Doppler Emboli Signals Vary According to Stroke Subtype

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Background and Purpose
Doppler ultrasound detection of emboli signals may assist in distinguishing embolic from thrombotic stroke. Selected patient groups have a high incidence of such signals. We have examined consecutive stroke cases to identify the incidence of Doppler emboli in different etiologic subtypes of stroke.

Methods
Forty-five patients presenting with first-ever acute carotid territory cerebral ischemia were studied prospectively. Transcranial Doppler examination of both middle cerebral arteries, carotid color duplex ultrasound, and transthoracic or transesophageal echocardiography were completed within 48 hours of deficit onset. Clinical and imaging data were interpreted independent of emboli data, and stroke etiology was classified according to recent multicenter trial criteria.

Results
Middle cerebral artery signals were identified in at least one cerebral hemisphere in 41 of the 45 patients. Emboli signals were present in 29 of these 41 cases (71%). These signals were bilateral in 22, within the affected (symptomatic) cerebral hemisphere only in 5, and contralateral only in 2 cases. No emboli signals were detected in any of 8 patients with lacunar stroke. The overall difference in emboli signal counts between etiologic subgroups was significant (P=.001, Kruskal-Wallis). A significantly higher emboli signal count was found within affected cerebral hemispheres than contralaterally in the 8 patients with large artery atherosclerosis (11.3 versus 1 signals per hour, median [95% confidence interval, 3 to 40 and 0 to 3, respectively], *P*=.02), but this interhemisphere difference was not present for other etiologic subgroups.

Conclusions
Emboli signals are common in patients with acute stroke, with the notable exception of lacunar stroke. This is consistent with the small vessel etiology for the latter group and provides support for the relevance of Doppler emboli signal detection in thromboembolic cerebrovascular disease.

Key Words • diagnostic imaging • stroke assessment • ultrasounds

The diagnosis of embolic stroke is currently made on detection of a potential embolic source, usually after the neurological event. Recently introduced criteria have updated and improved the etiologic classification of ischemic stroke, but the absence of a "test" for embolism means that definitive diagnosis of embolic versus thrombotic stroke is impossible. The detection of emboli signals by Doppler ultrasound may improve this situation; provisional reports indicate that persistent subclinical microemboli exist in patients with atrial fibrillation and stroke, and carotid stenosis, and other embolic sources. Experimental models confirm that embolic material produces ultrasound signals resembling those seen in patients. We have prospectively applied this technique in patients with acute stroke and in particular have compared patients with probable embolic stroke with those with lacunar stroke, because the latter represent a predominantly nonembolic category.

Subjects and Methods
Forty-five patients presenting with first-ever nonhemorrhagic acute anterior circulation cerebral ischemia were studied prospectively. The subjects were admitted consecutively to our Acute Stroke Unit. Cerebral computed tomography was performed routinely, and 8 patients with cerebral hemorrhage admitted during the study period were excluded from this study. Ultrasound studies were completed within 48 hours of the onset of acute neurological deficit. These consisted of echocardiography (transthoracic, with additional transesophageal testing in patients under 50 years) and color duplex carotid sonography, both using an Acuson 128 computed sonography system. In addition, a 30-minute transcranial Doppler (TCD) recording was made over each of the middle cerebral arteries (MCAs) at a depth of 46 to 54 mm from the temporal position, using a TC2000 with 2 MHz probe (Nicolet Instruments, Warwick, UK). Each MCA was examined sequentially, and an elastic headband was used to hold the transducer in position. The severity of carotid stenosis was estimated according to ultrasound findings of an increased internal-to-common carotid artery peak systolic velocity ratio and the presence of spectral broadening. MCA Doppler emboli signals were recognized by their characteristic short-duration high-intensity appearance (<200 milliseconds, at least 3 dB above background), and artifacts were excluded by their typically symmetrical appearance within the Doppler spectrum, often coincident with patient movement. All Doppler signals were recorded by a single observer. Clinical details and the results of other investigations were recorded independently by a second observer blinded to the Doppler emboli data, and these were used to define the etiologic classification of stroke according to the TOAST criteria. An important feature of this classification is the consideration and inclusion of patients with atrial fibrillation as having a cardioembolic source, whereas patients with more than one site of disease (eg, atrial fibrillation and carotid stenosis) are considered to have an uncertain stroke etiology.
Middle Cerebral Artery Doppler Emboli Signals in 45 Patients With Ischemic Stroke

<table>
<thead>
<tr>
<th>Etiology of Stroke</th>
<th>No. of Patients</th>
<th>Technical Failure (No.)</th>
<th>Patients With Emboli (No.)</th>
<th>Symptomatic Hemisphere</th>
<th>Asymptomatic Hemisphere</th>
</tr>
</thead>
<tbody>
<tr>
<td>Large artery atherosclerosis</td>
<td>8</td>
<td>0</td>
<td>7</td>
<td>11.3 (3-40)</td>
<td>1.0 (0-3)</td>
</tr>
<tr>
<td>Cardioembolic/probable embolic</td>
<td>12</td>
<td>1</td>
<td>9</td>
<td>2.2 (0.5-4)</td>
<td>2.0 (1-3.5)</td>
</tr>
<tr>
<td>Lacunar</td>
<td>9</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Uncertain</td>
<td>16</td>
<td>2</td>
<td>13</td>
<td>3.5 (2-7.5)</td>
<td>2.5 (0-3)</td>
</tr>
</tbody>
</table>

Data are median. CI indicates confidence interval.

Results

The 45 patients were aged 32 to 96 years, mean 67 years; 18 were women and 27 were men. TCD was technically unsuccessful (MCA not identified in either cerebral hemisphere) in 4 patients, and in a further 2 patients one MCA was not identified (in both cases in the asymptomatic cerebral hemisphere). Of the 41 patients we were able to evaluate, 8 had a final diagnosis of transient ischemic attack and 33 had ischemic stroke. Ten subjects were in atrial fibrillation and 31 were in sinus rhythm. Hemodynamically significant internal carotid artery (ICA) stenosis was present in 8 patients. This was unilateral in 7 and bilateral in 1 patient; 1 patient had contralateral ICA occlusion. The luminal reduction was estimated to be over 70% in 7 patients and to be 50% to 70% in 1 patient. The side of carotid stenosis did not always correspond to the presenting symptoms. Potential cardiac sources of embolism were present in 12 patients, of whom 11 had more than one abnormality identified (aortic or mitral valve disease in 6, aortic and mitral valve disease in 5, increased left atrial size and/or left ventricular dysfunction in 7, new lone atrial fibrillation in 1). One patient was on warfarin for mitral valve disease at the time of Doppler study. Stroke etiology according to the TOAST criteria was uncertain in 16 patients, of whom 2 had coincident carotid and cardiac disease.

Emboli signals were present in 29 of the 41 patients in whom at least one MCA was identified (71%); these signals were within the affected (symptomatic) cerebral hemisphere in 5 patients, bilateral in 22 patients, and contralateral only in 2 patients. No emboli signals were detected in either hemisphere in 8 patients with lacunar stroke. The overall difference in emboli signal counts between etiologic subgroups was significant ($P=.001$, Kruskal-Wallis). In the group with large artery atherosclerosis, the emboli signal count was significantly higher within affected cerebral hemispheres than in contralateral (unaffected) hemispheres (11.3 versus 1, median [95% confidence interval, 3 to 40 and 0 to 3, respectively], $P=.02$, Kruskal-Wallis, multiple comparisons with Bonferroni correction). Interhemispheric differences were not significantly different in any of the other patient groups (Table). No patient experienced new symptoms of cerebral ischemia during the TCD examination.

Thirty normal control subjects aged 22 to 38 years, mean 26 years, were examined for a total of 42 hours. No emboli signals were detected in any of these subjects.

Discussion

We have shown a high incidence of emboli signals in consecutive patients presenting with transient ischemic attack and acute stroke, matching the previous experience in selected patient groups with cardiac and carotid embolic sources. Against this background, the absence of emboli signals in patients with lacunar stroke is most significant, and is, to our knowledge, the first report of this finding. This observation lends support to the proposed small-vessel etiology of lacunar stroke, which is rarely due to embolic disease. We might have expected to find some emboli signals arising from coincidental atherosclerotic disease, which is known to occur in this patient group. The absence of these signals may be explained by our use of the TOAST criteria of etiologic classification; this excludes patients who would be classified as lacunar on standard clinical and/or radiological grounds, but who are identified as having a potential embolic source. We nevertheless expect that with increased numbers of lacunar strokes, emboli signals will be identified in a small proportion of patients. An additional possibility is that patients with lacunar stroke have a lower frequency of emboli, so recording for more than 1 hour may be required.

However, the clinical significance of continued microembolization remains to be established; even if this can be shown by proposed neuropsychological and cerebral imaging studies, it may remain difficult to classify embolic sources identified with Doppler studies as "causative" or "coincidental," especially in patients with two potential embolic sources. The clinical significance of Doppler emboli in patients with minor degrees of carotid stenosis also deserves more detailed study.

We found an approximately equal incidence of emboli signals in the symptomatic and asymptomatic hemispheres of patients with a probable cardioembolic source. This is consistent with results in patients with prosthetic cardiac valves, in whom there is no interhemispheric difference in emboli signal counts in either the extracranial or intracranial circulation. The problem of multiple embolic sources may be solved in part by further study using dual-probe recording on the common carotid arteries and MCAs: central (cardiac or aortic) embolic sources produce emboli at both sites, but ICA stenosis produces only intracranial signals. In patients with large artery atherosclerosis, higher signal...
counts would be expected in vessels distal to a stenosed carotid artery, resulting in hemisphere asymmetry, and this was confirmed in the present study. This offers a further potential approach in categorizing stroke patients on the basis of the Doppler emboli signal count distribution. However, this may be confounded by the presence of embolic signals in the cerebral hemisphere opposite from a stenosed carotid artery, which may arise from interhemispheric passage of emboli, as we have found in some patients in this study, or by the presence of an alternative embolic source.

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References


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