Usefulness of Transcranial Color-Coded Sonography in the Diagnosis of Cerebral Vasospasm

F. Proust, MD; F. Callonc, MD; E. Clavier, MD; J.P. Lestrat, MD; D. Hannequin, MD; J. Thiébot, MD; P. Fréger, MD

Background and Purpose—The noninvasive diagnosis of cerebral vasospasm with the use of conventional transcranial Doppler ultrasonography (TCD) is based on a velocity study of the middle cerebral artery (MCA). The authors report a prospective comparative study between transcranial color-coded sonography (TCCS), conventional transcranial Doppler (TCD), and angiography in the diagnosis of cerebral vasospasm after surgical treatment for aneurysm.

Methods—Thirty consecutive patients underwent routine angiography after surgical treatment for intracranial aneurysm. The distribution of vasospasm was determined after a prospective calculation of the angiographic diameter of the MCA, internal carotid artery (ICA), and anterior cerebral artery (ACA). The blood flow velocities (systolic and maximum) of the MCA, ICA, and ACA were evaluated by TCCS and TCD.

Results—The correlation between mean maximum velocity and angiographic diameter was significant for the MCA ($r = -0.637$, $P < 0.0001$), ICA ($r = -0.676$, $P < 0.0001$), and ACA ($r = -0.425$, $P < 0.01$). TCCS sensitivity and specificity were higher than those for TCD for MCA (100% and 93%, respectively) and ICA (100% and 96.6%, respectively). For ACA, the sensitivity and specificity were 71.4% and 84.8%, respectively.

Conclusions—The authors suggest that TCCS is useful for accurate monitoring of cerebral vasospasm in the MCA and ICA. In the ACA, TCCS monitors the hemodynamic state of the anterior part of the circle of Willis, which could expose the patient to a delayed ischemic deficit. (Stroke. 1999;30:1091-1098.)

Key Words: blood flow velocity ■ cerebral aneurysm ■ ultrasonography, Doppler, transcranial ■ vasospasm

The incidence of symptomatic cerebral vasospasm (delayed ischemic deficit) has been reported to vary between 12% and 57% in cases of subarachnoid hemorrhage (SAH).1-3 At the present time, monitoring of cerebral vasospasm has played a major role in the efficacy of preventive therapeutic methods.4-6 The usefulness of conventional transcranial Doppler ultrasonography (TCD) has been clearly demonstrated in the diagnosis of cerebral vasospasm by measurement of blood flow velocity in the middle cerebral artery (MCA).7-13

TCD alone is not sufficiently sensitive to detect cerebral vasospasm in the internal carotid artery (ICA) and the anterior cerebral artery (ACA),14-17 which is explained in part by difficulty in the detection of basal cerebral arteries. This obstacle could be avoided by using transcranial color-coded sonography (TCCS). TCCS is a procedure that visualizes the arteries and permits registration of angle-corrected flow velocities in basal cerebral arteries.18 This approach has been used in different clinical situations.19-24 To our knowledge, the use of TCCS has not been reported to evaluate cerebral vasospasm after surgical treatment of ruptured aneurysms. This comparative study evaluates the correlation between blood flow velocity calculated with TCCS, TCD, and angiographic diameter of the residual arterial lumen of the MCA, ICA, and ACA after surgical treatment of intracranial aneurysm. The main focus of this study is the ability of TCCS to determine cerebral vasospasm distribution and in particular to improve the diagnosis of cerebral vasospasm in the ICA and ACA by comparison with TCD.

Subjects and Methods

Population
Thirty consecutive patients who underwent surgery for an intracranial aneurysm (17 women, 13 men; mean age, 46 ± 14 years) were included in the study. SAH had occurred in 26 patients. According to the classification of Hunt and Hess,25 1 patient was classified as grade I (3.8%), 9 as grade II (34.6%), 13 as grade III (50%), and 3 as grade IV (11.5%). The aneurysm was incidentally discovered in the other 4 patients. The aneurysms were located at the MCA in 7 patients (23.3%), the ICA in 9 (3%), and the anterior communicating artery in 14 (46.7%). After SAH, the patients were managed according to a standard treatment that included the following: comprehensive intensive care; early surgical management depending on the date of hospitalization (surgical timing: 3.76 ± 5.98 days after SAH); ventricular derivation and intracranial pressure monitoring in 12 patients; and aggressive prevention and management of cerebral vasospasm, including nimodipine administration, postoperative hypervolemia, and daily TCD. At 6 months, according to the Glasgow
Outcome Scale (GOS) score,26 23 patients were in GOS 5 (76.7%), 6 in GOS 4 (20%), and 1 in GOS 2 (3.3%). In each patient, routine angiography was performed at postoperative day 10. Postoperative angiography consisted of unilateral carotid angiogram on the side of the clipped aneurysm in 20 patients and bilateral carotid angiograms in 10 patients. The 40 postoperative carotid angiograms constituted the control examination to evaluate the Doppler methods. TCCS and TCD were performed on the same day without reference to the angiography results.

Angiographic Cerebral Vasospasm

Carotid angiography was performed by the transfemoral route under local anesthesia by one of us (E.C.) using Selnderg’s technique. The larger diameter of 3 segments (horizontal portion of MCA, ICA, and horizontal portion of ACA) was measured at the anteroposterior location for each carotid angiogram by automatic evaluation (Siemens angiography system) both preoperatively and postoperatively (Figure 1). Our study concentrated on analysis of the postoperative cerebral vasospasm. The definition of the postoperative cerebral vasospasm was related to a preoperative cerebral vasospasm of the corresponding segment. On each of 60 preoperative carotid angiograms, a visual interpretation of cerebral vasospasm was performed by 3 independent experts (F.C., P.F., F.P.). Cerebral vasospasm was considered present only when all 3 experts agreed that the segment was abnormal.

On each of 60 preoperative and 40 postoperative carotid angiograms, we calculated the diameters of the MCA at 2 cm from the ICA bifurcation and of the ACA at 1 cm to compare the Doppler data at the same location. Postoperatively, cerebral vasospasm was defined as an arterial caliber reduction >25% as determined by calculating the caliber variation on each segment between preoperative and postoperative carotid angiograms. The reference caliber was the diameter of each preoperative segment evaluated without cerebral vasospasm by visual interpretation. In cases of preoperative segments with spasm or when the experts disagreed regarding visual interpretation, the value of caliber used as reference was the mean value measured with the segment without spasm.

Postoperative Blood Flow Velocity Measurements With TCCS

For TCCS, we used an ultrasound system with color-added blood flow representation (HDI 3000 ATL) and a transducer with aperture size of 19 mm. We applied the probe to the preauricular area of the skull, focusing the ultrasound beam and the depth at the point of insonation for each arterial segment was as follows: MCA, 60 mm; ICA, 60 mm; ACA, 40 mm. The Doppler sample volume (4 to 6 mm3) was placed within the color flow image of the artery under insonation (Figure 2). The mean depth of insonation in each artery was not reported because the vessels were frequently not seen in their entire length. The angle of insonation was 6° to 50°. The mean depth of insonation was arbitrarily fixed at 50°. The velocities recorded for the ICA were only an estimation of the blood flow from this segment because of its anatomic disposition, perpendicular to the ultrasound beam. These measurements were performed by 2 independent examiners (F.C., J.P.L.).

Postoperative Blood Flow Velocity Measurements With TCD

Comparative TCD was performed by a third examiner (I.F.). For TCD, we used a 2.0-MHz transducer of a pulsed-wave Doppler sonography instrument with an energy output level corresponding to 100 mW per squared spatial peak-temporal average intensity, according to the method of Aaslid et al.7 The velocities reported were obtained in the proximal portion of the MCA, ICA, and ACA. The sample volume size was 8 to 10 mm in the axial and 5 mm in the lateral direction at a depth of 40 mm for the MCA, 60 mm for the ICA, and 70 mm for the ACA. Vm, Vs, PL, and RI were measured.

Data Analysis

All parameters were reported as mean ± SD. For the angiographic diameter and the velocity measurements, the comparison TCCS/TCD between segments with and without spasm was performed with Kruskal-Wallis 1-way ANOVA. The correlations between angiographic diameters and TCD or TCCS measurements were tested with the Spearman rank correlation method. The correlation was considered significant at P<0.01. By performing a receiver operating characteristic curve, we determined the optimal threshold Vm values using TCCS and TCD for MCA, ICA, and ACA segments. These values permitted us to calculate the sensitivity and specificity for each segment with both Doppler methods.

Results

Angiographic Studies

By visual interpretation of 60 preoperative carotid angiograms, cerebral vasospasm was present in 3 patients (3 MCA, 5 ICA, and 3 ACA). Cerebral vasospasm in 40 postoperative carotid angiograms was present in 17 patients (7 MCA, 10 ICA, and 11 ACA). The angiographic diameters of the MCA, ICA, and ACA are reported in Table 1. The difference between arteries with and without spasm was significant for all 3 segments (F=45.26, df=38, P=10−6 for MCA; F=32.26, df=38, P=2.10−6 for ICA; F=20.66, df=38, P=6.10−3 for ACA).

Velocimetric Studies

In postoperative TCCS imaging, 95% (38/40) of MCA, 95% (38/40) of ICA, and 95% (38/40) of ACA were isonated. The poor acoustic bone window in 2 patients prevented insonation of their MCA, ICA, and ACA segments. The velocity index (Vm, Vs, PL, RI) was calculated in 100% of 38 MCA, 38 ICA, and 38 ACA isonated. The mean angle of insonation for each arterial segment was as follows: MCA, 21±14.6° (range, 0° to 46°); ICA, 50°; ACA, 28±15.6° (range, 0° to 50°). The mean depth of insonation in each artery was not reported because the vessels were frequently isonated more often by the operative flap side. With the TCD method, reliable flow signals could be obtained in the 40 M1, ICA, and ACA segments (100%).

With TCCS and TCD, there was no difference between segments with and without spasm for PL and RI. The mean Vm and Vs are reported in Table 1. The differences between arteries with and without spasm were significant for all 3 segments with TCCS (F=9.77, df=38, P=3.10−4 for Vm and F=55.32, df=38, P=10−6 for Vs for MCA; F=33.35, df=38,
Figure 1. On the anteroposterior view of the carotid angiogram, the larger angiographic diameter was automatically calculated for the horizontal portion of the MCA, the ICA, and the proximal portion of the ACA.
There were also significant differences with TCD, except for the $V_m$ for ACA ($F = 19.51, df = 38, P < 0.001$ for $V_s$ and $F = 30.91, df = 38, P = 0.0001$ for $V_m$ for MCA; $F = 19.03, df = 38, P = 0.001$ for $V_s$ and $F = 28.5, df = 38, P = 0.507, P < 0.0001$ for $V_m$, respectively). For the ICA, these correlations were also demonstrated after measurement by TCCS ($r = -0.463, P < 0.001$ and $r = -0.637, P < 0.0001$, respectively) and by TCD ($r = -0.507, P < 0.0001$ and $r = -0.599, P < 0.0001$, respectively). For the ACA, these correlations were also demonstrated after measurement by TCCS ($r = -0.711, P < 0.0001$ and $r = -0.676, P < 0.0001$, respectively) and by TCD ($r = -0.352, P < 0.01$ and $r = -0.430, P < 0.01$, respectively). At the ACA, $V_s$ and $V_m$ were correlated with the angiographic diameter after measurement by TCCS ($r = -0.416, P < 0.01$ and $r = -0.425, P < 0.01$, respectively). Conversely, there was no correlation for $V_s$ and $V_m$ after measurement by TCD. There was a significant correlation between TCCS and TCD measurements of $V_s$ and $V_m$ recorded in MCA and ICA ($r = -0.614, P < 0.001$ for $V_s$, and $r = -0.778, P < 0.001$ for $V_m$; $r = -0.465, P < 0.01$ for $V_s$, and $r = -0.608, P < 0.001$ for $V_m$, respectively). There was no correlation in the ACA for either $V_s$ or $V_m$. TCCS values were higher than those obtained by TCD, except for the $V_m$ of MCA, in which case they were equivalent.

### Sensitivity and Specificity

Measurements of $V_m$ by TCCS and angiographic diameters for the 3 segments are represented in Figures 3, 4, and 5. The

---

**TABLE 1. Comparison of Flow Velocity Recording by TCD and TCCS With Angle Correction in MCA, ICA, and ACA Segments**

<table>
<thead>
<tr>
<th>Vessels and Methods</th>
<th>No Spasm</th>
<th>Spasm</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$V_s$</td>
<td>$V_m$</td>
<td></td>
</tr>
<tr>
<td>MCA</td>
<td>n=31</td>
<td>n=7</td>
<td>$10^{-6}$</td>
</tr>
<tr>
<td>Diameter, mm</td>
<td>2.11±0.32</td>
<td>1.35±0.28</td>
<td></td>
</tr>
<tr>
<td>TCCS</td>
<td>119±32</td>
<td>73±20</td>
<td></td>
</tr>
<tr>
<td>TCD</td>
<td>105±33</td>
<td>73±26</td>
<td></td>
</tr>
<tr>
<td>ICA</td>
<td>n=28</td>
<td>n=10</td>
<td>$2.10^{-6}$</td>
</tr>
<tr>
<td>Diameter, mm</td>
<td>2.86±0.44</td>
<td>1.96±0.37</td>
<td></td>
</tr>
<tr>
<td>TCCS</td>
<td>110±40</td>
<td>66±26</td>
<td></td>
</tr>
<tr>
<td>TCD</td>
<td>87</td>
<td>60±19</td>
<td></td>
</tr>
<tr>
<td>ACA</td>
<td>n=27</td>
<td>n=11</td>
<td>$6.10^{-5}$</td>
</tr>
<tr>
<td>Diameter, mm</td>
<td>1.71±0.42</td>
<td>1.40±0.36</td>
<td></td>
</tr>
<tr>
<td>TCCS</td>
<td>90±0</td>
<td>56±22</td>
<td></td>
</tr>
<tr>
<td>TCD</td>
<td>77±15</td>
<td>54±13</td>
<td></td>
</tr>
</tbody>
</table>

Flow velocities with angle correction are expressed in centimeters per second.
sensitivity and specificity obtained for the 3 segments with each \( V_m \) threshold are reported in Table 2. Values were higher for TCCS than for TCD. With the use of TCCS for MCA and ICA, the sensitivity was 100%, with a specificity of 93.3% and 96.6%, respectively. For ACA, the sensitivity and specificity were 71.4% and 84.8%, respectively.

**ACA Velocities**

In 15 patients, the anterior portion of the circle of Willis was explored fully in the preoperative and postoperative periods: 10 patients with bilateral carotid angiograms and 5 with ACA agenesis (Table 3). A unilateral cerebral vasospasm of the ACA was present in 8 patients. With the use of TCCS, only 4 ACA presented a \( V_m > 100 \, \text{cm} \cdot \text{s}^{-1} \) (patients 1, 2, 3, and 4). These arteries all exhibited spasm; 3 contralateral ACAs had agenesis, and 1 was narrowed to 20%.

**Discussion**

**Angiographic Cerebral Vasospasm**

Since its initial description by Ecker and Riemenschneider,\(^{30}\) the angiographic presentation of cerebral vasospasm appears as a concentric, smoothly contoured narrowing of the implicated vessel.\(^{31,32}\) The development of a practical noninvasive method, such as ultrasonography, to diagnose cerebral vasospasm is necessary because angiography may be a source of complications in a segment with spasm. Calculation of the velocity threshold value of cerebral vasospasm by TCD (sensitivity/specificity) depends on arterial narrowing to define the angiographic cerebral vasospasm. The criteria for angiographic cerebral vasospasm reported in the literature have been extremely varied: (1) a scale of residual lumen on studied segments,\(^{33,34}\) (2) a subjective reduction of vascular lumen from 25% to 40% in comparison with an angiogram after it has disappeared,\(^{35,36}\) and (3) an external measurement margin of the dye column made by an optical device.\(^{8,15,37–39}\) According to these different criteria, the incidence of angiographic cerebral vasospasm varied between 23.7% and 85.2%.\(^{10,31,33–35,38,40}\) In our series, the diagnostic criteria of angiographic cerebral vasospasm were doubled to be as accurate as possible in the selection of segments with spasm (subjective evaluation by 2 independent experts and objective
Blood Flow Velocities and Correlation With Arterial Diameter

As reported by Martin et al., the window was found in 100% of young patients and 70% of those older than 60 years. This is slightly worse than is expected when conventional TCD is used. Arteries were insonated successfully at a rate of 96.6% for MCA and ICA and 85% for ACA. These rates were higher than those reported by Martin et al., who found a mean rate of 77%. This difference is explained by its postoperative realization. Indeed, the operative flap increased the quality of the used window in our series.

The blood flow for the MCA was estimated on the basis of the $V_m$ or the index of Lindegaard et al. ($V_{MCA}/V_{ICA \; extra}$). In healthy subjects, the $V_m$ for the MCA ranged from 43±8.1 to 78±5 cm·s$^{-1}$ for either recording Doppler method used: TCD. For the ICA, the $V_m$ was recorded at 60±15 cm·s$^{-1}$. The $V_m$ for the ACA in healthy subjects recorded by conventional TCD varied from 47±14 to 58±9 cm·s$^{-1}$. In this latter segment, when TCCS was used in healthy subjects, the $V_m$ was 58±10 to 66±4 cm·s$^{-1}$. For the segments without spasm, the mean velocities we found corresponded to the upper limits of the range reported in the literature. This is probably due to the fact that these measurements were performed during a postoperative period, during which the velocities are increased.

The correlation between the inner angiographic diameter and the $V_m$ recorded by TCD has been clearly demonstrated for the MCA ($r=0.54$, $P<0.001$). The correlation for the ICA with TCD ($r=0.758$, $P<0.001$) has only been reported by Harders and Gilbash. In our series, an excellent correlation between diameter and velocity for the MCA was the same for each Doppler method. For the ICA, the correlation with TCCS ($r=0.425$, $P<0.001$) was lower than those obtained for MCA and ICA ($P<0.001$).

TABLE 2. Sensitivity and Specificity of TCD and TCCS in Diagnosing Vasospasm in MCA and ICA in Our Series of 30 Patients

<table>
<thead>
<tr>
<th>Doppler Method</th>
<th>Threshold Value, cm/s</th>
<th>Sensitivity, %</th>
<th>Specificity, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>MCA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TCCS</td>
<td>120</td>
<td>100</td>
<td>93.3</td>
</tr>
<tr>
<td>TCD</td>
<td>122</td>
<td>83.3</td>
<td>93.7</td>
</tr>
<tr>
<td>ICA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TCCS</td>
<td>120</td>
<td>100</td>
<td>96.6</td>
</tr>
<tr>
<td>TCD</td>
<td>100</td>
<td>85.7</td>
<td>87.8</td>
</tr>
<tr>
<td>ACA</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TCCS</td>
<td>75</td>
<td>71.4</td>
<td>84.8</td>
</tr>
<tr>
<td>TCD</td>
<td>...</td>
<td>...</td>
<td>...</td>
</tr>
</tbody>
</table>

For the ACA, different reports of angiographic diameter and TCD velocity have previously shown no or low significant correlations. Lennihan et al. observed no significant difference between the $V_m$ of ACAs with and without spasm. In our series, with TCCS we obtained a correlation between velocity and diameter that was significant ($r=0.425$, $P<0.001$) but lower than those obtained for MCA and ICA ($P<0.001$). Lindegaard et al. registered a high velocity for ACA only when the cerebral vasospasm was bilateral. In our experience, the visualization of the ACA segment is fundamental to superimpose the sample volume on the studied vessel and the aliasing area. It represents significant progress compared with the previous difficulty in locating this segment with TCD. This difficulty is related to various factors: its short length, its small caliber, its sinuous course, and agenesis present in approximately 15% of patients. The use of echo-contrast agents may improve...
the visualization of the ACA and help in the location of the sample volume.

Sensitivity and Specificity
When only studied for cerebral vasospasm of the MCA segment, the values of sensitivity varied from 73% to 80%, and those for specificity varied from 89% to 100%.9,38,44,45 Since the MCA segment alone was studied, the overall sensitivity decreased to 58.6% because isolated cerebral vasospasm of other segments was not detected.38 This result prompted cerebral vasospasm diagnosis for other basal cerebral arteries. TCCS revealed cerebral vasospasm not only of the MCA but also of the ICA (see Results). Using TCCS, in this series we have determined the threshold for the MCA with the best sensitivity and specificity. This cerebral vasospasm threshold, 120 cm s⁻¹, is near those described for conventional TCD, reported to range from 100 to 140 cm s⁻¹.8,10,15,45 This is not surprising because Shöning et al,29 in healthy volunteers, showed a velocity flow for the MCA higher (61 ± 13 cm s⁻¹) by conventional TCD than that recorded by TCCS (58 ± 12 cm s⁻¹). There is a slight difference between the 2 methods for the MCA, because in standard TCD, the axis of the ultrasound beam is easily aligned with the axis of the MCA segment. Therefore, the angle correction for the MCA segment is very weak. A diagnostic problem remains for the ACA because the sensitivity and specificity were not sufficient at this segment. The correlation existed for the segments without spasm but disappeared when the vessel exhibited spasm. The false-negative TCCS results for ACA with spasm may be explained by different factors. As suggested by Lindegård et al,15 the functional role of anastomosis of the anterior communicating artery between the ACAs is a fundamental cause of the absence of correlation between diameter and some segments with spasm. In our series we observed either acceleration flow or absorption flow in the ACA with spasm. The acceleration flow seemed to occur in a single ACA with contralateral agenesis, and the absorption flow seemed to occur when the contralateral ACA was functional. Moreover, the agitation of the patients sometimes made the progress of the examination difficult.

In practice, diagnosis of unilateral cerebral vasospasm of the ACA is not obligatory because its hemodynamic consequences for the downstream flow are generally not a cause for concern. Conversely, unilateral cerebral vasospasm of a single ACA or bilateral cerebral vasospasm may cause hemodynamic consequences. In these latter situations, the TCCS demonstrates variations of velocity and may help to monitor cerebral vasospasm that extends to the anterior portion of the circle of Willis.

Conclusion
TCCS is a practical noninvasive bedside method that provides an accurate evaluation of distribution of cerebral vasospasm in the MCA and ICA segments. TCCS is also useful to detect acceleration of velocity in the ACA despite the fact that this type of acceleration was only observed in the adverse situation in which all segments composing the anterior portion of the circle of Willis exhibited spasm. TCCS, with sensitivity and specificity higher than those provided by TCD, should be considered the method of choice in the noninvasive diagnosis of cerebral vasospasm.

Acknowledgments
We are grateful to Richard Medeiros for advice in editing the manuscript, to Jean-François Menard for assistance with statistical analysis, and to Isabelle Fedina for technical expertise in TCD examination.
References
30. Ecker A, Riemschneider P. Arteriographic demonstration of spasm of the intracranial arteries: with special reference to saccular arterial aneu-
37. Grosset DG, Straiton J, McDonald I, Bullock R. Angiographic and Dopp-
ler diagnosis of cerebral artery vasospasm following subarachnoid haem-
42. Crompton JS, Redmond S, Symon L. Cerebral blood velocity in subarachnoid haemorrhage: a transcranial Doppler study. J Neurol Neu-
Usefulness of Transcranial Color-Coded Sonography in the Diagnosis of Cerebral Vasospasm

F. Proust, F. Callonec, E. Clavier, J. P. Lestrat, D. Hannequin, J. Thiébot and P. Fréger

Stroke. 1999;30:1091-1098
doi: 10.1161/01.STR.30.5.1091

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/30/5/1091