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Transcranial Color-Coded Duplex Sonography in Unilateral Flow-Restrictive Extracranial Carotid Artery Disease

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PURPOSE: To provide transcranial color-coded duplex flow-velocity data for the basal cerebral arteries in patients with unilateral flow-restrictive extracranial carotid artery disease, and to compare these data with the flow velocities obtained in healthy control subjects. **METHODS:** Transcranial color-coded duplex sonography was performed in 78 patients with different patterns of cross flow through the anterior and posterior communicating arteries associated with unilateral obstruction (70% to 100%; 46 stenoses and 32 occlusions) of the internal carotid arteries. Peak systolic, mean, and end diastolic velocities were measured in the anterior, middle, and precommunicating and postcommunicating posterior cerebral arteries. These measurements were compared with the values obtained in 125 age- and sex-matched healthy control subjects. **RESULTS:** Patients with anterior communicating artery cross flow to the middle cerebral artery (63%) had increased peak velocity in the anterior cerebral artery and decreased peak velocity in the middle cerebral artery on the obstructed (ipsilateral) side, and increased peak velocity in the anterior cerebral artery on the unobstructed (contralateral) side. Patients with anterior communicating artery cross flow to the pericallosal artery (19%) had increased contralateral peak systolic velocity and mean anterior cerebral artery velocities. Patients without anterior communicating artery cross flow (18%) had normal peak velocities in the anterior and middle cerebral arteries. Patients with posterior communicating artery cross flow (42%) had ipsilaterally decreased peak systolic and mean middle cerebral artery velocities and increased peak velocities in the precommunicating posterior cerebral artery. Patients without posterior communicating artery cross flow (58%) had ipsilaterally decreased peak systolic and mean middle cerebral artery velocities. **CONCLUSION:** Our findings suggest that typical abnormalities of basal cerebral artery flow velocities occur in patients with unilateral 70% to 100% obstruction of the internal carotid arteries resulting in different patterns of cross flow through the circle of Willis.

Index terms: Arteries, stenosis and occlusion; Arteries, ultrasound; Ultrasound, Doppler

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Transcranial color-coded duplex sonography is a noninvasive, cerebrovascular imaging technique (1). This method adds B-mode imaging and color coding of the Doppler signal to transcranial Doppler sonography. These additions permit correct identification of the insonated artery and direction of flow and determination of angle-corrected velocities. Whereas several

studies have established the value of transcranial Doppler sonography for the diagnosis of collateral flow through the circle of Willis concomitant velocity changes in the basal cerebral arteries have not been reported (2-7). The lack of reference velocity values prevents reliable sonographic detection of stenoses of the major basal cerebral arteries in such patients. Moreover, delineation of typical velocity abnormalities in the major basal cerebral arteries may improve the diagnostic confidence of transcranial sonographic examinations. Using transcranial color-coded duplex sonography, we measured velocities in the intracranial cerebral arteries of patients with angiographically identified different patterns of cross flow through the anterior and posterior communicating arteries

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and unilateral 70% to 100% obstructions of the extracranial internal carotid artery. The purpose of this study was to provide flow velocity data gathered by using transcranial color-coded duplex sonography for the major basal cerebral arteries in patients with unilateral flow-restrictive extracranial carotid artery disease, and to compare these data with the flow velocities obtained from healthy control subjects.

Material and Methods

We prospectively studied 78 patients (18 women, 60 men; mean age \pm SD, 57 years \pm 13) with unilateral extracranial stenoses of more than 69% ($n = 46$) and occlusions ($n = 32$) of the internal carotid artery using color-coded duplex sonography, which serves as a screening method in our neurovascular laboratory. The internal carotid artery obstructions were asymptomatic in 8 patients and symptomatic in 70. Three symptomatic patients had experienced transient blindness and 5 had permanent monocular blindness; 20 patients had cerebral transient ischemic attacks, and 42 had minor cerebral strokes. All patients were examined before surgery with cerebral angiography. Patients with additional stenoses or occlusions of other cerebral arteries and inadequate temporal sonographic windows were not included.

To reduce the effect of abnormal cerebral artery velocities resulting from ischemic strokes, patients with lesions involving more than 25% of the territory of a cerebral artery or with multiple cerebral infarcts, and patients with an interval between cerebral angiography and stroke of less than 28 days were not included.

Sonographic Studies

The extracranial cerebral arteries were examined by means of a color duplex sonographic device equipped with a 5.0 to 7.0-MHz linear scan. Sonographic evaluation of arterial obstructions was performed according to previously published criteria (8).

The intracranial cerebral arteries were studied using a 2.5-MHz 90° sector scan. Doppler energy output had a maximal in situ intensity of 262 mW/cm² spatial peak time average intensity, corresponding to 120 W/cm² spatial peak pulse average intensity. Peak systolic, mean, and end diastolic velocities were measured in the anterior, middle, and precommunicating and postcommunicating posterior cerebral arteries using the temporal bone window with the patient in a supine position. The sonographer was aware of extracranial sonographic findings, but was blinded to the results of cerebral angiography.

Sex- and age-matched reference velocity values for the anterior, middle, and precommunicating and postcommunicating posterior cerebral arteries were obtained from 125 healthy subjects (29 women, 96 men; mean age \pm SD, 57 years \pm 18). The subjects had no cerebrovascular risk factors and no history of neurologic disease. Among the

patients, the interval between transcranial color-coded duplex sonography and cerebral angiography was maximally 24 hours in 19 patients with an interval between stroke and sonography of less than 28 days. The interval between sonography and cerebral angiography in the other 59 patients was 0 to 4 days (median, 1 day).

Angiographic Studies

Selective intraarterial digital subtraction angiography was performed via a femoral artery approach in both internal carotid arteries ($n = 156$) and in at least one vertebral artery ($n = 123$) of every patient. Standard anteroposterior and lateral views of the extracranial and intracranial circulation were obtained routinely. The neuroradiologist was blinded to the results of the sonographic study.

Angiography was used for grading stenoses of the internal carotid artery according to the North American Symptomatic Carotid Endarterectomy Trial criteria (9), and for evaluation of the circle of Willis cross-flow pattern. Occlusions were classified as obstructions with 100% diameter reduction.

Cross flow through the anterior communicating artery to the postcommunicating (A2) anterior cerebral artery was judged to be present if filling of the anterior communicating artery and the postcommunicating anterior cerebral artery on the obstructed (ipsilateral) side occurred with carotid injection on the nonobstructed (contralateral) side. Cross flow through the anterior communicating artery to the middle cerebral artery was judged to be present if filling of the anterior communicating artery, and ipsilateral anterior and middle cerebral arteries occurred with contralateral carotid injection. Cross flow through the posterior communicating artery to the middle cerebral artery was judged to be present if antegrade filling of the ipsilateral middle cerebral artery occurred with vertebral injection.

Statistics

Ipsilateral velocities in the anterior, middle, and precommunicating and postcommunicating posterior cerebral arteries were compared with those on the contralateral side for all types of cross flow through the anterior and posterior communicating arteries by using Wilcoxon's matched pairs signed rank sum test. Velocities measured in each cross-flow type were compared with the velocities in all other cross-flow types and in healthy subjects by means of nonparametric analysis of variance (Mann-Whitney U test). The degrees of internal carotid artery obstruction in each cross-flow type were compared with the corresponding values in the other cross-flow types by using nonparametric analysis of variance (Mann-Whitney U test). Two-sided P values of less than .05 were considered significant.

TABLE 1: Patterns of cross flow through the anterior and posterior communicating arteries, and the degree of unilateral internal carotid artery obstruction in 78 patients

| Cross Flow through the Anterior Communicating Artery | Cross Flow through the Posterior Communicating Artery | | | |
|--|---|---|-------------------------|---|
| | Present, no. of patients | Percentage of ICA Obstruction, median (range) | Absent, no. of patients | Percentage of ICA Obstruction, median (range) |
| To ACA and MCA | 20 | 100 (70–100) | 29 | 98 (71–100) |
| To postcommunicating ACA | 9 | 87 (70–100) | 5 | 74 (70–77) |
| Absent | 4 | 100 (83–100) | 11 | 73 (70–82) |

Note.—ACA indicates anterior cerebral artery; ICA, internal carotid artery; and MCA, middle cerebral artery.

TABLE 2: Anterior and middle cerebral artery velocities in 78 patients with different patterns of cross flow through the anterior communicating artery compared with healthy control subjects

| Cases | Cross Flow through the Anterior Communicating Artery (n) | Peak Velocity, cm/s | Anterior Cerebral Artery | | Middle Cerebral Artery | |
|------------------|--|---------------------|--------------------------|---------------|------------------------|---------------|
| | | | Ipsilateral | Contralateral | Ipsilateral | Contralateral |
| Patients | To ACA and MCA (49) | Systolic \pm SD | 93 \pm 36†¶ | 141 \pm 38‡ | 79 \pm 24 | 103 \pm 27* |
| | | Mean \pm SD | 64 \pm 25†¶ | 95 \pm 29‡ | 50 \pm 15 | 66 \pm 16* |
| | | Diastolic \pm SD | 44 \pm 20†¶ | 68 \pm 23‡ | 38 \pm 13§ | 43 \pm 10* |
| | To postcommunicating ACA (14) | Systolic \pm SD | 75 \pm 41 | 97 \pm 27† | 86 \pm 22 | 104 \pm 26 |
| | | Mean \pm SD | 48 \pm 23 | 66 \pm 20† | 57 \pm 16 | 64 \pm 20 |
| | | Diastolic \pm SD | 34 \pm 16 | 43 \pm 17* | 42 \pm 13 | 45 \pm 12 |
| | Absent (15) | Systolic \pm SD | 70 \pm 23 | 79 \pm 34 | 89 \pm 18 | 104 \pm 24 |
| | | Mean \pm SD | 46 \pm 18 | 48 \pm 21† | 57 \pm 14 | 62 \pm 15 |
| | | Diastolic \pm SD | 32 \pm 14 | 32 \pm 14 | 40 \pm 12 | 41 \pm 11 |
| Control subjects | Absent (125) | Systolic \pm SD | 79 \pm 20 | | 96 \pm 20 | |
| | | Mean \pm SD | 48 \pm 14 | | 59 \pm 13 | |
| | | Diastolic \pm SD | 33 \pm 10 | | 41 \pm 10 | |

Note.—ACA indicates anterior cerebral artery; contralateral/ipsilateral, on the side without/with internal carotid artery obstruction; and MCA, middle cerebral artery.

* $P < .05$ that velocities are greater than normal.

† $P < .01$ that velocities are greater than normal.

‡ $P < .001$ that velocities are greater than normal.

§ $P < .01$ that velocities are less than normal.

|| $P < .001$ that velocities are less than normal.

¶ Reversed flow; three patients with alternating flow are not reported.

Results

All patients were divided according to angiographic findings into six groups with three patterns of cross flow through the anterior communicating artery and two patterns of cross flow through the posterior communicating artery. These are given with the degree of internal carotid artery obstruction in Table 1. The degree of internal carotid artery obstruction was greater in patients with anterior and posterior communicating artery cross flow to the middle cerebral artery than in patients without such cross flow ($P < .001$).

Velocities in the anterior and posterior cerebral arteries depended on the pattern of cross flow through the anterior and posterior communicating arteries, respectively. Anterior and middle cerebral artery velocities of all 78 pa-

tients classified according to the pattern of cross flow through the anterior communicating artery, the corresponding values of 125 healthy subjects, and significant velocity differences compared with the healthy subjects are given in Table 2. Velocities in the middle and precommunicating and postcommunicating posterior cerebral arteries of all 78 patients classified according to the pattern of cross flow through the posterior communicating artery, the corresponding values of 125 healthy subjects, and significant velocity differences compared with the healthy subjects are given in Table 3.

The relative increase of flow velocities in cerebral arteries used as collateral channels were calculated as percentages of the values of 125 healthy subjects defined as 100%. In patients with anterior communicating artery cross flow

TABLE 3: Anterior and middle cerebral artery velocities in 78 patients with different patterns of cross flow through the posterior communicating artery compared with healthy control subjects

| Cases | Cross Flow through the Posterior Communicating Artery | Peak Velocity, cm/s | Middle Cerebral Artery | | Precommunicating Posterior Cerebral Artery | | Postcommunicating Posterior Cerebral Artery | |
|------------------|---|---------------------|------------------------|---------------|--|---------------|---|---------------|
| | | | Ipsilateral | Contralateral | Ipsilateral | Contralateral | Ipsilateral | Contralateral |
| Patients | Present (33) | Systolic \pm SD | 83 \pm 21§ | 110 \pm 26* | 107 \pm 31‡ | 69 \pm 13* | 69 \pm 20* | 66 \pm 12* |
| | | Mean \pm SD | 52 \pm 14§ | 67 \pm 17* | 70 \pm 22‡ | 44 \pm 8* | 45 \pm 14* | 43 \pm 9* |
| | | Diastolic \pm SD | 40 \pm 11 | 45 \pm 11 | 46 \pm 17† | 29 \pm 7* | 31 \pm 11† | 28 \pm 8 |
| | Absent (45) | Systolic \pm SD | 83 \pm 25§ | 100 \pm 24 | 69 \pm 17* | 65 \pm 14 | 66 \pm 14* | 64 \pm 14 |
| | | Mean \pm SD | 52 \pm 18§ | 61 \pm 14 | 44 \pm 11* | 40 \pm 9 | 43 \pm 10* | 39 \pm 9 |
| | | Diastolic \pm SD | 39 \pm 14 | 42 \pm 9 | 29 \pm 9* | 27 \pm 7 | 28 \pm 8* | 27 \pm 7 |
| Control subjects | Absent (125) | Systolic \pm SD | 96 \pm 20 | | 64 \pm 11 | | 62 \pm 12 | |
| | | Mean \pm SD | 59 \pm 13 | | 39 \pm 8 | | 38 \pm 8 | |
| | | Diastolic \pm SD | 41 \pm 10 | | 27 \pm 7 | | 26 \pm 6 | |

Note.—Contralateral/ipsilateral indicates on the side without/with internal carotid artery obstruction; MCA, middle cerebral artery; and PCA, posterior cerebral artery.

* $P < .05$ that velocities are greater than normal.

† $P < .01$ that velocities are greater than normal.

‡ $P < .001$ that velocities are greater than normal.

§ $P < .01$ that velocities are less than normal.

to A2 of the anterior cerebral artery, the contralateral anterior cerebral artery velocity increase was 23% for peak systolic and 30% for peak end diastolic values. In patients with anterior communicating artery cross flow to the middle cerebral artery, the contralateral anterior cerebral artery velocity increase was 78% for peak systolic and 106% for peak end diastolic values, and the ipsilateral anterior cerebral artery velocity increase was 18% for peak systolic and 33% for peak end diastolic values. In patients with posterior communicating artery cross flow to the middle cerebral artery, the ipsilateral precommunicating posterior cerebral artery increase was 67% for peak systolic and 70% for peak end diastolic values.

Ipsilateral middle cerebral artery velocity was lower compared with contralateral middle cerebral artery velocity in patients with (peak systolic and mean, $P < .001$; peak end diastolic, $P < .01$) and without (peak systolic $P < .001$) anterior communicating artery cross flow to the middle cerebral artery, but also in patients with and without a functional posterior communicating artery ($P < .001$). Contralateral middle cerebral artery velocity was higher in all patients, and in patients with cross flow to the middle cerebral artery ($P < .05$) compared with healthy control subjects.

In patients with a functional posterior communicating artery, the ipsilateral precommunicating posterior cerebral artery velocity was higher compared with the contralateral precom-

municating posterior cerebral artery velocity, and compared with the ipsilateral and contralateral precommunicating posterior cerebral artery velocities in patients without a functional posterior communicating artery ($P < .001$). Ipsilateral postcommunicating posterior cerebral artery velocity was higher in all patients compared with that of the healthy control subjects ($P < .01$).

Discussion

In the present study, velocity of the anterior cerebral artery was determined by the size of the perfusion territory that was supplied by this artery (Table 2). Contralateral anterior cerebral artery velocity was normal in patients without anterior communicating artery cross flow, and increased from patients with anterior communicating artery cross flow to A2 of the anterior cerebral artery ($P < .05$) to patients with cross flow to the middle cerebral artery ($P < .001$). In patients with anterior communicating artery cross flow to the middle cerebral artery also ipsilateral (reversed) anterior cerebral artery velocity was increased ($P < .01$), but remained lower than contralateral anterior cerebral artery velocity ($P < .001$). Flow direction in the ipsilateral anterior cerebral artery was related to the degree of internal carotid artery obstruction, because patients with antegrade ipsilateral anterior cerebral artery flow had lower degrees of internal carotid artery obstruction than did pa-

tients with reversed ipsilateral anterior cerebral artery flow ($P < .001$). Patients with lower degrees of internal carotid artery obstruction probably had lower downstream drops in pressure than did those with greater degrees of obstruction, and were thus able to maintain antegrade flow in the ipsilateral anterior cerebral artery. These results are in accordance with the findings of von Reutern et al (10), who showed that transitions from antegrade to reversed flow occur in the vertebral artery in response to progressive obstruction of the ipsilateral subclavian artery. Only three (6%) of our patients showed alternating flow in the ipsilateral anterior cerebral artery, whereas this flow pattern was more frequent in the vertebral arteries of patients in von Reutern's series.

Ipsilateral middle cerebral artery velocities showed decreased values in patients with anterior communicating artery cross flow to the middle cerebral artery, but not in the other two anterior communicating artery cross flow patterns (peak systolic and mean, $P < .001$; peak end diastolic, $P < .01$). It is likely that the greater degree of internal carotid artery obstruction in patients with anterior communicating artery cross flow to the middle cerebral artery caused greater pressure drops downstream that were insufficiently counterbalanced by cerebral autoregulation and caused low ipsilateral middle cerebral artery velocity. Contralateral middle cerebral artery tended to show increased values that were significant in patients with anterior communicating artery cross flow to the middle cerebral artery ($P < .05$), but not in the other two anterior communicating artery cross-flow patterns. Contralateral internal carotid artery flow and velocity are increased in patients with unilateral stenoses of more than 69% and occlusions of the internal carotid artery (11). In vitro color Doppler flow imaging studies using model stenoses with 13% to 94% cross-sectional area reduction have shown that increased intrastenotic velocities persist for a few centimeters in the poststenotic segment (12, 13). Since sonographic measurements of the velocity of the middle cerebral artery were performed near the origin of the artery, increased contralateral middle cerebral artery velocity may have resulted from continuation of increased contralateral internal carotid artery velocity. Increased contralateral peak end diastolic middle cerebral artery velocity may also have resulted

from mild vasodilatation in its territory that counterbalanced the steal of blood flow through the anterior communicating artery.

Ipsilateral peak systolic and mean precommunicating posterior cerebral artery velocities were higher in patients with cross flow through the posterior communicating artery than in patients without such cross flow, and compared with healthy control subjects ($P < .001$). This velocity increase resulted from the enhanced perfusion territory of the posterior cerebral artery that also supplied the middle cerebral artery (3-7). It is interesting that contralateral precommunicating posterior cerebral artery velocity was also increased in patients with cross flow through the posterior communicating artery ($P < .05$). It is well known that basilar artery velocity is increased in many patients with cross flow through the posterior communicating artery (3-7). The precommunicating segment has a short average length of 6.6 mm in patients with a normal configuration of the posterior part of the circle of Willis (14). Therefore, the same two mechanisms that were suggested for enhanced contralateral middle cerebral artery velocity in patients with anterior communicating artery cross flow to the middle cerebral artery may have been responsible for increased contralateral precommunicating posterior cerebral artery velocity—that is, a continuation of increased basilar artery velocity to the precommunicating posterior cerebral artery—and/or vasodilation in the contralateral posterior cerebral artery territory to compensate for the steal of blood through the posterior communicating artery. Increased ipsilateral postcommunicating posterior cerebral artery velocity in patients with cross flow through the posterior communicating artery ($P < .05$) may have resulted from continuation of increased precommunicating posterior cerebral artery velocity and/or leptomeningeal collaterals. In patients without cross flow through the posterior communicating artery, ipsilateral precommunicating and postcommunicating posterior cerebral artery velocities were increased ($P < .05$). This velocity increase probably resulted from leptomeningeal anastomoses, since sonographic and neuroradiologic studies excluded all other causes that might have increased ipsilateral posterior cerebral artery velocity.

In all patients with cross flow through the anterior and/or posterior communicating arteries, the relative velocity increases in the anterior

cerebral artery and the precommunicating posterior cerebral artery, assuming the velocities of healthy control subjects as 100%, were greater for peak end diastolic than for peak systolic values. These hemodynamic abnormalities are similar to those observed in cerebral arteries supplying arteriovenous malformations. In clinical practice, this means that sonographers should interpret their results cautiously and take the extracranial sonographic findings into consideration.

The ipsilateral middle cerebral artery was collateralized in 63% of patients through the anterior communicating artery, but only in 42% of patients through the posterior communicating artery. It is well known from anatomic studies that hypoplasia and aplasia are less frequent in the anterior than in the posterior communicating artery, and in the anterior than in the precommunicating posterior cerebral artery (15, 16). Therefore, it was more likely that in our patients cross flow would develop through the anterior rather than the posterior communicating artery. Moreover, the development of cross flow through the anterior communicating artery increases the perfusion pressure in the collateralized carotid system, and may thus prevent the development of cross flow through the posterior communicating artery.

For this study, cerebral angiography was used as the standard of reference, although we were aware that this technique may distort cerebral hemodynamics by virtue of the injection of significant volumes of contrast material or an increase in pressure in the injected vessel (17). However, we assume that this was not a relevant limitation of our study, because ipsilateral anterior cerebral artery flow direction was identical at transcranial color-coded duplex sonography and at angiography. Moreover, contralateral anterior cerebral artery velocity was increased in patients with anterior communicating artery cross flow to A2 of the anterior cerebral artery but not in patients without cross flow through the anterior communicating artery; and ipsilateral precommunicating posterior cerebral artery velocity was increased in patients with cross flow through the posterior communicating artery relative to these without such cross flow. The major shortcoming of transcranial sonographic studies is the acoustic window that becomes smaller or may prevent transtemporal insonation in as many as 30% of cases (18–20). This was not a problem in our study, because all

patients with insufficient bone windows were excluded.

The reference values for ipsilateral and contralateral anterior, middle, and precommunicating and postcommunicating posterior cerebral arteries obtained in the present study are useful for several clinical applications. First, the diagnosis of additional stenoses of the major intracranial cerebral arteries in patients with a functional circle of Willis becomes feasible. We propose the analogous diagnostic criteria used in patients without internal carotid artery obstructions; that is, delineation of a focal peak systolic velocity increase, low velocity, and reversed flow signals (21–26). The peak systolic values, however, have to be greater than the mean values plus two SDs obtained in patients with the corresponding cross-flow pattern. Second, previously, transcranial sonographic evaluation of cross flow through the anterior communicating artery was not feasible in patients with internal carotid artery obstruction and absent ipsilateral temporal insonation windows. Contralateral anterior cerebral artery velocity reference values reported in this study, however, may allow prediction of the functional state of the anterior communicating artery in such patients. False-positive results caused by anterior cerebral arteries that are stenosed or that selectively supply an arteriovenous malformation are unlikely to occur, because both diseases are rare (25, 27–29). Finally, our data may improve diagnostic confidence in transcranial sonographic studies that are performed in patients with suboptimal temporal windows.

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