Detection of Intracranial Emboli in Patients With Symptomatic Extracranial Carotid Artery Disease

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Background and Purpose: Cerebral embolism from extracranial sources is an important cause of ischemic stroke. The purpose of this limited study using long-term transcranial Doppler ultrasonographic monitoring was to estimate the frequency of clinically silent intracranial embolisms in patients with symptomatic extracranial carotid artery disease.

Summary of Report: By means of a 2-MHz pulsed-wave transcranial Doppler instrumentation, three consecutive patients with extracranial internal carotid artery stenosis (n=2) or occlusion (n=1) and recurrent ipsilateral ischemic events were monitored (19 hours total recording time). In addition, 10 control subjects without cerebrovascular disease were studied (25 hours total recording time). Formed-element emboli were defined as distinct signals within the fast Fourier-transform Doppler spectrum that were <70 msec in duration and >9 dB greater in intensity than the background signal. Clinically silent formed-element embolism of ophthalmic or cerebral arteries was demonstrated in all three patients. Embolic events occurred only in the territory of the symptomatic internal carotid artery. The average rate of cerebral embolization at transcranial Doppler ultrasonography was 4.1/hr, with a mean signal duration of 47 msec. No emboli were found in control subjects.

Conclusions: The observed high frequency of silent embolism of the intracranial arteries detected by transcranial Doppler monitoring in patients with recurrently symptomatic extracranial carotid artery disease should encourage studies of the prognostic and therapeutic implications of this method. (Stroke 1992;23:1652-1654)

KEY WORDS • cerebral ischemia • cerebrovascular diseases • embolism • ultrasonics
TABLE 1. Clinical and Radiological Characteristics of Three Patients With Recurrently Symptomatic Extracranial Carotid Artery Disease

<table>
<thead>
<tr>
<th>Patient/age (yr)/sex</th>
<th>Symptoms*</th>
<th>CT/MRI scans†</th>
<th>Intra-arterial angiography</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/42/M</td>
<td>7 TMBs, 1 TIA</td>
<td>Normal</td>
<td>L ICA occlusion collateralized via L OphA</td>
</tr>
<tr>
<td>2/72/F</td>
<td>2 TIAs, 1 minor stroke</td>
<td>Multiple R MCA territory infarcts</td>
<td>80% stenosis, R ICA origin</td>
</tr>
<tr>
<td>3/48/M</td>
<td>3 TMBs, 1 minor stroke</td>
<td>Multiple L MCA territory infarcts</td>
<td>40% stenosis, L ICA petrous portion</td>
</tr>
</tbody>
</table>

CT, computed tomography; MRI, magnetic resonance imaging; TMB, transient monocular blindness; TIA, transient ischemic attack (cerebral hemisphere); L, left; R, right; ICA, internal carotid artery; OphA, ophthalmic artery; MCA, middle cerebral artery.

*Symptoms within last 4 weeks attributable to territory of angiographically affected ICA.
†These scans were obtained before transcranial Doppler monitoring.

with a head tape to the lateral temporal region (MCA, ACA) or hand held during transorbital insonation (ophthalmic artery). The subjects were in a supine or sitting position. They were examined continuously over 1–2 hours per recording session. Two different probes were used to exclude possible technical artifacts originating from the transducer. The amplitude of frequencies in each vertical line of the fast Fourier–transform spectra (128 points) was color coded. Each of 15 steps in the color scale corresponded to a difference of 3 dB. The following criteria were used to define a signal as indicating a formed-element embolus: signal occurring within the blood flow velocity spectrum; signal intensity >9 dB compared with background intensity of the spectrum; signal duration <70 msec. Artifacts produced by dislocation of the Doppler probe, movement of the subjects, or twitches of the eyelids could be clearly distinguished from signals indicating emboli, the former being characterized by bidirectional Doppler signals, broader velocity spectra, or longer signal durations.

Results

In each of the three patients, intracranial emboli were detected in the MCA and ACA ipsilateral to the clinically symptomatic ICA (Figure 1, Table 2). This was the case on each of several days on which TCD monitoring was performed. No emboli occurred contralaterally. In patient 3, emboli were also observed in the ipsilateral ophthalmic artery (Figure 1). Total TCD recording times, numbers, and vascular distributions of

![Figure 1: Traces of Doppler velocity spectra obtained during long-term transcranial Doppler monitoring in three patients. Color scale at top illustrates relative grading of frequency amplitudes in steps of 3 dB, with blue corresponding to low dB values. Arrows indicate typical signals designating formed-element emboli. Panel a: Patient 1; transtemporal insonation of left anterior cerebral artery (ACA). Panel b: Patient 2; transtemporal insonation of right middle cerebral artery (MCA). Panel c: Patient 3; transorbital insonation of left ophthalmic artery (OphA). Panel d: Patient 3; transtemporal insonation of left MCA. Symbols at left indicate flow directions relative to Doppler probe.](http://stroke.ahajournals.org/)

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emboli are presented in Table 2. Intensity amplitudes of emboli in the fast Fourier–transform Doppler spectra exceeded background intensity by 12–21 dB. Mean ± SD signal duration was 47 ± 14 msec. Signals occurred randomly and seemed to be preferentially located in the upper range of the velocity spectrum (Figure 1). Features of the displayed signals, such as duration, intensity, or occurrence relative to cardiac cycle, were different for each embolic event. The average frequency of embolism was 4.1 events/hr recording time (averaged across all three patients for transtemporal TCD recordings ipsilateral to the clinically symptomatic ICA) (Table 2). None of the patients reported symptoms associated with TCD-detected embolic events. No emboli were detected in the 10 control subjects.

Discussion

There is strong evidence that the TCD signals observed in the present study represented intra-arterial emboli. First, the signals were detected only ipsilateral to recurrently symptomatic ICA stenosis or occlusion, never contralaterally or in control subjects. Second, signal intensity, duration, and location in the velocity spectra closely corresponded to the findings of Russell et al, who used the same TCD equipment in experimental arterial thromboembolism. Third, the TCD signals were similar to those attributed to formed-element emboli by Spencer et al, who examined humans before, during, and after carotid endarterectomy. Further validation studies are needed to establish correlations between TCD characteristics, size, and composition of cerebral emboli.

The results reported here suggest that even under anticoagulation, clinically silent embolism of the intracranial circulation may be common in symptomatic atherosclerotic ICA disease. The frequency and vascular distribution (Table 2) of embolic events may be substantially underestimated by clinical or radiological evaluation alone. Of course, our threshold criteria for identifying emboli did not exclude even higher rates of embolism by “smaller” formed elements. The high probability of detecting arterial emboli within only a few hours’ recording time promises that TCD monitoring will be a powerful diagnostic tool.

More than 30 years ago, Fisher and Ross Russell were the first to report direct observation of retinal emboli in transient monocular blindness. Since then, transient retinal ischemia has been viewed as the prototype of embolism as the cause of ischemia in the ICA territory. TCD monitoring now provides a noninvasive method for investigating embolic phenomena in cerebral ischemia as well. Further studies will have to consider the role of this method in identifying high-risk patients among those with carotid stenosis, in analyzing the pathogenesis of cerebral ischemia in symptomatic carotid occlusion, and in assessing the efficacy of antithrombotic treatment.

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References

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