Transcranial Doppler Correlation With Angiography in Detection of Intracranial Stenosis

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Background and Purpose The purpose of this study was to evaluate the use of velocity criteria applied to transcranial Doppler (TCD) signals in the detection of stenosis of the middle cerebral (MCA), distal vertebral, and basilar arteries.

Methods Sixty-five patients who underwent both cerebral angiography and transcranial Doppler examinations in the workup of acute cerebral ischemia were reviewed. Angiography was performed a mean of 7±5 days (range, 1 to 28 days) after TCD. Interpretation of the angiogram was performed without input regarding the TCD findings. TCD interpretation was performed according to standard criteria.

Results When we used a mean velocity (MV) cutoff of ≥80 cm/s in the MCA as the criterion for stenosis, 10 of 12 stenoses of any degree were detected by TCD, with 11 of 87 false-positives. Nine of 12 MCA stem (M1) stenoses were detected when a cutoff of ≥90 cm/s was used, with 7 of 87 false-positives. When we used an MV cutoff of ≥70 cm/s as the criterion for ≥50% stenosis of the vertebrobasilar system, 5 of 6 stenoses were detected, with 15 of 85 false-positives. The most important confounding factor was the presence of ≥75% stenosis of the extracranial internal carotid artery, resulting in both false-positive (from collateral flow) and false-negative (decreased volume flow from the proximal stenosis without adequate collateral flow) errors in TCD interpretation. When patients with ≥75% stenosis of the cervical internal carotid artery were excluded from analysis, a TCD MV cutoff of ≥80 cm/s identified 9 of 10 M1 lesions with 7 of 61 false-positives, and an MV of ≥70 cm/s identified 3 of 4 vertebrobasilar lesions causing ≥50% stenosis with 7 of 56 false-positives.

Conclusions TCD may be an effective screening test for M1 stenosis when velocity criteria alone are used. TCD may less reliably detect intracranial vertebral and basilar artery stenosis. (Stroke. 1994;25:1931-1934.)

Key Words • angiography • stenosis • ultrasonics • vertebrobasilar circulation

Subjects and Methods Adults (>18 years old) who had both TCD and cerebral angiography studies as part of a workup for acute cerebral ischemic events were identified by analysis of patient logs from the neurosonology laboratory and radiology department of the Medical College of Georgia Hospital. Patients were included in the study if TCD had been performed between May 1987 and July 1993 and cerebral angiography had been performed less than 28 days from the date of TCD. Patient referral information was used to exclude patients with intracranial hemorrhage, arteriovenous malformation, and sickle cell anemia.

The primary reasons for angiography included further evaluation of the carotid bifurcation in patients considered for carotid endarterectomy and evaluation for suspected intracranial stenosis. In both situations, TCD served as a screening test before angiography. In patients with suspected intracranial arterial stenosis and poor acoustic windows, TCD examination produced limited data, and these patients frequently had cerebral angiography. We do not know how many patients could not undergo angiography for reasons such as allergy, renal disease, technical problems, or refusal to have the procedure. As a result of these biases, true sensitivity and specificity data for TCD in the detection of intracranial stenosis cannot be determined from our data. We will present the data using descriptive statistics; these data can then be used to determine the usefulness of a normal or abnormal examination.

TCD studies were performed on either a TC2-64b or TC2000 machine (Eden Medical Electronics). We used a standard method of insonation without compression testing.10,11 We used the time-averaged maximum MV expressed in centimeters per second exclusively in our analyses; all further references to velocities in this report refer to the time-averaged mean maximum. TCD MV data were obtained

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from reports produced by a neurologist (R.J.A.) who was not aware of the angiography results. The primary analysis of TCD data was based on MV values recorded on the TCD reports, in most cases limited to single values for each artery. The MV on the report was the maximum recorded for that artery. Velocity criteria were chosen on the basis of published normal values and reported velocity criteria for M1 stenosis. For the effect of using two different MV criteria: ≥80 cm/s and ≥90 cm/s. For the analysis of intracranial vertebral and basilar artery segments, an MV ≥70 cm/s was abnormal.

Cerebral angiograms that used the Seldinger technique with conventional biplanar magnification were the source of angiographic correlation in most patients, with intra-arterial digital subtraction angiography in the remainder. Cerebral angiograms were evaluated independent of the TCD as the "gold standard" by two of the authors (F.T.N., M.B.R.), except in six cases in which only neuroradiologist reports were available. Each major arterial segment (M1, proximal anterior cerebral artery [A1], and intracranial vertebral and basilar arteries) was graded as follows: no stenosis, stenosis <50%, ≥50% and <75%, ≥75% and <99%, or occlusion. Extracranial carotid stenosis was measured by the North American Symptomatic Carotid Endarterectomy Trial technique. A modification of this was used for intracranial stenosis: the vessel being evaluated was measured at its point of maximal narrowing and compared with the angiographically normal section of vessel adjacent to the stenosis to determine the degree of stenosis (normal lumen diameter−residual lumen/normal lumen diameter). In the presence of occlusions, collateral flow patterns were noted.

**Results**

Sixty-five patients were included in the study group, which was 40% male and 42% black, with a mean age of 51 years (range, 21 to 73 years; SD, 12). The mean time interval between TCD and angiography was 7 days (maximum, 27; SD, 5), and, with one exception, all angiograms were performed after TCD. Twenty-three of 65 patients had stenosis or occlusion of one or more intracranial artery segments, yielding an intracranial arterial disease frequency of 35%. Eight occluded intracranial arterial segments (5 internal carotid artery [ICA], 1 MCA, 1 anterior cerebral artery, 1 vertebral artery) were excluded from analysis.

A poor or absent temporal acoustic window resulted in failure to detect MCA signals in 17 patients (5 of 38 whites, 12 of 27 blacks), yielding a failure rate of 26%. Usually when there was a poor or absent temporal acoustic window it was bilateral (10 patients accounted for 20 of 27 cases of unobtainable M1 data). The highest failure rate was in black women (37%), followed by black men (25%), white men (14%), and white women (10%).

TCD data were available for correlation in 12 of 18 angiographically identified M1 stenoses and were abnormal in 10 when we used a velocity cutoff of ≥80 cm/s (Table 1). In the two missed cases (one graded 50% to 74% and the other 75% to 99%), the stenotic M1 segments were ipsilateral to significant ICA stenosis (patients 8 and 11, Table 1), and there was either absent or very minimal collateral flow into the MCA. When we used ≥90 cm/s as the velocity cutoff, the number of M1 stenoses detected by TCD was 9 of 12. The average MV for 7 M1 lesions of <50% stenosis was 136 cm/s (range, 86 to 220 cm/s); for 3 M1 lesions of 50% to 75% stenosis it was 182 cm/s (range, 50 to 269 cm/s). Two M1 lesions causing 75% to 99% stenosis had velocities of 50 and 143 cm/s. One occluded M1 segment was read as patent by TCD, but MV was low.

TCD exceeded the cutoff of ≥80 cm/s in 11 of 87 normal M1 segments. Of these 11 M1 segments, 6 were ipsilateral to significant ICA stenosis (≥50%) or occlusion. When we used a cutoff of ≥90 cm/s, TCD was abnormal in only 7 M1 segments.

When we used the ≥80 cm/s velocity cutoff for the M1, the two false-negative studies and 4 false-positive MV elevations were ipsilateral to cervical ICA stenosis of ≥75%. When the data from all patients with cervical ICA stenosis of ≥75% are eliminated from analysis, positive predictive value and negative predictive value increase from 48% to 56% and from 97% to 98%, respectively. With the ≥90 cm/s velocity cutoff, when data from patients with cervical ICA stenosis of ≥75% are eliminated from analysis, positive predictive value and negative predictive value increase from 56% to 73% and from 96% to 97%, respectively.

**Table 1. Stenotic M1 Segments With Transcranial Doppler Correlation**

<table>
<thead>
<tr>
<th>Pt</th>
<th>M1, % stenosis</th>
<th>IICA, % stenosis</th>
<th>M1 MV, cm/s</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>&lt;50</td>
<td>0</td>
<td>86</td>
</tr>
<tr>
<td>2</td>
<td>&lt;50</td>
<td>0-74</td>
<td>90</td>
</tr>
<tr>
<td>3</td>
<td>&lt;50</td>
<td>&lt;50</td>
<td>108</td>
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<tr>
<td>4</td>
<td>&lt;50</td>
<td>0</td>
<td>115</td>
</tr>
<tr>
<td>5</td>
<td>&lt;50</td>
<td>50-74</td>
<td>123</td>
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<tr>
<td>6</td>
<td>&lt;50</td>
<td>&lt;50</td>
<td>210</td>
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</tr>
<tr>
<td>9</td>
<td>50-74</td>
<td>0</td>
<td>228</td>
</tr>
<tr>
<td>10</td>
<td>50-74</td>
<td>&lt;50</td>
<td>269</td>
</tr>
<tr>
<td>11</td>
<td>75-99</td>
<td>75-99</td>
<td>50</td>
</tr>
<tr>
<td>12</td>
<td>75-99</td>
<td>&lt;50</td>
<td>143</td>
</tr>
</tbody>
</table>

Pt indicates patient; M1, middle cerebral artery stem; IICA, ipsilateral internal carotid artery; and MV, maximum mean velocity.

**Table 2. Stenotic Intracranial Vertebral and Basilar Arteries With Transcranial Doppler Correlation**

<table>
<thead>
<tr>
<th>VA, % stenosis</th>
<th>MV, cm/s</th>
<th>BA, % stenosis</th>
<th>MV, cm/s</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;50</td>
<td>10</td>
<td>&lt;50</td>
<td>38</td>
</tr>
<tr>
<td>&lt;50</td>
<td>26</td>
<td>&lt;50</td>
<td>43</td>
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<tr>
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<td>&lt;50</td>
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<td>78</td>
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<td>114</td>
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<td>50-74</td>
<td>86</td>
<td>50-74</td>
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</tr>
<tr>
<td>50-74</td>
<td>&gt;200</td>
<td>50-74</td>
<td>264</td>
</tr>
<tr>
<td>75-99</td>
<td>10</td>
<td>50-74</td>
<td>124</td>
</tr>
</tbody>
</table>

VA indicates vertebral artery; MV, maximum mean velocity; and BA, basilar artery.
TCD was abnormal in 4 of 8. Of the 4 false-negative TCD studies of the vertebral artery stenoses, 3 had <50% luminal narrowing and 1 had 75% to 99% stenosis. One occluded vertebral segment had TCD MV that suggested patency. This occlusion was just distal to the origin of the posterior inferior cerebellar artery (PICA). TCD was abnormal (false-positive) in 3 of 39 normal intracranial vertebral arteries.

Of 8 basilar artery stenoses with TCD correlation available, TCD was abnormal in 6. The two basilar artery stenoses missed by TCD had <50% narrowing. Six of 30 patients with normal basilar arteries had abnormal MV.

TCD was abnormal in 2 of 3 vertebral artery lesions causing ≥50% stenosis and in 3 of 3 basilar artery lesions causing ≥50% stenosis. When patients with ≥75% stenosis of the cervical ICA are eliminated from analysis, the positive predictive value of TCD for the detection of posterior circulation lesions causing ≥50% stenosis increases from 25% to 30%, and negative predictive value remains at 98%.

Anterior cerebral artery disease was rare. TCD correlation was available in 4 A1 segments with stenoses, and in 3 of these MV was >80 cm/s. One patient had an occluded A1 segment with associated TCD finding of no flow.

**Discussion**

We correlated TCD and cerebral angiography in patients with transient ischemic attack or ischemic stroke symptoms. At our institution TCD has been used as a screening test for intracranial arterial stenosis. TCD was performed and initially interpreted separately from carotid duplex testing.

Our study shows that there was one fewer false-negative TCD study in M1 stenotic lesions with the 80 cm/s velocity cutoff than with the 90 cm/s cutoff. Post-stenotic flow reduction is the likely reason for the false-negative studies ipsilateral to high-grade ICA disease. 

Sensitivity of TCD for M1 lesions causing ≥50% stenosis has been reported to be 75% and 86%. There was not a clear correlation between angiographic estimation of the degree of M1 stenosis and MV. It should be recognized that angiographic assessment of the degree of stenosis of the M1 segment is problematic. Only one view (anteroposterior) of the M1 segment is routinely obtained on cerebral angiography. The lack of a second view may potentially result in either overestimation or underestimation of the degree of reduction of luminal area. The highest MV occurred with 50% to 74% M1 stenosis.

Use of the 90 cm/s velocity cutoff reduced the number of false-positive TCD studies. Specificity of TCD for detection of M1 stenosis has been reported to be 26% for patients with acute stroke and 99% for patients with acute stroke along with other indications. With a cutoff of ≥80 cm/s, 6 of the 11 false-positive M1 MV elevations occurred in the presence of ipsilateral ICA stenosis. The elevated velocities may have represented misidentification of the ipsilateral posterior communicating artery carrying collateral flow into the distal ICA as an abnormal M1 signal.

TCD detected 80% of posterior circulation intracranial stenoses causing ≥50% stenosis. TCD was previously reported to have a sensitivity of 74% for detection of posterior circulation stenosis of unspecified degree. Combining continuous-wave Doppler ultrasound of the extracranial arteries and TCD of intracranial vertebral arteries in another study did not improve sensitivity. Posterior circulation lesions may be missed as a result of inability to insonate the entire course of the vertebral and basilar arteries or suboptimal angle of insonation, resulting in underestimation of MV. The use of transcranial color-coded real-time ultrasonography in our patients could have potentially allowed correction for Doppler angle to eliminate underestimation of velocities in stenotic lesions. Angle correction may improve sensitivity, in that it is not possible using the blind TCD technique used in this study to overestimate flow velocity. Color-flow Doppler potentially could better define the exact site of the juncture of the vertebral arteries to form the basilar artery, allowing better identification of the site of abnormality, which may decrease the number of false-negatives. A previous study reported specificity of TCD for the posterior circulation to be 86%, with another reporting 98% when continuous-wave Doppler ultrasound of the extracranial arteries was combined with TCD.

The false-positives for the vertebrobasilar system in our study occurred in the presence of significant ICA occlusive disease, with collateral flow from the vertebrobasilar to the anterior circulation. Review of the angiograms in 4 patients (including 3 patients with MV elevated in the basilar artery and 1 patient with MV elevated in both vertebral arteries and basilar artery) showed that the vertebrobasilar system was supplying collateral flow to the anterior circulation via the posterior communicating artery, leptomeningeals, or both. In 1 other patient (with left vertebral artery with elevated MV), the innominate artery was occluded at its origin, with supply of the right vertebral, right subclavian, and right common carotid arteries from the left vertebral artery. Angiograms of two basilar arteries with elevated MV did not show a collateral flow pattern supplying the anterior circulation, even though both patients had associated ICA disease (bilateral in one, unilateral in the other). Collateral flow caused velocity elevation in the vertebrobasilar circulation has been previously noted.

Vertebral artery occlusions may potentially pose a particular problem for TCD. In cases of vertebral occlusion at the level of their origin from the subclavian, no flow may be identified distally by angiography. The distal vertebral artery may be reconstituted from muscular arterial branches of the external carotid artery or thyrocervical system or by a proatlantal branch, so that flow may be identified on TCD examination. In the case of an occlusion just distal to the PICA, there will be flow detected in the vertebral artery to the level of the PICA. The TCD sample volume may be long enough to insonate the proximal basilar artery distal to the PICA, so potentially no interruption of TCD signal would be detected.

Our study reported a higher rate (26%) of failure to obtain temporal bone signals than cited in most studies.

This rate may have been increased artificially because angiography was performed more often in neurologically symptomatic patients who did not have adequate TCD recordings, eg, those with poor acoustic windows. Anterior intracranial circulation TCD data were most often unobtainable in black women, a finding attributed by Halsey.
to increased temporal bone window thickness. Since blacks constituted 42% of the study group, this may account for the higher overall failure rate when compared with studies containing fewer blacks.

Using velocity criteria alone, our results suggest that TCD may miss up to 17% of M1 stenoses of any degree and up to 20% of intracranial vertebral and basilar stenoses causing ≥50% stenosis. We recognize that velocity analysis alone is not sufficient to describe a TCD study. The use of asymmetry in velocities, “focality” of velocity elevations, and disturbed flow characteristics may all improve the ability of TCD to discriminate stenosis from normal arterial segments. Extracranial ultrasound data were not used in interpretation of TCD findings. In practice, knowing the status of the extracranial arteries would likely affect the interpretation of TCD or even the decision to perform the test at all. Positive and negative predictive values of TCD in this sample can be improved by restricting the analysis to patients without high-grade cervical carotid stenosis; under current practice, the identification of a high-grade ICA stenosis ipsilateral to an ischemic cerebral event would prompt angiographic evaluation for possible carotid endarterectomy in many cases. With an MV cutoff of ≥80 cm/s and exclusion of patients with ≥75% cervical carotid stenosis, positive and negative predictive values of TCD for M1 stenosis are 56% and 98%, respectively. For vertebrobasilar lesions causing ≥50% stenosis, positive and negative predictive values of TCD are 30% and 98%, respectively. The observation of both false-positive and false-negative errors in TCD in the setting of high-grade cervical carotid disease points out a potential limitation of Doppler investigation of the intracranial arteries.

Our study demonstrates that when TCD is successfully performed on patients with <75% stenosis of the ICA, the finding of normal TCD flow velocities in the M1 segment reliably indicates the absence of stenosis in the M1. In this situation, a normal anterior circulation TCD has 98% negative predictive value, suggesting that angiography is not necessary to rule out M1 stenosis. However, an abnormal TCD study of the M1 segment does not reliably predict the presence of stenosis, with positive predictive value of only 56%. Our study also suggests that TCD may miss a number of stenotic lesions in the vertebrobasilar segments, particularly those lesions causing <50% stenosis. Larger studies, perhaps combining color Doppler or TCD with magnetic resonance angiography, are needed to develop and test a highly accurate noninvasive means to detect vertebrobasilar disease.

Acknowledgments

The authors thank Sharon Stephens and Vandy Haggard for technical work and Pamela Lewis for preparation of the manuscript. We also thank Dr James Schmidey for his critical review of the manuscript.

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Stroke. 1994;25:1931-1934
doi: 10.1161/01.STR.25.10.1931

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