Anterior Cerebral Artery Velocity Changes in Disease of the Middle Cerebral Artery Stem

L.M. Brass, MD, D.L. Duterte, MD, and J.P. Mohr, MD

Transcranial Doppler ultrasonography can map the changes in blood velocity that result from stenosis or occlusion of the middle cerebral artery. To evaluate patterns of collateral blood flow in disease of the middle cerebral artery stem, we used both cerebral angiography and transcranial Doppler ultrasonography to study the systolic blood velocities in both anterior cerebral arteries in 10 consecutive patients with middle cerebral artery stenosis or occlusion. Five patients had no evidence of hemodynamically significant carotid disease and good-quality measurements of systolic velocity in each anterior cerebral artery. Two of the five patients had middle cerebral artery stem stenosis and the other three had occlusion. The ratios of mean blood velocity in the normal compared with the abnormal side for the five patients (mean 1.34±0.23, range 1.15–1.74) were significantly higher than ratios for 10 controls (mean 1.04±0.12, range 0.76±1.19) using an unpaired *t* test (*t*=3.492, 0.0005 < *p* < 0.005). Our results suggest that transcranial Doppler ultrasound measurements of anterior cerebral artery blood velocity may be a useful index of collateral blood flow from the anterior cerebral artery territory into the middle cerebral artery territory. Changes in mean velocity ratio may document the evolution and adequacy of collateral blood flow over the cerebral convexity in middle cerebral artery stem disease. In addition, the changes in anterior cerebral artery blood velocity appear to be an important corroborative finding for middle cerebral artery stem occlusion. (Stroke 1989;20:1737–1740)

**Subjects and Methods**

We reviewed the TCD examinations performed at Columbia-Presbyterian Medical Center over 6 months and identified 10 patients in whom there was evidence of MCA stenosis or occlusion. From this group, we selected patients who had a technically adequate TCD examination that included both ACA and angiographic confirmation of their MCA stenosis or occlusion with no evidence of stenosis or hypoplasia of the ACA. Five patients met these criteria (three with MCA stem occlusion and two with stenosis). Ten additional patients with normal MCA
velocities on TCD examination were selected to serve as controls.

The device used was the Model TC2-64 (EME, Ueberlingen, F.R.G.). It is a microcomputer-controlled directional Doppler device operating at 2 MHz pulsed ultrasound, using a 64-point fast-Fourier transform spectrum analyzer. The transcranial probe is 22 mm in diameter and uses a focused beam operating at depths of 25–155 mm electronically adjustable in 5-mm steps. The pulse repetition frequency was 5–10 KHz, with a sound intensity range of 10–100 mW/cm², a pulse length of 13 μsec, a gate width of 13 μsec, a vessel wall filter of 150 Hz, and a low-pass filter of 10 kHz. The output is displayed as a time versus Doppler shift plot on a cathode ray terminal. A hard copy of the output was made on an Epson RX-80 dot matrix printer (Torrance, California) using a customized program developed by one of us for the Tandy Model 100 laptop computer (Fort Worth, Texas).

The technique has been described. All studies were performed with the patient in bed, at rest, with the head elevated no more than 30°. TCD recordings were made along both MCAs and both ACAs. The arithmetic mean of the systolic blood velocities in each ACA (MOSV) was calculated for each subject and used in all analyses.

In the patients, ipsilateral MOSV (on the side of the MCA disease) was divided by that of the contralateral (normal) side to calculate the ACA velocity ratio (ACAVR). In the controls, MOSV of the right side was divided by that of the left side to calculate ACAVR. Mean ACAVRs were calculated for each group and compared using the unpaired t test.

Results
A typical TCD study is illustrated in Figure 1. An angiogram and a computed tomogram (CT scan) from one patient are shown in Figures 2 and 3, respectively. The posterior communicating arteries were not reliably identified in 40% of the patients, and these measurements were not analyzed further. The two patients with MCA stenosis had each had 80–90% stenosis with peak velocities of 230 cm/sec (115 cm/sec on the contralateral side) and 160 cm/sec (78 cm/sec on the contralateral side) in the MCA stem.

In the patients (mean age 42 years), ipsilateral MOSVs were significantly higher than contralateral MOSV; therefore, ACAVRs were >1.00 (Table 1). Mean ACAVR for the group was 1.34 (range 1.15–1.74); that is, the blood velocities in the ACA on the side of the MCA disease were 35% higher than those of the contralateral side. In the control group (mean age 53 years), mean ACAVR was 1.04 (range 0.76–1.19); the groups were significantly different (t=3.492, p<0.005).

Discussion
An increase in ACA blood velocity with MCA disease has been noted. Mattle et al reported the results of TCD examination on 61 patients with a clinical diagnosis of MCA ischemia. Only one patient had angiographic demonstration of MCA stem occlusion with a normal ICA; four patients had MCA stem stenosis demonstrated by angiography and a normal ICA, but the degree of stenosis in these four was "less than 75% on average." In addition, the patients with angiographic demonstration of MCA stem disease were not considered separately in their analysis.

In our patients, the increased velocity in the ipsilateral Al segment of the ACA proved to be an important corroborating sign of MCA occlusion or stenosis. Because of technical difficulties in insonating the MCA in some individuals, the determination of MCA occlusion should not be based solely on the absence of a TCD signal. Most often these technical difficulties limit insonation of both the MCA and the ACA. It is conceivable that the MCA might not be identified because of a tortuous course, because it has moved out of the line of the ultrasonic beam, or because of variations in the skull contour that allow the distal Al segment to be insonated, but not the MCA stem. Increased blood velocity in the ipsilateral ACA should help in diagnosing MCA occlusion. The ability to determine MCA occlusion will be important in addressing the natural history of MCA disease. This occlusive state has a poor prognosis, with as few as 20% of patients having a good outcome. MCA occlusion can mimic lacunar syndromes and has a wide range of clinical presentations. Neither the clinical syndromes nor CT scans can reliably diagnose MCA disease, and until now angiography has been the required diagnostic step.
With the promising results of tissue plasminogen activator\textsuperscript{13} and some renewed interest in embolectomy,\textsuperscript{14–16} TCD may prove useful in screening patients with acute cerebral ischemia in the MCA distribution without the risks associated with angiography.

The changes in ACA blood velocity at the time of occlusion, or over time, may reflect the degree of collateral blood flow. The prognosis of patients with MCA occlusion depends on their age, location of the occlusion, and the collateral circulation.\textsuperscript{8} The degree of collateral blood flow also appears to correlate with the clinical outcome in patients with surgical revascularization.\textsuperscript{15} Siato et al\textsuperscript{8} measured collateral blood flow by timing the conduction of contrast material through the ACA into the M2 segment of the MCA. These authors correlated the extent collateral blood flow in the ACA with the size of the infarction. It may be possible to obtain similar information by TCD examination of the ACA blood velocity or pulsatility.

In animal models, it appears that collateral circulation may evolve over the first month after a MCA occlusion.\textsuperscript{17,18} Changes in the ACA blood velocity, and vascular reactivity, over time could provide

\textbf{TABLE 1. Anterior Cerebral Artery Blood Velocity Ratios by Transcranial Doppler Ultrasonography}

<table>
<thead>
<tr>
<th>Subject</th>
<th>Patients</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.74</td>
<td>1.05</td>
</tr>
<tr>
<td>2</td>
<td>1.15</td>
<td>0.94</td>
</tr>
<tr>
<td>3</td>
<td>1.22</td>
<td>1.11</td>
</tr>
<tr>
<td>4</td>
<td>1.27</td>
<td>1.00</td>
</tr>
<tr>
<td>5</td>
<td>1.33</td>
<td>1.15</td>
</tr>
<tr>
<td>6</td>
<td>1.02</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>1.07</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>1.08</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>0.76</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>1.19</td>
<td></td>
</tr>
</tbody>
</table>

Mean±SEM 1.34±0.23* 1.04±0.12

Data are ratio of mean systolic blood velocity in contralateral divided by that in ipsilateral side for patients, right divided by that in left side for controls. Blood velocity increases in proximal anterior cerebral artery ipsilateral to diseased middle cerebral artery.

*p<0.005 different from control by unpaired t test.
could conceivably occur in other settings, such as a
of almost 20%) occurred in one of our controls and
TCD examination. An ACAVR of 1.19 (asymmetry
clinical information and the technical quality of the
MCA disease), and the finding of ACA blood veloc-
be affected by other factors (such as the duration of
MCA disease and may prove useful in identifying
rent underway to investigate the relation between
Fisher CM: The circle of Willis: Anatomic variations. Vasc
Radiodensity in middle cerebral artery is
sion in patients before the CT scan becomes positive.
The changes in ACA blood velocity provide impor-
tant confirmatory evidence in cases of MCA occlu-
sion. These same changes may also prove to be a
useful index of collateral circulation from the ACA
territory and may help predict the extent of infarc-
tion in patients before the CT scan becomes positive.

References
1. Aaslid R, Markwalder TM, Nornes H: Noninvasive trans-
cranial Doppler ultrasound recording of flow velocity in
basal cerebral arteries. J Neurosurg 1982;57:569–574
2. Kirkham FJ, Neville BGR, Levin SD: Bedside diagnosis of
3. Bruno A, Biller J, Silvoti JA: A reason for failure to obtain
transcranial Doppler flow signals: Hyperostosis of the skull
4. Alpers BJ, Berry RG: Circle of Willis in cerebral vascular
The tPA-Acute Stroke Study Group: An open multicenter
study of the safety and efficacy of various doses of r-tPA in
symptomatic middle cerebral artery occlusion. J Neurosurg
1987;65:326–330
10. Adams HP, Damasio HC, Putman SF, Damasio AR: Middle
cerebral artery occlusion as a cause of isolated subcortical
infarction. Stroke 1983;44:948–950
Symptomatic middle cerebral artery stenosis. Ann Neurol
1979;5:152–157
12. Aoki N: Caudate head hemorrhage caused by asymptomatic
occlusion of the middle cerebral artery. Surg Neurol 1987;
27:173–176
study of the safety and efficacy of various doses of r-tPA in
patients with acute stroke: Preliminary results (abstract).
Stroke 1988;19:134
14. Meyer FB, Piepras DG, Sundt TM, Yanagihara T: Emer-
gency embolectomy for acute embolic occlusion of the
15. Meyer FB, Piepras DG, Sundt TM Jr, Yanagihara T:
Emergency embolectomy for acute occlusion of the middle
16. Risberg J, Smith P: Prediction of hemispheric blood flow from
17. Coyle P, Heistad DD: Blood flow through cerebral collateral
vessels one month after middle cerebral artery occlusion.
Stroke 1987;18:463–468
vessels one month after middle cerebral artery occlusion.
Stroke 1985;62:639–647
19. Diaz FG, Ausman JI, Mehta B, Dujovny, De Los Reyes RA,
Pearce J, Patel S: Acute cerebral revascularization. J Neu-
rosurg 1985;63:200–209

KEY WORDS • cerebral arteries • collateral circulation •
ultrasonics

FIGURE 3. Noncontrast computed tomogram of patient in Figure 2. Radiodensity in middle cerebral artery is highly suggestive of thrombosis there.
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