Comparison of Cerebral Angiography and Transcranial Doppler Sonography in Acute Stroke

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We compared digital intra-arterial angiography and transcranial Doppler sonography in acute cerebral ischemia as part of a wider study on a continuous series of 48 patients with acute focal cerebral ischemia in the carotid territory, observed within 4 hours of the onset of symptoms. The most significant Doppler findings of the middle cerebral artery included no detection of the artery when occlusion of the carotid siphon or the middle cerebral artery at its origin was shown by angiography and reduced flow velocities and asymmetry (symptomatic < asymptomatic) when the occlusion was located in the terminal tract of the middle cerebral artery mainstem or in numerous terminal branches. Higher flow velocities in the anterior cerebral artery or posterior cerebral artery, mostly in the symptomatic hemisphere, often accompanied middle cerebral artery pathology, probably indicating collateral compensatory pathways. (Stroke 1989;20:899–903)

The high frequency of acute intracranial occlusion and the therapeutic use of active thrombolytic agents stress the need to monitor the patency of intracranial cerebral arteries in acute ischemic strokes.1 Noninvasive exams should represent the first choice in detecting the occurrence of intracranial arterial occlusions in the very early phase and in following their natural course and responsiveness to therapies.2 The aim of this report is to validate transcranial Doppler (TCD) with respect to cerebral angiography, when both are performed within a short interval, and to define some guidelines for interpretation of TCD findings in acute cerebral ischemia.

Subjects and Methods

We examined 48 patients (22 men, 26 women; mean age±SD, 66.2±9.7 years) who had focal cerebral deficits of acute onset due to hemispheric cerebral ischemia. Clinical observation occurred within 4 hours of the onset of the stroke, and within the following 2 hours, all patients underwent a computed tomography (CT) scan, TCD, and intra-arterial cerebral angiography. The last two tests were scored independently and blindly.

Digital cerebral angiography3 was limited to the symptomatic carotid territory and performed either by direct injection or by humeral route. In four patients with occlusion of the internal carotid artery (ICA), an additional contralateral angiographic study was performed. Pathological angiographic findings were classified in the following six groups: 1) absence of lesions or presence of nonstenosing plaques at the origin of the ICA; 2) occlusion of the ICA in the neck; 3) "tandem occlusion," including ICA occlusion in the neck and middle cerebral artery (MCA) mainstem occlusion; 4) occlusion of the ICA in the siphon; 5) MCA mainstem occlusion; and 6) occlusion of MCA peripheral branches.

The TCD was performed with TC 2-64 EME equipment (Uberlingen, FRG) with a 2-MHz probe and a spectral analyzer to study the MCA, the anterior cerebral artery (ACA), and the posterior cerebral artery (PCA) through the temporal window. The examination was completed within 6 hours of the onset of symptoms and repeated after 24 hours, 48 hours, and 1 week with the technique described by Aaslid et al.4-7 Carotid compression was not performed on these patients, given the acute phase of their illness.

For each artery, systolic velocity (SV), diastolic velocity (DV), and mean velocity (MV) were measured. The pulsatility index (PI)8 was calculated as PI=(SV−DV)/MV. The pulsatility transmission index (PTI)9 was also calculated for the MCAs: PTI=(PI_{MCA}/PI_{REF})×100 where PI_{MCA} and PI_{REF} are,
was shown by contralateral angiography, whereas other. MCA mainstem occlusion was evident in four cases. Occlusion of the MCA branches was detected in 17 patients.

Carotid siphon occlusion was present in four patients, the supraclinoid tract was occluded; contralateral angiography did not show MCA and ACA in the symptomatic hemisphere in one case but did show a refilling of MCA by way of the ACA in the other. MCA mainstem occlusion was evident in four cases. Occlusion of the MCA branches was detected in 17 patients.

respectively, the PI of the symptomatic MCA and the PI of another basal cerebral artery in the same patient.

Our standard parameters were obtained from routine measurements in a sample of 60 subjects (mean age, 53.6±10 years) without intracranial and extracranial vascular pathology (Table 1). Velocity values higher or lower than the mean±2 SD were considered out of normal limits. An interhemispheric asymmetry index (AI) was also calculated according to the following formula

$$AI = \left| \frac{MV_1 - MV_2}{(MV_1 + MV_2)/2} \right| \times 100$$

where MV$_1$ and MV$_2$ represent the mean velocities of homologous arteries. The threshold values for the assessment of this asymmetry (21% for MCA, 27% for ACA, and 28% for PCA) were determined as upper limits of the confidence intervals (5% of the distribution in the right tail) in the reference sample of normal subjects.

Results

TCD was unsuccessful in nine cases because of a bilaterally undetectable temporal window. Therefore, the comparison was made in 39 cases.

Normal angiograms (n=5) or nonstenosing ICA plaques (n=4) were observed in nine cases. ICA isolated occlusion in the neck was observed in two patients, one of whom underwent contralateral angiography, which showed the patency of the symptomatic MCA. ICA occlusion in the neck plus MCA occlusion (tandem lesion) was detected in three patients. In one case, the ipsilateral MCA occlusion was shown by contralateral angiography, whereas in the other two patients, a collateral flow by way of the ophthalmic artery made this diagnosis possible. Carotid siphon occlusion was present in four patients. In two cases, the lesion was located in the intracavernous tract, and these patients did not have contralateral angiography. In two other patients, the supraclavicular tract was occluded; contralateral angiography did not show MCA and ACA in the symptomatic hemisphere in one case but did show a refilling of MCA by way of the ACA in the other. MCA mainstem occlusion was evident in four cases. Occlusion of the MCA branches was detected in 17 patients.

For the symptomatic MCA, TCD data (in 15 patients) were within normal limits and without asymmetry in relation to the contralateral side. In 13 patients, the MCA flow was reduced by more than 21% in relation to the contralateral side. The absolute values of MV (cm/sec) in the MCAs of these different groups are shown in Table 2. In the only two cases of ICA occlusion in the neck, the PTI was lower than 92 as previously described by Lindegaard et al.9 Among the other groups of patients, we found no significant differences of the PI and PTI calculated at the MCA level. In 11 patients, TCD was unable to detect the MCA of the affected side although the other ipsilateral arteries were detected in all these cases, indicating a good temporal window.

In the ACA, eight patients were found to have an increased flow velocity in the symptomatic side and two patients in the contralateral one. Inverted flow was observed in three cases in the ACA of the symptomatic side. In six patients, the PCA had an increased flow velocity in the symptomatic side.

When carotid angiography showed neither extracranial stenosing pathology nor intracranial pathology (nine cases), the TCD showed normal results. No false-positives were observed.

The nine patients with occlusion of the ICA showed different pictures in relation to the level of the occlusion and to the state of collateral pathways. When the occlusion was isolated and located at the cervical level (two cases), the values of the contralateral MCA prevailed, and a reverse flow in the ipsilateral ACA was found, with an increased flow in the contralateral ACA. When the occlusion was located in the carotid siphon (four cases), the ipsilateral MCA was not detected, even in the case in which contralateral angiography showed its refilling. The findings in the other arteries were variable. In two cases with siphon occlusion in the

### TABLE 1. Values of Blood Flow Velocity in 60 Normal Subjects

<table>
<thead>
<tr>
<th>Artery</th>
<th>SV</th>
<th>DV</th>
<th>MV</th>
</tr>
</thead>
<tbody>
<tr>
<td>MCA</td>
<td>81.6±16.3</td>
<td>40.1±9.3</td>
<td>55.7±11.6</td>
</tr>
<tr>
<td>ACA</td>
<td>73.8±17.2</td>
<td>38.1±9.8</td>
<td>49.7±10.1</td>
</tr>
<tr>
<td>PCA</td>
<td>65±15.5</td>
<td>33.5±7.8</td>
<td>42.8±7.3</td>
</tr>
</tbody>
</table>

Values are in cm/sec and are mean±SD.

SV, systolic velocity; DV, diastolic velocity; MV, mean velocity; MCA, middle cerebral artery; ACA, anterior cerebral artery; and PCA, posterior cerebral artery.

### TABLE 2. Values of Mean Velocity in Middle Cerebral Arteries as Measured by Transcranial Doppler Related to Angiographic Findings

<table>
<thead>
<tr>
<th>Angiographic findings</th>
<th>Symptomatic side</th>
<th>Asymptomatic side</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICA occlusion in the neck</td>
<td>35±7</td>
<td>66±2</td>
</tr>
<tr>
<td>MCA mainstem occlusion</td>
<td>32</td>
<td>52</td>
</tr>
<tr>
<td>MCA branch occlusion (&gt;3)</td>
<td>30.75±7.76</td>
<td>47.11±10.33</td>
</tr>
<tr>
<td>MCA branch occlusion (&lt;3)*</td>
<td>35±3</td>
<td>45±1</td>
</tr>
<tr>
<td>MCA branch occlusion (&lt;3)†</td>
<td>46.66±7.63</td>
<td>46.33±9.26</td>
</tr>
<tr>
<td>Normal</td>
<td>53.75±17.35</td>
<td>49.75±16.23</td>
</tr>
</tbody>
</table>

Values are in cm/sec and are reported as mean±SD except for MCA mainstem occlusion.

ICA, internal carotid artery and MCA, middle cerebral artery.

*Diagnosed by transcranial Doppler.
†Not diagnosed by transcranial Doppler.
intracavernous tract, a very high flow velocity was found in the contralateral ACA, whereas the ipsilateral ACA was not detected. In the other two cases with occlusion in the supraclinoid tract of the carotid siphon, the ipsilateral ACA was undetected, whereas the contralateral one was within normal range. In the three cases of tandem occlusion, the ipsilateral MCA was not found. In two of these cases, the ipsilateral ACA showed orthodromic flow, and angiography revealed a collateral circulation through the ophthalmic artery. In the third case, without collateral ophthalmic circulation, the flow in the ipsilateral ACA was inverted.

When the occlusion was located before the perforating branches of the MCA (four cases: one occlusion of the MCA alone and three cases of tandem occlusion previously described), we were unable to find the MCA. The tandem occlusion findings over the ipsilateral ACA have been described above. No asymmetry of the other cerebral arteries was detected in the patient with isolated MCA occlusion.

Two of the three patients in whom the occlusion was located after the perforating branches had an undetectable MCA. In the third case, the artery was found but showed very low values of flow velocity and a notable asymmetry when compared with the contralateral one. In two of these cases, the ipsilateral ACA had a higher flow velocity than the contralateral one (asymmetry >27%); in the third case, the ipsilateral PCA was significantly increased in relation to the contralateral one (asymmetry >28%).

When more than three superficial branches were occluded (nine cases), a significant asymmetry
between the MCAs was found in eight cases (Figure 1), and in one case, the symptomatic MCA was not found. A significant asymmetry existed between the ACAs (ipsilateral>contralateral) in five cases and between the PCAs (ipsilateral>contralateral) in three cases. MCA flow velocity was symmetrical in six of eight cases in which the occlusion involved two superficial branches (Figure 2). In only two cases did the contralateral MCA exceed the symptomatic one by more than 21%. In one of these patients, ipsilateral ACA was higher than the contralateral, with a significant asymmetry; in another, this asymmetry was present for the ipsilateral PCA.

Discussion

In this report, patients with acute hemispheric ischemic stroke were examined with CT scan, carotid angiography, and TCD within 6 hours of the onset of symptoms. The time interval between TCD and angiography was less than 30 minutes, making the present series a highly homogenous one. It is well known that strokes in the acute phase are characterized by an unstable perfusional picture. In this study, the short time interval makes a comparison between the two exams very reliable. Thus, the angiographic pattern actually represents a "gold standard" for the TCD findings.

TCD was recorded in 39 of 48 patients. The inability of TCD to explore the intracranial arteries successfully has already been described by other authors. Six of eight patients with occlusion of few (one or two) MCA superficial branches at angiography had normal TCD. Therefore, the occlusion of a limited number of branches does not seem to affect the hemodynamics of the MCA, whereas in nine cases with three or more occluded branches, TCD showed a reduction of the MCA flow velocity in relation to the contralateral side. When the intracranial tract of the ICA or the MCA mainstem was occluded (11 cases), TCD was unable to detect
the MCA flow in 10 of them. This flow was also undetected in one case of siphon occlusion in which angiography showed a refilling of MCA through the contralateral ACA. In this case, it can be assumed that angiography demonstrated ineffective collateral circulation.

The use of TCD is also useful in assessing collateral pathways. For example, in extracranial occlusions, when a collateral flow was present through the external carotid-ophthalmic-internal carotid arteries, the ipsilateral ACA was orthodromic. When this refilling through the ophthalmic artery was not present, the flow in the ipsilateral ACA was inverted, thus demonstrating a collateral circulation through the contralateral ACA. When the occlusion involved only the mainstem or superficial branches of the MCA, the TCD showed collateral flow originating from the ipsilateral ACA or PCA in the 13 cases in which a collateral pathway was demonstrated by angiography. In the remaining four cases in which angiography showed no compensatory circulation, no asymmetry of the ACA or PCA was found by TCD.

The occurrence of these collateral pathways can make identification of the MCA pathology more reliable. In fact, their presence will add importance either to the lack of detection or to the asymmetry of this artery. The collateral flows would also suggest an occlusion of few superficial branches when the MCA is in normal range and without asymmetry. It appears, therefore, that TCD is reliable even in the difficult clinical situation when studying acute stroke patients.

Despite a relatively high percentage (18.7%) of technically impossible exams in this series, TCD is still proposed as a useful bedside clinical tool for immediate and sequential monitoring of patency of the intracranial arteries in the acute phase of stroke.

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References


KEY WORDS • angiography • cerebrovascular disorders • ultrasonics
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