Insonation Method and Diagnostic Flow Signatures for Transcranial Power Motion (M-Mode) Doppler

Andrei V. Alexandrov, MD

Andrew M. Demchuk, MD, FRCPC

W. Scott Burgin, MD

ABSTRACT

Background and purpose. Power motion mode Doppler (PMD) simultaneously displays flow signal intensity and direction over several centimeters of intracranial space. Insonation protocol for PMD and spectral transcranial Doppler (TCD) with typical PMD flow signatures is described in serial patients with acute stroke symptoms examined via conventional windows with a PMD/TCD unit. *Results*. Thirty-five patients were studied within 12 hours after stroke onset (age 64 ± 15 years; 8 received intravenous and 3 intra-arterial thrombolysis). One patient had no temporal window, and 3 patients had suboptimal windows. In 90% of patients, PMD showed more than 1 ipsilateral temporal windows. In 63% of patients (n = 22), PMD simultaneously displayed the entire M1 (65-45 mm) and proximal M2 (45-30 mm) flows, leading to spectral TCD examination of the proximal M2 middle cerebral artery (MCA) in 28 of 35 patients (80%). All patients had sufficient foraminal (depth display = 60-110 mm) and orbital (depth display = 30-80 mm) windows. PMD displayed the entire basilar artery stem (75-100+ mm) in 69% (n = 24) of patients, and the distal basilar flow was detected in all patients by both PMD and TCD. TCD results were normal (12), proximal intracranial stenosis (5), large vessel occlusion (17), and cerebral circulatory arrest (1). Compared to spectral TCD, PMD signatures of similar diagnostic significance were low resistance (vessel identification and recanalization), high resistance (ophthalmic artery identification and distal obstruction), collateral (communicating arteries and leptomeningeal flow), reverberating (circulatory arrest), and branch embolization. *Conclusions*. PMD is a window-finding tool and a guide for spectral TCD gate placement. PMD facilitates flow detection in the M2 branches and the distal basilar artery. PMD can demonstrate recanalization of the entire MCA main stem and proximal branches, increase the yield of embolus detection and procedure monitoring, and facilitate abnormal flow pattern recognition.

Key words: Transcranial Doppler, thrombolysis, stroke, recanalization.

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Transcranial power motion mode Doppler (PMD) was recently invented by Mark Moehring.[1] PMD, or M-mode, uses 33 overlapping Doppler samples to simultaneously display flow signal intensity and direction over 6 cm of intracranial space. PMD provides a color-coded display of all flow signals detectable at a given position and direction of the transducer in real time. The brighter PMD colors reflect stronger intensities, and this "road map" can serve as a guide for proper spectral analysis. PMD promises to make a standard transcranial Doppler (TCD) examination[2,3] easy even for an inexperienced person. Instead of lengthy acquisition of skills to find windows of insonation with a single-channel spectral TCD, a clinician can search for a window of insonation relying less on sound recognition and arm coordination and not be locked in to a single spectrum depth. Furthermore, PMD flow patterns, or signatures, may have their own diagnostic significance, and these flow changes can be observed over large segments of intracranial vasculature in real time. PMD may prove helpful for thrombolysis monitoring and embolus detection by tracking time-space path of high-intensity signals traveling simultaneously to several major intracranial vessels.

Implementation of any ultrasound technology requires a laboratory to establish a standard insonation protocol and locally validate adopted diagnostic criteria.[4] We have previously validated our fast-track insonation protocol for spectral TCD and diagnostic criteria for stroke patients.[5,6] We aimed to evaluate feasibility of PMD-guided TCD studies in acute stroke patients. For the purposes of this study, we developed a standard insonation protocol for PMD combined with spectral TCD. We used a previously

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From the Center for Noninvasive Brain Perfusion Studies, Stroke Treatment Team, University of Texas–Houston Medical School, Houston, TX (AVA, WSB); and the Department of Clinical Neurosciences, University of Calgary, Calgary, Alberta, Canada (AMD).

Address correspondence to Dr Alexandrov, MSB 7.044, 6431 Fannin Street, University of Texas, Houston, TX 77030. E-mail: avalexandrov@att.net.

described spectral TCD technique by Aaslid et al[2,3] and aimed to determine typical flow signatures on PMD in patients with ischemic stroke. This report is a prospective study of PMD-guided TCD in stroke patients.

Subjects and Methods

Serial patients with acute stroke referred to the STAT Neurosonology Service were examined via conventional temporal, foraminal, and orbital windows[2,3] with a portable PMD/spectral TCD unit (TCD 100M, Spencer Technologies, Seattle, WA). A complete PMD/TCD insonation protocol is provided in the appendix. Briefly, using a standard 2 MHz pulsed-wave TCD transducer, a PMD/TCD digital Doppler device simultaneously displays in real time a multidepth power flow map. The examiner chose a single depth to display conventional spectral TCD waveforms (Fig 1). The examiner used a free-hand technique to obtain different PMD views of the flow signals, simultaneously display spectral waveforms, and switch between different depths as with routine TCD insonation. Temporal windows were determined as absent when no flow signals could be obtained after extensive search usually up to 2 minutes. Temporal windows were determined as suboptimal if assessment of the middle cerebral artery (MCA) M1 stem was incomplete (ie, flow signals obtained did not cover the range of 65-45 mm depths) or if no flow signals were detected in the anterior or posterior cerebral arteries on both PMD and TCD. Anterior, middle (preauricular), and posterior temporal windows were determined from standard transducer positions[2,3,6] when flow signals were obtained.

Mean flow velocity (MFV) and pulsatility index (PI) were obtained using a single-channel spectral display at assumed 0° of insonation at the depths previously defined by a standard protocol for spectral TCD insonation of the proximal branches of the circle of Willis.[2,3,5] For example, M1 MCA depths were arbitrarily assigned as 65 to 45 mm, M2 MCA depths as < 45 mm, and distal basilar as 100 mm for an average size adult skull (diameter = 15 cm). Waveforms and MFV/PI were analyzed using previously published and validated TCD criteria.[6] TCD spectral data were interpreted by an experienced sonographer and stroke neurologist with knowledge of PMD findings but without information on subsequent angiography. The spectral TCD results and cerebral angiograms (obtained when clinically indicated) were compared to PMD flow tracks.

Patients presenting within 3 hours of ischemic stroke symptoms were treated with a standard 0.9 mg/kg dose of intravenous tissue plasminogen activator (TPA),[7] and those presenting after the first 3 hours were treated with intra-arterial thrombolysis TPA when TCD in the emergency room showed the presence of a large-vessel intracranial occlusion.[6] This experimental protocol was approved by the University of Texas Committee for Protection of Human Subjects.

Invasive cerebral angiograms were obtained when clinically indicated. Because we have previously established the accuracy parameters of spectral TCD in our laboratory,[6] assessment of these parameters for PMD was not a goal in this study. Although patients with abnormal TCD were more likely to undergo angiography, our recent analysis indicates that TCD in the emergency room can have a 100% sensitivity and specificity of 90%+ for intracranial occlusion (Lee SY et al, unpublished data), and these parameters can minimize associated biases in this study. We performed an observational study and qualitatively compared PMD flow findings to spectral TCD. When obtained, the angiograms were interpreted by a neuroradiologist without knowledge of PMD/TCD results. PMD/TCD were performed and interpreted by experienced sonographers prior to angiography in these cases. Representative angiograms are included in Figures 1-10 to illustrate PMD findings. The goal of this study was to show the feasibility of PMD use in patients with ischemic stroke. We also aimed to prospectively compare flow information obtained by PMD and single-gate spectral TCD to describe typical PMD flow signatures and to present their interpretation and differential diagnosis for future users.

Results

A standard insonation protocol was applied in 35 acute stroke patients (age 64 ± 15 years, median = 66 years). All patients had a baseline PMD/TCD examination within the first 12 hours after stroke onset, repeated later in 5 patients when TCD follow-up was clinically indicated. The National Institutes of Health Stroke Scale (NIHSS) scores obtained in the emergency room ranged from 2 to 25 points. Eight patients were treated with a standard 0.9 mg/kg dose of TPA, and 3 patients underwent intra-arterial thrombolysis with TPA.

The PMD/TCD examination was performed by an experienced sonographer without contrast enhancement in all patients. One patient had no temporal window on both PMD and TCD, and 3 had suboptimal windows. These patients were older than 70 years. All patients had sufficient foraminal and orbital windows, and all segments prespecified by a complete insonation protocol were found.

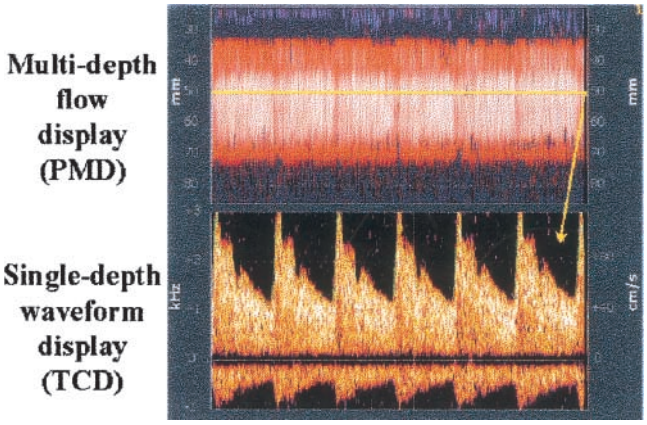

Fig 1. Simultaneous power motion mode Doppler/transcranial Doppler (PMD/TCD) display. The yellow line indicates depth selection for TCD display controlled by an operator. The PMD display shows depth of insonation in mm, and the TCD display shows frequency/velocity in kHz and cm/s.

Using the transtemporal window, PMD detected flow signals along the MCA course including M1 and proximal M2 segments at the depth range of 30 to 70 mm (Fig 2). In 90% of patients, PMD showed more than 1 ipsilateral temporal window: 22 patients, or 63%, had both middle and anterior temporal windows, and 18 patients, or 51%, had the posterior temporal window in addition to either anterior or middle temporal windows. In 63% of patients (22/35), PMD simultaneously displayed the entire M1 and proximal M2 subdivision flows. On PMD, the M2 flow was arbitrarily considered present when flow signals were obtained at 45- to 30-mm depths. PMD-guided spectral TCD of the proximal M2 MCA (depth $<$ 45 mm) was possible in 28 of 35 (80%) patients. PMD displayed low-resistance flow along the entire basilar artery (BA) stem depths of 75 to 100+ mm in 24 of 35 (69%) patients (Fig 3). A sequential PMD assessment of the proximal, mid, and distal BA flows (as directed away

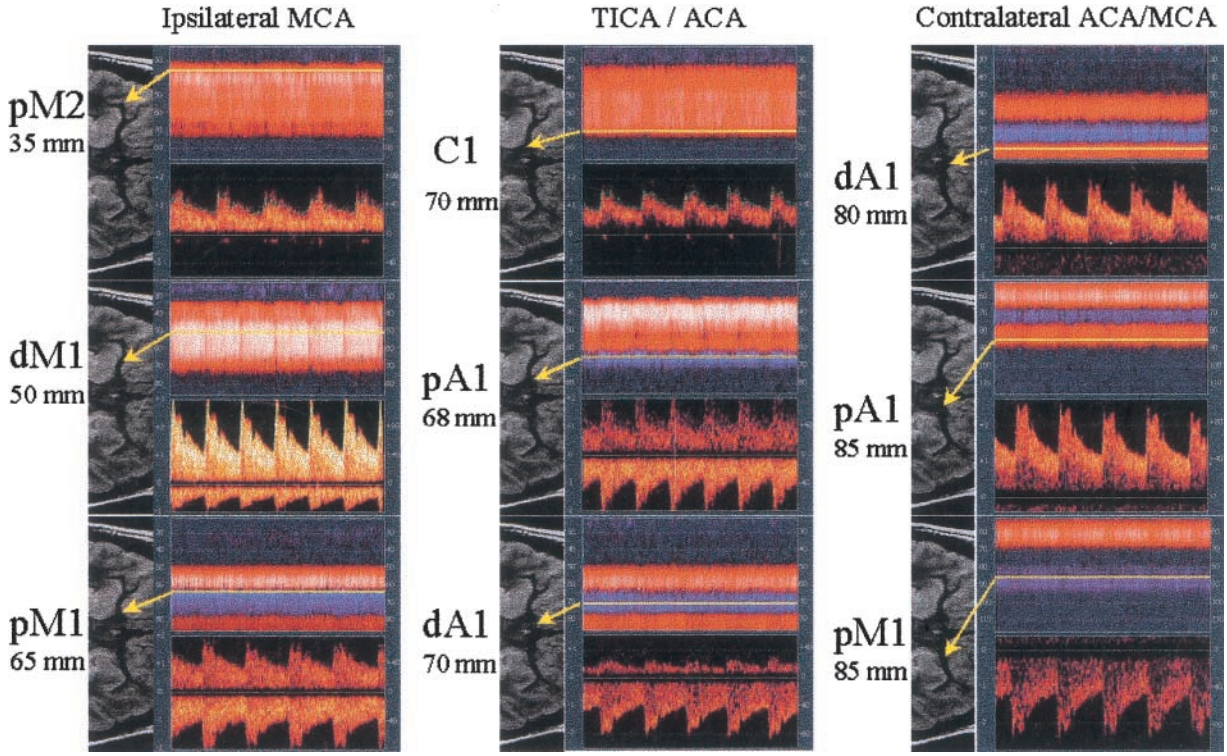

Fig 2. Normal examination of the anterior circulation vessels with power motion mode Doppler/transcranial Doppler (PMD/TCD). Nine simultaneous PMD flow track and single-gate TCD displays illustrate findings through the temporal window. Topography of the circle of Willis is shown on a magnetic resonance image that also illustrates presumable ultrasound beam path. Arrows indicate the origin of the TCD waveforms. The yellow line superimposed over the PMD color display indicates where in relationship to the depth of insonation and PMD flow tracks TCD spectral information was obtained. TCD waveforms were documented with a 9-mm sample volume. pM2 = proximal M2 middle cerebral artery (MCA) flow signal at 35 mm; dM1 = distal M1 MCA flow signal at 50 mm; pM1 = proximal M1 MCA flow signal, or internal carotid artery bifurcation (bidirectional signal), at 65 mm; C1 = terminal internal carotid artery (TICA) signal at 70 mm; pA1 = proximal A1 anterior cerebral artery (ACA) signal at 65 mm; dA1 = distal A1 ACA signal at 70 mm. Contralateral ACA/MCA was as follows: distal A1 ACA signals were located at 80 mm, proximal A1 ACA signals were located at 85 mm, and proximal M1 MCA signals were located at 85 mm.

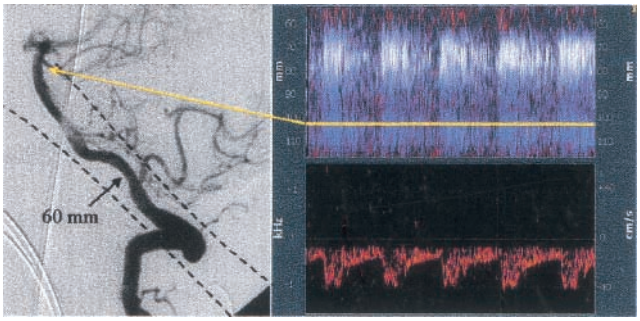

Fig 3. Transforaminal power motion mode Doppler/transcranial Doppler (PMD/TCD) examination. The dotted lines indicate the presumable ultrasound beam path; the depth scale value starts with 60 mm for PMD display. TCD spectra were obtained from the distal basilar artery (102 mm).

from the probe at 80, 90, and 100 mm depths) was possible in all patients.

TCD spectral data were interpreted as normal in 12 patients. In these patients, the M1 MCA MFVs were up to 2 times as high as the ipsilateral terminal ICA and M2 MCA MFVs when spectral analysis was performed at a steady transducer position and assumed 0° of insonation guided by PMD display (Fig 4). In this particular example, the proximal and mid M1 MCA segments had mean flow velocities up to 75 cm/s, whereas the terminal ICA and the M2 MCA segments had velocities as low as 40 cm/s (Fig 4).

TCD spectral data were interpreted as proximal intracranial stenoses in 5 patients and large-vessel occlusions in 17 patients. All patients treated with thrombolytics had an intracranial occlusion before TPA bolus diagnosed by TCD, and the presence of occlusion was confirmed in 3 cases when angiography was performed within 1 hour after TCD. Recanalization was detected in 4 patients treated with intravenous TPA and in both patients during intra-arterial thrombolysis. Compared to spectral TCD, PMD flow signatures of diagnostic significance were as follows:

1. *Normal flow.* A low-resistance flow signature, which shows a continuous flow signal during systolic and diastolic phases that can be used for vessel identification (Figs 2-4) and recanalization (Fig 5).
2. *Arterial occlusion.* A high-resistance signature showing absent diastolic flow. This pattern can also be used in vessel identification (normal ophthalmic artery flow or near 90° insonation of a branching vessel) as well as occlusion localization (Figs 5 and 6, middle frame). In the latter case, this flow track displays a Thrombolysis in Brain Ischemia (TIBI) grade I minimal flow signal and shows the extent of the abnormal residual flow in the MCA stem from 46 to 62 mm.
3. *Emboli.* A branch embolization signature, which shows embolic signatures traveling away from the direction of the main flow stream at depths corresponding to vessel branching (Fig 7). This case also illustrates PMD application for monitoring of intra-arterial thrombolysis when

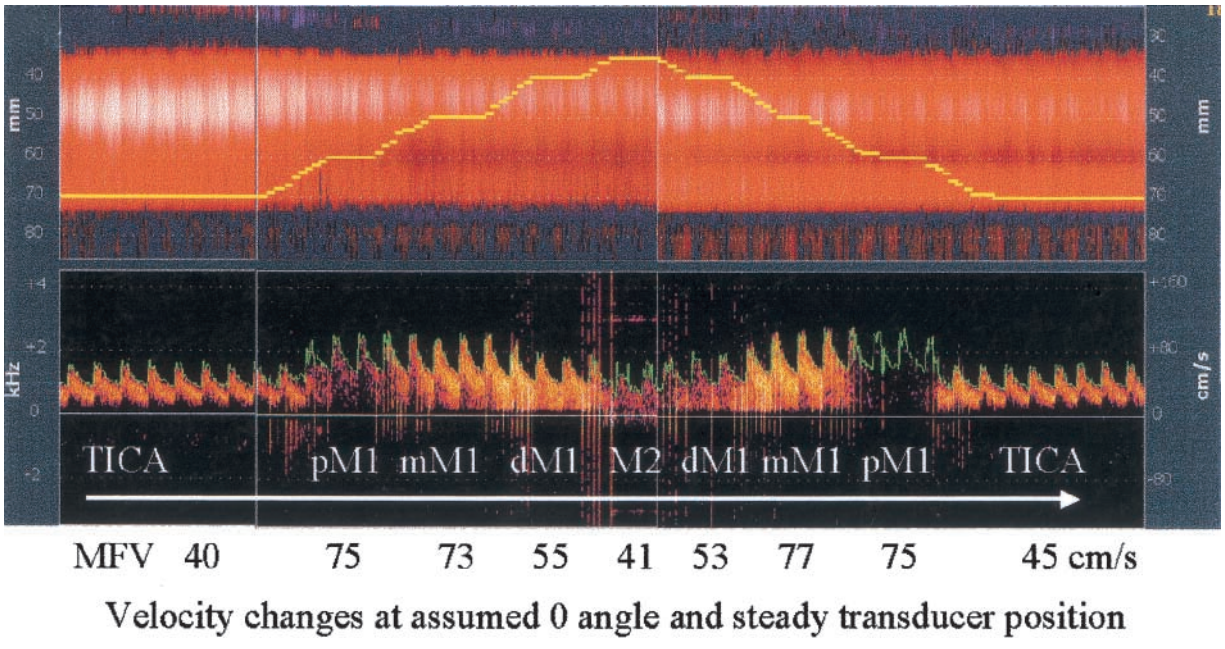

Fig 4. Power motion mode Doppler–guided transcranial Doppler (TCD) velocity measurements. TCD spectra were obtained at an assumed 0° of insonation at a steady transtemporal position of the transducer. The velocity changes in the same plane of insonation help one appreciate the tortuous course of the middle cerebral and other arteries. MFV = mean flow velocity, TICA = terminal internal carotid artery, PM1 = proximal M1, mM1 = mid-M1 segment, dM1 = distal M1 middle cerebral artery segment.

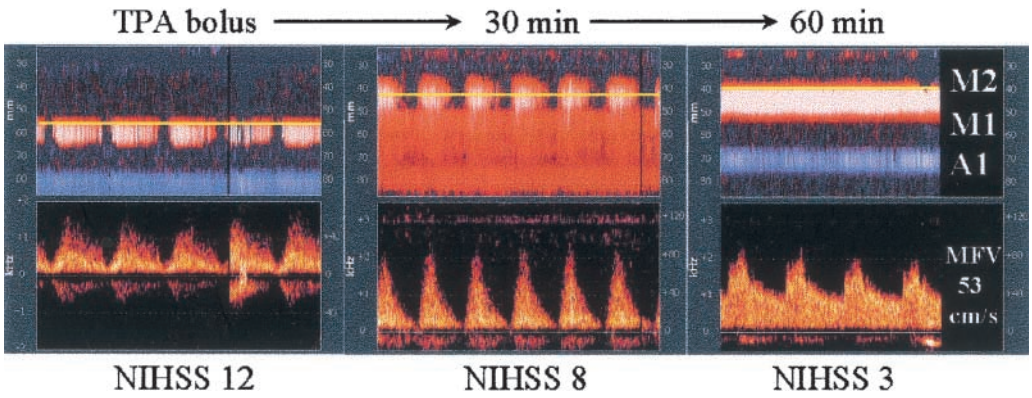

Fig 5. Middle cerebral artery (MCA) recanalization on power motion mode Doppler/transcranial Doppler (PMD/TCD). A 62-year-old man with a National Institutes of Health Stroke Scale (NIHSS) score of 12 points was treated intravenously with 0.9 mg/kg tissue plasminogen activator (TPA) at 112 minutes after stroke onset. At the time of TPA bolus, PMD showed a high-resistance flow track in the mid M1 MCA and detectable flow in the contralateral MCA, indicating a good transtemporal window of insonation. Spectral TCD showed a dampened Thrombolysis in Brain Ischemia (TIBI) grade III flow. At 30 minutes, PMD showed a recanalization process with a low-resistance flow track in the proximal and mid MCA and a high-resistance track in the distal M1 proximal M2 MCA. TCD spectral display showed improvement of the TIBI flow grade from dampened to a high-resistance stenotic signal. Microemboli can be seen on both PMD and TCD (arrows). At 60 minutes, PMD and TCD show low-resistance flows (TIBI grade V) in the distal M1-M2 subdivision, indicating completion of the MCA recanalization. The NIHSS score decreased to 3 points within minutes after recanalization. MFV = mean flow velocity.

information about flow intensity and vessel embolization can be obtained from the entire MCA stem.

4. *Collaterals.* A collateral flow signature. This pattern shows a reversal in the direction of flow in a collateral channel (ie, the anterior communicating artery [Fig 8] and filling of the proximal M2 MCA branches via leptomeningial collaterals in the presence of the terminal ICA occlusion documented by angiography). Differential diagnosis includes detection of venous flow signals.
5. *Circulatory arrest.* A reverberating flow signature. This pattern was found in 1 patient at follow-up PMD/TCD examination. It demonstrates changing flow direction between systolic and diastolic phases and indicates cerebral circulatory arrest (Fig 9). The left spectrum indicates the presence of reverberating (or oscillating) flow signature at a bifurcation leading to erroneous impression of a positive diastolic flow (note bidirectional systolic peaks and oscillations in opposite directions).

In patients with intracranial stenoses ≥ 50% by our spectral TCD criteria (n = 5),[8] PMD showed a flow signature consistent with a bruit (Fig 10). Bruits may be found in a vessel without stenosis, and we did not consider this signature to have any diagnostic significance without spectral measurements. This flow signature appears due to a low-frequency bidirectional turbulent flow during systoli, and PMD subtracts these signals from the display. This flow signature may help spectral interrogation because it topographically maps the location and extent of turbulence (Fig 10).

Discussion

Our study has shown feasibility of PMD use in patients with ischemic stroke. PMD has a potential to increase the yield of diagnostic TCD by showing more windows and more flow signals at depths and locations that may not necessarily be tested during a single standard TCD examination. Also, simultaneous display by PMD of the flow signals over long and tortuous arterial segments led to an observation that the flow velocities may vary up to 100% between the terminal internal carotid artery, M1 MCA, and M2 MCA in the same scanning plane with an assumed 0° of Doppler shift calculation. This finding is in agreement with previous observations with standard TCD[2,3] and is attributable to significant changes in the spa-

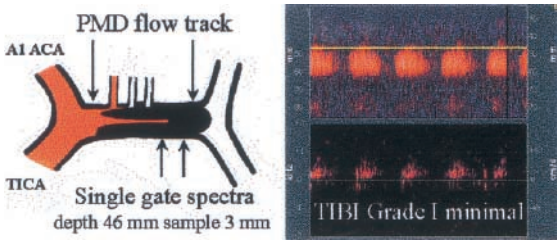

Fig 6. Middle cerebral artery (MCA) high-resistance flow signature and the residual flow signals at the clot location. An 80-year-old man had a total National Institutes of Health Stroke Scale score of 15 and confirmed proximal M1 MCA occlusion at digital subtraction angiography (see Fig 7). Power motion mode Doppler (PMD) shows a high-resistance MCA flow track with absent end-diastolic flow. A single-gate transcranial Doppler spectra shows a Thrombolysis in Brain Ischemia (TIBI) minimal flow signal at a depth of 46 mm. PMD flow tracks also indicate that this flow pattern is present through the M1 stem at depths of 45 to 62 mm. ACA = anterior cerebral artery, TICA = terminal internal carotid artery.

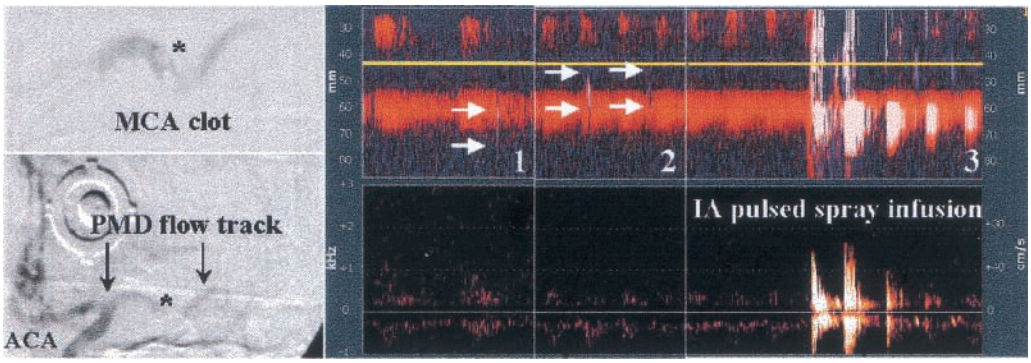

Fig 7. Branch embolization flow signature. An 80-year-old man with a total National Institutes of Health Stroke Scale score of 15 had an elongated clot in the M1 middle cerebral artery (MCA) stem (left panel). An asterisk shows the depth of a single-gate transcranial Doppler (TCD) spectral monitoring of the worst residual flow signals at the site of maximum occlusion (gate = 9 mm). Power motion mode Doppler (PMD) shows a proximal MCA flow and embolic signals traveling in the opposite direction to the anterior cerebral artery (ACA) (60- to 72-mm depths; frame 1) and perforators (48- to 60-mm depths; frame 2). When an intra-arterial pulsed-spray infusion was initiated (frame 3), multiple embolic signals were seen, with PMD displaying more signals than a single-gate spectral TCD. Also, spectral overload from the pulsed-spray injections creates saturation artifacts on PMD, decreasing its appearance (vertical length and brightness) after the first 2 infusion cycles. IA = intra-arterial.

tial course of the arteries and actual angles of insonation. PMD, therefore, may help TCD users to better appreciate the angle changes and potentially reduce errors in velocity comparisons between homologous arterial segments.

Our study has limitations, as this is a pilot clinical investigation of the PMD/TCD method. For instance, the question of whether PMD can help reduce the number of patients with no temporal windows on TCD cannot be answered from the sample studied. However, our findings that most patients with temporal windows had more than 1 window to locate flow are promising. As our and other centers' experience grows, more data will become available to determine whether PMD application expands the yield of TCD. Because we studied a relatively small number of patients and PMD comparison was limited to spectral TCD information and only a few angiograms, a prospective validation and determination of accuracy parameters for PMD-guided spectral TCD examination should be a goal of future studies.

In our initial experience, PMD changed the window-finding strategy. Instead of relying on a single-gate spectral analysis to produce sound from a preselected depth,

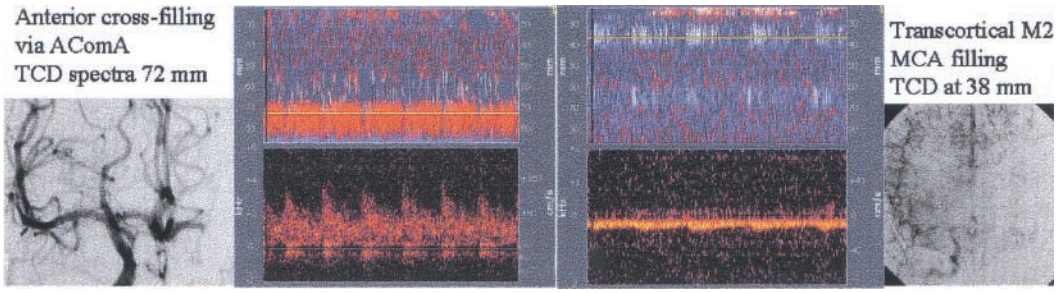

Fig 8. Collateral flow signature. A 17-year-old woman with a cardioembolic left terminal internal carotid artery (ICA) occlusion and a National Institutes of Health Stroke Scale (NIHSS) score of 14 points was brought to the hospital 18 hours after symptom onset. Her power motion mode Doppler/transcranial Doppler (PMD/TCD) showed a reversed left A1 anterior cerebral artery, indicating anterior cross-filling (left panel), and a retrograde blunted flow signal at the depths of the M2 middle cerebral artery (MCA) subdivision, suggesting transcortical flow collateralization (right panel). These findings were confirmed at digital subtraction angiography performed within 1 hour of PMD/TCD. Note that PMD also displays a weak high-intensity signal at the depths of ICA bifurcation (left panel) and reversed flow at this location on the right panel. These flow signatures may represent stenotic flow signals and partial retrograde filling of the terminal ICA that together with transcortical collateral flow explains the relatively low NIHSS scores in the presence of a terminal ICA occlusion.

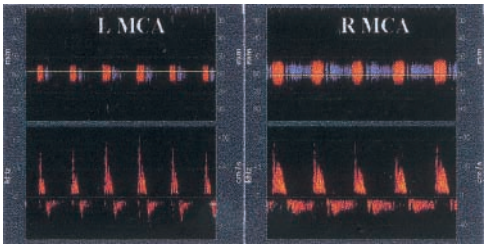

Fig 9. Reverberating flow signature. Both middle cerebral arteries (MCAs) have reverberating flow patterns on both power motion mode Doppler (PMD) and transcranial Doppler (TCD). PMD displays the strongest flow signals (red-to-blue), whereas spectral TCD shows an oscillating flow with a 9-mm gate. The patient had a large right MCA stroke, and 4 days after onset of symptoms had clinical signs of brain death due to supratentorial mass effect and herniation.

the examiners easily found several flow signals at multiple depths and, with less concern of losing the signal, continued to explore the region over the temporal bone to find the most optimal position. At first, this may create an impression of lengthening TCD examination. The benefit, however, is finding a better window for a more complete spectral TCD assessment with better appreciation of flow location. This raises an intriguing possibility that PMD may be the first technology to make TCD easy to use for clinicians, particularly in the emergency situations or at bedside, and to help develop operator-independent TCD devices for noninvasive diagnosis and monitoring.

We described several diagnostic PMD flow signatures that can be helpful for "at a glance" vessel identification, diagnosis of occlusion, better visualization of the process of embolization, and overall interpretation of TCD findings due to simultaneous assessment of flow through large arterial segments and branches. The first and most common finding of a low-resistance flow signature can be used for vessel identification. In 80% of our adult patients, the M2 MCA flow was located by PMD at depths of 30 to 40 mm. It is possible that PMD may improve diagnostic value of TCD in the detection of the M2 lesions, often regarded as unassessable by a simple spectral method.[9] Hennerici and Neuerburg-Heusler[9] stated that changes in the ascending arteries such as M2 MCA can be detected only if color flow imaging and spectrum analysis are used. Although PMD cannot substitute for a color-coded duplex examination, our experience suggests that PMD can serve as a road map for spectral analysis to evaluate flow in the proximal M2 segments.

Another PMD flow signature that can be useful in vessel identification and diagnosis of an abnormal flow is a high-resistance flow signature. With suppression of the end-diastolic frequencies in a high-resistance vascular bed (ie, the ophthalmic artery or preocclusion), PMD displays mostly the systolic flow signals. This pattern is easy to recognize visually. However, the differential diagnosis should include a suboptimal angle of insonation that leads to underestimation of flow velocity, particularly of its diastolic component, and can produce this PMD signature in a normal vessel. The arterial segments particularly prone to this artifact are the vertebral arteries and M2 MCA. Proper transducer alignment and spectral analysis help to avoid misinterpretation. PMD does not substitute for spectral TCD just as color flow imaging does not replace angle-corrected spectral analysis in duplex scanners.[10] Spectral waveform analysis and velocity measurements

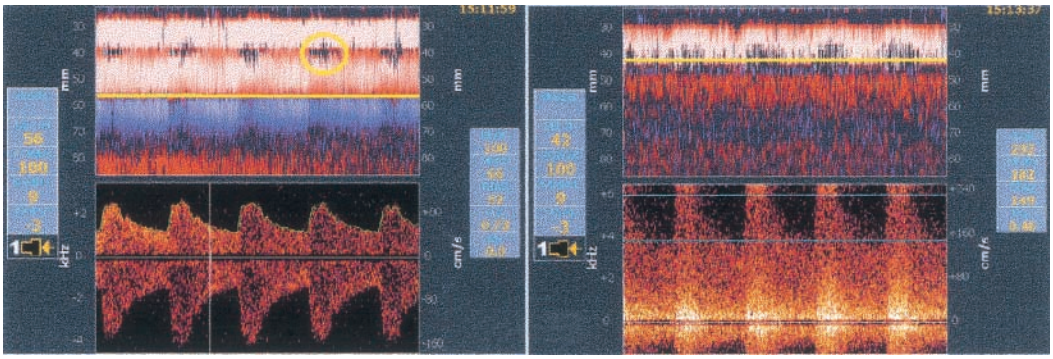

Fig 10. Intracranial stenosis on power motion mode Doppler (PMD) and transcranial Doppler (TCD). A 64-year-old woman with a right middle cerebral artery (MCA) stroke (National Institutes of Health Stroke Scale = 9) was admitted 10 hours after the onset of symptoms. Spectral TCD showed the mean flow velocity difference anterior cerebral artery (ACA) > MCA (left panel), indicating flow diversion to the right ACA. PMD simultaneously showed the presence of a bruit (yellow circle) at a depth of 40 mm. The transducer was repositioned over the temporal window to obtain the highest velocity signal at this location (right panel). The mean flow velocities were 66 cm/s for the proximal right MCA and 182 cm/s for the distal M1/proximal M2 segments of the right MCA, indicating severe intracranial stenosis.

should remain the mainstay of intracranial TCD examination.

After the diagnosis of an arterial occlusion is confirmed by spectral analysis,[11] PMD may help a clinician to appreciate the location or arterial occlusion and extent of the residual flow signals around the clot. The residual flow signals determined by TCD have diagnostic and prognostic significance for an acute stroke patient.[11–13] Future studies will determine whether PMD helps to advance our knowledge about the process of arterial recanalization with thrombolytic therapy and to better study the relationship between the TIBI flow grades[11] and stroke recovery. PMD can detect collateral flow in the major communicating arteries and has the potential to assess the presence of M2 subdivision flow delivered by the leptomeningeal collaterals. Previously, only indirect TCD findings of flow diversion at the MCA/anterior cerebral artery division were considered an indicator of the leptomeningeal or transcortical flow collateralization.[12,13] PMD/TCD has the potential to directly detect transcortical flow collateralization with retrograde filling of the MCA branches. This information can be obtained with a portable unit that does not use transcranial duplex technology. However, any flow detected by PMD has to be thoroughly evaluated by spectral TCD because the waveform analysis and breath-holding maneuvers will help differentiation with venous flow.

PMD can detect emboli and track their spatial course[1] and may visualize branch embolization and help to localize the recipient vessel, as the embolic signatures are depth specific. During intra-arterial thrombolysis, PMD can simultaneously detect emboli and show flow changes around and distal to the catheter as well as recanalization with drug infusion. This real-time information may reduce the number of diagnostic infusions of contrast and help select catheter position for drug infusion that allows maximum residual flow to and around the clot.

The reverberating PMD flow track corresponds to a similar spectral pattern previously documented with spectral TCD.[14,15] An advantage that PMD may offer in this clinical situation is visualization of any flow signals besides the MCA, particularly positive diastolic flow tracks that would necessitate spectral assessment. PMD may also help to detect better windows of insonation that may be difficult to find in patients with suspected brain death if flow is markedly reduced.

The role of TCD has been rapidly evolving from a simple screening tool to a diagnostic modality with a broad spectrum of clinical applications and direct impact on patient management.[16] PMD is the newest addition to our TCD arsenal. In our experience, PMD can be used first as a window-finding tool, then as a guide for arterial segment selection for spectral analysis with TCD. PMD can broaden the range of spectral TCD studies by facilitating flow detection in the M2 branches and the distal basilar artery, increase the yield of embolus detection and periprocedural monitoring, and facilitate abnormal flow signature recognition. PMD obtained in addition to spectral TCD may become an investigational and diagnostic tool for studies of cerebral hemodynamics.

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References

1. Moehring MA, Spencer MP. Power M-mode transcranial Doppler ultrasound and simultaneous single gate spectrogram. *Ultrasound Med Biol* 2002;28:49–57.
2. Aaslid R, Markwalder TM, Nornes H. Noninvasive transcranial Doppler ultrasound recording of flow velocity in basal cerebral arteries. *J Neurosurg* 1982;57:769–774.
3. Otis SM, Ringelstein EB. The transcranial Doppler examination: principles and applications of transcranial Doppler sonography. In: Tegeler CH, Babikian VL, Gomez CR, eds. *Neurosonology*. St Louis, MO: Mosby; 1996:140–155.
4. Katanick SL. Accreditation of vascular ultrasound laboratories. In: Tegeler CH, Babikian VL, Gomez CR, eds. *Neurosonology*. St Louis, MO: Mosby; 1996:484–488.
5. Alexandrov AV, Demchuk AM, Wein TH, Grotta JC. The yield of transcranial Doppler in acute cerebral ischemia. *Stroke* 1999;30:1604–1609.
6. Demchuk AM, Christou I, Wein TH, Felberg RA, Malkoff M, Grotta JC, Alexandrov AV. Accuracy and criteria for localizing arterial occlusion with transcranial Doppler. *J Neuroimaging* 2000;10:1–12.
7. National Institutes of Neurological Disorders and Stroke rt-PA Stroke Study Group. Tissue plasminogen activator for acute ischemic stroke. *N Engl J Med* 1995;333:1581–1587.
8. Felberg RA, Christou I, Demchuk AM, Malkoff M, Alexandrov AV. Screening for intracranial stenosis with transcranial Doppler: the accuracy of mean flow velocity thresholds. *J Neuroimaging* 2002;12:9–14.
9. Hennerici M, Neuerburg-Heusler D. *Vascular Diagnosis With Ultrasound*. Stuttgart, Germany: Thieme; 1998.
10. Polak JF. Color flow imaging of the carotid arteries. In: Tegeler CH, Babikian VL, Gomez CR, eds. *Neurosonology*. St Louis, MO: Mosby; 1996:68–82.
11. Demchuk AM, Burgin WS, Christou I, et al. Thrombolysis in Brain Ischemia (TIBI) TCD flow grades predict clinical severity, early recovery and mortality in intravenous TPA treated patients. *Stroke* 2001;32:89–93.
12. Ringelstein EB, Biniek R, Weiller C, Ammeling B, Nolte PN, Thron A. Type and extent of hemispheric brain infarctions and clinical outcome in early and delayed mid-

dle cerebral artery recanalization. *Neurology* 1992;42:289–298.
13. Kaps M, Teschendorf U, Dorndorf W. Haemodynamic studies in early stroke. *J Neurol* 1992;239:138–142.
14. Petty GW, Mohr JP, Pedley TA, et al. The role of transcranial Doppler in confirming brain death: sensitivity, specificity, and suggestions for performance and interpretation. *Neurology* 1990;40:300–303.
15. Ducrocq X, Hassler W, Moritake K, et al. Consensus opinion on diagnosis of cerebral circulatory arrest using Doppler-sonography: Task Force Group on Cerebral Death of the Neurosonology Research Group of the World Federation of Neurology. *J Neurol Sci* 1998;159:145–150.
16. Babikian VL, Feldmann E, Wechsler LR, et al. Transcranial Doppler ultrasonography: year 2000 update. *J Neuroimaging* 2000;10:101–115.

Appendix

A standard power motion mode Doppler (PMD)/spectral transcranial Doppler (TCD) insonation protocol for an average-sized adult patient includes the following steps.

Transtemporal Insonation

1. Set PMD display depths at 30 to 80 mm.
2. Apply transducer to the middle aspect of the temporal window above zygomatic arch and close to the ear lobe.
3. Maintain slightly upward and anterior angulation of the probe.
4. Set noise levels to allow minimal background signal on PMD display.
5. If no flow signals appear, advance transducer in slow circular movements toward the anterior temporal window.
6. While advancing the transducer, keep changing the probe angulation from the anterior to perpendicular direction relative to the temporal bone.
7. Find a window with maximum spatial presence of the middle cerebral artery (MCA) flow signature between 30- and 70-mm depths. In other words, attempt to fill the PMD screen with color flow signals over the MCA depth range.
8. Readjust the probe angulation to detect flow signals in the M2 MCA segments (30- to 40-mm depths), terminal internal carotid artery (ICA) (60- to 70-mm depths), and anterior cerebral artery (60- to 75-mm depths).
9. Return to the view of the MCA origin and slightly rotate the transducer 10° to 30° posteriorly and downward to detect flow in the posterior cerebral artery (60- to 70-mm depths).
10. At all transducer positions, note whether the contralateral flow signals can be displayed (depth ≥ 75 mm in most adults).
11. Sequentially advance the TCD sample volume with 1-mm steps over all arterial segments detected by PMD to display spectral information.

Transorbital Insonation

1. Decrease the power output of the unit to 10%.
2. Set the PMD depth at 30 to 80 mm.
3. Place the transducer over closed eyelid and angle it slightly medially.
4. Align the transducer position to display flow signatures at the depth of 40 to 70 mm.
5. Determine pulsatility and direction of flow in the ophthalmic artery.
6. Sample spectral information from the distal ophthalmic artery (40- to 55-mm depths) and ICA siphon (55- to 70-mm depths).

Transforaminal Insonation

1. Set the PMD depth at 60 to 110 mm.
2. Place the transducer suboccipitally at midline and aim toward the bridge of the nose, and detect any flow signal moving away from the probe.
3. Align the transducer position to display maximum flow signatures between 75 and 100 mm.
4. Sample spectral information from the proximal (80 mm), middle (90 mm), and distal (100+ mm) portions of the basilar artery.
5. To find the terminal vertebral arteries, set the PMD depth at 30 to 70 mm.
6. Place the transducer laterally 0.5 to 1 in. off midline and aim toward orbits. Avoid angulation to the contralateral side.
7. Sample spectral flow signals from all segments of the terminal vertebral artery and repeat examination on the other side.

Note that small sample volume for spectral TCD delivers higher intensities with a PMD/spectral TCD unit. The trade-off between sensitivity and spatial resolution may remain optimal even with a small 3-mm spectral gate.