Effect of Internal Carotid Artery Occlusion on Intracranial Hemodynamics

Transcranial Doppler Evaluation and Clinical Correlation

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Neurologic deficits that occur simultaneously with or subsequent to internal carotid artery occlusion may be influenced by the adequacy of the intracerebral collateral circulation. Transcranial Doppler ultrasonography was used to evaluate mean middle cerebral artery blood velocity and blood flow in major collateral arteries in 78 patients, including 39 patients with 40 internal carotid artery occlusions and 39 control patients with less severe extracranial cerebrovascular disease, matched for age and sex distribution. Middle cerebral artery blood velocity was 38.9 ± 17.9 cm/sec ipsilateral to an occlusion, 50.9 ± 18.3 cm/sec contralateral to an occlusion (p<0.01), and 56.8 ± 14.4 cm/sec in the controls (p<0.01). Pulsatility index ipsilateral to an occlusion (0.86 ± 0.32) was reduced compared with contralateral and control pulsatility indexes (1.05 ± 0.33 and 1.03 ± 0.18, respectively; p<0.05). Major intracerebral collateral arteries were detectable in 94.9% of occlusion patients and in 53.8% of controls (p<0.01). A posterior communicating artery was demonstrated ipsilateral to an occlusion in 80.0% of the patients and contralateral to an occlusion in 39.5% (p<0.01). An ipsilateral posterior communicating artery was identified in all 10 asymptomatic occlusions and in 75.8% of the symptomatic ones. Pulsatility index was 1.02 ± 0.34 for asymptomatic occlusions and 0.76 ± 0.30 for symptomatic occlusions (p<0.01). Transcranial Doppler ultrasonography permits noninvasive quantification of the cerebral hemodynamic consequences of internal carotid artery occlusion and direct evaluation of the collateral blood supply, which can be correlated with symptomatology. (Stroke 1988;19:589-593)

The severity of neurologic deficits associated with internal carotid artery (ICA) occlusion is highly variable and may be related to the collateral blood supply within the cerebral circulation.1-3 In addition, later cerebral events ipsilateral to an ICA occlusion may be due to continuing local cerebral hypoperfusion.4-6 The pathophysiology of the cerebral circulation in patients with extracranial cerebrovascular disease may be further delineated through an understanding of the perfusion patterns in patients with this severe pattern of carotid occlusive disease. Assessment of the cerebral hemodynamic consequences of ICA occlusion may contribute to the optimal evaluation and treatment of such patients.

Transcranial Doppler ultrasonic evaluation (TCD) of the major intracranial arteries and their collaterals provides a new and important noninvasive diagnostic capability in patients with extracranial cerebrovascular disease.7-9 Measurement of the blood velocity of the middle cerebral artery (MCA) and other intracranial arteries and identification of the collaterals comprising the circle of Willis can be accomplished using a 2-MHz Doppler probe through a transcranial approach.9,10

TCD has previously demonstrated a diminished response to hypercapnia in patients with ICA occlusion.6 We describe other TCD findings in patients with ICA occlusion and demonstrate the utility of this technique in delineating collateral patterns, in identifying hypoperfusion, and in evaluating intracerebral hemodynamics following a severe arterial inflow disturbance.

Subjects and Methods

Patient and Control Populations

Between August 1986 and June 1987, 39 consecutive patients with 40 ICA occlusions were referred to the vascular laboratory and examined using TCD. These were compared with 39 age- and sex-matched controls with less severe extracranial cerebrovascular disease (ICA stenosis of <50% bilaterally, diseased but patent carotid arteries). All subjects underwent concurrent duplex ultrasonic examination of the extracranial carotid vessels. Carotid arteriography was performed in 25 of the patients. Information was recorded concerning the relevant clinical history and neurologic deficits. Two patients were examined both before and after ICA occlusion.

Transcranial Doppler Examination Technique

Evaluation was performed using a 2-MHz pulsed-wave, range-gated Doppler (TC 2-64, Carolina Medical Electronics, Inc., King, North Carolina). Spectral analysis was performed with 64-point fast-Fourier transformation and displayed as velocity, assuming an
angle between the probe and the blood column of 0°. The transtemporal window was located superior to the zygomatic arch and anterior to the ear. Major factors assessed in our study included quantification of the mean MCA blood velocity (MCA-V) and the identification of blood flow in the major collateral arteries, including the anterior communicating artery (ACoA) and the posterior communicating artery (PCoA).

The MCA was insonated with the probe perpendicular to the skull or angled slightly anteriorly at its position over the transtemporal window. The MCA was usually tracked from a depth (or distance from the probe) of 45–60 mm. MCA blood flow toward the probe was shown as upwardly deflected pulse waves. Small incremental changes in probe position were made until the optimal MCA signal was achieved. The systolic, diastolic, and mean MCA-V were quantified, and the pulsatility index ([MCA-V\text{max} - MCA-V\text{min}) / MCA-V \text{mean}] (PI) was calculated for each MCA signal.

The ACoA was not insonated directly but was identified in its capacity as a collateral pathway between the two hemispheres. The anterior cerebral artery (ACA) was located from the transtemporal window by the probe with blood flowing away from the probe (downward deflection of the signal) at a depth of 65–75 mm with a slight anterior angling of the probe. Either of the following criteria were accepted as evidence of the presence of an ACoA: 1) momentary interruption of ACA blood flow by compression of the nonoccluded carotid artery usually caused a reversal of blood flow, diverting blood from the contralateral hemisphere or 2) in some patients, proximal ACA blood flow ipsilateral to an ICA occlusion was continuously reversed due to persistent blood flow through the ACoA from the side of the nonoccluded ICA to the compromised hemisphere. Insonation of the MCA or ACA ipsilateral to the ICA occlusion with simultaneous compression of the contralateral nonoccluded carotid artery demonstrated a decrease in blood flow when an ACoA was present.

The PCoA was located at a depth of 60–75 mm with blood flow usually directed toward the probe. After identifying the point at which the ICA branched into the MCA and the ACA, the probe was directed slightly posterior and inferior. PCoA blood velocities were highly variable, and signals were frequently hypodynamic. The presence of a PCoA was indirectly verified using compression maneuvers. Common carotid artery compression on the nonoccluded side caused an increase in PCoA blood flow, while blood flow in the ACA, ICA, and MCA decreased. Bilateral vertebral artery compressions were also routinely employed to further confirm the origin of the signals in question. Vertebral artery compressions were performed immediately inferior and medial to the mastoid process with an extended index finger. Care was taken not to influence the signal by movements of the patient’s head. A concurrent decrease in PCoA blood flow was seen with vertebral artery compression. Standard TCD has been reported in detail.

### Statistical Analysis

The mean age of patients was 63.9 ± 9.9 years and that of controls was 62.9 ± 11.0 years. There were 31 men and 8 women in each group. One patient had bilateral ICA occlusions.

Mean MCA-V ipsilateral to an ICA occlusion was less than that for the contralateral patent ICA and less than that in controls (p < 0.01, Table 1). PI for MCA signals distal to an ICA occlusion was less than that for the contralateral patent carotid artery and less than that in controls (p < 0.05, Table 1).

Patients were evaluated for the degree of stenosis in the contralateral carotid artery (Figure 1). MCA-V ipsilateral to an ICA occlusion was less than that on the contralateral side regardless of the degree of stenosis of the contralateral ICA. MCA-V distal to both the occluded and the nonoccluded carotid arteries was inversely related to stenosis of the contralateral ICA; MCA-V was lower when the nonoccluded ICA was more severely diseased. There was no apparent relation.

<table>
<thead>
<tr>
<th>Study group</th>
<th>No. observations (arteries)</th>
<th>Velocity (cm/sec)</th>
<th>Pulsatility index</th>
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<tr>
<td>Ipsilateral</td>
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<td>0.86 ± 0.32</td>
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<td>1.05 ± 0.33*</td>
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<tr>
<td>Controls</td>
<td>78</td>
<td>56.6 ± 14.41</td>
<td>1.03 ± 0.18*</td>
</tr>
</tbody>
</table>

* p < 0.05, 0.01, respectively, different from ipsilateral group.

**Figure 1.** Effect of internal carotid artery (ICA) stenosis contralateral to ICA occlusion. Patients with ICA occlusion were divided into three groups based on degree of stenosis in patent contralateral ICA. Middle cerebral artery blood velocity (MCA-V) (± SD) contralateral to ICA occlusion was higher than that ipsilateral in all groups. MCA-V both ipsilateral and contralateral to ICA occlusion was higher if contralateral ICA stenosis was < 50% than if it was more severe.
between PI and the degree of stenosis in the ICA contralateral to an occlusion.

Major collateral arteries were demonstrated in more patients than controls ($p < 0.01$, Table 2). A PCoA was identified more frequently in patients than in controls ($p < 0.01$, Table 2). Among patients, an ipsilateral PCoA was demonstrated in 80.0% and a PCoA accompanying a contralateral patent ICA was identified in 39.5% ($p < 0.01$). When a PCoA was located ipsilateral to an ICA occlusion, MCA-V was $39.9 \pm 17.7$ cm/sec compared with $35.0 \pm 20.9$ cm/sec when there was no ipsilateral PCoA ($p > 0.5$).

There were 10 patients with asymptomatic occlusions. The remaining 29 patients developed either transient or permanent neurologic deficits; neurologic deficits were appropriate to lesions ipsilateral to ICA occlusions in 23 patients and contralateral in six. Eleven of the 29 symptomatic patients had transient ischemic attacks (TIAs) and 18 had strokes. Of the 18 stroke patients, 10 had cerebral infarcts documented by computed tomography (CT scans). MCA-V was $41.0 \pm 14.9$ cm/sec in asymptomatic patients compared with $38.6 \pm 19.9$ cm/sec in symptomatic patients ($p > 0.7$). PI in asymptomatic patients was $1.02 \pm 0.34$ compared with $0.76 \pm 0.30$ in symptomatic patients ($p < 0.01$). Most symptomatic patients had ICA occlusion at the time of initial evaluation, but five patients with previously documented occlusion developed subsequent cerebrovascular events. All five patients with recurrent events after occlusion had ipsilateral TIAs, and their mean MCA-V was $30.3 \pm 24.3$ cm/sec. Major collateral arteries were identified in all but two patients; both had profound deficits, and mean MCA-V was 10 cm/sec. An ipsilateral PCoA was present in all 10 asymptomatic patients and in 22 of 29 (75.8%) symptomatic patients ($p < 0.10$).

Two patients who were examined both before and after ICA occlusion are described in more detail:

**Case 1**

A 71-year-old man had six episodes of left leg weakness and numbness over a 3-month period. Carotid bruises were present bilaterally. Noninvasive extracranial carotid evaluation demonstrated stenotic lesions of >80% in the right ICA and of >50% in the left ICA. TCD showed MCA-V of 32 cm/sec on the right and 60 cm/sec on the left. No intracranial collateral arteries could be identified. Formal contrast arch and carotid arteriography was performed in preparation for surgery, which confirmed the presence of a severe right ICA stenosis. Several hours after angiography, he developed a dense left-sided hemiplegia. Cerebral CT scan revealed an infarct in the right MCA distribution. Repeat duplex examination showed occlusion of the right ICA. TCD demonstrated MCA-V of 20 cm/sec on the right and of 64 cm/sec on the left. In addition, a right PCoA (mean blood velocity 86 cm/sec) was present, which was not identified during the initial TCD (Figure 2).

**Comment.** Despite severe carotid disease, no hemodynamically significant collateral blood flow could be identified before ICA occlusion. After ICA occlusion and cerebral infarction, right MCA-V was further diminished, and the right PCoA was recruited as a collateral channel.

**Case 2**

A 71-year-old asymptomatic woman had longstanding bilateral ICA stenoses. Duplex evaluation showed >80% stenosis in the left ICA and <50% in the right ICA. TCD showed MCA-V of 24 cm/sec on
Before Occlusion

Right MCA-V 54 cm/sec
ICA Stenosis <50%

Left MCA-V 24 cm/sec
ICA Stenosis >80%

Left PCoA 44 cm/sec

After Occlusion

Right MCA-V 50 cm/sec
ICA Stenosis <50%

Left MCA-V 24 cm/sec
ICA Occlusion

Left PCoA 54 cm/sec

FIGURE 3. Transcranial Doppler signals from Case 2. Middle cerebral artery blood velocity (MCA-V) ipsilateral to long-standing severe left internal carotid artery (ICA) stenosis was 24 cm/sec. Ipsilateral posterior communicating artery (PCoA) was identified (mean blood velocity 44 cm/sec). After asymptomatic occlusion of left ICA left MCA-V was unchanged and left PCoA blood velocity increased to 54 cm/sec.

the left and of 54 cm/sec on the right. A left PCoA was identified (mean blood velocity 44 cm/sec). Follow-up evaluation 3 months later demonstrated a left ICA occlusion, but she remained asymptomatic. MCA-V was unchanged, and left PCoA mean blood velocity was 54 cm/sec (Figure 3).

Comment. The presence of long-standing severe ICA stenosis and major identifiable collateral blood flow (i.e., left PCoA) in this case was associated with the maintenance of adequate MCA blood flow and no neurologic deficit when ICA occlusion occurred.

Discussion

MCA-V and PI associated with an ICA occlusion were decreased compared with both the contralateral patent carotid arteries in the same patients and with controls (Table 1). Although the loose inverse relation between MCA-V distal to an ICA occlusion and the degree of contralateral ICA stenosis suggests the importance of collateral perfusion from the contralateral hemisphere, the fact that this trend was not more striking suggests that other collateral pathways may also contribute significantly (Figure 1). Major collateral arteries were identified in most patients (Table 2) and were seen more frequently in asymptomatic than in symptomatic patients.

Current data regarding the natural history of ICA occlusion do not explain the wide variability observed in both presenting symptoms and subsequent neurologic events.1-3,12 Both embolic and ischemic mechanisms have been invoked, but these are difficult to differentiate.6,16,17 Many patients with ICA occlusion have diminished ipsilateral resting regional cerebral blood flow (rCBF), which may be accentuated with hypercapnia because of the loss of CO2-induced reactivity.16 Although the capacity to recruit collateral perfusion to the depleted MCA territory through the circle of Willis may be estimated using rCBF, it does not allow direct identification of specific intracranial arteries.6,18 TCD has been used to assess the cerebral hemodynamic consequences of carotid artery disease.7-9 MCA-V can be evaluated and collateral arteries can be examined directly.10 Bishop et al found that resting systolic MCA-V was reduced ipsilateral to ICA occlusion and that the expected increase in blood flow in response to hypercapnia was markedly diminished. In further studies, MCA-V correlated with rCBF during hypercapnia in patients with symptomatic extracranial cerebrovascular disease.19 Repeated noninvasive evaluations using TCD may help to delineate the natural history of carotid disease both before and after occlusion.

The reduction in pulsatility distal to an ICA occlusion demonstrates vasodilatation within the MCA territory accompanied by reduced peripheral resistance. This effect suggests that MCA collateral perfusion is insufficient in some patients. Indeed, PI was significantly higher with asymptomatic occlusions than with symptomatic ones. Most of the patients with an ICA occlusion developed specific collateral arteries that were directly identifiable by TCD. Our preliminary data suggest that the development of collateral arteries before occlusion of the ICA may reduce the risk of symptoms due to hypoperfusion.

Well-developed collateral blood flow demonstrated by TCD preoperatively appears to be a determinant of the hemodynamic significance of an extracranial carotid lesion and a strong predictor of cerebral protection during carotid cross-clamping.6 Follow-up evaluations of patients with progressive carotid disease and occlusion may enhance our understanding of the pathophysiology of neurologic deficits in these patients as related to collateral artery formation. Consideration of contralateral internal carotid endarterectomy or other therapeutic measures may be aided by the assessment of MCA-V and the presence of collateral arteries.

TCD permits quantitative noninvasive evaluation of the cerebral hemodynamic consequences of ICA occlusion and the direct examination of cerebral collateralization, which can be correlated with symptomatology. TCD may help to identify those patients at risk for cerebral ischemia due to ICA occlusion and may provide assistance in the management of therapeutic interventions in the future.
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References

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