Transhemispheric Passage of Microemboli in Patients With Unilateral Internal Carotid Artery Occlusion

Dimitrios Georgiadis, MD; Donald G. Grosset, MD; Kennedy R. Lees, MD

Background and Purpose: Ischemic episodes distal to an internal carotid artery occlusion are common. We undertook this study to look for evidence of transhemispheric passage of embolic material in this patient category as a mechanism for embolic events.

Methods: Seven symptomatic patients with unilateral internal carotid artery occlusion and contralateral stenosis were examined by transcranial Doppler ultrasonography with 2-MHz probe (average monitoring time, 2.5 hours per patient). Both middle cerebral arteries and (if present) the reverse-flow anterior cerebral artery ipsilateral to the occluded internal carotid were monitored. Three patients were reexamined 1 month after carotid endarterectomy.

Results: Embolic signals were detected in the middle cerebral artery ipsilateral to the stenosed internal carotid artery in all seven patients and in the opposite middle cerebral artery in four patients. In these four patients, a reverse-flow anterior cerebral artery was observed in which embolic signals were detected. No embolic signals were detected after surgery in any of the three patients who underwent carotid endarterectomy.

Conclusions: Transhemispheric passage of embolic material occurs in patients with unilateral internal carotid artery occlusion and contralateral stenosis. Endarterectomy of the stenosed internal carotid artery may eliminate the detected embolic signals in both hemispheres. Transcranial Doppler ultrasonography could be used as a diagnostic tool to identify the embolic source in patients with unilateral carotid occlusion. (Stroke. 1993;24:1664-1666.)

KEY WORDS • carotid artery diseases • embolism • ultrasonics

Ischemic symptoms distal to an occluded internal carotid artery (ICA) are common. The failure of extracranial-intracranial bypass to reduce the risk of ischemic stroke in this patient category argues against such symptoms being caused by hypoperfusion, leaving embolism as the most probable cause. The source of embolic material could be the contralateral ICA, the ipsilateral external carotid artery (ECA), or the proximal (carotid stump) or distal end of the occluded ICA. The importance of accurate localization of the embolic source is obvious, particularly in planning surgical intervention.

Transcranial Doppler ultrasonography (TCD) has been successfully used in the detection of emboli in several patient categories and in experimental models.

We report a group of seven patients who were monitored with TCD to identify the occurrence of transhemispheric passage of embolic material.

Subjects and Methods

Seven consecutive symptomatic patients referred for carotid color duplex examination in whom unilateral ICA occlusion with contralateral stenosis was diagnosed were examined in this study. Selected patients underwent additional intra-arterial angiography as part of a preoperative workup. Color duplex ultrasonography was performed with an Acuson 128 with a 5-MHz probe (Acuson, Calif). The degree of carotid stenosis was measured according to the European Carotid Surgery Trial criteria in the patients who underwent carotid angiography and standard ultrasonographic criteria in the remaining patients, measuring the peak end-diastolic velocity, the systolic velocity ratio (internal to common carotid artery), and the spectral broadening. Bilateral transcranial Doppler studies were performed with a pulsed Doppler ultrasound machine (TC-2000; Nicolet, Warwick, UK) with a 2-MHz probe. The emitted ultrasonic power used was 50 mW/cm², and the sample volume was 15 mm. These settings were kept constant throughout the study. After localization of the temporal window, the probe was fixed with an elasticated band to ensure a constant insonation angle and minimize movement artifacts.

The following vessels were studied: bilateral middle cerebral arteries (MCA) at a depth of 40 to 50 mm and reverse-flow anterior cerebral artery (ACA) ipsilateral to the occluded ICA (when present) at a depth of 70 to 74 mm for 1 hour per vessel. Reverse-flow ACA was identified according to standard criteria. Abnormal high-pitched signals representing microemboli were recognized by their acoustic (harmonic sound) and visual...
(unidirectional within the advancing or receding velocity spectrum, duration <0.1 second inversely proportional to their velocity, random occurrence within the cardiac cycle) qualities7 and stored for later off-line evaluation. Artifacts were distinguished both by their different visual and acoustic qualities (bidirectional, coincidental with probe or patient movement or electrical switching transients, and nonharmonic sound) online7 and their spectral characteristics.12

Cranial computed tomography (CT) was performed in three patients. Three of the seven patients underwent carotid endarterectomy and were reexamined 1 month after the operation by use of the same technique.

### Results

Clinical data of the seven patients examined are summarized in Table 1. Five of the seven patients had a severe (>70%) and two a moderate (50% to 70%) stenosis contralateral to the occluded ICA. All patients were on aspirin, between 75 and 300 mg/d. In four patients, the symptomatic cerebral hemisphere was ipsilateral to the stenosed ICA and in two, to the occluded ICA; one patient had bilateral symptoms (Table 1).

Reverse flow in the ACA ipsilateral to the carotid occlusion was identified in five patients. Emboli were detected within this vessel in four of these patients and were also present within the adjacent MCA. The embolic signal counts are shown in Table 2. The mean number of embolic signals in the MCA ipsilateral to the occluded ICA was 3.2 per hour; in the contralateral MCA, 11.8 per hour; and in the reverse-flow ACA, 2.2 per hour.

Three patients underwent carotid endarterectomy. Two had bilateral preoperative embolic signals, one only contralateral to the occluded ICA (Table 2). Histological examination of the endarterectomy specimens revealed severe atheroma, ulceration of the endothelium, fibrin deposits within the vessel wall, and cholesterol clefts in all three cases. Additionally, mononuclear inflammatory cells were diagnosed in two patients (patients 2 and 3). There was no evidence of thrombus within the lumen of any of the three specimens. One month after surgery, no embolic signals were detected in the MCA or the reverse-flow ACA in any of the three patients.

### Discussion

Abnormal high-pitched signals detected with TCD have been documented to represent microemboli, particularly by the reproduction of similar signals by introducing embolic material in vitro9 and animal models.10 There are no definitive guidelines on the optimal monitoring time for recording Doppler emboli signals. The variability in the number of detected embolic signals is high, particularly when their frequency is low. In particular, failure to detect embolic signals during the monitoring time adopted in this and other studies8 does not necessarily exclude their presence.

Despite these limitations, we believe that TCD could be used as a diagnostic tool in distinguishing between sites of potential embolic sources in patients with unilateral carotid occlusion. Embolic signals would be expected in the ipsilateral reverse-flow ophthalmic artery if their source were ipsilateral to the occluded ICA (plaque in the ECA or the common carotid artery or arising from the carotid stump). Embolic signals arising from the distal part of the occluded ICA would be detectable in the carotid siphon. The ophthalmic artery and the carotid siphon were not monitored in this study; therefore, neither the extracranial

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### Table 1. Clinical Details and Imaging Results in Seven Patients With Carotid Occlusion and Contralateral ICA Stenosis

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, y</th>
<th>Sex</th>
<th>Symptoms, Onset, Frequency, Duration</th>
<th>CT Scan</th>
<th>Angiography, Color Doplex</th>
<th>Plaque Morphology</th>
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<tbody>
<tr>
<td>1</td>
<td>70</td>
<td>F</td>
<td>L TMB, 8 months, 2 episodes, 10 and 30 minutes</td>
<td>Normal</td>
<td>L ICA occlusion*</td>
<td>Smooth, homogeneous</td>
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<tr>
<td>2</td>
<td>65</td>
<td>M</td>
<td>R TIA, pure motor, arm&gt;leg, 2 months, 3 episodes, 1/2, 2, and 6 hours</td>
<td>†</td>
<td>L ICA occlusion*</td>
<td>Smooth, heterogeneous‡</td>
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<tr>
<td>3</td>
<td>62</td>
<td>M</td>
<td>R+L TMB, 3 months, 3 episodes, 10 to 15 minutes</td>
<td>†</td>
<td>R ICA occlusion*</td>
<td>Irregular, heterogeneous‡</td>
</tr>
<tr>
<td>4</td>
<td>78</td>
<td>M</td>
<td>R TMB, 3 months, 1 episode, 5 minutes</td>
<td>Normal</td>
<td>L ICA occlusion*</td>
<td>Smooth, homogeneous</td>
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<tr>
<td>5</td>
<td>73</td>
<td>F</td>
<td>R cerebral infarction (L arm, leg weakness, homonymous hemianopia, neglect), acute</td>
<td>R cortical/subcortical infarct</td>
<td>L ICA occlusion R ICA &gt;70% stenosis</td>
<td>Smooth, homogeneous</td>
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<tr>
<td>6</td>
<td>74</td>
<td>M</td>
<td>R TIA (L arm, leg weakness, dysarthism), 2 months, 1 episode, 3 hours</td>
<td>†</td>
<td>R ICA occlusion*</td>
<td>Irregular, heterogeneous</td>
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<tr>
<td>7</td>
<td>66</td>
<td>M</td>
<td>R cerebral infarction (L arm, leg weakness, homonymous hemianopia), 6 months</td>
<td>R cortical infarct</td>
<td>L ICA occlusion R ICA 50-70% stenosis</td>
<td>Smooth, homogeneous</td>
</tr>
</tbody>
</table>

ICA indicates internal carotid artery; ECA, external carotid artery; CT, computed tomography; L, left; TMB, transient monocular blindness; R, right; and TIA, transient ischemic attack.

*Patients who underwent intra-arterial angiography.
†CT not performed.
‡Ulcereated plaque confirmed intraoperatively.
TABLE 2.  Number and Site of Doppler Emboli Signals in Seven Patients With Unilateral Carotid Occlusion

<table>
<thead>
<tr>
<th>No. of Embolic Signals per Hour</th>
<th>Reverse-Flow Anterior Cerebral Artery</th>
<th>Middle Cerebral Artery (ip)</th>
<th>Anterior Cerebral Artery (rf)</th>
<th>Middle Cerebral Artery (co)</th>
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</thead>
<tbody>
<tr>
<td>Patient</td>
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<td>1</td>
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</table>

ip indicates ipsilateral to the occluded internal carotid artery; co, contralateral to the occluded internal carotid artery; and rf, reverse flow.

*Patients who underwent carotid endarterectomy.

carotid system ipsilateral to the occluded ICA can be excluded as potential embolic sources. The demonstrated presence of embolic signals in the reverse-flow ACA identifies the contralateral stenosed ICA as an embolic source. To the best of our knowledge, this is the first documentation of such a phenomenon.

The five patients with severe contralateral ICA disease had higher embolic signal counts than the remaining two patients. This could be a result of the high incidence of irregular plaque in this group. It has been suggested that the embolic risk of patients with carotid stenosis depends on the plaque subtype. Quantification of emboli with TCD could provide valuable information in this regard.

Although tranhemispheric passage of emboli was detectable in four patients in this study, only one had potentially related symptoms. Clinically silent emboli have been detected in patients with prosthetic cardiac valves or carotid stenosis and also intraoperatively during carotid surgery. The clinical relevance of the detected intracranial embolic signals remains to be determined.

Long-term monitoring of the ophthalmic artery, the carotid siphon, and the reverse-flow ACA could localize the site of the embolic source and lead to appropriate surgical treatment. Surgical intervention might improve the outcome of patients with unilateral carotid occlusion and contralateral ICA stenosis that is identified as the source of the embolic material. This theory is supported by the postoperative results of the three patients in our study in whom no embolic signals were detected in either MCA. Further studies with greater numbers of patients with unilateral carotid occlusion might improve our understanding of the pathogenesis of the ischemic events occurring in this patient group and, subsequently, their treatment.

References

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