Specific Transcranial Doppler Flow Findings Related to the Presence and Site of Arterial Occlusion

Andrew M. Demchuk, MD, FRCPC; Ioannis Christou, MD; Theodore H. Wein, MD, FRCPC; Robert A. Felberg, MD; Marc Malkoff, MD; James C. Grotta, MD; Andrei V. Alexandrov, MD, RVT

Background and Purpose—Transcranial Doppler (TCD) can localize arterial occlusion in stroke patients. Our aim was to evaluate the frequency of specific TCD flow findings with different sites of arterial occlusion.

Methods—Using a standard insonation protocol, we prospectively evaluated the frequency of specific TCD findings in patients with or without proximal extracranial or intracranial occlusion determined by digital subtraction or MR angiography.

Results—Of 190 consecutive patients studied, angiography showed occlusion in 48 patients. With proximal internal carotid artery (ICA) occlusion, TCD showed abnormal middle cerebral artery (MCA) waveforms (AMCAW) in 66.7%, reversed ophthalmic artery (OA) in 70.6%, anterior cross-filling via anterior communicating artery (ACoA) in 78.6%, posterior communicating artery (PCoA) in 71.4%, and contralateral compensatory velocity increase (CVI) in 84.6% of patients. With distal ICA occlusion, TCD showed AMCAW in 88.9%, OA in 16.7%, ACoA in 50%, PCoA in 60%, and CVI in 88.9% of patients. With MCA occlusion, TCD showed AMCAW in 100%, OA in 23.5%, ACoA in 31.3%, PCoA in 23.1%, and CVI in 62.5%. With no anterior circulation occlusion at angiography, TCD showed these parameters in 1.8% to 17.9%, \( \chi^2 P \# 0.003. \) Parameters localizing anterior circulation occlusion were stenotic terminal ICA velocities 46% versus 10% in patent vessels; flow diversion to perforators 73% versus 1.8%; OA 70.6% versus 5.6%; ACoA 78.6% versus 8.2%; PCoA 71.4% versus 8.5%, all at \( P < 0.05. \) In patients with basilar artery (BA) occlusion, ABAW were found in 80% versus 3% (patent BA); flow diversion to anterior vessels in 60% versus 5.7%; BA flow reversal in 20% versus 0%; and PCoA in 100% versus 13.7%, all at \( P < 0.001. \) No individual parameters differentiated BA from the terminal vertebral occlusion.

Conclusions—Specific TCD findings are common with major arterial occlusion and can be used to broaden diagnostic batteries and improve the predictive value of noninvasive screening in stroke patients. TCD findings useful to localize anterior circulation occlusion include collaterals, abnormal waveforms or velocities, and flow diversion to perforators. (Stroke. 2000;31:140-146.)

Key Words: occlusion ■ stroke ■ ultrasonography, Doppler

The presence and location of arterial occlusion helps to determine likelihood of recanalization with thrombolysis as well as underlying stroke mechanism, secondary stroke prevention options, and prognosis. Transcranial Doppler is a noninvasive method that can be used to identify intracranial and extracranial arterial occlusion. Wilterdink et al described a battery of TCD findings that identified a >70% proximal internal carotid artery (ICA) stenosis or occlusion with sensitivity of 95% and specificity of 42%. The battery included reversed ipsilateral ophthalmic artery (OA), reversed ipsilateral anterior cerebral artery (ACA), elevated flow velocity in the contralateral ACA, absence of flow signal in the ipsilateral OA or carotid siphon, and diminished pulsatility or flow acceleration in the ipsilateral middle cerebral artery (MCA). Others reported TCD flow findings with the middle cerebral and posterior circulation arterial occlusions, including absent or diminished flow signals and flow diversion to branching vessels. However, the data regarding frequency of specific flow findings such as abnormal waveforms, posterior communicating artery (PCoA) flow, compensatory flow increase, or diversion are largely lacking. In our prospective series, TCD had specificity of 94% with sensitivity of 83% to identify the presence of any proximal extracranial or intracranial arterial occlusion compared with angiography. TCD sensitivity for the anterior circulation occlusions exceeded 90% due to acquisition of more physiological data compared with previously described batteries.

In the present study we aimed to evaluate the frequency of specific TCD flow findings in patients with angiographically proved arterial occlusion. The data on frequency of these flow findings in angiographically positive and negative cases will...
aid application of diagnostic criteria for TCD and will help develop a role for TCD in future stroke evaluation and treatment. Our secondary goal was to identify TCD flow findings that could help to localize arterial occlusion.

### Subjects and Methods

We evaluated all bedside TCD studies in patients with symptoms of cerebral ischemia referred between September 1997 and November 1998 by the Stroke Treatment Team to the STAT Neurosonology Service, University of Texas–Houston. Approximately half of the referred patients were evaluated by TCD within 24 hours of symptom onset, with the remainder evaluated >24 hours from symptom onset. A complete transcranial insonation protocol with 2-MHz pulsed-wave Doppler was used as outlined previously. Specific flow parameters were selected on the basis of previous publications indicating their importance for noninvasive vascular diagnosis of arterial occlusion and flow collateralization. During TCD examination we documented the following parameters:

### Anterior Circulation

#### Normal ICA, MCA, or ACA Findings

A low-resistance flow (PI 0.6 to 1.1) with normal flow direction, age-expected mean flow velocities (MFVs) ≤80 cm/s, unilateral velocity ratio MCA≥ACA≥ICA, and normal systolic flow acceleration, ie, arrival of maximum velocity during early systole.

#### Stenotic Velocities

A significant focal MFV increase of >30% compared with a neighboring or homologous arterial segment, and/or MCA MFV ≥80 cm/s, ACA MFV ≥80 cm/s, and ICA MFV ≥70 cm/s in adults.

M1 MCA waveforms were determined from 65- to 45-mm depths as unidirectional signals toward the probe; M2 MCA waveforms were obtained from 45- to 35-mm depths as unidirectional or bidirectional signals.

#### Abnormal ICA, MCA, or ACA Waveforms (Figure 1)

1. **Dampened signal**: Pulsatile flow with normal flow acceleration and decreased MFV (>30% difference between hemispheres); any PI values.
2. **Blunted signal**: Delayed flow acceleration with stepwise maximum velocity arrival during mid to late systole compared with contralateral side and focal decreased MFV and positive end-diastolic flow (low PI≤1.1).
3. **Minimal signal**: Presence of a flow signal with no end diastolic flow; PI≥1.2.
4. **Absent signal**: No detectable flow at 40- to 65-mm depths (toward the probe) via transtemporal window (double-checked with insonation from contralateral window across midline at depths of 80 to 100 mm).

### Findings Suggesting Flow Diversion in the Anterior Circulation

1. **Flow diversion to ACA or posterior cerebral artery (PCA)** (Figure 2): Increased velocities in A1 ACA or P1-P2 PCA segments located at 60- to 74-mm depths (MFV ACA>MCA or PCA>A CA or PCA>MCA by ≥10%).
2. **Flow diversion to perforators**: A low-resistance flow at proximal M1 MCA in the presence of abnormal or absent distal M1 or M2 MCA signals.

### Posterior Circulation

#### Normal Posterior, Basilar, and Vertebral Artery Findings

A low-resistance (PI 0.6 to 1.1) antegrade flow with age-expected MFVs <60 cm/s and velocity ratio basilar artery (BA)≥PCA≥vertebral artery (VA).

#### Stenotic Velocities

A focal significant MFV increase >30% compared with proximal or contralateral arterial segments, and/or PCA MFV≥50 cm/sec, BA MFV≥60 cm/sec, and VA MFV≥50 cm/s in adults.
Abnormal PCA, BA, or VA Waveforms

1. **Dampened signal**: Pulsatile flow with normal flow acceleration and decreased MFV (≥30% difference between proximal and distal arterial segments); any PI values.
2. **Blunted signal**: Arterial flow with delayed flow acceleration and decreased MFV and positive end-diastolic flow (low PI ≤1.1).
3. **Minimal signal**: Presence of a flow signal directed away from the probe with no end-diastolic flow.
4. **Absent signal**: No detectable flow at 58- to 70-mm depths for P1-P2 PCA via transtemporal window, no antegrade flow at 75- to 100-mm depths (BA) and 40- to 75-mm depths (VA) via transforaminal window.

Reversed Basilar Artery Flow

Retrograde low- or high-resistance flow identified in the BA along with abnormal proximal arterial signals indicating terminal VA or proximal BA obstruction.

Findings Suggesting Flow Diversion in the Posterior Circulation

Flow diversion to contralateral VA, PICA, or other cerebellar arteries:

1. Antegrade flow in V4 VA segment located at 40- to 75-mm depths or retrograde flow located at 50 to 70 mm with velocities above age-expected values;
2. Normal or increased velocity in the proximal BA and low-resistance flow in the presence of abnormal distal basilar signals.

Flow Velocity Asymmetry

1. **Velocity increase in the anterior circulation vessels**: Flow velocities above age-expected values, in vessels of both hemispheres.
2. **Velocity increase in the contralateral hemispheric vessels**: Flow velocities above age-expected values, in vessels of the nonaffected side (>20% asymmetry between hemispheres).
3. **Velocity increase in the posterior circulation vessels**: Flow velocities above age-expected values, in the BA and at least 1 VA (BA>MCA or ICA).

Collateral Signals

**OA Reversal (Figure 3)**

1. A low-resistance (PI ≤1.1) unidirectional or bidirectional flow found at 50- to 60-mm depths transorbitally;
2. No velocity acceleration found in the ICA siphon at 60- to 65-mm depths compared with OA FVs;
3. Siphon flow signals directed away from the probe;
4. Delayed systolic flow acceleration in the siphon compared with the contralateral side.

Anterior Cross-Filling (Figure 4)

1. Elevated A1 ACA MFVs on the donor side presenting as ACA>MCA and/or donor ACA MFVs are ≥1.2 times greater than contralateral ACA;
2. Possible stenotic-like flow at depths of 72 to 78 mm directed away from the donor side;
3. A normal or low MFV in A1 ACA of the recipient side with A1 flow reversal.

Figure 4. Anterior cross-filling via ACoA (solid arrow). TCD shows elevated velocities in the contralateral ACA>MCA (upper Doppler spectra) of the donor site (dotted arrow) and elevated velocities at midline depths with reversed unilateral A1 ACA flow direction (lower spectra).

PCoA Flow (Figure 5)

1. A low-resistance flow directed mostly toward the probe and located posterior to ICA bifurcation (consistently detected at depths of 58 to 68 mm);
2. Velocities equal or greater to that of the M1 MCA.

TCD findings were interpreted by an investigator (A.V.A., A.M.D., or J.C.G.), independent of angiographic findings, following the predefined set of flow parameters presented above. Angiographic investigations included digital subtraction (DSA) in 40% of patients and magnetic resonance (MRA) in 60%. These studies were performed when clinically indicated and interpreted by a neuroradiologist without knowledge of specific TCD flow findings. Angiographic results were used as the gold standard to evaluate specific TCD findings for differentiating each site of arterial occlusion.

Results

A total of 190 patients underwent TCD and DSA or MRA. Angiography was performed at median 8 hours later than TCD examination (1 hour, 25th percentile; 27 hours, 75th percentile). Angiography was performed before TCD in 41 cases. However, a standard TCD insonation was performed, and TCD was interpreted independent of angiographic results in each of these cases. TCD failed to completely insonate via the transtemporal window in 28 of 190 patients (14.7%). These patients were excluded from further analysis.

Angiographic occlusion was seen in 48 patients (including 12 patients with multiple sites of occlusion). There were 17 proximal ICA occlusions, 13 distal ICA occlusions, 17 MCA occlusions, 1 ACA occlusion, 1 PCA occlusion, 9 distal VA occlusions, and 5 BA occlusions. TCD findings in ACA and PCA occlusions were not analyzed because of the small numbers of both types of occlusion.

With proximal ICA occlusion, TCD showed abnormal MCA waveforms (AMCAW) in 66.7%, reversed OA in 70.6%, anterior cross-filling via the anterior communicating artery (ACoA) in 78.6%, PCoA in 71.4%, and contralateral compensatory velocity increase (CVI) in 84.6% of patients (Tables 1 to 2). Only 1 of 17 patients with proximal ICA occlusion had no abnormal TCD findings. With distal ICA occlusion, TCD showed AMCAW in 88.9%, OA in 16.7%, ACoA in 50%, PCoA in 60%, and CVI in 88.9% of patients. All 13 patients with distal ICA occlusion had 1 or more abnormal TCD findings. With MCA occlusion, TCD showed AMCAW in 100%, OA in 23.5%, ACoA in 31.3%, PCoA in 23.1%, and CVI in 62.5%. Collaterals were mostly found in patients with multiple occlusion sites (ie, MCA and ICA). All TCD parameters listed above were found with patent anterior vessels in 1.8% to 17.9% (all associations tested separately using a 2x2 matrix χ² analysis, P≤0.003).

Parameters localizing anterior circulation occlusion were as follows: stenotic terminal ICA velocities 46% versus 10% in patent vessels; flow diversion to perforators 73% versus 1.8%; OA 70.6% versus 5.6%; ACoA 78.6% versus 8.2%; and PCoA 71.4% versus 8.5%, all at P<0.05. A positive trend at P<0.1 was found for the parameters abnormal distal M1-M2 MCA waveforms and flow diversion to ACA.

In patients with BA occlusion, ABAW were found in 80% versus 3% (patent BA); flow diversion to anterior vessels in 60% versus 5.7%; BA flow reversal in 20% versus 0%; and PCoA in 100% versus 13.7%, all at P<0.001. Two of 5 patients with BA occlusion had no abnormal TCD findings.
except a slight decrease in the distal BA velocities. These patients had distal BA occlusion. All patients with proximal BA occlusion had 1 or more abnormal TCD findings. TCD findings indicating the presence of a posterior circulation occlusion were abnormal VA or BA waveforms, flow diversion to contralateral VA/PICA or anterior circulation vessels, and BA flow reversal and PCoA flow (all parameters at P < 0.001; Table 3). No individual flow parameters differentiated BA from the terminal VA occlusion. Abnormal BA waveforms and PCoA flow (P < 0.20) were seen more frequently with BA occlusion than VA occlusion; however, because of the small numbers, there was no statistical difference. The presence of BA flow reversal was seen in only 1 patient, yet represented a dramatic sign of proximal basilar occlusion.

Discussion
We prospectively identified the frequency of specific TCD findings helpful to unmask arterial occlusion and to differentiate its level. Our findings are consistent with those in previous reports describing similar pathophysiological findings2–9 and provide further data on the frequency of abnormal TCD flow findings with angiographically proved occlusion compared with patent vessels. Specific TCD flow findings are common with proximal arterial occlusion, and these parameters can be applied for emergent evaluation of acute stroke patients and monitoring therapies.

Our study has limitations before it can be extrapolated to the acute stroke setting. We included patients at any time point from symptom onset, thus explaining the relatively low 25% (48/190) incidence of occlusion. When TCD and angiography had been performed within the first few hours of ischemia, the incidence of occlusion was likely to be much higher.12,13 The incidence of arterial occlusion in rtPA-eligible patients on TCD was 69% with sensitivity of 88% compared with angiography.13 Another limitation of any study of this kind is the variable time between TCD and angiography, yet this is commonplace in clinical practice.

Our data show that abnormal MCA, ICA, or posterior circulation waveforms (not just the velocities) are the key parameters to unmask arterial occlusion. Proximal ICA occlusion commonly produces abnormal waveform at the ICA siphon or terminal ICA. Its effect on MCA waveform will largely depend on collateralization of flow and arrival of

TABLE 1. Abnormal Waveforms and Stenotic Velocities in the Anterior Circulation Occlusion

<table>
<thead>
<tr>
<th>Occlusion Site</th>
<th>Siphon</th>
<th>tICA</th>
<th>pM1</th>
<th>dM1</th>
<th>M2</th>
<th>MCA</th>
<th>pM1</th>
<th>dM1</th>
<th>M2</th>
<th>MCA</th>
</tr>
</thead>
<tbody>
<tr>
<td>MCA</td>
<td>60</td>
<td>66.7</td>
<td>86.7</td>
<td>100*</td>
<td>100*</td>
<td>0</td>
<td>6.7</td>
<td>0</td>
<td>0</td>
<td>0</td>
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<tr>
<td>dICA</td>
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<td>58.3</td>
<td>80</td>
<td>80</td>
<td>88.9</td>
<td>7.7</td>
<td>33.3</td>
<td>10</td>
<td>10</td>
<td>0</td>
</tr>
<tr>
<td>pICA</td>
<td>76.5</td>
<td>53.8</td>
<td>66.7</td>
<td>66.7</td>
<td>66.7</td>
<td>17.6</td>
<td>46.2</td>
<td>8.3</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>None</td>
<td>6.6</td>
<td>4.5</td>
<td>3.6</td>
<td>1.8</td>
<td>2.7</td>
<td>5.8</td>
<td>10</td>
<td>12.5</td>
<td>15</td>
<td>13.6</td>
</tr>
<tr>
<td>P (χ²)</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>NS</td>
<td>0.001</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
</tbody>
</table>

*χ² P < 0.1 proximal ICA vs MCA; †χ² P < 0.05 proximal ICA vs MCA.

Figure 5. Collateralization of flow via PCoA. PCoA flow signals are found at 60- to 70-mm depths with posterior angulation of the probe when switching from ICA bifurcation to PCA. Under normal circulatory and anatomic conditions, this area has no detectable flow signals. PCoA MFV is usually greater than MFV of the recipient artery, where the delayed systolic flow acceleration can also be found. DSA shows PCoA flow from the BA to MCA distribution (arrow).
maximum systolic velocity (flow acceleration). Other findings helpful to indicate arterial occlusion include flow diversion or compensatory velocity increase in branching vessels.

The level of anterior circulation obstruction is confirmed by detection of collateral channels, such as reversed OA or communicating arteries. Occasionally, OA may have no detectable flow or normal flow direction if a distal ICA or MCA occlusion is also present. OA may also have a normal flow direction with proximal ICA occlusion if siphon is filled retrograde via communicating arteries.

Isolated occlusion at the MCA level produces abnormal waveforms without flow collateralization via OA or communicating arteries. If present, these collaterals usually indicate tandem ICA/MCA lesions. TCD detected abnormal MCA waveforms or no signals in all patients with angiographically proved MCA occlusion. The highest sensitivity of TCD with MCA lesions has been established previously. Other factors that help to localize MCA occlusion include flow diversion to the ACA, perforators, or PCA. Tandem MCA/ICA occlusion can be diagnosed by detecting abnormal MCA waveform or asonic MCA segment and major collateral channel, which indicate the presence of a proximal lesion in the feeding vessel.

The presence of abnormal VA or BA waveforms should signal the possibility that occlusion of either of these vessels is present. These findings need to be interpreted cautiously and confirmed by identifying flow diversion or compensatory flow increase. Differential diagnosis should include a flow-limiting stenosis and velocity asymmetry due to changes in the insonation angle. TCD accuracy for posterior circulation lesions is lower than for anterior occlusion, and TCD can be used as an adjunct to other vascular imaging tests in patients with verteobasilar ischemia.

In our study the frequency of specific TCD flow findings in patients with no arterial occlusion was low (0% to 17.9%). Such findings as stenotic velocities and flow velocity increase can be found in 12.5% to 17.9% of patients with no occlusion. These findings are mostly attributable to stenotic lesions or velocity asymmetry and should not be overinterpreted without abnormal waveforms and evidence of flow collateralization. The frequency of abnormal waveforms in patients with no occlusion was 1.8% to 6.6%, which indicates that if TCD findings are normal there is a >90% chance that angiography will show no proximal arterial obstruction. Although MRA had 100% sensitivity and 95% specificity for intracranial occlusion in 1 study, its overall accuracy is limited compared with DSA due to appearance of flow gaps with critical stenoses and difficulties in differentiating slow flow from occlusion. Nevertheless, in clinical practice TCD is often compared with MRA for the presence of these lesions. Our results indicate that an acceptable agreement between these modalities can be achieved.

A better understanding of the specific flow findings is a key step in clinical acceptance of this modality, because these findings can broaden diagnostic batteries for TCD. An experienced TCD user can rapidly identify patients with M1 MCA occlusion with sensitivity and specificity exceeding 90%. With these accuracy parameters, TCD screening can help to reduce the number of negative invasive angiograms. Although MRA had 100% sensitivity and 95% specificity for intracranial occlusion in 1 study, its overall accuracy is limited compared with DSA due to appearance of flow gaps with critical stenoses and difficulties in differentiating slow flow from occlusion. Nevertheless, in clinical practice TCD is often compared with MRA for the presence of these lesions. Our results indicate that an acceptable agreement between these modalities can be achieved.

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In conclusion, specific TCD findings are common with major arterial occlusion and can be used to broaden diagnostic batteries and improve the predictive value of this noninvasive screening tool. TCD findings useful to localize anterior circulation occlusion include collaterals, abnormal waveforms or velocities, and flow diversion to perforators.

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