Transcranial Doppler in Cerebrovascular Disease

Lawrence R. Wechsler, M.D.,* Allan H. Ropper, M.D.,†‡ and J. Philip Kistler, M.D.†

SUMMARY Doppler analysis of flow in intracranial arteries is now possible using a 2 MHz probe allowing sufficient penetration of bone to obtain signals noninvasively. Thirty-two normal subjects, and 11 patients with cerebrovascular diseases including vasospasm following subarachnoid hemorrhage, middle cerebral artery stenosis, and extracranial internal carotid artery stenosis were studied by transcranial Doppler. Increased peak velocity and spectral broadening of the reflected signal corresponded to clinical and angiographic evidence of middle cerebral artery vasospasm or stenosis. Decreased peak velocity and blunted waveforms occurred in the middle cerebral artery ipsilateral to severe extracranial internal carotid stenosis with poor crossfilling from the contralateral carotid artery. Abnormalities resolved following carotid endarterectomy. Transcranial Doppler identifies vasospasm or stenosis of the middle cerebral artery and may allow noninvasive evaluation of collateral flow across the anterior circle of Willis in patients with extracranial carotid artery stenosis.

Stroke Vol 17, No 5, 1986

DOPPLER EXAMINATION of the extracranial internal carotid artery permits noninvasive detection of extracranial stenosis with considerable accuracy.1 Studying intracranial vessels is now possible using a 2 MHz probe that allows sufficient ultrasound to penetrate bone.2 This report describes preliminary results of transcranial Doppler (TCD) studies, performed in a systematic and uniform fashion, in patients with vasospasm following subarachnoid hemorrhage, middle cerebral artery stenosis, and extracranial internal carotid artery stenosis. Each group includes only a few patients, however, characteristic abnormalities are identified, in most cases correlating with clinical and angiographic evidence of vascular disease. These results provide an indication of the clinical utility of TCD for studying patients with cerebrovascular disease.

From the Department of Neurology,* University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania, and from the Department of Neurology,† and the Neurological/Neurosurgical ICU,‡ Massachusetts General Hospital, Boston, Massachusetts.

This work was supported in part by the Elliot B. Shoolman Fund. Address correspondence to: Lawrence R. Wechsler, M.D., Department of Neurology, University of Pittsburgh, 322 Scaife Hall, Pittsburgh, Pennsylvania 15261.

Received February 6, 1986; revision #1 accepted April 1, 1986.

Instrumentation

A 2 MHz pulsed Doppler instrument with an external probe diameter of 22 mm (TC2-64, EME, Oberlingen, W. Germany) was used for transcranial Doppler examinations. Focal depth of the Doppler signal varied in 5 mm increments from 25 to 150 mm. Pulse repetition frequencies included 5, 8 or 10 KHz depending on depth. Bidirectional signals were recorded with a 10 KHz low-pass filter and a 150 Hz high-pass filter. Spectral analysis was accomplished with fast fourier transformation and 64 point resolution. Spectral information was displayed either as KHz frequency shift or velocity. Calculation of velocity from Doppler shift frequency assumed an angle between probe and blood column of 0 degrees.2

Technique

Doppler signals from the middle cerebral artery were obtained by placing the probe over the temple and adjusting its position for a maximal reflected signal at a depth of 45 to 55 mm. Depth of focus was then increased until bidirectional flow appeared from the terminal bifurcation of the carotid artery, confirming that the original signal was from the MCA. Recordings were obtained from all depths at which an MCA signal could be detected. Depth of focus was then further
increased until flow was predominantly away from the probe, indicating flow in the anterior cerebral artery (ACA). ACA Doppler signals were usually obtained at depths of 65–75 mm. An adjustable cursor identified the peak velocity (or frequency) for each recording. Spectral broadening was judged by widening of the band of recorded frequencies. Minor degrees of spectral broadening were often seen in normal patients, and this was not considered abnormal.

Patient Selection

Patients with subarachnoid hemorrhage, MCA stenosis or carotid artery stenosis were selected from the inpatient population at the Massachusetts General Hospital for TCD examination. Studies were performed with the patient supine and the head at zero or 30 degrees. Normal values were obtained from 2 groups of control subjects. Group 1 consisted of 20 healthy volunteers with mean age 31 (range 18–69). Group 2 included 12 hospitalized patients, mean age 45 (range 22–69), with medical and neurological illnesses not involving the central nervous system.

Results

Controls

Sixty-two middle cerebral arteries (32 patients) and 40 anterior cerebral arteries were studied in normal subjects. In Group 1, peak MCA velocity was 105 ± 16 cm/sec (mean ± SD for 38 arteries) and 85 ± 17 cm/sec (21 arteries) for the ACA. Mean left to right MCA peak velocity ratio was 0.99 ± 0.14 (18 patients). Peak velocity in group 2 for the MCA was 94 ± 16 cm/sec (24 arteries), and 73 ± 16 cm/sec (19 arteries) for the ACA. Mean left to right MCA peak velocity ratio in this group was 1.03 ± 0.17 (12 patients).

Subarachnoid Hemorrhage

Case 1

A 40 year old woman suddenly developed light-headedness, severe headache and vomiting. There was diffuse blood in basal cisterns and interhemispheric fissure on CT scan a few hours later. Angiography on day 2 demonstrated a left carotid bifurcation aneurysm, and she underwent surgery on day 3. Ten days postoperatively and 13 days after the hemorrhage, she developed word finding difficulty and right hand weakness. Neurological signs resolved after increasing systolic blood pressure to 190 mm/Hg. TCD examination of the left MCA on day 13 showed increased peak velocity (284 cm/sec) and spectral broadening (fig. 1-A). Pressors were used to keep blood pressure elevated. On day 16, speech difficulty and right hand weakness recurred when pressors were tapered, resolving again with return of blood pressure to the previous range. TCD continued to show increased velocities and spectral broadening, although peak velocity (262 cm/sec) was slightly lower (fig. 1-B). There was no return of neurological symptoms when blood pressure was again lowered on day 21. TCD then was improved (fig. 1-C); peak systolic velocity decreased to 170 cm/sec. She remained asymptomatic and had an otherwise uneventful recovery. On day 26, TCD of the left MCA appeared normal. TCD abnormalities of the right MCA paralleled those of the left MCA despite the lack of symptoms referable to the right MCA territory, although peak systolic velocity was consistently lower on the right (table 1 — Pt. 1).

Comment

Increased peak velocity and spectral broadening in the left MCA correlated with symptoms of vasospasm. When neurological deficits recurred with reduction of
TABLE 1  Transcranial Doppler Studies of MCA Vasospasm

<table>
<thead>
<tr>
<th>PT</th>
<th>Peak vel*</th>
<th>Peak vel</th>
<th>Peak vel</th>
<th>Peak vel</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>MCA (Day #)</td>
<td>vel (Day #)</td>
<td>vel (Day #)</td>
<td>vel (Day #)</td>
</tr>
<tr>
<td>1</td>
<td>L</td>
<td>284 (13)</td>
<td>262 (16)</td>
<td>170 (21)</td>
</tr>
<tr>
<td></td>
<td>R</td>
<td>224</td>
<td>230</td>
<td>208</td>
</tr>
<tr>
<td>2</td>
<td>L</td>
<td>306 (10)</td>
<td>184 (13)</td>
<td>148 (20)</td>
</tr>
<tr>
<td></td>
<td>R</td>
<td>302</td>
<td>188</td>
<td>122</td>
</tr>
<tr>
<td>3</td>
<td>L</td>
<td>108 (3)</td>
<td>122 (7)</td>
<td>164 (9)</td>
</tr>
<tr>
<td></td>
<td>R</td>
<td>178</td>
<td>178</td>
<td>240</td>
</tr>
<tr>
<td>4</td>
<td>L</td>
<td>75 (4)</td>
<td>209 (10)</td>
<td>177 (12)</td>
</tr>
<tr>
<td></td>
<td>R</td>
<td>104</td>
<td>300</td>
<td>260</td>
</tr>
<tr>
<td>5</td>
<td>L</td>
<td>74 (2)</td>
<td>128 (8)</td>
<td>82 (10)</td>
</tr>
<tr>
<td></td>
<td>R</td>
<td>60</td>
<td>160</td>
<td>198</td>
</tr>
<tr>
<td>6</td>
<td>L</td>
<td>224 (5)</td>
<td>290 (6)</td>
<td>302 (7)</td>
</tr>
<tr>
<td></td>
<td>R</td>
<td>80</td>
<td>—</td>
<td>—</td>
</tr>
</tbody>
</table>

*Velocities in cm/sec.
†Days after hemorrhage (day 1 = day of bleed).
MCA = middle cerebral artery; L = left; R = right.

blood pressure, indicating persistent severe vasospasm, TCD peak velocity remained elevated. TCD peak velocity later diminished and pressors were successfully withdrawn. TCD may be useful in monitoring the development and resolution of symptomatic vasospasm following subarachnoid hemorrhage and guiding the timing of therapeutic measures.

Including the previous patient, six patients with subarachnoid hemorrhage were studied serially between 3 and 21 days after hemorrhage (table 1). Two were symptomatic; 4 had no neurological symptoms or signs. Symptomatic patients (Pt. 1, 2) had MCA peak velocities of 306 cm/sec and 284 cm/sec on the symptomatic side and 302 cm/sec and 224 cm/sec on the asymptomatic side respectively, at the time neurological deficits first appeared. Highest MCA peak velocities in asymptomatic patients were 302, 300, 240 and 198 cm/sec. The symptomatic patients became asymptomatic associated with decreases in peak velocity from 262 to 170 cm/sec in one and 306 to 184 cm/sec in the other. Angiography was performed in two patients (Pts. 3, 4) when TCD peak velocity was increased. MCA vasospasm was confirmed angiographically in both studies. No vasospasm was seen on angiography in one patient (Pt. 2) after TCD peak velocity had returned to normal.

Middle Cerebral Artery Stenosis

Case 2

A 70 year old man had several 5 minute spells over 4 days of left facial weakness and clumsiness of the left hand. He did not appear to be aware of his deficit during the spells. The following day, he dropped a paper out of his left hand and suddenly fell to the ground with pronounced left sided weakness. This cleared over the next 24 hours except for mild residual left hand clumsiness and left facial weakness. He had no further spells on heparin until 4 days later when he again developed left face, arm and leg weakness, slurred speech and left arm numbness. He was unaware of the deficit. Two hours later strength returned and dysarthria cleared. TCD examination of the right MCA at a depth of 50 mm showed increased peak velocity (290 cm/sec) with spectral broadening (fig. 2-A). At 60 mm, peak velocity (82 cm/sec) was normal (fig. 2-B); left MCA peak velocity was also normal (90 cm/sec). An arterial digital subtraction angiogram (fig. 3) demonstrated a focal area of stenosis in the distal right MCA near its bifurcation. The more proximal MCA was not appreciably narrowed.

Comment

MCA stenosis presumably due to atherosclerotic disease was detected by TCD at a depth of 50 mm. Normal peak velocity at 60 mm suggested the lesion was confined to the distal MCA and this was confirmed angiographically. Localization of an MCA lesion may be possible when a focal segment of stenosis is confined to the proximal or distal segment of the MCA.

Case 3

An 80 year old man had two episodes of speech difficulty and right arm weakness, the second resolving slowly over several days. Carotid noninvasive studies of the extracranial vessels showed no hemodynamically significant stenosis. Peak velocity in the left MCA was 100 cm/sec at depths of 40 to 60 mm.

FIGURE 2.  Transcranial Doppler studies of the right middle cerebral artery at depths of 50 (top) and 60 (bottom) mm in a patient with symptoms of right hemisphere ischemia (case 2). Increased peak systolic velocity and spectral broadening were seen at 50 mm but not 60 mm. Cursor indicates peak systolic velocity for each trace.
On the right, peak velocity at 60 mm was 98 cm/sec, but at 40 mm peak velocity was 164 cm/sec with spectral broadening (fig. 4). Angiography demonstrated several small branch occlusions in the distal left MCA territory suggesting previous embolism. An area of stenosis was seen in the proximal superior division of the right MCA. The lumen was narrowed by approximately 50% (fig. 5).

**Comment**

The abnormal right MCA Doppler signal was an incidental finding with stenosis later confirmed angiographically. The stenosis was asymptomatic and did not delay filling of distal MCA branches. TCD may allow detection of less severe MCA stenosis prior to development of symptoms or reduction of cerebral blood flow.

**Extracranial ICA Stenosis**

**Case 4**

A 65 year old man had 4 episodes in 5 days of transient blindness in the left eye associated with numbness over the left cheek. Angiography demonstrated severe stenosis of the left extracranial internal carotid artery (ICA) with residual lumen diameter less than 1 mm. The left external carotid artery filled before the internal carotid artery (fig. 6). The right extracranial ICA was also stenotic with a residual lumen diameter of 2.5 mm and no delay of flow. Both anterior cerebral arteries were visualized from the right carotid injection but there was no crossfilling of the left MCA. TCD examination initially showed peak velocity of 52 cm/sec in the left MCA and 106 cm/sec in the right MCA (fig. 7-A). Left to right MCA peak velocity ratio was greater than 2 standard deviations below the mean (0.49). One day following left carotid endarterectomy peak velocity increased from 52 to 126 cm/sec in the left MCA, and from 106 to 132 cm/sec in the right MCA (fig. 7-B). Left to right MCA peak velocity ratio increased to 0.95.

**Comment**

MCA peak velocity was reduced ipsilateral to a severe carotid stenosis, when compared with the contralateral MCA. Peak systolic MCA velocities equalized following endarterectomy. This suggests the reduction in ipsilateral peak MCA velocity prior to endarterectomy was due to carotid stenosis.

**Case 5**

A 56 year old woman awoke with speech difficulty and right arm paralysis. Her symptoms improved over

---

**Figure 3.** Arterial digital study of the right middle cerebral artery in the same patient (case 2) showing stenosis in the distal segment.

**Figure 4.** Transcranial Doppler study from the right middle cerebral artery in case 3. Increased peak systolic velocity occurred at depths of 40 mm and 50 mm but not at 60 mm. Cursor indicates peak systolic velocity in each trace.
FIGURE 5. Right carotid angiogram from the same patient (case 3) demonstrating 50% stenosis of the proximal superior division of the middle cerebral artery. The artery appeared normal in the middle cerebral artery stem.

the next several hours, however, some residual aphasia persisted. CT scan demonstrated a small left frontal infarct. Carotid noninvasive studies suggested bilateral hemodynamically significant extracranial ICA stenosis. On angiography, both ICAs were narrowed to approximately 1 mm at the bifurcation. The external carotid artery filled before the internal carotid artery bilaterally. Both anterior cerebral arteries filled from the left carotid injection indicating a patent anterior communicating artery. Only the right anterior cerebral artery filled from the right carotid injection. TCD at a depth of 55 mm initially showed peak velocity of 58 cm/sec in the left MCA and 66 cm/sec in the right MCA (fig. 8-A). She underwent left carotid endarterectomy and postoperatively had paralysis of the left leg with a new right anterior cerebral artery territory infarct on CT scan. TCD of the MCA one day and 6 days postoperatively showed an increase in peak velocity (182 cm/sec) on the left and a smaller increase (98 cm/sec) on the right (fig. 8-B). Left to right MCA peak velocity ratio was reduced (0.54). Eight days after left endarterectomy she underwent right carotid endarterectomy without complication. TCD one day after the second procedure showed a further increase in peak velocity on the right to 142 cm/sec (fig. 8-C). Peak systolic velocity on the left decreased from 182 to 144 cm/sec. Left to right MCA ratio became normal (1.01).

Comment

Initial MCA peak systolic velocities were in the low range of normal bilaterally. Left carotid endarterectomy increased both ipsilateral and contralateral peak MCA systolic velocities; more on the operated side. Following the second endarterectomy, peak MCA systolic velocities equalized. The initial reduced peak MCA velocity bilaterally possibly reflected lack of crossflow from the contralateral carotid despite a patent anterior communicating artery. Following left carotid endarterectomy, left MCA peak velocity increased as expected but right MCA peak velocities also increased, perhaps because of additional crossflow from the left carotid. Left to right MCA peak velocity ratio was reduced postoperatively because of the remaining severe stenosis of the right internal carotid artery.

Case 6

A 56 year old woman had sudden onset of global aphasia and right hemiplegia preceded by several brief episodes of right hand numbness. Over the next several weeks there was return of strength in the right leg but little improvement in speech. Angiography two months later demonstrated left internal carotid artery occlusion and severe stenosis (residual lumen 1 mm) of the right internal carotid artery at the bifurcation. There was no crossfilling of the left MCA from the right carotid injection. The left MCA filled from the posterior communicating artery and from reconstitution of the left carotid via muscular branches of the left vertebral. Transcranial Doppler studies prior to right endarterectomy demonstrated peak systolic velocities of 84 cm/sec on the right and 45 cm/sec on the left. Left to right MCA ratio was 0.54. Following endarterectomy peak MCA velocities increased on the right to 118 cm/sec. On the left there was only a small increase to 57 cm/sec. Left to right MCA ratio remained low (0.48).

Comment

The right carotid artery did not appear to contribute to left MCA flow across the anterior circle of Willis. Following right carotid endarterectomy right MCA peak velocity increased appropriately but left MCA peak velocity increased only slightly and remained

FIGURE 6. Left carotid arteriogram in case 4. There is severe stenosis of the internal carotid artery at the bifurcation (left). Lateral head view (right) shows filling of external carotid artery before the internal carotid artery.
FIGURE 7. Transcranial Doppler studies of the middle cerebral arteries from case 4 before (7A) and after (7B) left carotid endarterectomy. Before surgery, peak systolic velocity was reduced on the left. Left/right middle cerebral artery peak velocity ratio (0.49) was reduced. Following left endarterectomy ipsilateral middle cerebral artery peak velocity more than doubled (126 cm/sec), and left/right ratio became normal (0.95). Cursor indicates peak systolic velocity for each trace.

Discussion

Our preliminary experience suggests that TCD may become a useful, noninvasive method for diagnosing and following arterial spasm and MCA stenosis. TCD may also provide an independent measure of the severity of hemodynamic change in patients with extracranial carotid disease, and allow noninvasive detection of collateral flow across the anterior circle of Willis. The combination of TCD and extracranial noninvasive studies in some cases may avoid the need for angiography.

Few normal values for blood flow velocities in the intracerebral arteries have been published2-4 and most have been mean velocities rather than peak systolic velocities. Peak velocity should be more reproducible since it is visually easy to identify on an oscilloscope sweep of Doppler shift frequencies. When reflected signal quality is poor, determination of mean velocity may be particularly difficult. Variability in TCD values over time or between examiners has not been tested, although such studies are necessary. Our experience with a few patients indicates that similar values can be obtained by repeated studies in normal and stenotic vessels. Increases in MCA peak velocity in patients with vasospasm, and changes following endarterectomy are large enough to indicate a physiologic change rather than intertrial variability.

Increased peak systolic velocities were detected by TCD in patients with subarachnoid hemorrhage and MCA vasospasm. Doppler abnormalities were similar to abnormalities produced by extracranial stenosis.4 In two asymptomatic patients, vasospasm was confirmed by angiography and in two others Doppler abnormalities correlated with clinical evidence of vasospasm. Vasospasm could be followed from severe narrowing to eventual resolution. In two of our patients, velocities decreased over several days associated with resolution of symptoms. Initial TCD was performed within the first few days after subarachnoid hemorrhage in 4 patients. Abnormalities were detected as early as 3 days after hemorrhage confirming that mild vasospasm may begin earlier than clinical symptoms of vasospasm appear. Detection of vasospasm before symptoms develop may allow initiation of therapy at a time when intervention is more effective.

In a previous study of patients with vasospasm after subarachnoid hemorrhage,6 MCA peak velocity correlated well with residual lumen diameter. Angiographic data in our patients was insufficient to assess this correlation. Severity of Doppler abnormalities, however, did not consistently predict development of symptoms.
Some asymptomatic patients had peak velocities that were as high or higher than symptomatic patients. In one patient, peak velocities were as high in the MCA on the asymptomatic side, as the MCA on the side appropriate to symptoms. Patients with severe vasoelastic do not always develop symptoms and this may account for the observed discrepancy. Collateral flow from the anterior cerebral artery might provide enough blood flow in some patients to prevent ischemia.

MCA stenosis due to atherosclerosis or other pathologic processes can also be detected by TCD. Peak systolic velocities were increased with spectral broadening in stenotic arteries. TCD should provide information regarding the natural history of MCA stenosis, and the relationship between severity of stenosis and production of symptoms. Progression or regression of stenosis after treatment can be monitored by TCD.

Peak systolic MCA velocities in patients with severe extracranial ICA stenosis were mostly in the low range of normal or below. Systolic peaks appeared blunted, reducing the peak systolic and end diastolic difference. Increases in ipsilateral MCA peak velocity in two patients following endarterectomy indicated that initial
low peak velocities were related to the stenosis. MCA peak velocity was low normal or below in the absence of angiographic crossflow from the contralateral ICA, and well within the normal range in one patient (case 5) at a time when crossflow should have been present. Abnormal left to right MCA peak velocity ratio identified unilateral ICA stenosis when peak velocity was not reduced on either side.

Lindegaard et al.5 studied patients with extracranial carotid artery stenosis of varying severity. MCA mean velocity did not strongly correlate with severity of stenosis. A broad range of MCA mean velocities was found in patients with severe ICA stenosis or occlusion. Our studies suggest that MCA peak systolic velocities may be influenced by collateral flow from the contralateral ICA across the anterior circle of Willis. Lindegaard et al did not stratify MCA mean velocities according to collateral flow, perhaps accounting for the large variability in MCA mean velocities in their patients with severe stenosis or occlusion.5 Increased ACA and posterior cerebral artery mean velocities were found when these arteries were major sources of collateral flow.5 TCD studies of these vessels, in addition to MCA peak velocities, may allow noninvasive determination of patterns of collateral flow in patients with extracranial ICA disease.

Our experience with TCD includes only a few cases, and angiographic correlation in patients with vasospasm has been minimal. More studies are needed to document the diagnostic accuracy of TCD and to test the validity of these initial impressions in larger groups of patients. TCD should have other applications in patients with cerebrovascular disease such as defining pathophysiology (low flow vs. embolic) of stroke or TIA, and detecting lysis of MCA stem emboli. We have also found the technique useful in clinical situations not directly related to cerebrovascular disease, such as raised intracranial pressure and brain death. Patients in this report illustrate the potential usefulness of this noninvasive technique and, hopefully, will stimulate additional investigations.

Acknowledgments
The authors would like to thank Dr. Rakish Agrawal and Ms. Kim Layckx for assistance with collection of normal data. We are also grateful to Dr. Laligam Sekhar for his helpful comments and Ms. Linda Szalla for preparing this manuscript. Teca Corp. provided the transmural Doppler for research purposes.

References
Transcranial Doppler in cerebrovascular disease.
L R Wechsler, A H Ropper and J P Kistler

*Stroke*. 1986;17:905-912
doi: 10.1161/01.STR.17.5.905

Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1986 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/17/5/905

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org/subscriptions/