Detection of Reversed Basilar Flow With Power-Motion Doppler After Acute Occlusion Predicts Favorable Outcome

Marc Ribo, MD; Zsolt Garami, MD; Ken Uchino, MD; Joon Song, MD; Carlos A. Molina, MD; Andrei V. Alexandrov, MD

Background and Purpose—Power-motion transcranial Doppler PMD-TCD is a new method for simultaneous display of flow at multiple depths. We aimed to determine clinical significance of PMD-TCD demonstration of reversed basilar flow in patients with basilar artery (BA) occlusion.

Methods—We prospectively evaluated patients with acute vertebrobasilar ischemia using PMD-TCD. Using a predefined set of TCD depth criteria and specific flow findings, occlusion was localized to the proximal, middle, or distal BA stem. The National Institutes of Health Stroke Scale was used to measure stroke severity and the modified Rankin Scale (mRS) to assess outcome at 3 months.

Results—BA occlusion was diagnosed in 16 patients (3 women, mean age 65, median NIHSS 8, mean time from symptoms to onset 8.5 hours). PMD-TCD diagnosis of BA occlusion was confirmed in 11 of 12 patients who underwent invasive angiography. Reversed BA flow on PMD-TCD was identified in 8 patients (50%). Angiography confirmed flow from carotid system in 6 of these 8 patients (κ=0.87). Patients with reversed BA flow showed lower NIHSS scores on admission (median 4 versus 15.5, \( P=0.009 \)), on discharge (2 versus 21.5, \( P=0.03 \)) and did not experience neurological deterioration during hospital stay (n=0 versus 4, \( P=0.05 \)). There was a trend toward better outcome at 3 months (mRS 1 versus 4, \( P=0.07 \)).

Conclusion—Detection of reversed flow in the distal BA with PMD-TCD is associated with lower stroke severity and better outcome after acute basilar artery occlusion. (Stroke. 2004;35:79-82.)

Key Words: basilar artery ischemia ■ power-motion Doppler ■ stroke ■ ultrasonography, Doppler, transcranial...
80 to 100 mm in the absence of antegrade basilar flow signals. If extracranial ultrasound examination confirmed the absence of any significant stenosis or hypoechoic plaques in carotid arteries, carotid tapping could be performed to confirm anterior circulation flow (Figure 2). Presumed thrombus location and residual flow signals were determined by the presence of abnormal flow signals using the Thrombolysis In Brain Ischemia (TIBI) flow grading system. Complete occlusion was considered when TIBI grades were 0 to 1. Location of occlusion was determined: proximal, mid, and distal.

We obtained a detailed history of vascular risk factors from each patient. Urgent CT angiography, MRI/MR angiography (MRA), or digital subtraction angiography (DSA) was performed shortly after PMD-TCD examination when clinically indicated. DSA with selective injection of the common carotid and vertebral arteries was performed with femoral artery approach. Sites of segmental BA occlusion were classified according to Archer and Horenstein anatomy-based criteria: proximal, from the vertebral artery junction to the origin of the anterior inferior cerebellar arteries; mid, from the origin of the anterior inferior cerebellar arteries to the origin of the superior cerebellar arteries; and distal, at the top of the BA. Reversed flow in BA was identified when injection of contrast in the carotid arteries allowed visualization of collateral flow in the distal BA.

Patients were treated according to standard guidelines with intravenous, intraarterial or combined intravenous/intraarterial tPA; experimental intraarterial interventions were performed when consented patients were eligible according to Institutional Review Board–approved research protocol.

Neurological status was assessed on patient’s arrival and discharge from hospital using the National Institutes of Health Stroke Scale (NIHSS) by a neurologist who was not aware of the purposes of this study. Neurological deterioration or improvement was defined as an increase or decrease of ≥4 points on the NIHSS score. Modified Rankin scale (mRS) was used to assess clinical outcome at 90 days. We defined poor outcome as mRS ≥3 points or death.

Statistical analysis was performed with the use of the software Analyze-it 1.67. Statistical significance for intergroup differences were assessed by the 2-tailed Fisher’s exact test or χ² test for categorical variables and Student’s t test or Mann-Whitney U for continuous variables. A level of P < 0.05 was accepted as statistically significant. Agreement between PMD-TCD and DSA was assessed with Cohen’s κ statistic. Values between 0.4 and 0.8 indicated a moderate agreement; values >0.8 were considered excellent; and perfect value was 1.

Figure 1. Normal flow in basilar artery. Right, image shows ultrasound probe and beam path. Left, PMD-TCD display shows flow in BA away from the probe at all depths from 60 to 110 mm. Right bottom, TCD spectra were obtained from the mid basilar artery (90 mm).

Figure 2. Reversed distal basilar flow in a patient with proximal BA occlusion. Upper image, PMD-TCD display shows red signal in distal BA (94 to 101 mm), indicating reversed flow. Intermittent blue signal in proximal BA (60 to 75 mm) is highly suggestive of vessel occlusion. Lower image, TCD spectra with carotid tapping is performed (arrows), indicating that flow in BA responds to tapping of the anterior circulation vessel.


Results

Of a total of 763 consecutive acute stroke patients who underwent emergent transcranial ultrasound examination, 73 (9.6%) presented with a posterior circulation stroke. Of these 16 (2.1%) had a documented BA occlusion on PMD-TCD (3 women; mean age, 65). Mean time from symptoms onset to ultrasound examination was 8.5 hours and median baseline NIHSS was 8 (range, 0 to 29) points. All TCD-PMD tests were performed and interpreted at bedside within 10 minutes after initiation of ultrasound examination. BA occlusion location was assessed: proximal, 9 patients (56.2%); mid, 4 patients (25%); and distal, 3 patients (18.8%) Of the total 16 patients, 12 underwent DSA, 3 were examined with MRA, and 1 did not receive any additional imaging. In 11 of the 12 patients who underwent DSA, basilar occlusion was confirmed. In the remaining patient who underwent DSA, the study revealed left posterior cerebral artery occlusion without evidence of BA occlusion. In this patient, intravenous tPA was initiated prior to angiography. For the 3 patients who underwent MRA, BA occlusion was also confirmed. Reversed flow in BA was detected in 8 patients with PMD-TCD. Seven patients (87.5%) presented a proximal occlusion, 1 patient (12.5%) a mid occlusion, and none of them presented a distal BA occlusion. No differences could be observed in the retrograde-flow Doppler spectrum according to occlusion location. Six of these patients underwent DSA. In all of them, reversed BA flow from carotid system was confirmed. One of the remaining 2 patients underwent MRA that confirmed the presence of reversed flow in BA. Angiographic imaging did not show reversed BA flow in any of the remaining patients ($\kappa=0.87$).

Patients with reversed basilar flow on PMD-TCD showed lower NIHSS scores on admission (median, 4 versus 16; $P=0.009$) and on discharge (2 versus 22; $P=0.03$) and were less likely to present neurological deterioration (n=0 versus 4; $P=0.05$) or poor outcome (mRS at 3 months, 1 versus 4; $P=0.07$) (Table).

Discussion

Our study showed that PMD-TCD can be used for quick and reliable detection of BA occlusion and flow reversal in its distal portion. This finding indicates collateralization of flow through the posterior communicating arteries and accounts for lower NIHSS scores at baseline. These patients also had better outcomes at 3 months.

Previous studies showed limited feasibility and low sensitivity of TCD in BA occlusion compared with CT angiography. Because of its ability to simultaneously display the power and direction of the blood flow signatures over a wide range of depth, PMD-TCD facilitates location of transcranial window and rapid vessel identification and thus may be useful in detection of distal basilar flow. The screening of a wide range of depths also permits simultaneous display of the flow patterns in proximal and distal BA, showing at the same time the occlusion signatures and the reversal flow (Figure 2).

It seems counterintuitive that a reversed flow distal to occlusion has low resistance. This reversed flow develops because of a pressure gradient. During a cardiac cycle, there is never enough pressure to push flow into a completely occluded vessel. However, if occlusion develops in the proximal basilar, a pressure gradient develops between carotid circulation and posterior cerebral arteries, superior cerebellar arteries, and perforating vessels. If a thrombus or embolus in the proximal BA does not completely occlude basilar all the way immediately, the patient has a chance to recruit posterior communicating arteries and deliver blood from carotids via the reversed basilar stem to parts of the cerebellum and smaller distal basilar branches. This collateral flow reaches the low-resistance system of cerebellar and brain stem parenchyma, and that is why it has good diastolic frequencies and low-resistance signatures on Doppler. Identification of low-resistance flow moving toward the probe, ie, reversed basilar at 80 to 100 mm, thus indicates continuing perfusion of vital brain structures and explains often partial deficits despite the presence of a proximal basilar obstruction.

Previous studies demonstrated that length of basilar occlusion and state of collaterals determined by DSA are independent variables affecting survival. Our study showed that similar information can be obtained rapidly and noninvasively. Identification of reversed flow in distal BA with PMD-TCD was associated with lower baseline stroke severity and better outcome. On the other hand, in patients in whom collateral flow cannot be established after acute BA occlusion, stroke severity and risk of neurological deteriora-

![Table](https://example.com/table.png)

### Demographic Data, Risk Factor Profile, and Baseline Clinical Findings

<table>
<thead>
<tr>
<th>Variable</th>
<th>Reversed Flow (n=8)</th>
<th>Nonreversed Flow (n=8)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex (M)</td>
<td>8 (100)</td>
<td>15 (63)</td>
<td>0.246</td>
</tr>
<tr>
<td>Age, y</td>
<td>63 (12.1)</td>
<td>66 (18.3)</td>
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<td>Hypertension</td>
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<td>Coronary disease</td>
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<tr>
<td>Atrial fibrillation</td>
<td>2 (29)</td>
<td>1 (12)</td>
<td>0.892</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>4 (57)</td>
<td>4 (57)</td>
<td>1</td>
</tr>
<tr>
<td>Obesity</td>
<td>0 (0)</td>
<td>2 (25)</td>
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<tr>
<td>Hyperlipidemia</td>
<td>4 (57)</td>
<td>1 (12)</td>
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<tr>
<td>Onset PMD interval, h</td>
<td>9 (7)</td>
<td>10.3 (11)</td>
<td>0.851</td>
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<tr>
<td>Previous stroke</td>
<td>3 (37)</td>
<td>1 (14)</td>
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<td>Anterior stenosis on PMD-TCD</td>
<td>0 (0)</td>
<td>3 (37)</td>
<td>0.2</td>
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<td>Blood glucose, g/dL</td>
<td>168 (62)</td>
<td>136 (54.1)</td>
<td>0.259</td>
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<td>Systolic blood pressure</td>
<td>175 (46.8)</td>
<td>161 (38.4)</td>
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<td>Diastolic blood pressure</td>
<td>92 (16.2)</td>
<td>78 (10.8)</td>
<td>0.136</td>
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<td>TPA treatment</td>
<td>2 (25)</td>
<td>5 (62.5)</td>
<td>0.43</td>
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<td>Recanalization (on 24-h PMD-TCD)</td>
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<td>6 (75)</td>
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<td>Deterioration</td>
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<td>4 (50)</td>
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<td>Baseline NIHSS</td>
<td>4 (2.5–7.5)</td>
<td>15.5 (8–23)</td>
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<td>Discharge NIHSS</td>
<td>2 (0.5–5)</td>
<td>21.5 (3–28)</td>
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<td>Third-month Rankin</td>
<td>1 (0–3)</td>
<td>4.5 (1–6)</td>
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<tr>
<td>Mortality</td>
<td>1 (12)</td>
<td>3 (37)</td>
<td>0.569</td>
</tr>
</tbody>
</table>

Values are mean (SD), median (interquartile range), or n (%) as appropriate.

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tion are higher. PMD-TCD can demonstrate favorable collateralization of flow, despite persistence of arterial occlusion, through posterior communicating arteries or even cross-cerebellar flow in some other cases. This information can explain heterogeneity of clinical syndromes associated with BA occlusion and may provide some insights as to how patients respond to intraarterial rescue or conservative management. PMD-TCD is intended not to replace other angiographic imaging techniques but to offer a wider diagnostic arsenal available for the physician in the acute management of suspected BA occlusion.

There are some potential limitations related to the application of PMD-TCD. First, although this procedure seems to improve some limitations of conventional TCD in obtaining a proper sonographic window, this technology is still likely to require skilled operators. The presence of other retrograde-flow signatures such as venous vessels or the superior cerebellar arteries could be misinterpreted by an inexperienced technician as a reversed BA flow. The absence of an antegrade arterial flow, the presence of a proximal signal compatible with BA occlusion, and a positive carotid tapping test help to rule out pitfalls. Second, because of the design of this study, real sensitivity of PMD-TCD in detection of BA occlusion compared with DSA cannot be assessed. New prospective studies with different inclusion criteria should be initiated to answer this question. In addition, the capability of this new system to assess very relevant issues in terms of clinical outcome as the length of the retrograde perfused portion or the length of the occlusion should also be tested.

In conclusion, identification of reversed flow in distal BA with PMD-TCD can be accurately performed at bedside, and this finding is associated with lower stroke severity and better outcomes.

Acknowledgments

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References

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