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.

This study was performed to evaluate the relationship between the cross-circulation through the circle of Willis and cerebrovascular resistance (CVR). Cross-circulation via the circle of Willis was evaluated by an ultrasonic Doppler flowmeter measuring changes of velocity of blood flow in the internal carotids and vertebrals during carotid compression.

ELEKTRONEPHALOGRAPHIC monitoring,1-3 measurement of carotid artery back pressure,5-6 measurement of jugular venous blood gases,7-8 and of regional cerebral blood flow1-8 are used for assessing the potential of cerebral collateral circulation.

In a previous report,9 cross-circulation via the circle of Willis was examined by ultrasonic Doppler flowmetry and was compared with angiographic variations of the circle of Willis. It was suggested that a peripheral vascular factor distal to the circle might play an important role in regulating increases in carotid and vertebral blood flow.

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TABLE Angiographic Findings of Patients with Cerebrovascular Disease

<table>
<thead>
<tr>
<th>Unilateral CAG (n = 6)</th>
<th>Wall irregularity of ICA</th>
<th>1</th>
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<tbody>
<tr>
<td></td>
<td>Wall irregularity of ACA &amp; MCA</td>
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<tr>
<td></td>
<td>Wall irregularity or mild stenosis of ICA, ACA &amp; MCA</td>
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<tr>
<td></td>
<td>Wall irregularity of ICA &amp; MCA</td>
<td>2</td>
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<tr>
<td></td>
<td>No evidence of vascular disease</td>
<td>1</td>
</tr>
<tr>
<td>Bilateral CAG (n = 2)</td>
<td>Wall irregularity or mild stenosis of bilateral ICA</td>
<td>2</td>
</tr>
<tr>
<td>Unilateral CAG &amp; VAG (n = 1)</td>
<td>Wall irregularity of ICA and kinking of VA</td>
<td>1</td>
</tr>
<tr>
<td>Bilateral CAG &amp; VAG (n = 2)</td>
<td>Wall irregularity of peripheral branch of unilateral MCA &amp; VA</td>
<td>1</td>
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<td></td>
<td>No evidence of vascular disease</td>
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ICA = Internal Carotid Artery; MCA = Middle Cerebral Artery; ACA = Anterior Cerebral Artery; VA = Vertebral Artery.

Cerebral Blood Flow Measurement

Cerebral blood flow (CBF) measurements were performed by the 133Xe intracarotid injection method. Clearance of the radioisotope was recorded by an Anger type gamma camera (Hitachi RI-IC-1205, Hitachi Medical Corporation, Tokyo). This system has been reported in detail.19 Approximately 4 mCi of 133Xe in 2 ml of saline is injected through a catheter in the internal carotid artery. Regional CBF was calculated using a computer from the initial slope of the clearance curves obtained in the first 2 minutes. The mean hemispheric CBF was calculated based on the sum of the clearance curves from all areas of interest. CBF was measured in the symptomatic hemisphere in the patients with CBVD, and in the nondominant hemisphere in the patients with normal brain in both the resting state and following hyperventilation. The CBF at 40 mm Hg of Paco2 (i.e., corrected CBF) was calculated from the CBF in both the resting state and following hyperventilation.

Cerebral vascular resistance (CVR) was calculated from the corrected mean hemispheric CBF and the mean arterial blood pressure. CVR which was under 2.2 mm Hg/ml/100g/min was specified as low and bilateral hemispheric cerebral blood flow was measured only unilaterally. For patients without cerebrovascular disease, cerebral blood flow was assumed to be equal, and bilateral hemispheric cerebral vascular resistance was believed to be equal. Accordingly, the percent increase of velocity of blood flow in patients without cerebrovascular disease was related to bilateral internal carotid and vertebral arteries. It is known that in ischemic cerebrovascular disease, blood flow on the healthy side may recover to nearly normal levels.14

In this study, the increase in velocity of flow in the internal carotid artery on the healthy side during a carotid compression on the ischemic side was used to determine the percent of increased blood flow in patients with cerebrovascular disease (fig. 1). This percent increase is believed to correspond to the degree of the cross-circulation via the anterior communicating artery from the healthy to the ischemic side. The blood flow increase in the vertebral artery on the ischemic side during a carotid compression was used to determine the percent increase of blood flow in patients with ischemic cerebrovascular disease (fig. 2). This increase is believed to...
**Figure 1.** The blood flow velocity increase of the internal carotid artery of the non-ischemic side during a digital compression of the common carotid artery of the ischemic side. The dotted area represents the ischemic hemisphere. The black shaded artery represents the vessel on which measurement of the flow velocity is performed. The dotted artery is the collateral channel. The big dotted arrow shows the velocity increase during a carotid compression. The small dotted arrow shows the flow direction through the circle of Willis.

The data also correspond to the degree of cross-circulation through the posterior communicating artery from the vertebral artery on the ischemic side to the internal carotid artery on the ischemic side.

**Results**

1. **Flow Velocity Increase in Internal Carotid Artery**

   The relationship between the CVR and the percent increase of velocity of blood flow was investigated in 7 internal carotid arteries in 4 patients without CBVD and in 10 internal carotid arteries on the ischemic side in 10 patients with CBVD and normal anterior communicating artery. There was no correlation between the percent increase of the velocity of flow in internal carotid arteries and CVR in 17 arteries among 14 patients (fig. 3). The percent increase in 5 arteries among those 5 patients with the high CVR (above 2.2 mm Hg/ml/100g/min) was 18.8 ± 11.6 (SD). In 12 arteries among those 9 patients with low CVR (i.e. lower than 2.2 mm Hg/ml/100g/min) it was 36.3 ± 17.3 (SD)%%. This is a significant difference between the 2 groups (p < 0.02, fig. 4).

2. **Flow Velocity Increase in Vertebral Artery**

   The relationship between the CVR and the percent increase of vertebral blood flow velocity was investigated in bilateral vertebral arteries in the 3 patients with normal brain and in 11 vertebral arteries on the ischemic side in 11 patients with CBVD. There was no correlation between the CVR and the percent increase of velocity of flow in the vertebral artery (fig. 5). The percent increase of the 11 vertebral arteries among the 8 patients with low CVR and the 6 arteries among 6 with the high CVR were respectively 34.5 ± 17.0 (SD) and 13.8 ± 11.3 (SD)%%. This difference between the 2 groups was significant (p < 0.05, fig. 6).

**Discussion**

Our previous study suggested that no increase in velocity of flow in the internal carotid artery during a contralateral compression occurred in patients with no anatomical connection between the internal carotid arteries via the anterior communicating artery. The data also suggested that a peripheral vascular factor distal to the circle of Willis might also play a significant role in regulating cross-circulation through the
FIGURE 3. Relationship between percent increase of the internal carotid blood flow velocity and cerebral vascular resistance in 17 internal carotid arteries of 14 patients. There was no correlation.

FIGURE 4. Percent increase of the internal carotid blood flow velocity of the patients with low cerebral vascular resistance (CVR) under 2.2 mm Hg/ml/100g/min (n=12) and high CVR over 2.2 mm Hg/ml/100g/min (n=5). The percent increase of patients with low CVR and with high CVR was 36.3 ± 13.7 (sd) and 18.8 ± 11.6 (sd)% respectively. There was a significant difference between the 2 groups (p < 0.02).

FIGURE 5. Relationship between percent increase of the vertebral blood flow velocity and cerebral vascular resistance in 17 arteries of the 14 patients. There was no correlation.

The percent increase in velocity of flow in the internal carotid artery in patients with low CVR, however, was significantly higher than that in those with high CVR (fig. 4). It is believed that the flow velocity increase of the internal carotid artery is in part dependent on CVR.

Jawad et al. demonstrated that the cerebral vascular resistance which determines collateral supply in the brain lies distal to the circle of Willis.

It has been shown that the pressure drop that occurred between the aorta and the circle of Willis and its large distributing vessel is small, probably less than 30 mm Hg, and that there is a pressure drop 3 to 4 times greater in the vascular bed distal to the circle of Willis. Thus, only a small part of the total resistance resides in the circle of Willis proper.

In this study there was no correlation between the percent increase in velocity of flow in the internal carotid and the cerebral vascular resistance in 14 patients with a normal anterior communicating artery (fig. 3). The percent increase of velocity of flow in the internal carotid artery in patients with low CVR, however, was significantly higher than that in those with high CVR (fig. 4). It is believed that the flow velocity increase of the internal carotid artery is in part dependent on CVR.

The percent increase in velocity of flow in the vertebral artery in patients with low CVR was significantly higher than that for patients with high CVR (fig. 6).
This suggests that cross-circulation through the posterior communicating artery is in large part dependent on CVR.

These results also suggest a significant role of peripheral cerebral vascular resistance distal to the circle of Willis for the regulation of cross-circulation via the circle of Willis.

This study did not allow identification of the level at which this resistance occurred. Several reports indicated that cerebrovascular response to a decrease in systemic blood pressure occurred predominantly downstream from the cortical surface vasculature and that the resistance of the arterioles was greatest. Pathological change at this level might be related to high vascular resistance in patients in this study.

The investigation of the cross-circulation by ultrasonic Doppler flowmetry is an excellent non-invasive method for the detection of changes in cerebral vascular resistance.

References


Cerebral Vascular Resistance (mmHg/ml/100g/min)

FIGURE 6. Percent increase of the vertebral blood flow velocity of the patients with low cerebral vascular resistance (CVR) below 2.2 mm Hg/ml/100g/min (n=11) and with high CVR above 2.2 mm Hg/ml/100g/min (n=6). The percent increase of patients with low CVR and with high CVR was 34.5 ± 17.0 (SD) and 13.8 ± 11.3 (SD)% , respectively. There was a significant difference between the 2 groups (p < 0.05).
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