Collateral Variations in Circle of Willis in Atherosclerotic Population Assessed by Means of Transcranial Color-Coded Duplex Ultrasonography

A.W.J. Hoksbergen, MD; D.A. Legemate, MD, PhD; D.T. Ubbink, MD, PhD; M.J.H.M. Jacobs, MD, PhD

Background and Purpose—Transcranial color-coded duplex ultrasonography combined with common carotid artery (CCA) compression can be used to assess the collateral function of the circle of Willis. The aim of this study was to assess the unknown fraction of hemodynamic functional anterior and posterior communicating arteries (AcoA and PcoA, respectively) in an atherosclerotic population with no cerebrovascular symptoms.

Methods—In 76 patients with a mean age of 61 (35 to 89) years, the blood flow velocity changes in the precommunicating parts (A1 and P1, respectively) of the anterior and posterior cerebral arteries were measured during CCA compression. The AcoA was defined as functional if blood flow was reversed in the ipsilateral A1 and enhanced in the contralateral A1 during CCA compression. The PcoA was defined as functional if the flow velocity in the P1 was enhanced >20% during ipsilateral CCA compression.

Results—It was possible to assess cross flow through the anterior part of the circle of Willis in 95% of the subjects. Failure of this collateral pathway was caused by a hypofunctional AcoA in 4% and a hypofunctional A1 in 1% of the subjects. Anomalies in the posterior part of the circle of Willis hampering collateral flow from the basilar to the internal carotid artery were found in 45% of the hemispheres. Thirty-eight percent of PcoAs were hypofunctional, and 7% of the posterior cerebral arteries had a persistent fetal anatomy.

Conclusions—We found that in subjects with no cerebrovascular symptoms, the anterior collateral pathway of the circle of Willis was nearly always functional. In contrast, the posterior collateral pathway was nonfunctional in almost half of the total number of hemispheres. Comparing these basic data with data from patients with cerebral ischemic disease might further help to elucidate the importance of the collateral capacity of the circle of Willis.

(Stroke. 2000;31:1656-1660.)

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

Many anatomic1–4 and radiological5–7 studies involving the configuration of the circle of Willis (Figure 1) in subjects with no cerebrovascular disease have been published, with special emphasis on the integrity of the collateral vessels. However, assessment of the true collateral potential of the circle of Willis requires the use of common carotid artery (CCA) compression tests to simulate carotid occlusion. With transcranial color-coded duplex ultrasonography (TCCD), the real-time reaction of the intracranial circulation to the CCA compression can be examined. Basic knowledge of the hemodynamic integrity of the circle of Willis is important because a correlation between anomalies of the circle of Willis (occurring in 50% to 80% of individuals)2,4,8 and stroke risk has been shown.4,9–14

Data on the hemodynamic potential of the circle of Willis in subjects without cerebrovascular symptoms are largely lacking. Therefore, the aim of the present study was to establish the range of collateral variations in the circle of Willis as determined by TCCD and CCA compression tests in atherosclerotic subjects without cerebrovascular symptoms.
systolic velocity [PSV] > 1.25 m/s) or occlusions of the internal carotid artery (ICA) or vertebral arteries were excluded to rule out any possible influence on the enlarging of collateral pathways.\textsuperscript{15} TCCD was performed by use of a low-frequency (2.0- to 2.5-MHz) transducer. Insonation of the main trunk of the middle cerebral artery and the precommunicating parts (A1 and P1, respectively) of the anterior and posterior cerebral arteries through the temporal window was performed in the standard manner, the details of which are reported elsewhere.\textsuperscript{16,17} In the case of unilateral window failure, investigation of the A1 and P1 through the opposite temporal window was attempted. A routine transcranial examination also included insonation of the vertebrobasilar arteries through the foramen magnum, but these data are not considered for further analysis here.

For reliable assessment of the functional patency of the anterior and posterior communicating artery (AcoA and PcoA, respectively), CCA compression tests are required.\textsuperscript{18,19} Collateral supply through the AcoA was demonstrated by reversal of blood flow in the A1 segment of the anterior cerebral artery ipsilateral to the compressed CCA, combined with an enhanced blood flow velocity in the contralateral A1 (Figure 2). Both A1 segments were routinely investigated by use of ipsilateral and contralateral CCA compression. Functional patency of the PcoA was defined by a PSV increase of > 20% in the P1 segment of the posterior cerebral artery during ipsilateral CCA compression (Figure 2), with this value being twice as much as expected from normal variation and measurement error.\textsuperscript{11,19} The PSV increase was always measured over the highest peaks on the Doppler spectrum. If the PSV increase in the P1 was < 20%, the PcoA was defined as hypofunctional. In the case of a fetal posterior cerebral artery, the main stem of the posterior cerebral artery arises from the ICA instead of from the basilar artery. In such cases, the PcoA, which is now the main stem of the posterior cerebral artery, is enlarged and is accompanied by a thin or hypoplastic P1. Such a large PcoA can be detected by TCCD, enabling direct velocity measurements. If ipsilateral CCA compression caused a velocity decrease in the PcoA instead of flow reversal, then the P1 was defined as hypofunctional. To avoid artifacts due to turbulence near the origins of the communicating arteries on provoking collateral flow, velocity measurements were taken proximally in the A1 and P1 with the sample volume set as narrowly as possible.

Compressions of the CCA were applied for 3 to 5 cardiac cycles, low in the neck just proximal to the sternal head of the clavicle, to avoid a systemic cardiovascular reaction. 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 photoplethysmograph that generated pulse tracings on a separate monitor was attached to the earlobe on the side of the compressed artery. Flattening of this pulse wave indicated cessation of blood flow through the CCA and, thus, an adequate compression. To assess the collateral function of the AcoA and PcoA, a minimum of 3 compressions of both CCAs was needed.

**Results**

**General**

From the initial 99 patients, we were finally able to determine the patency of the collateral vessels in 76. The reasons for exclusion are listed in Table 1. No ischemic complications of carotid compression occurred. Adverse reactions were local pain at the site of compression and coughing due to irritation of the trachea during compression. In patients with large necks and deeply located carotid arteries and in patients with very high systolic blood pressure, the CCA had to be firmly compressed to stop blood flow. One patient noted a shooting pain in the ipsilateral shoulder and arm during compression, which was probably due to stimulation of the brachiovascular plexus. In another patient, compression caused a short-lasting bradycardia, probably caused by compression too close to the carotid sinus. In 9 women and 2 men, we were unable to visualize the intracranial arteries because of unsuitable tem-

![Figure 1. Typical normal polygon configuration of the circle of Willis. M1 indicates main trunk of the middle cerebral artery; A2, postcommunicating part of the anterior cerebral artery; P2, postcommunicating part of the posterior cerebral artery; and Ant., anterior.](image1)

![Figure 2. Schematic drawing of blood flow reversal in the right A1 and blood flow velocity enhancement in the left A1 and right P1 during compression of the right carotid artery (expressed by the black square), indicating a functional AcoA and PcoA. MCA indicates middle cerebral artery; BA, basilar artery; and VA, vertebral artery.](image2)

**TABLE 1. Excluded Patients**

<table>
<thead>
<tr>
<th>Excluded Patients</th>
<th>No. of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initial patients, N</td>
<td>99</td>
</tr>
<tr>
<td>Excluded patients, n</td>
<td></td>
</tr>
<tr>
<td>ICA stenosis &gt; 50%</td>
<td>7</td>
</tr>
<tr>
<td>ICA occlusion</td>
<td>1</td>
</tr>
<tr>
<td>VA occlusion</td>
<td>2</td>
</tr>
<tr>
<td>CCA plaques</td>
<td>2</td>
</tr>
<tr>
<td>Unsuitable temporal windows</td>
<td>11</td>
</tr>
<tr>
<td>Remaining patients, n</td>
<td>76</td>
</tr>
</tbody>
</table>
poral bone windows, a well-known problem of transcranial ultrasound investigations, particularly in elderly women. 20 –22

Collateral Variations
In Figure 3, the collateral variations of the circle of Willis with frequency of occurrence are shown. In 22 (29%) of the patients, the AcoA and both PcoAs were functionally patent, resulting in a hemodynamically complete circle of Willis. In none of the patients could the AcoA be visualized in the physiological state, and visualization could not be determined during CCA compression. Unilateral hypofunctional PcoAs were found in 34 (22%) of the hemispheres, and bilateral hypofunctional PcoAs were found in 12 (16%) of the patients. In 10 (7%) of the hemispheres, persistence of the fetal origin of the posterior cerebral artery was found. In Table 2, the precompression and postcompression velocities in the ipsilateral and contralateral A1 and ipsilateral P1 in cases of functional AcoA and PcoAs are shown. The median PSV enhancement during CCA compression was significantly higher in the A1 segments than in the P1 segments ($P < 0.001$, Mann-Whitney $U$ test). The 90% central range of the postcompression velocities is very wide for both A1 and P1 segments, reflecting the large spread of the collateral capacity.

Discussion
Because of the obvious role of the AcoAs and PcoAs in preventing neurological damage from obstructive cerebrovascular disease,11 –14 methods of determining their collateral potential are currently much in discussion.5 –7, 23, 24 We found a functional AcoA in 95% of an atherosclerotic population with no cerebrovascular disease. Anomalies in the posterior part of the circle of Willis hampering collateral flow from the basilar artery to the ICA were found in 45% of the hemispheres. Because of the character of the present study, we could not confirm our findings by angiography. Comparative studies between transcranial ultrasound combined with CCA compression and angiography in patients with cerebrovascular disease showed that transcranial ultrasound has a high level of sensitivity and specificity in detecting AcoA and PcoA patency.23 –25 However, it should be noted that these studies were performed in patients with carotid artery obstructive disease and that patients with insufficient collateral capacity were probably underrepresented because the severity of their stroke precluded inclusion in the study. The AcoA is commonly recognized as the most important collateral pathway in the event of severe ICA stenosis or occlusion.13, 26 –29 The importance of a functional PcoA in ICA obstructive disease is not yet clear, although a hypoplastic PcoA might increase the risk of developing cerebral ischemia in patients with ICA occlusion.12

![Figure 3. Schematic drawings of the collateral variations found in the circle of Willis in the present study. Numbers and percentages of patients are shown in parentheses for the following conditions: A, complete circle; B, hypofunctional right PcoA; C, hypofunctional left PcoA; D, bilateral hypofunctional PcoAs; E, fetal right posterior cerebral artery; F, fetal left posterior cerebral artery; G, hypofunctional left PcoA and fetal right posterior cerebral artery; H, hypofunctional right PcoA and fetal left posterior cerebral artery; I, hypofunctional AcoA; J, hypofunctional AcoA and hypofunctional left PcoA; and K, hypoplasia right A1 and hypofunctional right PcoA. R indicates right; L, left.](http://stroke.ahajournals.org/)

**TABLE 2. PSV Changes in the Presence of a Functionally Patent AcoA or PcoA**

<table>
<thead>
<tr>
<th>Velocities Ipsilateral A1</th>
<th>Contralateral A1</th>
<th>Ipsilateral P1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Precompression, cm/s</td>
<td>80 (50–127)</td>
<td>84 (54–131)</td>
</tr>
<tr>
<td>Postcompression, cm/s</td>
<td>97 (27–200)*</td>
<td>166 (96–261)</td>
</tr>
<tr>
<td>Increase, cm/s</td>
<td>86% (38–237)†</td>
<td>47% (23–319)†</td>
</tr>
</tbody>
</table>

Velocities and velocity increases are given as medians with 5th and 95th percentiles.

*Reversed blood flow velocities.
†$P < 0.001$ by Wilcoxon signed-rank test.

therefore, we assumed that a hypofunctional AcoA was the reason for the absence of collateral flow.

We were able to visualize 13% of the functional PcoAs. In all of these PcoAs, an antegrade flow from the ICA to the posterior cerebral artery was detected, which reversed during ipsilateral CCA compression. Unilateral hypofunctional PcoAs were found in 34 (22%) of the hemispheres, and bilateral hypofunctional PcoAs were found in 12 (16%) of the patients. In 10 (7%) of the hemispheres, persistence of the fetal origin of the posterior cerebral artery was found. In Table 2, the precompression and postcompression velocities in the ipsilateral and contralateral A1 and ipsilateral P1 in cases of functional AcoA and PcoAs are shown. The median PSV enhancement during CCA compression was significantly higher in the A1 segments than in the P1 segments ($P < 0.001$, Mann-Whitney $U$ test). The 90% central range of the postcompression velocities is very wide for both A1 and P1 segments, reflecting the large spread of the collateral capacity.
TABLE 3. Intracranial Collateral Variations

<table>
<thead>
<tr>
<th>Reference</th>
<th>Brains, n</th>
<th>AcoA</th>
<th>A1</th>
<th>PcoA</th>
<th>P1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anatomic studies</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Krayenbühl and Yaşargil</td>
<td>400</td>
<td></td>
<td></td>
<td>38%</td>
<td>14%</td>
</tr>
<tr>
<td>Alpers et al</td>
<td>350</td>
<td></td>
<td>1%</td>
<td></td>
<td>16%</td>
</tr>
<tr>
<td>Yaşargil</td>
<td>200</td>
<td>3%</td>
<td>1%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Battacharji et al</td>
<td>88</td>
<td>1%</td>
<td>1%</td>
<td>30%</td>
<td>9%</td>
</tr>
<tr>
<td>MR angiographic studies</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Macchi et al</td>
<td>100</td>
<td>5%</td>
<td>3%</td>
<td>27%</td>
<td>12%</td>
</tr>
<tr>
<td>Stock et al</td>
<td>62</td>
<td>40%</td>
<td>4%</td>
<td>39%</td>
<td>13%</td>
</tr>
<tr>
<td>Krabbe-Hartkamp et al</td>
<td>100</td>
<td>22%</td>
<td>5%</td>
<td>30%</td>
<td>22%</td>
</tr>
<tr>
<td>Transcranial ultrasound studies</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>*Bass et al</td>
<td>10</td>
<td>0%</td>
<td></td>
<td>30%</td>
<td></td>
</tr>
<tr>
<td>*Chaudhuri et al</td>
<td>11</td>
<td>18%</td>
<td></td>
<td>18%</td>
<td></td>
</tr>
<tr>
<td>Present study</td>
<td>76</td>
<td>4%</td>
<td>1%</td>
<td>38%</td>
<td>7%</td>
</tr>
</tbody>
</table>

Percentages are calculated per artery and not per circle of Willis.

*Studies performed with conventional transcranial Doppler.

In Table 3, the state of the collateral vessels as found by anatomic, ultrasound, and magnetic resonance (MR) angiography studies is reported. Our findings resemble the results from anatomic studies in normal control subjects, particularly with regard to the patency of the anterior collateral pathway.1–4 With respect to the posterior collateral pathway, more variability between studies was found. This is most likely caused by the different criteria used in the anatomic studies for the definition of a hypoplastic PcoA. Unfortunately, the patient numbers of the transcranial Doppler studies are too small to make a reliable comparison with our data.19,30 Moreover, TCCD is considered a technique superior to conventional transcranial Doppler when exact measurements in small arterial segments are required.16,31,32

It is of interest to compare our findings with the results of MR angiography, another new noninvasive technique for establishing the collateral integrity of the circle of Willis.5–7 A striking difference appears in the detection and assessment of AcoA function with the studies of Stock et al and Krabbe-Hartkamp et al (Table 3). A much higher frequency of AcoA hypoplasia is found in these studies than in our own duplex study and reported anatomic studies. A possible explanation might be that in the physiological state, the pressure equilibrium in the anterior part of the circle of Willis results in a negligible cross flow through the AcoA, which hampers its detection by MR angiography.7 Furthermore, one third of the patients in the study of Stock et al suffered from cerebrovascular steno-occlusive disease, which might have influenced collateral flow patterns. The MR angiographic results of Macchi et al,5 who, like Krabbe-Hartkamp et al,7 studied healthy volunteers, are more in agreement with our results, but their study included younger subjects. There are indications that the collateral function of the circle of Willis decreases with advancing age. One limitation of MR angiography is that it is a static technique. It can show patency of collateral vessels but does not measure quantitative flow through them. Volume-flow calculations with dynamic MR inflow tracking is also a promising technique.33 Nevertheless, flow measurement in tiny vessels such as the AcoA and PcoA can be very difficult, especially when these vessels have not (yet) been recruited as significant collaterals.34 To date, it is not clear whether MR angiography or TCCD provides the best information on the collateral potential of the circle of Willis.

The main limitation of the use of TCCD for establishing collateral function is temporal window failure, which is caused by the decreasing acoustic quality of the temporal bone during aging, particularly in elderly women.20–22 Vessel discrimination problems might also be a source of error in testing circle of Willis collateralization with TCCD. Although the AcoA is too small to visualize, the indirect assessment of AcoA patency should not present the examiner with too many technical difficulties. However, the investigation of the collateral function of the PcoA is more susceptible to errors. It requires measurement of blood flow velocity changes in the P1 segment, which in our experience does not show a velocity enhancement during ipsilateral CCA compression, can be easily mistaken for the P1 segment. This technical difficulty might have caused some overestimation of hypofunctioning PcoAs in the present study.

In summary, we showed that in atherosclerotic subjects with no cerebrovascular symptoms, the anterior collateral pathway of the circle of Willis is nearly always functional as opposed to the posterior collateral pathway, which is nonfunctional in almost half of the hemispheres. TCCD probably gives a more reliable insight into the collateral ability of the circle of Willis than does MR angiography or conventional angiography because of the triggering of collateral flow with carotid compression tests. Furthermore, it is a relatively inexpensive and simple technique, which makes it an attractive method of studying intracranial hemodynamics. Comparing our basic data with data from patients with cerebral ischemic symptoms might further help to elucidate the importance of the collateral capacity of the circle of Willis.
Acknowledgments
The authors wish to thank the vascular technologists Johan van Gurp, Henk de Vos, and Leo Nagel for their technical advice, theoretical support, and practical assistance. Vascular surgeons Ron Balm and Steven van Sterkenburg are thanked for enlisting additional patients to the study.

References
Collateral Variations in Circle of Willis in Atherosclerotic Population Assessed by Means of Transcranial Color-Coded Duplex Ultrasonography

A. W. J. Hoksbergen, D. A. Legemate, D. T. Ubbink and M. J. H. M. Jacobs

Stroke. 2000;31:1656-1660
doi: 10.1161/01.STR.31.7.1656

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2000 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/31/7/1656

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/