Collateral Configuration of the Circle of Willis
Transcranial Color-Coded Duplex Ultrasonography and Comparison
With Postmortem Anatomy

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Background and Purpose—The anterior communicating artery (AcoA) and posterior communicating arteries (PcoA) of the circle of Willis provide the main route for collateral blood flow in cases of carotid artery obstruction. Transcranial color-coded duplex ultrasonography (TCCD) allows real-time measurement of the collateral function of the AcoA and PcoA. The primary objective of this study was to determine the collateral artery threshold diameters for supplying collateral flow.

Methods—In 12 acute stroke patients with a median age of 75 years (51 to 91 years), the collateral integrity of the circle of Willis as assessed by TCCD and carotid compression tests was compared with their postmortem anatomy. The lengths and diameters of the collateral arteries were measured.

Results—TCCD demonstrated absent anterior collateral flow in 3 patients. In 1 of these patients, absence of anterior cross-flow was due to an occluded anterior cerebral artery, which was revealed at autopsy. Absent posterior collateral flow was found in 14 hemispheres. In 2 of these hemispheres, autopsy revealed a fetal configuration of the posterior cerebral artery hampering posterior collateral flow. The median (range) diameters as found at autopsy of the functional (n=19) and nonfunctional (n=16) collateral arteries of the circle of Willis were 1.1 (0.4 to 2.0) and 0.5 (0.3 to 0.7) mm, respectively (P=0.003). PcoA diameters were found to correlate negatively (ρ=−0.50, P=0.01) to the diameters of their accessory P1 segments.

Conclusions—The threshold diameter allowing for cross-flow through the primary collateral arteries of the circle of Willis is between 0.4 and 0.6 mm. (Stroke. 2000;31:1346-1351.)

Key Words: cerebral arteries ■ collateral circulation ■ hemodynamics ■ ultrasonography ■ Doppler ■ duplex ■ autopsy

The anterior communicating artery (AcoA) and posterior communicating arteries (PcoA) of the circle of Willis provide the main route for collateral blood flow in cases of severe internal carotid artery (ICA) stenosis or occlusion.1 Absence of collateral function due to hypoplasia (Figure 1) or atherostenosis of collateral arteries may lead to a higher risk of stroke in patients with severe ICA occlusive disease.2-4 For this reason, insight into the collateral function of the circle of Willis is of clinical importance. Transcranial color-coded duplex ultrasonography (TCCD) combined with common carotid artery (CCA) compression tests allows real-time evaluation of the collateral ability of the circle of Willis.5,6 Comparative studies between transcranial ultrasound techniques and angiography have demonstrated that transcranial ultrasound has a high level of sensitivity and specificity in the assessment of the patency of the AcoA and PcoA.5,7,8 This is the first study comparing hemodynamic data on the collateral function of the circle of Willis with anatomic findings obtained at autopsy. One goal was to investigate whether the commonly used ultrasound criteria for the definition of AcoA and PcoA collateral patency6,7,9,10 reflect true differences in arterial size, but our main goal was to determine the collateral artery threshold diameters for supplying collateral flow through the circle of Willis.

Subjects and Methods

Patients
The study was performed at the Stroke Unit of the Department of Neurology and at the Department of Pathology of the Debrecen University Medical School in Hungary after being approved by the local ethics committee. This study was part of a larger study performed to assess the collateral function of the circle of Willis in acute stroke patients by means of TCCD and CCA compression tests. Because of the severity of stroke, a number of patients included in this larger study died. The circle of Willis of these patients was removed at autopsy, and the size of the collateral arteries was measured.

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Carotid Occlusion

In patients with unilateral ICA occlusion, AcoA function was proven if the A1 ipsilateral to the occluded ICA demonstrated spontaneous reversed flow. PcoA function on the side of the ICA occlusion was demonstrated if the mean blood flow velocity in the ipsilateral P1 was more than the mean blood flow velocity + 2 SD from an age- and sex-matched group of atherosclerotic patients with no ICA occlusive disease or cerebral symptoms. PcoA function contralateral to the occluded ICA was tested by compression of the nonocluded CCA. The presence of leptomeningeal collaterals could not be assessed by TCCD.

Compression Tests

To avoid a systemic cardiovascular reaction, compressions of the CCA were applied low in the neck just proximal to the sternal head of the clavicle for a maximum of 4 cardiac cycles. To minimize the risk of embolus, compressions were performed only in those patients with no atherosclerotic plaques in the proximal CCA as judged by the B-mode image of the duplex scan. To ensure the efficacy of the compression, a pulse oximeter (Eagle 3000, Marquette), which generated pulse tracings on a separate monitor, was attached to the earlobe on the same side as the compressed artery. Flattening of this pulse wave indicated cessation of blood flow through the CCA and thus an adequate compression.

Autopsy

The circle of Willis was dissected out after removal of the brain from the cranial cavity. All minute branches arising from the main vessels were carefully cut away. Pathology of the cerebral vessels was judged by a neuropathologist familiar with the medical history of the patient. A rough drawing of the anatomy was made to prevent mixing up the right and left sides of the circle of Willis during further procedures. Blood was carefully washed out from the circle of Willis with isotonic saline. Photographs were taken before and after dissection from the brain and after the blood was washed out.

Measurements

To measure the lengths and diameters of the arteries, the circle of Willis was put on a glass plate. After the arteries had been straightened out, the lengths of the PcoAs and A1 segments were measured with a ruler with a millimeter scale. These measurements were rounded off to whole millimeters. For the measurement of the length of the AcoA and P1 segments, a transparent 10 × 10-cm sheet with a millimeter scale was placed on top of the circle. Under a microscope with a ×10 magnification, AcoA and P1 lengths were measured with an approximation of 0.1 mm. For the measurement of the arterial diameters, a second glass plate was clamped on top of the first, with the circle of Willis and the transparent sheet in between. Sufficient force was applied to induce complete obliteration of the vessel lumen but not to flatten the arterial walls. If atherostenosis was found to be present in the middle cerebral artery or the basilar artery, this was cut away first. This was done to allow application of equal pressure on all 4 corners of the glass plates for obliteration of the vessel lumen. The set of glass plates was put under the microscope again, and in this way, readings representing half of the arterial circumference were obtained. Measurements were taken at proximal, middle, and distal sites of the AcoA, A1, PcoA, and P1 and approximated to the nearest 0.1 mm. The narrowest part of each artery was used for further analysis, because we considered that the width at this part determined the collateral ability. Assuming the arteries to be circular, their external diameter could be calculated by the formula diameter = circumference/π. All data in this study are reported as medians with ranges. Nonparametric tests were used to analyze the data. Significance was assumed at the 5% level.

Results

The study comprises TCCD and autopsy data from 12 patients, 8 men and 4 women, with a median age of 75 years (51 to 91 years). The median time between TCCD and death was more than the mean blood flow velocity ± 2 SD from an age- and sex-matched group of atherosclerotic patients with no ICA occlusive disease or cerebral symptoms. PcoA function contralateral to the occluded ICA was tested by compression of the nonocluded CCA. The presence of leptomeningeal collaterals could not be assessed by TCCD.
was 53 hours (4 hours to 36 days), and the median time between death and autopsy was 22 hours (5 hours to 3.5 days). Six patients had a unilateral ICA occlusion without significant contralateral ICA stenosis (Table 1). In Table 2, the TCCD results are listed together with the arterial sizes obtained at autopsy. With the exception of patient 3 (thrombus in the left anterior cerebral artery), no patients were found to have severely stenosed or occluded collateral arteries. Patient 10 showed a large thrombus occluding the left middle cerebral artery.

### Anterior Part of Circle of Willis

In 2 patients (patients 2 and 3) with ICA occlusion, spontaneous collateral flow through the anterior part of the circle of Willis toward the M1 was absent. In patient 3, the ipsilateral A1 could not be visualized by TCCD, and the velocity in the contralateral A1 was not enhanced. In patient 12, who had no significant ICA stenosis, cross-flow through the AcoA could not be provoked by CCA compressions. At autopsy, a single AcoA was found in all cases; no duplications or triplications were found. Three AcoAs had a typical hourglass shape.

### Table 1. Patient Characteristics

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Extracranial ICA</th>
<th>Stroke Type</th>
<th>Cause of Death</th>
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<tbody>
<tr>
<td>1</td>
<td>69</td>
<td>M</td>
<td>Both normal</td>
<td>...</td>
<td>Pulmonary embolism</td>
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<tr>
<td>2</td>
<td>88</td>
<td>M</td>
<td>L occlusion, R normal</td>
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<td>Myocardial infarction</td>
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<tr>
<td>3</td>
<td>72</td>
<td>M</td>
<td>L occlusion, R normal</td>
<td>L ACA/MCA territorial</td>
<td>Brain herniation</td>
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<tr>
<td>4</td>
<td>80</td>
<td>F</td>
<td>R occlusion, L normal</td>
<td>R MCA territorial</td>
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</tr>
<tr>
<td>6</td>
<td>66</td>
<td>F</td>
<td>R &gt;-50% st, L normal</td>
<td>L hemorrhagic</td>
<td>Brain herniation</td>
</tr>
<tr>
<td>7</td>
<td>73</td>
<td>M</td>
<td>Both normal</td>
<td>L MCA territorial</td>
<td>Pulmonary embolism</td>
</tr>
<tr>
<td>8</td>
<td>91</td>
<td>M</td>
<td>Both normal</td>
<td>R MCA territorial</td>
<td>Heart failure</td>
</tr>
<tr>
<td>9</td>
<td>76</td>
<td>M</td>
<td>Both normal</td>
<td>R MCA territorial</td>
<td>Heart failure</td>
</tr>
<tr>
<td>10</td>
<td>51</td>
<td>M</td>
<td>L occlusion, R normal</td>
<td>L MCA territorial</td>
<td>Brain herniation</td>
</tr>
<tr>
<td>11</td>
<td>77</td>
<td>M</td>
<td>R occlusion, L normal</td>
<td>L MCA territorial</td>
<td>Pulmonary embolism</td>
</tr>
<tr>
<td>12</td>
<td>76</td>
<td>F</td>
<td>Both normal</td>
<td>R hemorrhagic</td>
<td>Myocardial infarction</td>
</tr>
</tbody>
</table>

R indicates right; L, left; normal, no significant stenosis; st, stenosis; MCA, middle cerebral artery; and ACA, anterior cerebral artery.

### Table 2. Functional and Metric Values of Circle of Willis Collaterals for Individual Patients and for Groups Based on Functional and Nonfunctional AcoA and PcoA

<table>
<thead>
<tr>
<th>Patient</th>
<th>Autopsy</th>
<th>AcoA</th>
<th>TCCD Functional</th>
<th>A1</th>
<th>PcoA</th>
<th>P1</th>
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<td>Length</td>
<td>Diameter</td>
<td>Length</td>
<td>Diameter</td>
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<td>2.0</td>
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<td>1.6</td>
<td>1.5</td>
<td>1.4</td>
<td>1.5</td>
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<tr>
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<td>No</td>
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<td>1.3</td>
<td>1.4</td>
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<td>1.3</td>
</tr>
<tr>
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<td>1.8</td>
<td>2.2</td>
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<td>2.4</td>
<td>3.0</td>
<td>1.2</td>
<td>3.0</td>
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<tr>
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<td>1.8</td>
<td>1.3</td>
<td>1.8</td>
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<tr>
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<td>Yes</td>
<td>1.0</td>
<td>1.8</td>
<td>1.7</td>
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<td>1.7</td>
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<tr>
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<td>Yes</td>
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<td>2.2</td>
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<tr>
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<td>Yes</td>
<td>2.0</td>
<td>1.6</td>
<td>1.6</td>
<td>1.5</td>
<td>1.6</td>
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<tr>
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<td>Yes</td>
<td>1.8</td>
<td>2.1</td>
<td>1.0</td>
<td>1.4</td>
<td>1.0</td>
</tr>
<tr>
<td>11</td>
<td>Yes</td>
<td>1.5</td>
<td>1.3</td>
<td>1.9</td>
<td>1.2</td>
<td>1.5</td>
</tr>
<tr>
<td>12</td>
<td>No</td>
<td>5.5</td>
<td>1.5</td>
<td>1.9</td>
<td>1.4</td>
<td>1.5</td>
</tr>
</tbody>
</table>

R indicates right; L, left. Diameter and length are in mm.
form, with a smaller diameter in the midsection of the artery than at the site of junction with the A1 segments. In 1 case, a third anterior cerebral artery originated from the AcoA. All A1 segments had a diameter of $\approx 1.0$ mm (Table 2). Patients 2 and 12 showed very small AcoA diameters, 0.3 and 0.5 mm, respectively, which had impeded interhemispheric cross-flow. In patient 3, a large thrombus in the left anterior cerebral artery was found that blocked outflow from the AcoA, explaining the absence of anterior cross-flow on TCCD. The median AcoA diameter in patients with a nonfunctional anterior collateral pathway ($n = 2$, patient 3 excluded) was 0.4 mm, and the median AcoA diameter of patients with a functional anterior collateral pathway ($n = 9$) was 1.1 mm (Table 2).

**Posterior Part of Circle of Willis**

In 14 hemispheres, spontaneous collateral flow through the posterior part of the circle of Willis either was absent or could not be provoked by CCA compressions. In 2 hemispheres (right hemispheres of patients 6 and 8), we were able to measure the blood flow velocity in the PcoA directly. Instead of reversal of flow, ipsilateral CCA compression caused a PSV decrease of 89% and 61%, respectively. Autopsy revealed a fetal configuration of the posterior cerebral artery (Figure 2), consisting of a wide PcoA (diameter of 2.1 mm in both) combined with a narrow ipsilateral P1 (0.5 and 0.6 mm, respectively; Table 2). PcoAs classified as functional by TCCD had significantly larger diameters than nonfunctional PcoAs, 0.9 versus 0.6 mm ($P = 0.008$). PcoA diameters were found to correlate negatively ($\rho = -0.50$, $P = 0.01$, Spearman’s rank-order correlation) to the diameters of their ipsilateral P1 segments (Figure 3).

The diameters of all functional collateral arteries of the circle of Willis were significantly larger than the diameters of all nonfunctional collateral arteries (including the diameters of the P1 segments of the 2 fetal posterior cerebral arteries), 1.1 versus 0.5 mm ($P = 0.003$, Figure 4).

**Discussion**

Despite the limited number of patients and the relatively simple method of measuring the external arterial diameters, we believe our results to be unique, because this is the first time this type of information has been obtained from a study population as opposed to a model. The TCCD criteria for determining the collateral function of the circle of Willis used in the literature$^{6,7,9,10}$ truly correspond with significant differences in arterial diameter, despite the slight overlap in diameters of functional and nonfunctional collateral arteries (Figure 4). Overall, the arterial diameters found in this study are very similar to the diameters reported by Baptista$^{14}$ and Murray$^{16}$ but slightly smaller than the diameters reported by other authors.$^{15,17–19}$ Studies should be compared only with caution because of the difference in study populations and methods of measurement. How far postmortem arterial diameters are equal to in vivo diameters is unknown. The absence
of perfusion pressure and possible shrinking of the arterial wall due to muscle cell decay might result in the measurement of smaller diameters. Whether this process had a significant influence on our results remains speculative. Because of the nature of our study population, we assume that we found smaller diameters than some other authors. It has been shown that stroke patients have less well-developed circles of Willis because of a higher prevalence of thread-like vessels.15,23 Another reason is that we used the narrowest parts of the arteries in the analysis, because these parts probably determine collateral ability. The AcoA in particular can have a widely differing diameter along its course because of an hourglass (3 cases in this study) or triangular shape.15,23

The reported negative correlation between PcoA and P1 diameters (Figure 3) refutes the findings of Puchades-Orts et al and Orlandini et al; however, Kamath found the same negative correlation, and in the detailed analysis by Hillen, comparable correlation coefficients were found.

An important finding in this study is that AcoAs and PcoAs with a diameter considerably less than 1 mm can still supply collateral flow, which can be detected by TCCD. Our results indicate that the threshold diameter for collateral function lies between 0.4 and 0.6 mm (Table 2, Figure 4). Because of its greater length, it seems valid that the PcoA threshold diameter for collateral function is slightly higher than the AcoA threshold diameter, because the resistance to blood flow is higher in longer vessels as a result of the larger area of endoluminal vessel wall. Our data (Table 2) endorse this hypothesis, but numbers are too small to draw a definite conclusion. A threshold diameter for supplying collateral flow of between 0.4 and 0.6 mm is probably also applicable to the P1. In the hemispheres with a fetal posterior cerebral artery, the ipsilateral P1 diameters were 0.5 and 0.6 mm, respectively. CCA compression could not provoke a flow reversal in their wide accessory PcoAs (diameter of 2.1 mm in both).

Up to now, a threshold of 1 mm to define hypoplasia or inadequacy of collateral vessels has been widely used in anatomic studies.17,19,22,26–30 In clinical studies, an increased risk of ischemic cerebral infarction after ICA occlusion and an increased risk of brain stem ischemia after basilar artery occlusion have been associated with PcoA diameters <1 mm. Only in a minority of studies was a threshold of 0.5 mm used to define hypoplasia of collateral arteries.18,20

The varying definitions of hypoplasia of circle of Willis collaterals and the different populations studied have resulted in a large variability of anomalous or “incomplete” circles of Willis throughout the literature.14,18,20,22,26–32 The prevalence of the “normal” textbook polygon ranges from 21% to 76%. However, the arbitrary diameters of 0.5 or 1 mm have never been discussed in terms of functional significance. Fluid-dynamic mathematical models have been developed to study the effect of collateral artery diameter on cerebral blood flow after ICA stenosis or occlusion. Cassot et al and Dickey et al independently showed that the smallest luminal diameter allowing for cross-flow through the AcoA was 0.4 mm. Moreover, Dickey et al found that in patients with ICA occlusion and a well-functioning AcoA, the collateral supply from the PcoA to the deprived hemisphere fell to zero when its diameter was set at levels <0.5 to 0.6 mm. The results of our study confirm those of Cassot et al and Dickey et al. Therefore, we suggest that the term hypoplasia be reserved for those vessels that cannot supply collateral flow. Our results indicate that in practice, communicating arteries with a diameter <0.5 mm should be labeled hypoplastic. This may result in greater uniformity in radiological studies on the collateral integrity of the circle of Willis. This is important, because there is increasing evidence that a well-functioning, complete circle of Willis plays a protective role against cerebral ischemia in patients with carotid artery occlusive disease. Furthermore, it is quite possible that in the future, particularly in patients with an asymptomatic severe carotid stenosis, the decision whether to operate or not will be influenced in part by the collateral ability of the circle of Willis. Therefore, uniformity in the definition of a complete circle of Willis is a first requirement.

Although TCCD can be used to assess the presence of cross-flow through the AcoA and PcoA, it should be kept in mind that volumetric blood flow cannot be measured by this technique. A TCCD diagnosis that collateral flow to a deprived hemisphere is present or can be provoked does not guarantee that this is sufficient to protect the hemisphere against ischemia. Moreover, the presence of leptomeningeal collaterals, which might be vital for hemispherical perfusion in some cases, cannot be assessed by TCCD. One method of assessing the amount of collateral flow via the AcoA and/or PcoA to a deprived hemisphere is measurement of the proportional velocity decrease in the middle cerebral artery after carotid compression or carotid cross-clamping during carotid endarterectomy. The degree of velocity decrease is correlated with the frequency of electroencephalographic changes and the development of cerebral ischemia during carotid clamping.

In summary, the ultrasound criteria used in the literature to discern functional from nonfunctional collateral arteries reflect significant differences in arterial size. The arterial threshold diameter allowing for collateral flow through the circle of Willis lies between 0.4 and 0.6 mm. This threshold diameter might be used in prospective studies evaluating the influence of the collateral ability of the circle of Willis on the development of ischemic strokes in patients with carotid artery occlusive disease.

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