were studied. In the digital atlas of the PCA, the highest frequency of infarction was within the medial temporal lobe and lingual gyrus (probability = 0.60 to 0.70). The mean and maximal PCA infarct volumes were 55.1 and 128.9 cm³, respectively. Comparison with published maps showed greater agreement in the anterior and medial boundaries of the PCA territory compared with its posterior and lateral boundaries.

Conclusions—We have created a probabilistic digital atlas of the PCA based on subacute magnetic resonance scans. This approach is useful for establishing the spatial distribution of strokes in a given cerebral arterial territory and determining the regions within the arterial territory that are at greatest risk of infarction. (Stroke. 2007;38:1805-1811.)

Key Words: middle cerebral artery ■ digital techniques ■ atlas ■ stroke
affected by arterial occlusion in subjects with ischemic stroke because of the younger age of subjects used to create the cadaver and MR perfusion maps. Atherosclerotic disease in stroke patients may also lead to the development of leptomeningeal collaterals, which may influence the region at risk of infarction. Hence, mapping of the arterial infarct territory in cases of known occlusions of specific arteries may provide a more realistic understanding of the region at risk of infarction than do cadaver-based maps. The correspondence between these mapping modalities is unknown for the PCA but may not be close, based on our observations of MCA infarction. Previously published maps have a limited number of slices and may be difficult to use in clinical practice when the site of infarction is not at the same level as the one depicted.

We have created a digital probabilistic atlas of PCA infarcts (DA-PCA) from T2-weighted MR imaging scans in patients with occlusion of the PCA trunk and/or P1 or P2 segments on MR angiograms. This map was then compared with existing maps of the PCA territory.

Subjects and Methods

Patient Selection

MR scans performed in our institution between January 1999 and June 2006 with reported PCA trunk or branch occlusion on MR angiography (MRA) were collected. The MR images were inspected and clinical notes were reviewed. Patients were included in the study if the images demonstrated “occlusion” of the PCA trunk (P1) and/or P2 or P3 branches (nomenclature of Zel and Rhoton[16]) on the initial MRA, as defined by the loss of signal on the maximum-intensity projection and the corresponding source images of the MRA. T2-weighted MR scans were performed >24 hours after stroke onset.

Patients who had new or concurrent infarcts in the MCA and anterior cerebral artery (ACA) territory were excluded. The mechanism of infarction was determined according to the Trial of Org 10172 in Acute Stroke Treatment criteria as large-artery atherosclerotic disease.12–14 Atherosclerotic disease in stroke patients may also lead to the development of leptomeningeal collaterals, which may influence the region at risk of infarction. Hence, mapping of the arterial infarct territory in cases of known occlusions of specific arteries may provide a more realistic understanding of the region at risk of infarction than do cadaver-based maps. The correspondence between these mapping modalities is unknown for the PCA but may not be close, based on our observations of MCA infarction. Previously published maps have a limited number of slices and may be difficult to use in clinical practice when the site of infarction is not at the same level as the one depicted.

We have created a digital probabilistic atlas of PCA infarcts (DA-PCA) from T2-weighted MR imaging scans in patients with occlusion of the PCA trunk and/or P1 or P2 segments on MR angiograms. This map was then compared with existing maps of the PCA territory.

Imaging Techniques

MR imaging scans were performed on a 1.5-T superconducting imaging system (General Electric Medical Systems, Milwaukee, Wis, and Siemens Medical Solutions, Malvern, Pa) with echoplanar imaging capabilities. T1 images were acquired with a thickness of 6 mm/1.7 mm; matrix size of 256×256; and a repetition time/echo time of 2000 ms/102 ms. The 3-dimensional time-of-flight MRA was performed with a repetition time/echo time of 38/6.9 ms; 25° flip angle; thickness of 1.4 mm; slab thickness of 60 mm; matrix size of 256×224; and field of view of 180 mm.

Registration and Segmentation

Alignment of corresponding anatomical structures in images (before segmentation) from different subjects was achieved by registration to a standard brain template (MNI template available at http://www. bic.mni.mcgill.ca/software/). Manual registration was performed by identifying homologous internal and external anatomic landmarks on the standard brain template and patient image. Landmarks were individualized for each patient and were chosen with use of an interactive display package that allowed the user to ensure that landmark selection progressively improved image alignment. Manual registration was used rather than image intensity–based automated methods because of potential inaccuracies in the latter introduced by the presence of a lesion. These steps led to the creation of a 9-parameter linear transformation matrix that allowed for rotation, translation, and scaling of the patient image along each of the 3 principal axes. In a previous study that included the same methodology, we had shown that the registration error was small. The registration step also had the effect of correcting for overall differences in brain size. Segmentation was performed on the MR scans performed >24 hours after ischemic stroke onset. Cerebral infarcts were manually segmented on T1-weighted images with the use of interactive mouse-driven software and standardized intensity windows.

Creation of the DA-PCA

To create a composite map, the images from subjects with infarcts in the right hemisphere were also “flipped” along the y axis so that all infarcts lay on the left side of the image, according to radiologic convention. Registered binary images of the infarcts were averaged to create the DA-PCA. Anatomic interpretation was facilitated by the use of an existing database that related Talairach coordinates to anatomic structures (Talairach Daemon; available at http://ric.uthscsa.edu/projects/talairachdaemon.html).

Summary statistics were calculated for volumes obtained in individual subjects. From the probability maps, the volume of tissue at a number of probability thresholds was calculated. The maximum infarct volume is defined by the voxels with probabilities exceeding the minimum probability. The mean infarct volume is defined by the voxels with probabilities exceeding the mean probability. The standard error of the mean of these measurements of PCA territory infarct volume was calculated by a “leave one out” or jack knife analysis. Volumes for patients with P1 (trunk) occlusion were compared with those for P1–P3 (trunk-branch) occlusion.

Comparison With Existing Maps

Comparisons with existing vascular territory maps were performed both qualitatively and quantitatively. Maps chosen for comparison were those widely cited in the literature. Maps by van der Zwan et al, Beevor, and Tatoo et al were based on arterial injection studies in cadavers unaffected by stroke. The map by Damasio is a composite map derived from textbooks on the cerebral circulation. The map by Tatoo et al provides a stylized depiction of the variability of the arterial territory, taking into account the description of van der Zwan and Beevor. The map of van Laar et al is based on MR perfusion (arterial spin labeling) of the basilar arteries and PCAs.

Results

Clinical Characteristics

There were 30 patients (24 men) with a median age of 61 years (range, 22 to 86 years). All patients had MR imaging performed as part of the routine stroke work-up. As a result of this protocol, the T2-weighted MR imaging scan that was used for segmentation was performed at a median interval of 5 days (range, 1 to 224 days) after stroke onset. The stroke mechanisms and sites of arterial occlusion are displayed in Table 1. The mean (±SD) infarct volume in individual patients was 26.2±22.8 cm³, and the maximum volume was 92.3 cm³. According to infarct probabilities, the mean and maximum infarct volumes obtained from the 20 patients with P1 occlusion and the 30 patients with P1–P3 occlusion are shown in Table 2.

The regions with the highest probability of PCA infarcts were the parahippocampal and lingual gyrus (0.60 to 0.70; Figure 1). The probability of infarction in the posterolateral thalamus was 0.33. The probability of in-
fraction in the occipital pole varied from 0.10 inferiorly (fusiform gyrus) to 0.43 at the level of the lingual gyrus and 0.10 superiorly (cuneus). The regions with a low probability of PCA infarction were the splenium (0.10), posterior limb of the internal capsule and posterior part of the lentiform nucleus (0.04), and tail of the caudate nucleus (0.04). The mean and median probabilities of infarction were 0.16 and 0.10, respectively.


<table>
<thead>
<tr>
<th>Age, y</th>
<th>Sex</th>
<th>Time of MR Scans, d</th>
<th>NIHSS Score on Admission</th>
<th>Infarct Volume, cm³</th>
<th>Side of Infarction</th>
<th>TOAST Classification</th>
<th>Ipsilateral Fetal PCA</th>
<th>Vertebal Artery Occlusion</th>
<th>PCA Trunk Occlusion</th>
<th>P2 Occlusion</th>
<th>P3 Occlusion</th>
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<td>53.1</td>
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</tr>
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<td>2</td>
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<td>–</td>
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</tr>
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<td>34</td>
<td>M</td>
<td>2</td>
<td>4</td>
<td>69.3</td>
<td>Right</td>
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<td>+</td>
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<td>+</td>
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<td>7</td>
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<td>+</td>
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<td>+</td>
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<td>–</td>
<td>–</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>51</td>
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<td>4</td>
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<td>+</td>
</tr>
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<td>7</td>
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<td>–</td>
<td>+</td>
<td>+</td>
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</tr>
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<td>M</td>
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<td>2</td>
<td>18.3</td>
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<td>–</td>
<td>–</td>
<td>+</td>
<td>+</td>
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</tr>
</tbody>
</table>

**NIHSS** indicates National Institute of Health Stroke Scale; **TOAST**, Trial of Org 10172 in Acute Stroke Treatment.

### TABLE 2. Comparison of PCA Territory Infarct Volumes Obtained by Using Predetermined Probability Thresholds for the DA-PCA and for the Map by van der Zwan et al²²

<table>
<thead>
<tr>
<th>PCA Territory Volume</th>
<th>DA-PCA, P1 occlusion (n=20)</th>
<th>DA-PCA, P1–P3 Occlusion (n=30)</th>
<th>Map of van der Zwan et al²² (n=23)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Method</td>
<td>MR imaging and MRA based</td>
<td>MR imaging and MRA based</td>
<td>Cadaver</td>
</tr>
<tr>
<td>Maximal extent: voxel probability &gt;0 (mean volume±SD)</td>
<td>128.9±14.3 cm³</td>
<td>152.6±43.3 cm³</td>
<td>228.6 cm³</td>
</tr>
<tr>
<td>Maximum volume in individual subjects</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Voxel probability&gt;mean probability (mean volume±SD)</td>
<td>55.1±8.7 cm³</td>
<td>56.6±43.3 cm³</td>
<td></td>
</tr>
<tr>
<td>Mean volume in individual subjects (±SD)</td>
<td></td>
<td></td>
<td>118.7±34.7 cm³</td>
</tr>
<tr>
<td>Minimum volume</td>
<td></td>
<td></td>
<td>69.1 cm³</td>
</tr>
</tbody>
</table>
Comparison With Existing Maps
The maximum infarct volume of the DA-PCA was much lower than that of van der Zwan et al22 (Table 2). The anatomic location of the boundaries of the PCA infarct territory in each map is provided in Table 3 and shown in Figure 2. The maximal extent of the DA-PCA map resembles that of Damasio15 and the minimum arterial map of Beevor10 and van der Zwan et al6 but not the maximum arterial map of Beevor,10 van der Zwan et al9 and Tatu et al11 because of the lack of lateral extension in the superior and inferior aspects of the probabilistic map. The superior extent of the map produced by van Laar et al12 is more congruent with the maximal map by Beevor,10 van der Zwan et al,9 and Tatu et al11 than our DA-PCA in terms of the lateral extent of the arterial territory.

Discussion
This study provides information on the regions at risk of infarction in the PCA territory. More accurate determination of infarct territory should aid topographic diagnosis and assist in formulating infarct mechanisms. As in our approach to creating the digital atlas of MCA territory infarcts,5 we based the identification of PCA territory infarcts on MR images taken from patients with occlusion of this artery. One advantage of this approach is the absence of a priori constraints on infarct size, shape, and location. The probabilistic approach used here realistically captures the variability in infarct location compared with earlier maps by showing the probability of infarction after PCA occlusion.

Methodologic Issues
Differences between previous maps of the PCA territory8 presumably influence the variety of injection techniques used, overflow of injection material through anastomosing arteries, variability in the physical properties of the injectate, and the effect of different injection pressures on the flow of contrast material in the case of MR perfusion maps. The MR perfusion map by van Laar et al12 depicts perfusion of the entire posterior circulation (basilar artery and both PCAs) rather than individual PCA territory. The resultant map may also be affected by overflow of endogenous contrast material through anastomosing arteries, leading to overestimation of the arterial territory. Additionally, MR images were obtained only from the level of the upper lateral ventricle, omitting the lower part of the PCA territory.12 In this study, PCA occlusion was defined by MRA. Although it is possible that in some cases PCA occlusion was incomplete and there was trickle flow, this is not likely to have significantly affected the results of the study, given that it is still indicative of severe hypoperfusion. A potential concern of this study is the timing
of the MR scans and the effect of infarct shrinkage on the size of the infarct territory. Although the majority of patients had their scans performed within 5 days of stroke onset, there were 3 patients whose scans were performed at 3 months and 1 patient whose scans were performed at 224 days. This patient had a small stroke (7.6 mL). Even allowing for shrinkage in infarct volume, it would not have affected the result of this study.

**Vascular Anatomy of the PCA**

We briefly summarize the origin and branches of the PCA according to the nomenclature of Zeal and Rhoton.\(^{16}\) The PCA originates from the basilar artery. Its proximal (P1) and distal (P2) segments are defined by the posterior communicating artery.\(^{16}\) The proximal P1 segment is hypoplastic in 17%, in which case the P2 and its branches arise from the anterior circulation via a large posterior communicating artery.\(^{16}\) The proximal P1 segment is hypoplastic in 17%, in which case the P2 and its branches arise from the anterior circulation via a large posterior communicating artery.\(^{16}\)

**TABLE 3. Differences in PCA Territory Boundaries Between the DA-PCA and Other Published Maps**

<table>
<thead>
<tr>
<th></th>
<th>DA-PCA</th>
<th>van der Zwan et al(^9)</th>
<th>Tatu et al(^{11})</th>
<th>Beevor(^{10})</th>
<th>Damasio(^{15})</th>
<th>van Laar et al(^{12})</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>N=30</td>
<td>N=23</td>
<td>NA</td>
<td>N=87</td>
<td>NA</td>
<td>N=115</td>
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<tr>
<td>Age, mean (range), y</td>
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<td>48 (15–100)</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Method</td>
<td>MR imaging and MRA based</td>
<td>Cadaver injection</td>
<td>Cadaver injection</td>
<td>Cadaver injection</td>
<td>Combined maps from in vivo cerebral angiographic studies</td>
<td>MR perfusion by arterial spin labeling</td>
</tr>
<tr>
<td>Superior and lateral</td>
<td>Extends to superior occipital sulcus but spares superior parietal lobule</td>
<td>Superior parietal lobule</td>
<td>Superior parietal lobule</td>
<td>Superior parietal lobule</td>
<td>Superior parietal lobule</td>
<td>Superior parietal lobule</td>
</tr>
<tr>
<td>Lateral and inferior</td>
<td>Does not extend to superior temporal sulcus</td>
<td>Extends to superior temporal sulcus</td>
<td>Extends to superior temporal sulcus</td>
<td>Extends to superior temporal sulcus</td>
<td>Extends to superior temporal sulcus</td>
<td>NA</td>
</tr>
</tbody>
</table>

*NA indicates that the axial slice at this level is unavailable for comparison. Data on the inferior extent of the PCA territory is unavailable in the map by van Laar et al.\(^{12}\)*
artery (fetal PCA variant).\textsuperscript{16} In the majority of cases, the P1 segment divides into the thalamoperforating arteries (to the thalamus), ventricular and choroid plexus branches (posterior choroidal arteries), and brainstem branches (short and long circumflex arteries). The P2 segment divides into the posterior or inferior temporal (branches include the anterior, middle, posterior, common temporal, and hippocampal arteries) artery and internal occipital arteries (calcarine and parieto-occipital branches). The P2 segment also branches to the thalamus, such as the thalamogeniculate arteries. The P3 segment begins at the posterior aspect of the midbrain, and the P4 segment, at the anterior part of the calcarine fissure.\textsuperscript{16}

The most common discrepancy between our map and those of previous studies was the superolateral and inferolateral extent of the PCA infarcts compared with those in cadaver maps.\textsuperscript{11,15,22} This is in accord with the smaller territorial infarct volume in this study compared with published maps in cadaver studies. This discrepancy may be related to the pattern of collateral blood supply. Brozici et al\textsuperscript{16} and Liebeskind\textsuperscript{23} have highlighted the possible relation between the leptomeningeal collateral blood supply and variability in the extent of stroke. These regions of low probability of infarction correspond to regions of leptomeningeal collateral blood supply depicted in perfusion maps.

The DA-PCA map extends as far superomedially as the cuneus and precuneus (probability = 0.10). The map by Tatu et al\textsuperscript{11} depicts the leptomeningeal blood supply by the PCA and ACA to this region. Despite the fact that in a cadaver study\textsuperscript{7} this region was found to have received the majority of its blood supply from the parieto-occipital artery, the relatively low probability of infarction involving this region is possibly due to anastomoses with the posterior pericallosal artery (branch of the ACA via the pericallosal artery).\textsuperscript{14,24,25} Unlike the cadaver\textsuperscript{9,10} and MR perfusion\textsuperscript{12} maps, the PCA infarct territory did not extend as far laterally as the superior parietal lobule in any patient. The map by Tatu et al\textsuperscript{11} depicts a variable leptomeningeal blood supply by the PCA and MCA to this region. Anastomoses are likely to form between the parieto-occipital artery (branches of the PCA) and the angular artery or anterior and posterior parietal arteries (branches of the MCA).\textsuperscript{14,24,25} Inferiorly, the lack of lateral extension to the middle temporal and lateral occipital gyri in the digital map compared with published maps is likely to be due to anastomoses between the anterior and/or middle temporal arteries (branch of the PCA) and the middle and/or posterior arteries (branches of the MCA).\textsuperscript{24} The presence of this leptomeningeal collateral network among the branches of the PCA\textsuperscript{7} and, to a lesser extent, from the MCA depicted by Tatu et al\textsuperscript{11} and van der Zwan et al\textsuperscript{9} may also explain the lower frequency of infarction in the occipital pole after PCA occlusion (probability = 0.12 to 0.41). This area receives its blood supply from several branches of the PCA, such as the calcarine artery, the parieto-occipital artery, and/or the posterior temporal artery\textsuperscript{7,26} and, to a lesser extent, from the MCA via the temporoparietal artery.\textsuperscript{26}

At the level of the corpus callosum, the infratentorial involvement of the splenium (probability = 0.10) is possibly due to the splenial artery (branch of the parieto-occipital, calcarine, or medial posterior choroidal artery) forming interarterial anastomoses with branches of the pericallosal artery (branch of the ACA).\textsuperscript{24} Similarly, the PCA (likely via the posterior choroidal arteries) has been described to perfuse the posterior limb of the internal capsule and the posterior part of the lentiform nucleus.\textsuperscript{9,11} However, the low risk of infarction in this region due to PCA occlusion suggests that this risk is modified by branches of the lenticulostriate arteries, which dominate the vascular supply of this region.\textsuperscript{27}

**Collateral-Poor Zone**

In the DA-PCA, the highest probability of involvement was in the parahippocampus and medial occipital lobe (probability = 0.60 to 0.70). This area is supplied by the hippocampal artery, the posterior temporal artery, or the common temporal artery (branches of the PCA).\textsuperscript{7} Branches from the MCA or ACA to this region have not been described. PCA infarction commonly involves the posteroslateral thalamus (probability = 0.33), presumably from a lack of perfusion of the thalamogeniculate branches.

In conclusion, we have created a DA of PCA territory infarction with the use of MR imaging techniques. The DA-PCA can be used to objectively define the regional risk of infarction and has been compared with existing maps of the PCA territory, as well as current knowledge of collateral supply. This approach may be useful to establish the distribution of the PCA and other arterial territories and the border zones between them.

**Disclosures**

None.

**References**

Digital Map of Posterior Cerebral Artery Infarcts Associated With Posterior Cerebral Artery Trunk and Branch Occlusion

Thanh G. Phan, MBBS, FRACP, Ashley C. Fong, Geoffrey Donnan and David C. Reutens

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