Applications and Advantages of Power Motion-Mode Doppler in Acute Posterior Circulation Cerebral Ischemia

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**Background and Purpose**—Evaluation of posterior circulation with single-gate transcranial Doppler (TCD) is technically challenging and yields lower accuracy parameters in comparison to anterior circulation vessels. Transcranial power motion-mode Doppler (PMD-TCD), in addition to spectral information, simultaneously displays in real-time flow signal intensity and direction over 6 cm of intracranial space. We aimed to evaluate the diagnostic accuracy of PMD-TCD against angiography in detection of acute posterior circulation stenoocclusive disease.

**Methods**—Consecutive patients presenting to the emergency room with symptoms of acute (<24 hours) cerebral ischemia underwent emergent neurovascular evaluation with PMD-TCD and angiography (computed tomographic angiography, magnetic resonance angiography, or digital subtraction angiography). Previously published diagnostic criteria were prospectively applied for PMD-TCD interpretation independent of angiographic findings.

**Results**—A total of 213 patients (119 men; mean age 65±16 years; ischemic stroke 71%, transient ischemic attack 29%) underwent emergent neurovascular assessment. Compared with angiography, PMD-TCD showed 17 true-positive, 8 false-negative, 6 false-positive, and 182 true-negative studies in posterior circulation vessels (sensitivity 73% [55% to 91%], specificity 96% [93% to 99%], positive predictive value 68% [50% to 86%], negative predictive value 95% [92% to 98%], accuracy 93% [90% to 96%]). In 14 patients (82% of true-positive cases), PMD display showed diagnostic flow signatures complementary to the information provided by the spectral display: reverberating or alternating flow, distal basilar artery flow reversal, high-resistance flow, emboli tracks and, bruit flow signatures.

**Conclusions**—PMD-TCD yields a satisfactory agreement with urgent brain angiography in the evaluation of patients with acute posterior circulation cerebral ischemia. PMD display can depict flow signatures that are complimentary to and can increase confidence in standard single-gate TCD spectral findings. (Stroke. 2008;39:1197-1204.)

**Key Words:** angiography ■ ischemia ■ power motion-mode Doppler ■ stroke ■ transcranial Doppler

Posterior circulation ischemia is an important cause of acute neurological disease and accounts for up to 20% of ischemic stroke events. Delayed, incorrect, or missed diagnosis of vertebrobasilar ischemia is frequent and may result in serious morbidity or mortality. The benefit of intraarterial thrombolytic therapy in patients with basilar2–4 (BA) or vertebral4 (VA) artery occlusions has been established as potentially lifesaving if given within 12 to 24 hours after symptom onset. Therefore, emergent noninvasive neurovascular assessment of posterior circulation is critical not only for rapid confirmation of vertebrobasilar stenoocclusive disease, but also for identification of patients at higher risk of poor outcome as candidates for interventional treatment.

Bedside transcranial Doppler (TCD) is a fast, noninvasive, widely available diagnostic tool that can detect, localize, and grade the severity of intracranial arterial obstruction in the setting of acute ischemic stroke. However, the ultrasonographic assessment of the vertebrobasilar system is complex due to difficult access (insufficient ultrasound penetration to the distal parts of BA or in cases of adipose necks), anatomic variability, and tortuosity of this vascular region. In addition, evaluation of posterior circulation vessels with single-gate TCD is technically challenging because it relies on one-depth-at-a-time waveform display without any image guidance and thus yields lower accuracy parameters in comparison to anterior circulation vessels.

Transcranial power motion-mode Doppler (PMD-TCD) was recently invented by Mark Moehring and Merrill Spencer. In its present configuration, PMD-TCD uses 33 overlapping Doppler samples to simultaneously display flow...
signal intensities and direction over 6 cm of intracranial space. PMD-TCD 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 more complete spectral analysis. Our group and other investigators have described potential applications and advantages of PMD over single-gate TCD in detecting acoustic windows and microembolic flow signatures, in diagnosing acute middle cerebral artery occlusions, and for evaluating cervical internal carotid artery stenosis.

In the present study, we aimed to evaluate the diagnostic accuracy of PMD-TCD against angiography for the detection of acute posterior circulation stenoocclusive arterial disease.

Subjects and Methods

Transcranial Power Motion-Mode Doppler

Our stroke treatment team routinely uses portable 2-MHz PMD-TCD (Spencer Technologies, Inc) as a noninvasive screening test in the emergency department for the evaluation of patients with acute (<24 hours) stroke. Bedside TCD is carried out simultaneously with clinical assessment and blood draws by experienced sonographers using a standardized fast-track (<15 minutes) insonation protocol to identify suspected arterial obstruction. All TCD studies are performed and interpreted by stroke neurologists (V.K.S., G.T., A.Y.L., M.D.M., A.V.A.) with specialized training and credentials in cerebrovascular ultrasound (certified by the American Society of Neuroimaging and/or the American Registry for Diagnostic Medical Sonographers).

Ultrasound results were interpreted at bedside by the members of our neurosonology team who were blinded to angiography results using previously published diagnostic criteria. An insonation depth of 40 to 79 mm and of 80 to 105 mm was used for the identification of VA and BA stenoocclusive disease, respectively, during transforaminal or suboccipital insonation (Figures 1 to 5). An insonation depth of 58 to 70 mm with posterior angulation of the probe during transtemporal insonation was used for identification of posterior cerebral artery (PCA) stenoocclusive disease.

Posterior circulation occlusions were diagnosed using the Thrombolysis in Brain Ischemia (TIBI) flow-grading system. Occlusion was defined as the absence of flow (TIBI 0) or the presence of minimal (TIBI I), blunted (TIBI II), or dampened (TIBI III) flow signals throughout the VA, BA (Figure 3, Case 2), and PCA at the respective insonation depths. Normal vertebral artery (if available) was used as a comparison vessel in cases of suspected basilar artery occlusion. TIBI grade was compared with the contralateral vertebral in patients with suspected VA occlusions. The presence of reverberating (oscillating) flow signals in the VA (Figure 2, case 3 and BA (Figure 3, Case 3) both in the PMD and spectral display was coded as TIBI I.

Maximum mean flow velocities (MFV) were obtained from the BA, VA, and PCA with a 4-second spectral Doppler data acquisition sweep. A stenosis of ≥50% was identified when MFV exceeded 80 cm/s and when the stenotic-to-normal MFV ratio was ≥2. For the calculation of the stenotic-to-normal MFV ratio, the following algorithm was used. If a focal stenotic velocity was found in the distal VA (50 to 79 mm), BA (90 to 105 mm), or distal PCA (65 to 70 mm), a more proximal prestenotic segment was chosen as normal (40 to 49 mm for VA, 80 to 89 mm for BA [if unaffected in the presence of a more distal BA lesion], 58 to 64 mm for PCA). If a proximal VA (40 to 49 mm) or proximal PCA (58 to 64 mm) stenotic flow velocity was found, a contralateral depth-corresponding segment was used to calculate the ratio. In case of proximal BA (80 to 89 mm) stenosis, the highest MFV velocity documented in the right or left VA was used for the calculation of the stenotic-to-normal MFV. In case of bilateral proximal PCA stenosis (Figure 4, Case 2),
Figure 2. Diagnostic PMD flow signatures in patients with VA stenoocclusive disease. Case 1: Stenotic PMD flow signatures in a patient with angiographically proven VA stenosis (A). Note that the stenotic (C) to normal (B) MFV ratio is 2.7 (162/60). Also note the presence of bruits on the PMD display depicted as flow gaps during systol (yellow circles) that correspond to a bidirectional appearance of systolic bruits (blue circles) on the spectrogram. Case 2: PMD high-resistance flow signatures in a patient with right extracranial VA occlusion (A) and reconstitution (B) of the distal intracranial segment on CTA. Note the difference in PMD flow signatures between the right (high-resistance flow on PMD display with delayed systolic flow acceleration on spectrogram) and left VA (low-resistance flow on PMD display with sharp systolic flow acceleration on spectrogram). Case 3: Stenotic (A) and occlusive (B) PMD flow signatures in a patient with right VA stenosis (C; injection of contrast into the right extracranial VA) and left VA occlusion (C, inset; injection of contrast into the left extracranial VA) on DSA. PMD showed reverberating flow (red to blue) signals that correspond to oscillating flow on spectrogram of the left VA (B). The BA has a blunted flow with delayed systolic acceleration (D; compare with normal BA systolic flow on spectrogram of the left VA (B). The BA has a blunted flow with delayed systolic acceleration (D; compare with normal BA systolic flow acceleration in Figure 1C) and a lower resistance PI of 0.55 (compare with Figure 1C; PI ~0.7) that indicates potential compensatory vasodilatation. Embolic track signatures (white circles) on PMD and high-intensity transient signals on spectrogram were observed during insonation of the VA and PICA (E). PI indicates Gosling-King pulsatility index; PICA, posterior inferior cerebellar artery.

The presence of the following collateral patterns was identified during the emergent PMD-TCD evaluation:

1. Collateralization through the posterior communicating (PCom) artery (normal, increased velocity or stenotic-like low-resistance flow directed either away from or toward the probe in case of BA or internal carotid artery/middle cerebral artery occlusion, respectively [Figure 4, Case 1] found at depths of 58 to 70 mm with posterior angulation of the transducer over the temporal window).9,16 In case of proximal BA occlusion with distal flow reversal through PCom collateralization, the MFV in the PCom was expected to be equal or greater than the highest MFV velocity documented in the BA, whereas in cases with middle cerebral artery/internal carotid artery occlusion, the MFV in the PCom was expected be equal or greater than the highest MFV velocity documented in the middle cerebral artery.

2. Reversed flow in basilar artery (low-resistance flow moving toward the probe at depths of 80 to 105 mm in the absence of antegrade basilar flow signals during suboccipital insonation with or without confirmation of anterior circulation flow with carotid tapping [Figure 4, Case 3]).17 Of note, carotid tapping was performed only if extracranial ultrasound examination previously confirmed the absence of significant stenosis or hypoechoic plaques in carotid arteries.

3. Compensatory flow increase in the contralateral VA or cerebellar collaterals (arterial flow directed toward the probe with higher MFV than in VA at depths of 55 to 70 mm for the posterior inferior cerebellar artery, 75 to 85 mm for the anterior inferior cerebellar artery, and >90 mm for superior cerebellar artery in the absence of PCom flow or suspicion of the reversed BA flow signature) in case of terminal vertebral artery or BA occlusions.9,18

The presence of the subclavian steal phenomenon was diagnosed if an alternating flow with reversal of direction toward the probe at the beginning of the cardiac cycle was displayed both in PMD and spectral displays.23 In cases in which flow reversal was incomplete (ie, “camelback” waveform), the ischemia–hyperemia test was performed either to provoke the steal or to augment flow reversal. The cuff was inflated to oversystolic blood pressure values and flow reduction to the arm was maintained for at least 0.5 to 1 minute with patients opening and closing their fists. The cuff was then quickly released and any augmentation of flow was monitored (Figure 5, Case 1). In all patients with no temporal windows, a noncontrast-enhanced TCD examination of the orbital (transorbital insonation) and vertebrobasilar (transforaminal insonation) vessels was routinely performed. These limited studies were also included in the present analysis.

Angiography

Computed tomography angiography (CTA) was obtained routinely as part of our standard neuroimaging assessment of patients with acute cerebral ischemia and was used as the gold standard for comparison with TCD in determining the presence of stenoocclusive intracranial lesions. The elapsed time between symptom onset and CTA was less than 48 hours for all study subjects and less than 24 hours in 90% of cases. Patients with contraindications to CTA (renal failure or creatinine levels >1.5 mg/dL, contrast allergy) were
significant intracranial artery stenosis was considered when the narrowest diameter of the residual lumen was less than 50%. Intracranial artery occlusion was diagnosed when no reconstitution of distal flow was detected. An attending-level staff neuroradiologist and stroke neurologist evaluated neurodiagnostic imaging studies independently of TCD results. The neuroradiological interpretation in the medical records was considered the official reading for the purposes of the study. This study was approved by the Institutional Review Board of our institution.

Statistical Analysis

Continuous variables are presented as mean (SD) or as median (range). Noncontinuous variables are presented as percentages. The accuracy parameters (sensitivity, specificity, positive predictive value, negative predictive value, and overall accuracy) with their corresponding 95% CI of the screening test (TCD) against angiography were calculated after computation of true positive, false-positive, true-negative, and false-negative values. Patients with absent temporal windows were included in the computation of accuracy parameters with only results in the posterior circulation assessment being considered for the analysis. The Statistical Package for Social Science (SPSS Inc, version 11.5 for Windows) was used for statistical analyses.

Results

A total of 213 patients (119 men, mean age 65 ± 16 years) presenting with symptoms of acute (<24 hours) cerebral ischemia underwent emergent neurovascular assessment with PMD-TCD within 24 hours from symptom onset and angiography (CTA, MRA, or DSA) within 48 hours from ictus (Table 1). Thirty-eight (18%) patients received intravenous (n = 29) or intraarterial thrombolysis (n = 9) (median National Institutes of Health Stroke Scale score 10; range, 4 to 26 points). No delay in treatment resulted from ultrasound testing. One hundred thirteen (53%) patients were found ineligible for thrombolysis and 62 patients (29%) were diagnosed with transient ischemic attacks.

Bedside PMD-TCD revealed intracranial artery stenosis or occlusion in the posterior circulation in 24 cases: VA (n = 11), BA (n = 8), and PCA (n = 5). Temporal acoustic windows were absent in 20 cases (9%). Angiography findings were positive for the posterior circulation stenoocclusive disease in 12% (n = 26) of the study population and revealed unremarkable...
able posterior circulation vessels in the remaining cases (n=187 [88%]). DSA was performed in 16 cases, whereas a total of 31 patients with contraindications to CTA were evaluated with MRA. Compared with angiography, PMD-TCD showed 17 true-positive, 8 false-negative, 6 false-positive, and 182 true-negative studies (sensitivity 73% [55% to 91%], specificity 96% [93% to 99%], positive predictive value 68% [50% to 86%], negative predictive value 95% [92% to 98%], accuracy 93% [90% to 96%]). The accuracy parameters with their corresponding CIs for TCD in detecting arterial stenosis or occlusion in VA, BA, and PCA are presented in Table 2.

Absent temporal windows led to one false-negative PCA study. Three of the remaining 7 false-negative studies occurred in the distal BA (n=2) or in the distal P2-PCA segment (n=1). Also, incorrect insonation of a single existing VA (VA was insonated from both sides during suboccipital insonation) led to 2 other false-negative studies. Finally, in cases of missed VA stenosis, the MFVs were within the normal range (72 cm/s and 67 cm/s) despite an abnormal stenotic-to-normal ratio (2.2 and 2.4, respectively). These patients were 72 and 81 years old.

Two of 6 false-positive TCD cases were attributed to the minimal flow signals due to suboptimal angle of insonation in tortuous distal VA (n=1) and distal BA (n=1). Also, the presence of a blunted flow signal (TIBI grade II) with low MFV throughout a dolichoectatic BA led to another false-positive study.

Misinterpretation of collateral flow signals in PCom (n=1) in a patient with extracranial internal carotid artery occlusion led to the diagnosis of a false-positive PCA stenosis. In a patient with dampened flow signals (TIBI grade III) in the intracranial VA, CTA findings were attributed to hypoplasia and not an acute occlusion. Finally, in one patient, the stenotic VA velocities were not confirmed by CTA after intravenous thrombolytic therapy. This patient had a 4-point reduction in the National Institutes of Health Stroke Scale score during thrombolysis with complete recanalization and time delay of 105 minutes between TCD and CTA causing this discrepancy.

In 16 cases (8%), PMD showed findings complementary to brain CTA or MRA: (1) flow reversal in the distal BA with the proximal BA occlusions; (2) flow reversal in the distal VA with the proximal intracranial VA occlusions; (3) real-time embolization distal to a stenoocclusive disease (Figure 2, Case 3); (4) blunted flow signals throughout intracranial VA in patients with extracranial severe stenoocclusive disease (both cases confirmed by subsequent neck CTA; Figure 2, Case 2); and (5) alternating PMD flow signals indicative of subclavian steal phenomenon (Figure 5, Case 1).

Furthermore, in 14 patients (82% of true-positive cases), PMD flow signatures were also complementary to the findings of the spectral TCD display: (1) reverberating flow signatures (Figure 2, Case 3, Figure 3, Case 3); (2) alternating flow signature (Figure 5, Cases 1 and 2); (3) high-resistance
flow signature (Figure 3, Cases 2 and 3); (4) embolic tracks flow signature (Figure 2, Case 3); and (5) disturbed flow (bu-ruit) signature (Figure 2, Case 1).

Explanations as to how these PMD findings provide additional diagnostic value are given in the figure legends.

Discussion
Our study showed that bedside PMD-TCD examination yields satisfactory agreement with urgent brain angiography in patients presenting with symptoms of acute posterior circulation cerebral ischemia. Moreover, in a substantial number of patients (82% of the true-positive cases), PMD display showed diagnostic flow signatures complementary to the information provided by the spectral display: reverberating or alternating flow, distal BA flow reversal, high-resistance flow, emboli-tracking, and disturbed flow signatures.

The accuracy parameters of PMD-TCD in the present study are higher than those of previous reports on the diagnostic accuracy of single-gate TCD in the evaluation of posterior circulation vessels. According to the recently published results of the Stroke Outcomes and Neuroimaging of Intracranial Atherosclerosis (SONIA) trial, the positive predictive value of single-gate TCD for detection of stenosis in the intracranial VA and BA were 45% and 36%, respectively (diagnostic cutoff for MFV was 80 cm/s) when compared with DSA with negative predictive value of 84% and 90%, respectively.24 We hypothesize that the higher yield of PMD-TCD may be related to its ability to visualize flow on the PMD display along tortuous and long arterial segments (Figure 1) that may not be readily appreciated by sonographers during a single-gate TCD examination.12 In addition, the ease of identification of turbulent flow on PMD display (flow gaps during systole; Figure 2, Case 1) prompts sonographers to perform a more thorough spectral sample volume interrogation because it topographically maps the location and extent of turbulence. Finally, PMD permits easy identification of retrograde flow in the BA that may be caused either by retroperfusion of the distal BA through the anterior circulation in cases of proximal BA occlusion (Figure 4, Case 3) or by BA dissection (retrograde high-resistance flow in the false lumen; Figure 3, Case 1).

**Table 1. Baseline Characteristics of the Study Population (n=213)**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age, years (SD)</td>
<td>65 (16)</td>
</tr>
<tr>
<td>Male sex</td>
<td>56</td>
</tr>
<tr>
<td>Hypertension</td>
<td>68</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>32</td>
</tr>
<tr>
<td>Ischemic heart disease</td>
<td>27</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>49</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>19</td>
</tr>
<tr>
<td>Median National Institutes of Health Stroke Scale score (range)*</td>
<td>12 (4–25)</td>
</tr>
</tbody>
</table>

Continuous variables are presented as mean (SD) or as median (range). Noncontinuous variables are presented as percentages.

*Tissue plasminogen activator-treated patients.
In the present report, we implemented a stenotic-to-normal MFV ratio ≥ 2 in addition to standard MFV thresholds in our diagnostic criteria for the detection of intracranial stenosis, because the addition of this ratio to the MFV cutoff improved TCD accuracy in diagnosing anterior circulation stenoses. These ratios were not taken into account in the analysis of WASID-SONIA findings. Because PMD simultaneously shows vessel segments with turbulent and laminar flow over a wide range of depths, it permits better interrogation of the affected and nonaffected vessel segments. This, in turn, facilitates a more reliable estimation of stenotic-to-normal MFV ratios and possibly decreases erroneous vessel identification while resulting in a more thorough vessel interrogation compared with a single-gate TCD examination.

Detection of flow reversal in retroperfused BA on PMD display may constitute another factor contributing to the higher diagnostic accuracy of this neurovascular imaging modality in the evaluation of posterior circulation stenoocclusive disease. According to our previous experience, BA flow reversal can be missed with the single-gate TCD, whereas its detection with PMD correlated with better outcomes after the proximal BA occlusion. Similar to our present findings, 2 recent studies of posterior circulation vessels with transcranial color-coded duplex ultrasound imaging, which allows simultaneous visualization of anatomic structures in color-coded B-mode images and Doppler flow, also reported satisfactory agreement with angiography in cases of suspected BA occlusion.

A plausible alternative explanation for the different accuracy parameters between our present report and previous single-gate TCD studies may be related to the elapsed time interval between the screening test and the gold standard. Longer time delays between ultrasound and angiography may provide ample time for thrombus propagation, dissolution, or reocclusion to occur, accounting for the lower predictive values in previous reports. Notably, in one study, 25% of middle cerebral artery stenoses had completely disappeared 6 months after stroke, suggesting an embolic rather than atherothrombotic process.

In our series, PMD display depicted diagnostic flow signatures that were complementary to the information provided by the spectral display in the majority of the true-positive cases. The presence of reverberating or high-resistance flow signatures on PMD can facilitate the rapid diagnosis and localization of vertebrobasilar artery occlusion. This observation is in keeping with the findings by Saqur et al who reported that absent or high-resistance PMD flow signatures can reliably diagnose 87% of angiographic-proven proximal middle cerebral artery occlusions. Furthermore, subclavian steal phenomenon can be easily identified by alternating PMD flow signatures (Figure 5, Case 1). Of note, however, a differential diagnosis that needs to be kept in mind is dissection of intracranial VA, because it can also present with alternating flow signatures (Figure 5, Case 2). Also, the PMD display has been shown to be more sensitive for the detection of embolic flow signatures than the single-gate spectral display, because it allows simultaneous display of flow over long arterial segments as well as more than one vessel. In our series, we noted PMD embolic tracks in the cerebellar arteries (depicted as red bands on PMD display over different depths), whereas our sample volume interrogation was set in depths corresponding to VA or BA. Because the cerebellar arteries are not routinely insonated during the assessment of posterior circulation vessels with single-gate TCD, the presence of embolization distal to an extracranial or intracranial stenoocclusive arterial lesion may have not been identified without PMD-TCD.

Our study has limitations, including the need for considerable sonographer expertise to complete and interpret testing promptly and efficiently. Our study also identified clinical situations when PMD-TCD could not be reliably interpreted, including limited visualization of the distal BA, incorrect insonation through both suboccipital windows of a single existing VA, and absent temporal windows rendering unfeasible the insonation of PCA. Moreover, it needs to be acknowledged that PMD cannot reliably differentiate a hypoplastic intracranial segment of VA with low-velocity, high-resistance flow signals from that of an acutely occluded VA with dampened residual flow signals. In addition, to avoid interobserver variation, angiographic measurements of the degree of stenosis by a second investigator would have been methodologically advantageous should they had been performed. MFV values also decrease with increasing age and the fact that we did not use age-dependent thresholds for detection of intracranial stenoocclusive disease should be acknowledged as another methodological shortcoming of the present report. Finally, the present study was not designed to compare the accuracy parameters of single-gate TCD with PMD in the evaluation of posterior circulation ischemia and therefore, direct conclusions regarding the potential superiority or inferiority of one imaging modality over another cannot be inferred. In fact, PMD is complementary to spectral TCD, which provides waveforms and velocities used for diagnosis of arterial patency and lesions.

On the other hand, a methodological strength of the present report is that both neurovascular assessments (PMD and angiography) were performed in the emergency setting with a relatively narrow time window. We also attempted to minimize selection bias by including consecutive patients presenting with symptoms of acute cerebral ischemia who under-
went angiographic and PMD-TCD neurovascular assessment irrespective of whether their clinical findings were indicative of an anterior or posterior circulation ischemic event and of whether their symptoms resolved or persisted during the first 24 hours of ictus.

In conclusion, emergent PMD-TCD yields a substantial proportion of posterior circulation stenoocclusive arterial lesions with satisfactory agreement with angiography in the acute stroke setting. In addition, in selected cases, it offers information complementary to noninvasive angiography or to single-gate TCD. These findings provide preliminary evidence indicating that PMD-TCD is a promising and valuable noninvasive imaging modality for the emergent bedside assessment of posterior circulation that can be used as a rapid vascular screening method to reliably exclude vertebrobasilar artery occlusion and to select potential candidates for invasive angiography that may ultimately undergo subsequent endovascular interventions if the ultrasound findings are confirmed by DSA.

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Disclosures

None.

References

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