Evaluation of Cross-Circulation Through Circle of Willis Using An Ultrasonic Doppler Technique

Part I. Comparison Between Blood Flow Velocity by Ultrasonic Doppler Flowmetry and Angiogram

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SUMMARY In 24 patients with cerebrovascular disease and 6 without organic brain lesions, the increased velocity of blood flow in both the internal carotid and vertebral arteries during a contralateral carotid compression was compared with the angiographic appearance of the circle of Willis. The flow velocity was measured using ultrasonic Doppler flowmetry.

It was not possible to investigate fully the relationship between the increase of velocity of blood flow in the internal carotid artery and the anatomical variations of the circle of Willis, specifically the anterior cerebral and communicating arteries. The velocity of flow in the patients with an aplastic proximal portion of the anterior cerebral artery showed no increase.

There were no differences in the increase of velocity of flow in the vertebral artery in patients with hypoplastic, normal and fetal posterior communicating arteries.

It is considered that although anatomical variations of the circle vessels influence the cross-circulation via the circle of Willis, peripheral vascular factors distal to the circle also play an important role in the quantity of cross-circulation through the circle.

Materials and Methods

This study included 24 patients with chronic ischemic cerebrovascular disease (CBVD) due to carotid system disease and 6 without organic brain lesions (normal brain). The 30 patients consisted of 28 men and 2 women with an age range from 15 to 70 years (average age 50.0 ± 12.3 SD). Among the 24 patients with cerebrovascular disease there were 17 with completed stroke and 7 with transient ischemic attack.

Angiography

Positive cross-circulation was believed present when in the antero-posterior projection of the angiogram branches of the contralateral carotid artery filled; it was believed to be absent when contralateral branches were not shown.

The proximal segment of the anterior cerebral
TABLE 1  Angiographic Findings of Patients with Cerebrovascular Disease

<table>
<thead>
<tr>
<th>Group</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unilateral CAG (n = 12)</td>
<td>Wall irregularity or mild stenosis of ICA</td>
</tr>
<tr>
<td></td>
<td>Wall irregularity of ACA &amp; MCA</td>
</tr>
<tr>
<td></td>
<td>Wall irregularity or mild stenosis of ICA, ACA &amp; MCA.</td>
</tr>
<tr>
<td></td>
<td>Branch occlusion of MCA</td>
</tr>
<tr>
<td></td>
<td>No evidence of vascular disease</td>
</tr>
<tr>
<td>Bilateral CAG (n = 3)</td>
<td>Wall irregularity or mild stenosis of ICA</td>
</tr>
<tr>
<td></td>
<td>No evidence of vascular disease</td>
</tr>
<tr>
<td>Unilateral CAG &amp; VAG (n = 1)</td>
<td>Wall irregularity of ICA and kinking of VA.</td>
</tr>
<tr>
<td>Bilateral CAG &amp; VAG (n = 8)</td>
<td>Wall irregularity of unilateral ICA.</td>
</tr>
<tr>
<td></td>
<td>Wall irregularity of bilateral ICA.</td>
</tr>
<tr>
<td></td>
<td>Wall irregularity of peripheral branch</td>
</tr>
<tr>
<td></td>
<td>of unilateral MCA and of VA</td>
</tr>
<tr>
<td></td>
<td>Wall irregularity of unilateral ICA, MCA &amp; ACA and stenosis of branch of PCA</td>
</tr>
<tr>
<td></td>
<td>No evidence of vascular disease</td>
</tr>
</tbody>
</table>

ICA; Internal Carotid Artery  
MCA; Middle Cerebral Artery  
ACA; Anterior Cerebral Artery  
VA; Vertebral Artery

artery was judged to be hypoplastic when this vessel was less than 1 mm in diameter. The caliber of the posterior communicating artery was determined on a lateral projection of the angiogram by the ratio of the diameter of that artery to the diameter of the posterior cerebral artery. It was said to be hypoplastic when the caliber of the posterior communicating artery was less than 25 percent of that of the posterior cerebral artery or when the posterior communicating artery was revealed as a string-like vessel by carotid angiogram. The posterior communicating artery was labeled fetal when its caliber was either equal to or approached that of either the anterior or the middle cerebral artery.

**Doppler Flowmetry**

**Apparatus and Basis Examination Technique**

The blood flow velocity was measured by a directional Doppler flowmeter (Hitachi EUD-3B, 5MHz, Hitachi Medical Corporation, Tokyo). All examinations were performed with the patient supine. The technique for internal carotid detection was as follows: The probe was directed to the internal carotid artery and was positioned so as to obtain a maximum audible audio signal from a speaker. To be sure that the signal came from the internal carotid artery the contralateral carotid artery was compressed. This resulted in an increase in the flow velocity signal.

The technique for vertebral flow detection is as follows: The patient's head was turned away from the side of the examination. The probe was placed beneath the posterior-inferior margin of the mastoid process and the ultrasonic beam directed medially and slightly superiorly. To determine that the signal came from the vertebral artery the ipsilateral carotid artery was compressed. This resulted in no change or consequential increase in the flow velocity signal. The signal from the external carotid artery was abolished by this maneuver.

The blood flow velocity (mean velocity) curve obtained from the directional Doppler flowmeter was integrated every 3 seconds. This value was employed so as to simplify the calculation of blood flow velocity increase during a carotid compression. The blood flow velocity, the electrocardiogram, and the ear plethysmogram were all recorded simultaneously on a polygraph (Hitachi ECP-1, Hitachi Medical Corporation, Tokyo) (fig. 1).

**Examination Procedure**

Cross-circulation via the circle of Willis was evaluated from the flow velocity changes in the internal carotid and vertebral arteries by an ultrasonic Doppler flowmeter.

The changes in flow velocity in both internal carotid and vertebral arteries were measured before and during digital compression of the common carotid artery (fig. 2). The changes in blood flow velocity were expressed as percent change from the blood flow velocity in the steady state. The flow change in the internal carotid artery was measured during a compression of the contralateral common carotid artery which redistributes blood flow in the internal carotid artery to the contralateral cerebral hemisphere through the anterior communicating artery. Flow change in the vertebral artery was measured during a compression of the ipsilateral common carotid artery which redistributes blood flow from the vertebral artery to the ipsilateral cerebral hemisphere of the carotid system through the posterior communicating artery.

Complete compression of the common carotid artery was confirmed by a plateau wave in the ipsilateral ear plethysmogram. Patients who had a significant change in blood pressure and heart rate during the carotid compression were excluded.

**Results**

1. **Percent Increase in Blood Flow Velocity**

The percent increase in the velocity of blood flow in the internal carotid artery was measured on 11 arteries.

![Figure 1. Schematic layout of Doppler ultrasonic system employed in this study.](http://stroke.ahajournals.org/fig-1)
from 6 patients with normal brain function and on 45 arteries from 23 patients with CBVD (fig. 3). The percent increase of the internal carotid blood flow velocity was $36.7 \pm 0.8$ (sp) % in 6 patients with normal brain function and $22.1 \pm 12.7$ (sd) % in 23 with CBVD (table 2). The difference is significant ($p < 0.005$). The mean ages in the normal brain function group and in the CBVD group were respectively $44.0 \pm 16.2$ (sd) and $51.3 \pm 11.2$ (sd) years. There was no significant difference in the mean ages between these 2 groups.

The percent increase of the vertebral blood flow velocity was measured on 9 arteries from 5 patients with normal brain function and on 42 arteries in 23 patients with CBVD (fig. 3). The percent increase of the velocity of blood flow in the vertebral artery in patients with normal brain function and CBVD was respectively $41.6 \pm 16.0$ (sd) and $18.9 \pm 12.0$ (sd) %, the difference being highly significant ($p < 0.001$). The mean ages in the normal brain function group and in the CBVD group were respectively $45.6 \pm 17.6$ (sd) and $52.4 \pm 10.3$ (sd) years. There was no significant difference in the mean (table 2).

2. Flow Velocity Increase in Internal Carotid Artery

There was spontaneous angiographic cross-circulation through the anterior communicating artery observed in 27 out of 28 patients who had angiograms. Bilateral anterior cerebral arteries were found in 20 patients. The relation between the percent increase in velocity of flow in the internal carotid artery and the findings from angiograms was examined in those 20 patients.

Thirty-four internal carotid arteries in 17 patients had a bilaterally normal proximal segment in their anterior cerebral artery (normal ACA) and 4 internal carotid arteries in 2 patients had a unilateral hypoplastic proximal segment in the anterior cerebral artery (hypoplastic ACA). The percent increase of the velocity of blood flow in the internal carotid was $24.0 \pm 10.8$ (sd) and $20.9 \pm 15.9$ (sd) % (figs. 4, 5). The patients with a normal ACA had higher mean values than those with a hypoplastic ACA. Because of the paucity of patients with hypoplastic ACA it was not possible to draw firm conclusions concerning the significance.

Flow studies from bilateral internal carotid arteries in one patient with a unilateral aplastic proximal segment of the anterior cerebral artery (aplastic ACA), showed no increase of the velocity of blood flow in the internal carotid artery.

3. Flow Velocity Increase in Vertebral Artery

Thirty-five posterior communicating arteries of 23 patients were indirectly evaluated by study of the flow velocity increase in the ipsilateral vertebral artery during ipsilateral carotid compression. Fifteen arteries in
TABLE 2 Percent Increase in Blood Flow Velocity in Internal Carotid and Vertebral Arteries in Normal Brain and in Cerebrovascular Disease

<table>
<thead>
<tr>
<th></th>
<th>Internal carotid artery</th>
<th>Vertebral artery</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of patients</td>
<td>Number of arteries</td>
</tr>
<tr>
<td>Normal Brain</td>
<td>6</td>
<td>11</td>
</tr>
<tr>
<td>CBVD</td>
<td>23</td>
<td>45</td>
</tr>
</tbody>
</table>

*: Significant difference between normal brain and CVD at p < 0.005  
**: Significant difference between normal brain and CVD at p < 0.001

CBVD Cerebrovascular disease.

Discussion

A variety of methods — electroencephalographic monitoring,** measurement of carotid artery back increase when compared to the other 2 groups. In patients with a fetal PCA and carotid compression one had high increase and 4 low increases.

Figure 4. The blood flow velocity increase in the internal carotid artery during digital compression of the contralateral common carotid artery. The black shaded artery represents the vessel on which measurement of the flow velocity was performed. The dotted artery is the collateral channel. The large dotted arrow represents the velocity increase during a carotid compression. The small dotted arrow shows the flow direction through the circle of Willis.

Figure 5. Percent increase of the velocity of flow in the internal carotid artery of the patients with a normal anterior cerebral artery (n = 34) and with a hypoplastic anterior cerebral artery (n = 4). The values in patients of these 2 groups was 24.0 ± 10.8 (SD) and 20.3 ± 13.9 (SD) %, respectively.
The blood flow velocity increase in the vertebral artery during digital compression of the ipsilateral common carotid artery. The black shaded artery represents the vessel on which measurement of the flow velocity was performed. The dotted artery is the collateral channel. The large dotted arrow represents the velocity increase during a carotid compression. The small dotted arrow shows the flow direction through the circle of Willis.

Pressure, jugular venous blood gases measurement, and regional cerebral blood flow are being used to assess the potential of cerebral collateral circulation.

In our previous report, a non-invasive method for evaluating both the efficiency and the adequacy of collateral circulation via the circle of Willis by the ultrasonic Doppler technique was presented.

In the present study, it was found that the percent increase in flow velocity in both the carotid and vertebral arteries in patients without an organic brain lesion during digital carotid compression was significantly higher than that of those patients with CBVD.

Spontaneous angiographic cross-circulation through the anterior communicating artery was observed in 27 out of 28 patients. This ratio is higher than that in previous reports. It is known that several technical factors influence the distribution of radiographic contrast medium injected into the carotid artery. These are related to both the pressure and the volume of injection, the arterial pressure, and the point of the arterial pulse cycle at the time of the injection of the contrast medium. The demonstration of cross-circulation through the anterior communicating artery on angiography is not a particularly reliable guide to the tolerance of unilateral carotid ligation.

It is not possible to visualize the anterior communicating artery on the antero-posterior angiogram projection and absence of the anterior communicating artery is seen only rarely. All but one of the patients studied who had cerebral angiograms had cross-filling of the anterior cerebral arteries. Based on this finding it was concluded that there was no aplasia of the anterior communicating artery. As to the one exceptional patient, the percent increase of the internal carotid blood flow velocity was 39 and 23% and was not necessarily a low increase.

It has been reported in a previous study that a hypoplastic ACA was accompanied with poor cross-filling through the circle of Willis. The percent increase in the velocity of blood flow in the carotid artery of those 2 patients with a hypoplastic ACA was 6 to 35%, and in 2 arteries it was within normal limits. The number of patients with the hypoplastic ACA studied is small and it is difficult to assess the significance of the changes. No flow increase in the internal carotid artery following carotid compression in the contralateral side was observed in patients with an aplastic proximal segment of the anterior communicating artery. This suggests that in patients with no anatomical connection between the internal carotid arteries via the anterior communicating artery, con-
tralateral carotid compression is not accompanied by increased collateral circulation.

The flow velocity of the patients with a normal ACA had a wide range of increase from 0 to 61% with carotid compression. This result may suggest that the peripheral vascular factors distal to the circle of Willis play a role in determining the difference in increased velocity.

The percent increase in flow velocity in the vertebral artery in patients with hypoplastic, normal, and fetal PCA were 20.9 ± 15.9 (sd), 24.0 ± 10.8 (sd) and 21.7 ± 19.9 (sd) % respectively. The differences between these groups were not significant and there were no differences in the angiographic variations of the PCA.

Levy et al. reported that vasodilation of the circle of Willis occurred immediately after a bilateral carotid occlusion. It is suspected that the PCA may possibly dilate during carotid compression, making it difficult to clarify the differences in the percent increase of the blood flow and the angiographic variations of the artery.

Beatty and Richardson reported that the presence of a bilateral fetal PCA was an indicator of poor tolerance for carotid ligation and that the presence of a unilateral fetal PCA was not associated with ischemia. Jawad et al. stated that most patients with this anomaly satisfactorily tolerated carotid ligation without showing a significant increase in the incidence of cerebral ischemia following the ligation. In our study, one patient with a unilateral fetal PCA had a high flow velocity increase and 2 others with a bilateral fetal PCA had a low increase. There was no difference in the percent increase in flow velocity between patients with a fetal PCA or the other patients.

A pressure drop of probably less than 30 mm Hg occurs between the aorta and the circle of Willis and its large distributing branches. There is a pressure drop 3 to 4 times this magnitude in the vascular bed distal to the circle of Willis and only a small part of the total vascular resistance comes from the circle of Willis itself. Jawad et al. demonstrated that most resistance in cerebral vessels, which determines collateral supply in the brain, lies distal to the circle of Willis.

This study indicates that although anatomical variations of the circle of Willis influence cross-circulation through the circle, a peripheral vascular factor distal to the circle of Willis also plays an important role.

References

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Evaluation of Cross-Circulation Through Circle of Willis Using An Ultrasonic Doppler Technique

Part II. Comparison Between Blood Flow Velocity by Ultrasonic Doppler Flowmetry and Cerebrovascular Resistance

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SUMMARY The correlation between the increase in velocity of blood flow in both the internal carotid and vertebral arteries during a carotid compression and the cerebrovascular resistance (CVR) was investigated in 11 patients with chronic ischemic cerebrovascular disease and 4 without organic brain lesions. The velocity of blood flow was measured by an ultrasonic Doppler flowmeter. CVR was calculated from cerebral blood flow and arterial blood pressure. There was no correlation between the increased velocity of blood flow in the internal carotid and vertebral arteries and CVR. The increased velocity of blood flow in patients with low CVR was, however, significantly higher than that of patients with high CVR. The investigation of cross-circulation by ultrasonic Doppler flowmetry is a useful non-invasive method for the detection of changes in cerebral vascular resistance.

Materials and Methods

Eleven patients with chronic ischemic cerebrovascular disease (CBVD) in the brain supplied by the carotid system without an aplastic anterior cerebral artery on angiogram, and 4 without organic brain lesions (normal brain) were examined. The ages of patients varied from 15 to 70 years with an average age of 48.9 years. Among the 11 patients with cerebrovascular disease there were 10 with completed stroke and one with transient ischemic attacks.

Unilateral carotid angiography was performed in 7 patients and bilateral angiography in 3. Unilateral carotid and vertebral angiograms were made in one patient, and bilateral carotid and vertebral angiograms were made in the other 2 patients. Angiography was not carried out on the 2 patients without cerebrovascular disease.

Angiographic findings of the 11 patients with CBVD are summarized in the table. Patients were excluded if they had moderate or severe stenotic and occlusive lesions of either the carotid and vertebral arteries, or of the anterior, middle, and posterior cerebral arteries.
Evaluation of cross-circulation through circle of Willis using an ultrasonic Doppler technique. Part I. Comparison between blood flow velocity by ultrasonic Doppler flowmetry and angiogram.

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