Transcranial color-coded duplex ultrasonography of the circle of Willis
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Chapter 5

Influence of the collateral function of the circle of Willis on hemispherical perfusion during carotid occlusion as assessed by transcranial colour-coded duplex ultrasonography

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Abstract

Objectives
To investigate the collateral potential of the circle of Willis with transcranial colour-coded duplex ultrasonography and common carotid artery (CCA) compression.

Materials and methods
In 46 atherosclerotic patients without cerebrovascular disease, the functional patency of the collaterals of the circle of Willis, the anterior and posterior communicating arteries, was assessed. The Peak-Systolic Velocity (PSV) decrease in the middle cerebral artery (MCA) during CCA compression between complete and incomplete circles was compared.

Results
In 10 (22%) patients a complete and in 36 (78%) patients an incomplete circle of Willis was found, mainly due to nonfunctioning posterior communicating arteries. In hemispheres with collateral supply through both the anterior and the posterior communicating artery, the median PSV decrease in the MCA during CCA compression was 43%. When the posterior, anterior or both communicating arteries (1 hemisphere) were missing the PSV decrease was 58% (P=0.003), 70% (P=0.001) and 75%, respectively.

Conclusions
Collateral flow from the basilar to the carotid territory is often hampered by nonfunctioning posterior communicating arteries. A nonfunctioning anterior communicating artery is rare. A complete collateral circulation provides better perfusion of the MCA during carotid occlusion as compared with collateral supply through only the anterior or the posterior communicating artery in the case of an incomplete circle of Willis.
**Introduction**

Collateral pathways can protect brain perfusion in the event of severe carotid artery disease. In patients with carotid artery disease, an important route of collateral circulation is provided by the circle of Willis at the base of the brain. The circle of Willis has the ability to function as an anastomosis between the left and right carotid territories via the anterior communicating artery and between the basilar and carotid circulation via the posterior communicating artery. There is evidence that patients with progressive carotid artery disease and an incomplete circle of Willis have an increased stroke risk. Variations of the "normal" Willisian polygon, interfering with adequate collateral function, are frequently found. Various authors have shown through anatomical studies of normal brains that a typical polygon configuration (Figure 1) occurs in only about 20% to 50% of individuals.

Transcranial colour-coded duplex ultrasonography (TCCD) is a relatively new, brain-imaging technique which allows noninvasive assessment of cerebral haemodynamics. The aim of this study was to establish the influence of collateral deficiency on middle cerebral artery (MCA) blood flow during carotid occlusion. To answer this question, we used TCCD to measure the decrease of MCA blood flow velocity during manual compression of the ipsilateral common carotid artery (CCA), simulating carotid artery occlusion. The velocity decrease in the MCA was compared between complete and incomplete circles of Willis in a group of atherosclerotic vascular patients without extracranial cerebrovascular disease.

**Patients and Methods**

**Patients**

The patients included in the study, 44 males and 17 females with a mean age of 61 years (range: 35-89), were randomly chosen from the surgical ward or outpatient clinic. All subjects suffered from peripheral arterial disease, but had specifically denied a history of cerebrovascular disease. Informed consent was obtained from each patient in accordance with the requirements of the local ethics committee. Routine extracranial duplex scanning preceded transcranial investigations to exclude patients with significant stenosis of the extracranial carotid and/or vertebral arteries. This was done to rule out the influence of extracranial stenoses on intracranial velocities and development of collateral pathways. The remaining patients underwent TCCD in combination with CCA compression tests. Compression of the common carotid artery to simulate carotid occlusion was performed for two reasons: first to detect and quantify the collateral ability of the anterior and posterior
communicating arteries and, second, to measure the decrease in peak-systolic velocity (PSV) in the MCA, which is the efferent vessel for both collaterals in the presence of carotid artery occlusion.

![Diagram of the circle of Willis](image)

**Figure 1.** Typical normal polygon configuration of the circle of Willis. M1, main trunk of the middle cerebral artery; A1, precommunicating segment of the anterior cerebral artery; A2, postcommunicating segment of the anterior cerebral artery; P1, precommunicating segment of the posterior cerebral artery; P2, postcommunicating segment of the posterior cerebral artery.

**Technique**
Transcranial investigation was performed with a low-frequency (2.0 MHz-2.5 MHz) probe (Hewlett Packard Sonos 2000) which emits high-output energies to achieve the tissue penetration that is needed to insonate the deep-set basal cerebral arteries. TCCD makes use of relatively thin areas of the skull or natural foramina, which can be penetrated with ultrasound, the so-called acoustic windows. For this study only the temporal windows were used. The temporal window is situated above the zygomatic arch immediately anterior and slightly superior to the tragus of each ear conch. This window was used to insonate the middle cerebral artery, the anterior cerebral artery and the posterior cerebral artery. A routine transcranial examination included insonation of the vertebrobasilar arteries as well, but these data were not considered for further analysis in this study.
Due to their minimal size, low-flow state and unfavourable position with respect to the ultrasound beam, the anterior and posterior communicating arteries usually cannot be visualised. For reliable assessment of their functional patency, CCA compression tests are needed. Compressions of the CCA were applied for 3-5 cardiac cycles, low in the neck, i.e. away from the carotid sinus. To minimise the risk of embolisation, compressions were performed only in patients without atherosclerotic plaques in the proximal CCA. To ensure the efficacy of carotid compression, a photoplethysmograph, which 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. Each carotid artery was compressed at least four times, to assess the collateral function of the anterior and posterior communicating arteries and to determine the PSV decrease in the MCA in both hemispheres.

Collateral supply through the anterior communicating artery is indicated by the reversal of blood flow in the precommunicating part (A1) of the anterior cerebral artery ipsilateral to the compressed CCA, in combination with an enhanced blood flow velocity in the contralateral A1 (Figure 2a). Functional presence of the posterior communicating artery can be demonstrated if the blood flow velocity in the precommunicating part (P1) of the posterior cerebral artery is significantly enhanced during ipsilateral CCA compression (Figure 2b). This velocity enhancement is the result of the pressure gradient between the carotid and vertebrobasilar circulations caused by the compression manoeuvre. A significant velocity increase in the precommunicating parts of the anterior and posterior cerebral arteries during compression was defined as an increase of more than 20% from precompression levels, this value being twice as much as expected from normal variation and measurement error. Persistence of the fetal origin of the posterior cerebral artery from the internal carotid artery

Figure 2a. Schematic drawing of blood flow reversal in the precommunicating segment (A1) of the left anterior cerebral artery and blood flow velocity enhancement in the right A1 during ipsilateral common carotid artery compression (indicated by the black square in the drawing), indicating a functionally patent anterior communicating artery. AcoA, anterior communicating artery; A1, precommunicating segment of the anterior cerebral artery; MCA, middle cerebral artery; ICA, internal carotid artery; PcoA, posterior communicating artery; P1, precommunicating segment of the posterior cerebral artery; BA, basilar artery; VA, vertebral artery.
(10-32% of circles of Willis\textsuperscript{12}) was revealed when the blood flow velocity in the posterior cerebral artery decreased more than 50% during ipsilateral CCA compression.

After determination of the physiological presence of both collateral vessels the proportional decrease in PSV in the main trunk of the MCA during ipsilateral CCA compression was calculated. Subsequently, arterial circles were classified according to the presence or absence of the anterior and posterior communicating arteries. In each patient, both hemispheres were considered as separate units and a subdivision was made regarding the presence of both the anterior and posterior communicating artery or absence of one or both collateral vessels. This subdivision produces four types of Willis circulation (Figure 3). As a fetal posterior cerebral artery will also hamper adequate collateral flow from the basilar to the carotid territory, hemispheres with this variation were classified as having an absent posterior communicating artery. The PSV decrease in the MCA during CCA compression between hemispheres with complete (type I), partially complete (type II and III) and incomplete (type IV) collateral pathways was compared.

Statistics
For analysing study results, the Statistical Package for the Social Sciences (SPSS) for Windows was used. Blood flow velocities (cm/s) are given as medians with their 5% to 95% ranges. The PSV decrease in the MCA during CCA compression was defined as $\frac{\text{PSV}_{\text{precompression}} - \text{PSV}_{\text{postcompression}}}{\text{PSV}_{\text{precompression}}} \times 100\%$. The PSV decrease between complete and incomplete circles was compared using the two-tailed Mann-Whitney $U$ test. Statistical significance was assumed at the 5% level.
Collateral function of the circle of Willis

Figure 3. Variations in the circle of Willis regarding collateral potential. Type I: complete circle of Willis, anterior and posterior communicating artery both functional; Type II: anterior type, anterior communicating artery functional, posterior communicating artery nonfunctional; Type III: posterior type, anterior communicating artery nonfunctional, posterior communicating artery functional; Type IV: incomplete circle, anterior and posterior communicating artery both nonfunctional.

Results
Of the 61 patients who participated five were excluded because of severe carotid artery stenosis, which was revealed during routine extracranial duplex scanning. Of the remaining 56 patients, seven were excluded because of inadequate temporal windows and three were excluded because of atherosclerotic plaques at the site of compression. As a consequence, in 46 patients the collateral integrity of the circle of Willis could be adequately determined (Table 1). No neurological or cardiovascular complications occurred during CCA compression. In 43 patients cross-flow through the anterior communicating artery was provoked during CCA compression. In the other three patients the blood flow velocity in both left and

Table 1. Variations in the Circle of Willis as Found by TCCD and Common Carotid Artery Compression Tests in 46 Patients without Cerebrovascular Disease.

<table>
<thead>
<tr>
<th>Circles</th>
<th>Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Complete</td>
<td>Type I</td>
</tr>
<tr>
<td>Partially complete</td>
<td>Type II</td>
</tr>
<tr>
<td></td>
<td>Type III</td>
</tr>
<tr>
<td>Incomplete</td>
<td>Type IV</td>
</tr>
</tbody>
</table>

Type I: anterior and posterior communicating artery functional; Type II: anterior communicating artery functional, posterior communicating artery nonfunctional; Type III: anterior communicating artery nonfunctional, posterior communicating artery functional; Type IV: anterior communicating artery and posterior communicating artery both nonfunctional.
right anterior cerebral arteries fell to zero upon ipsilateral CCA compression. Although ipsilateral anterior cerebral blood flow in these patients disappeared during this manoeuvre, the PSV in the contralateral anterior cerebral artery increased on the average with 30%.

In contrast with the high number of patent anterior communicating arteries, adequate collateral flow through the posterior communicating artery, which was defined as an increase in PSV of more than 20% in the ipsilateral P1-segment during CCA compression, could only be demonstrated in 48 out of the 92 examined hemispheres. In eight hemispheres in eight patients the PSV in the P1-segment markedly decreased (median 70%) during CCA compression, which proved that in these patients blood flow in the posterior cerebral artery was mainly depending on internal carotid artery blood supply (fetal origin). In nine hemispheres a slight (<20%) increase in PSV in the P1-segment during CCA compression was found. These circles were classified as type II, i.e. absence of a functional posterior communicating artery. In 27 hemispheres no velocity change in the P1-segment could be observed during ipsilateral CCA compression. In Table 2 the pre and postcompression velocities in the ipsilateral and contralateral A1 and P1-segment in the presence of functioning collaterals are shown.

Table 2. Peak-Systolic Velocity Changes in the Presence of a Functionally Patent Anterior or Posterior Communicating Artery.

<table>
<thead>
<tr>
<th>Velocities (cm/s)</th>
<th>Ipsilateral A1</th>
<th>Contralateral A1</th>
<th>Ipsilateral P1</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-compression</td>
<td>80 (49-126)</td>
<td>82 (51-126)</td>
<td>57 (36-77)</td>
</tr>
<tr>
<td>Post-compression</td>
<td>96 (22-197)‡</td>
<td>169 (75-292)</td>
<td>85 (49-173)</td>
</tr>
<tr>
<td>Increase</td>
<td>96% (38-239)*</td>
<td></td>
<td>44% (22-176)*</td>
</tr>
</tbody>
</table>

A1, precommunicating segment of the anterior cerebral artery; P1, precommunicating segment of the posterior cerebral artery. Velocities (cm/s) and velocity increases are given as medians with 5% to 95% ranges. ‡Indicates reversed blood flow velocities, *p<0.001 (Wilcoxon signed-rank test).

The PSV decrease in the MCA during CCA compression is shown in Table 3. A significant difference was found between complete and incomplete circles. Incomplete circles showed a greater decrease in MCA blood flow velocity as compared with complete circles during CCA compression (Figure 4). In three hemispheres in three patients a unilateral velocity increase in the MCA upon CCA compression was found. Each of these three hemispheres received collateral blood supply through both a functional anterior and posterior communicating artery. The velocity enhancements during CCA compression in the contralateral A1-segment and ipsilateral P1-segment in these hemispheres were among the
Table 3. Circle Type and Influence on Peak-Systolic Velocity in the Middle Cerebral Artery During Common Carotid Artery Compression.

<table>
<thead>
<tr>
<th>Circle type</th>
<th>Hemispheres (n=92)</th>
<th>PSV-decrease</th>
<th>p*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type I</td>
<td>43</td>
<td>43% (25-67)</td>
<td></td>
</tr>
<tr>
<td>Type II</td>
<td>43</td>
<td>58% (27-88)</td>
<td>0.003</td>
</tr>
<tr>
<td>Type III</td>
<td>5</td>
<td>70% (60-76)</td>
<td>0.001</td>
</tr>
<tr>
<td>Type IV</td>
<td>1</td>
<td>75%</td>
<td></td>
</tr>
</tbody>
</table>

PSV, peak-systolic velocity; PSV-decreases as medians with 5% to 95% ranges.
* As compared with type I circles.

largest of the total patient group. In one patient with a type II circle of Willis the MCA peak-systolic velocity fell to zero during CCA compression, in spite of cross-flow through the anterior communicating artery. A possible explanation for this phenomenon is that we might have mistaken the distal internal carotid artery (in which the blood flow ceases during CCA compression) for the MCA trunk in this particular patient.

Discussion

Using the criteria as described above, most circles of Willis in the patients included in this study are functionally incomplete, which is comparable with earlier anatomical studies.\(^{5-7}\) The most frequently found deviation was a physiologically absent posterior communicating artery. Truly morphological absence of posterior communicating arteries cannot be assessed with TCCD. Anatomical studies have shown that, although posterior communicating arteries might be hypoplastic or string-like, most of them contain a very small lumen when examined in cross-section.\(^{5}\) The question is whether these tiny channels might develop into vessels providing adequate collateral flow in the presence of severe carotid disease. It is not unthinkable that the collateral “performance” of the posterior communicating artery (and perhaps also the anterior communicating artery) increases during the slow progression of a carotid artery stenosis. A study performed in rats supports this hypothesis.\(^{13}\)

The anterior communicating artery, which provides cross-flow between the cerebral hemispheres, was found to be present in nearly all subjects in this study. This finding feeds the assumption of many authors that the anterior communicating artery is the most important collateral vessel in the presence of a carotid occlusion.\(^{3,14-16}\) Our study supports this theory, since the largest decrease in MCA peak-systolic velocity was found in hemispheres without collateral flow through this vessel, illustrating that residual MCA blood flow velocity was
mainly dependent upon a well-functioning anterior communicating artery. Nevertheless, some additional remarks have to be made. When an acute occlusion of the carotid artery was simulated by CCA compression, the posterior communicating artery had a significant influence on MCA velocity as well. This was shown in type I circles (anterior and posterior communicating artery both present), in which the PSV decrease was significantly less as compared with type II circles with an anterior collateral pathway only (Table 3, Figure 4). So, if the posterior communicating artery was functionally present, it significantly contributed to the maintenance of MCA velocity during sudden occlusion of the CCA. Although speculative, this might just hold the difference between adequate brain perfusion and cerebral ischemia in some patients with severe carotid artery stenosis or occlusion. This hypothesis is supported by a recent study in which the importance of a well-functioning posterior collateral pathway was illustrated by the finding that hypoplastic or absent ipsilateral posterior communicating arteries represent a risk factor for ischaemic cerebral infarction in patients with internal carotid artery occlusion.\(^2\) It was already known that in patients with severe carotid disease the cerebral vasomotor reactivity, which reflects the capacity of the cerebral precapillary vessels to dilate upon a reduced perfusion pressure, is significantly lower in incomplete circles as compared with complete circles.\(^1,3\)

The TCCD criteria, used for assessment of collateral flow, may be debatable. No one will argue that reversed flow in the ipsilateral anterior cerebral artery during carotid compression is not a reliable indicator for a patent anterior communicating artery.\(^10,17,18\) How should we interpret the velocity enhancement of 30% in the contralateral anterior cerebral artery upon CCA compression in the absence of an anterior communicating artery, which was shown in three patients in this study? Chaudhuri et al showed a rather similar case, with
absent unilateral reversal of blood flow in the A1-segment during CCA compression but true velocity enhancement in the same vessel upon contralateral CCA compression. They attributed this to a hypoplastic A1-segment, which was actually shown on angiography. In our three cases, however, the phenomenon was bilateral and TCCD clearly showed both A1-segments, making the assumption of bilateral hypoplastic A1-segments very unlikely. Probably, a contralateral velocity enhancement in the A1-segment during CCA compression is not as reliable an indicator of cross-flow through the anterior communicating artery as reversed flow in the ipsilateral A1-segment.

In the current literature no clear criteria are described for the detection of adequate posterior communicating artery flow using transcranial ultrasound and CCA compression tests. Keumen and Chaudhuri et al used a velocity increase of more than 20% in the P1-segment and Bass et al used a 30% increase as threshold for functional collateral flow through the posterior communicating artery. Other authors do not specify the increased velocities in the P1-segment during CCA compression. Whether a small increase in P1 velocity during CCA compression is caused by measurement error, physiological fluctuation, cardiac output variation or a hypoplastic but patent posterior communicating artery is not clear. Theoretically, secondary collateral vessels such as the leptomeningeal vessels, which can also provide collateral flow from the posterior to the anterior circulation, could also cause a slight velocity enhancement in the posterior cerebral artery during CCA compression. It seems unlikely, however, that in patients without extracranial cerebrovascular disease these vessels develop to significant collateral pathways.

Finally, there is no adequate gold standard for the quantification of blood flow in the collateral vessels of the circle of Willis. It is acknowledged that selective cerebral angiography can only demonstrate the patency of collateral channels, but does not measure flow through them. To date, transcranial ultrasound is the only modality which offers real-time measurement of collateral flow. Nevertheless, measuring velocity changes in the basal cerebral arteries is an indirect way of estimating changes in volumetric flow. Quantification of volumetric blood flow with TCCD is not possible, as this requires determination of vessel diameters, which cannot be done by means of TCCD. Extrapolation of velocity recordings to volumetric blood flow is still a matter of controversy, fueled by a considerable number of reports. Under the assumption that the arterial lumen area of the MCA and its perfusion territory remain constant during compression (which is probable when no leptomeningeal collaterals have developed), it is very likely that both parameters are related, but it is unknown whether this relationship is linear.

It should be realised that TCCD is a new advanced technique to study intracerebral
haemodynamics. It is noninvasive and can be performed as an outpatient procedure. The integration of TCCD in future research might open new horizons in the care for patients with cerebrovascular disease. One of the areas of interest is the ongoing problem of optimal treatment of asymptomatic carotid artery stenosis. In our opinion it would be interesting to study in a prospective way the relation between the functional integrity of the circle of Willis and the development of cerebral ischemia in patients with asymptomatic carotid artery stenosis. If it can be proven that a true relation exists between collateral deficiency of the circle of Willis and the risk of stroke, which is suggested by many authors, then TCCD can be used to select stroke-prone asymptomatic patients who will profit most from a surgical intervention.

In conclusion, collateral flow from the basilar to the carotid territory is often hampered by non-functioning posterior communicating arteries. A non-functioning anterior communicating artery is rare. A complete collateral circulation provides better perfusion of the MCA during carotid occlusion as compared with collateral supply through only the anterior or the posterior communicating artery in the case of an incomplete circle of Willis.

References


