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Transcranial Color-Coded Duplex Sonography in the Evaluation of Collateral Flow through the Circle of Willis

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PURPOSE: To determine the sensitivity, specificity, and positive and negative predictive values of transcranial color-coded duplex sonographic (TCCD) evaluation of cross flow through the anterior (ACoA) and posterior (PCoA) communicating arteries in patients with occlusive cerebrovascular disease. **METHODS:** We studied prospectively 132 patients (37 women, 95 men; mean age, 60 years) with stenoses of more than 69% reduction in vessel diameter ($n = 93$) and occlusions ($n = 52$) of the internal carotid artery, and three occlusions of the basilar artery. The sonographer was aware of extracranial sonographic findings but was blinded to the results of cerebral angiography. **RESULTS:** Nine patients (7%) with thick bones preventing transtemporal insonation and three patients (3%) with occlusions of the middle ($n = 3$) and anterior ($n = 1$) cerebral arteries were excluded. Sensitivity of TCCD for detection of collateral flow through the ACoA in patients with occlusive carotid artery disease was 98%, specificity was 100%, positive predictive value was 100%, and negative predictive value was 98%. The corresponding values for the PCoA were 84%, 94%, 94%, and 84%, respectively. All three functional PCoAs were identified in patients with occluded basilar arteries. **CONCLUSION:** TCCD is a valuable method for noninvasive evaluation of cross flow through the ACoA in patients with adequate sonographic windows. However, TCCD evaluation of cross flow through the PCoA is less reliable, because hemodynamic criteria may cause falsely positive and falsely negative results.

Index terms: Arteries, collateral; Arteries, ultrasound; Cerebral blood flow

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Since Thomas Willis's description of the arterial circle at the base of the brain, many studies have shown its role as a principal collateral channel to compensate for stenoses and occlusions of the carotid arteries (1–4). Impaired collateralization may be associated with greater risk of cerebral infarction during natural progression of extracranial carotid artery disease and during revascularization by carotid endarterectomy (5–7). Therefore, reliable evaluation of collateral flow through the circle of Willis is important in such patients.

Transcranial color-coded duplex sonography (TCCD) is a sonographic method that 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 (8) (Fig 1). The purpose of this prospective study was to assess the accuracy of TCCD for evaluation of collateral flow through the anterior (ACoA) and posterior (PCoA) communicating arteries in patients with occlusive cerebrovascular disease.

Subjects and Methods

We prospectively studied 132 patients (37 women, 95 men; mean age $\pm$ SD, 60 ± 15 years) who had a total of 93 stenoses with more than 69% reduction in vessel diameter and 52 occlusions of the internal carotid arteries (ICA), and three occlusions of the basilar arteries as assessed with extracranial color-coded duplex sonography and TCCD, which serve as screening methods in our neurovascular laboratory. All patients were enlisted to undergo cerebral angiography. In 117 of 132 patients, cerebral angiography was performed for presurgical evaluation of asymptomatic ($n = 19$) and symptomatic ($n = 98$)

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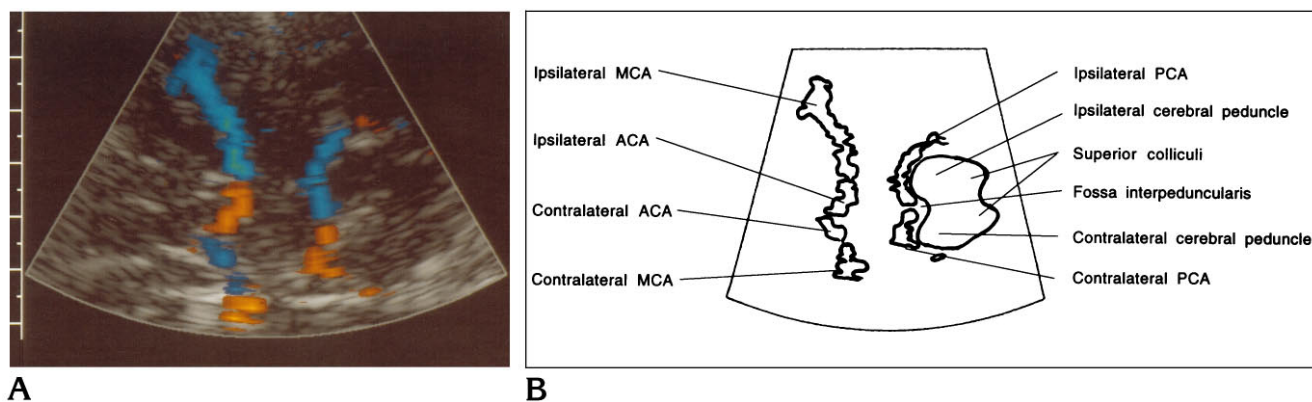


Fig 1. Normal TCCD findings obtained by using transtemporal insonation and an axial plane (A) and corresponding diagram (B).

stenoses and occlusions of the extracranial ICA. Eleven of the 98 patients with symptomatic stenoses and occlusions of the extracranial ICA had experienced transient and six were left with permanent monocular blindness, 27 had had cerebral transient ischemic attacks, and 54 had had cerebral ischemic strokes. Twelve of 132 patients underwent cerebral angiography for presumed symptomatic stenoses with more than 69% reduction in vessel diameter or occlusions of the intracranial ICA producing transient ischemic attacks ($n = 2$) and strokes ($n = 10$), and three of 132 patients for presumed basilar artery occlusions causing stroke. Definite classification of cerebral artery stenoses and occlusions was made according to angiographic findings.

Sonographic Studies

The extracranial cerebral arteries were examined with a color duplex device equipped with a 5.0 or 7.0 MHz linear scan. Sonographic evaluation of arterial stenoses and occlusions was performed according to previously published criteria (9, 10). The intracranial cerebral arteries were studied with a 2.5 MHz 90° sector scan. Doppler energy output had a maximal in situ spatial peak time average intensity of 262 mW/cm², corresponding to a spatial peak pulse average intensity of 120 W/cm². Flow toward the probe was displayed in blue and flow away in red. TCCD examinations of the anterior (ACA), middle (MCA), P1 (precommunicating), and P2 (postcommunicating) segments of the posterior (PCA) cerebral arteries, ACoA, and PCoA were performed through the temporal window with the patient supine. Vertebral and basilar arteries were insonated through the foramen magnum with the patient in a sitting position. All subjects were told to close their eyes and relax. Attention was given to maintaining a calm environment.

The interval between the TCCD study and cerebral angiography was less than 24 hours in 35 patients who had had a stroke not more than 28 days previously (median, 6 days; range, 0 to 27 days). The interval between the TCCD study and cerebral angiography of the other 97 patients (32 patients who had had a stroke more than 28 days

earlier, 29 patients who had had a transient ischemic attack, 11 patients who had experienced transient and six who had permanent monocular blindness, and 19 patients with asymptomatic ICA stenoses and occlusions) ranged from 1 to 4 days (median, 1 day). The sonographer was aware of extracranial sonographic findings, but was blinded to the results of cerebral angiography.

Presence of collateral flow through the ACoA to the MCA (functional ACoA) was established if the color-coded Doppler signals and Doppler spectra showed reversed flow in the ACA on the obstructed (ipsilateral) side (11) (Fig 2). If the ipsilateral ACA was missed, presence of a functional ACoA was established if velocity in the ipsilateral MCA decreased during manual compression of the contralateral common carotid artery for a few heart cycles (6, 11–15). Compression tests were performed only when B-mode sonography did not show atherosclerotic plaques in the common carotid artery.

Presence of collateral flow through the PCoA to the MCA (functional PCoA) was established if color-coded Doppler signals and Doppler spectra showed the PCoA with flow directed from the P1 segment of the PCA to the ICA (Fig 3). When the PCoA was not detected, collateral flow through the PCoA was determined to be present if peak systolic velocity in the ipsilateral P1 segment was more than 2 SD above the mean value of sex- and age-matched control subjects (6, 11–15).

Presence of collateral flow through the PCoA to the PCA and basilar artery (functional PCoA) in patients with occlusion of the basilar artery was established if color-coded Doppler signals and Doppler spectra showed the PCoA with flow directed from the ICA to the P1 segment of the PCA (reversed flow) and to the P2 segment of the PCA (Fig 4). Sex- and age-matched reference values for peak systolic velocities in the P1 segment of the PCA were obtained from 158 healthy control subjects who had no cerebrovascular risk factors and no history of neurologic disease.

Angiographic Studies

Selective intraarterial digital subtraction angiography was performed via a femoral artery approach in both ICAs

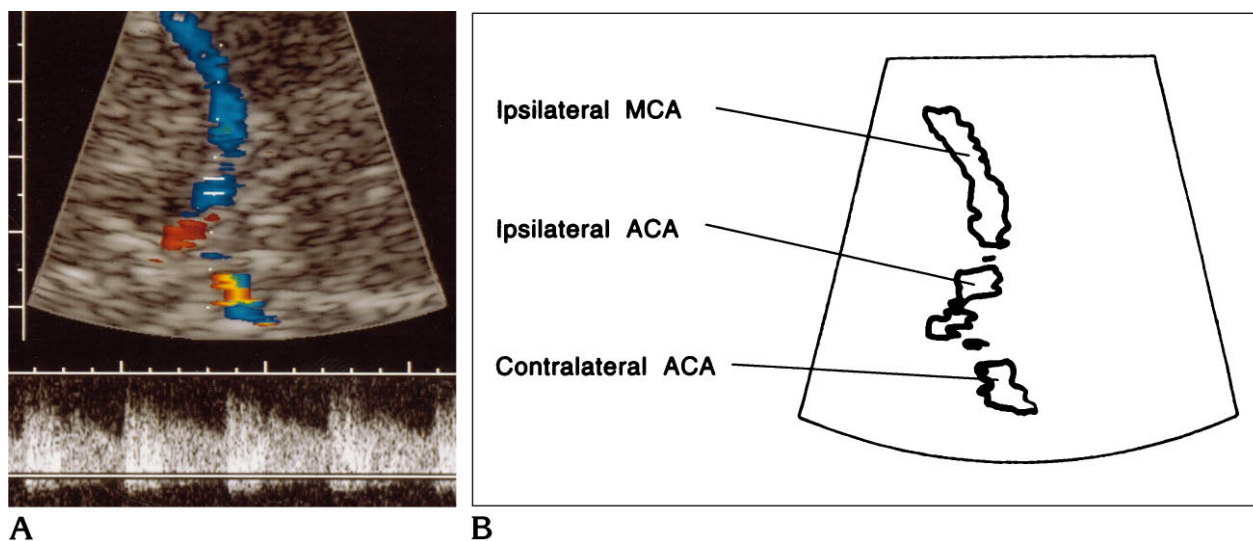


Fig 2. A, Cross flow through the ACoA to the MCA is shown on TCCD obtained by using transtemporal insonation in the axial plane on the side of a 90% stenosis of the extracranial ICA. Both ACAs and the MCA on the side of ICA stenosis are depicted in blue, indicating cross flow through the ACoA to the ACA and MCA. Doppler spectra obtained from the ACA on the side of ICA stenosis confirm its reversed flow direction. Corresponding diagram is shown in B.

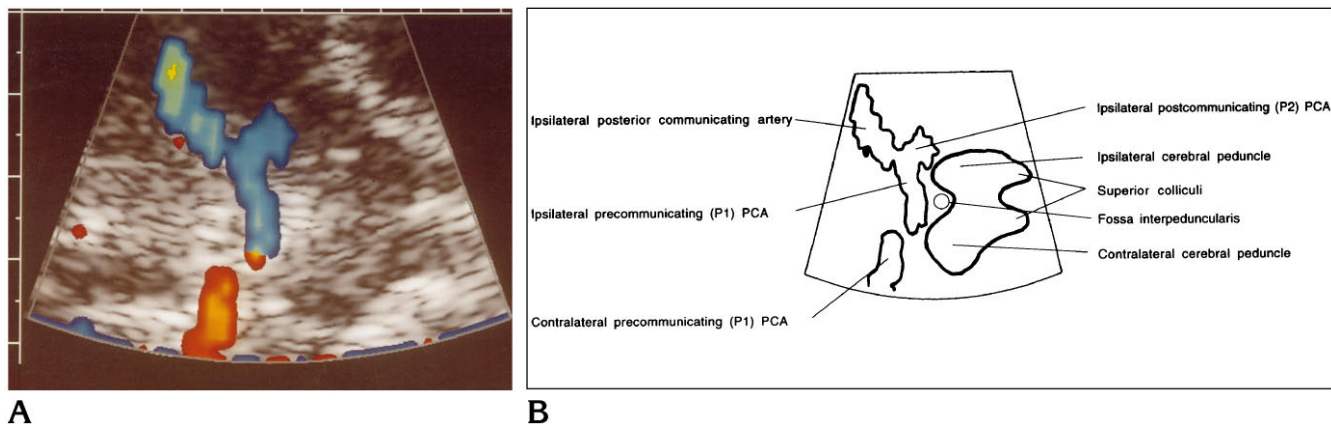


Fig 3. A, Cross flow through the PCoA to the MCA is shown on TCCD obtained by using transtemporal insonation in the axial plane on the side of an occluded extracranial ICA. The PCoA and P1 segment of the PCA are depicted in blue, indicating collateral flow from the P1 segment through the PCoA to the ICA. Corresponding diagram is shown in B.

($n = 258$) and in at least one vertebral artery ($n = 204$) of 129 patients with presumed obstructive carotid artery disease. Both vertebral arteries and four ICAs were examined in three patients with presumed occlusion of the basilar artery. The injected volume of contrast medium was 5 to 8 mL. Standard anteroposterior and lateral views (512×512 matrix) of the extracranial and intracranial circulation were obtained routinely. The neuroradiologist was blinded to the results of TCCD. The criteria of the North American Symptomatic Carotid Endarterectomy Trial (NASCET) were used for grading ICA stenoses (16).

Collateral flow through the ACoA to the MCA was judged to be present if filling of the ACoA, both ACAs, and both MCAs occurred with unilateral carotid injection. Collateral flow through the PCoA to the MCA was judged to be present if antegrade filling of the MCA occurred with vertebral injection. Presence of collateral flow through the

PCoA to the PCA and basilar artery was established if filling of the PCA and basilar artery occurred with carotid injection.

Results

TCCD showed 221 (84%) of 263 unoccluded ACAs, 248 (95%) of 261 unoccluded MCAs, 243 (92%) of 264 unoccluded P1 segments of the PCA, 248 (94%) of 264 unoccluded P2 segments of the PCA, 253 (96%) of 264 unoccluded vertebral arteries, and 121 (94%) of 129 unoccluded basilar arteries in 132 patients. Twelve patients were excluded: nine (7%) because bilateral insufficient acoustic windows prevented transtemporal insonation, and three

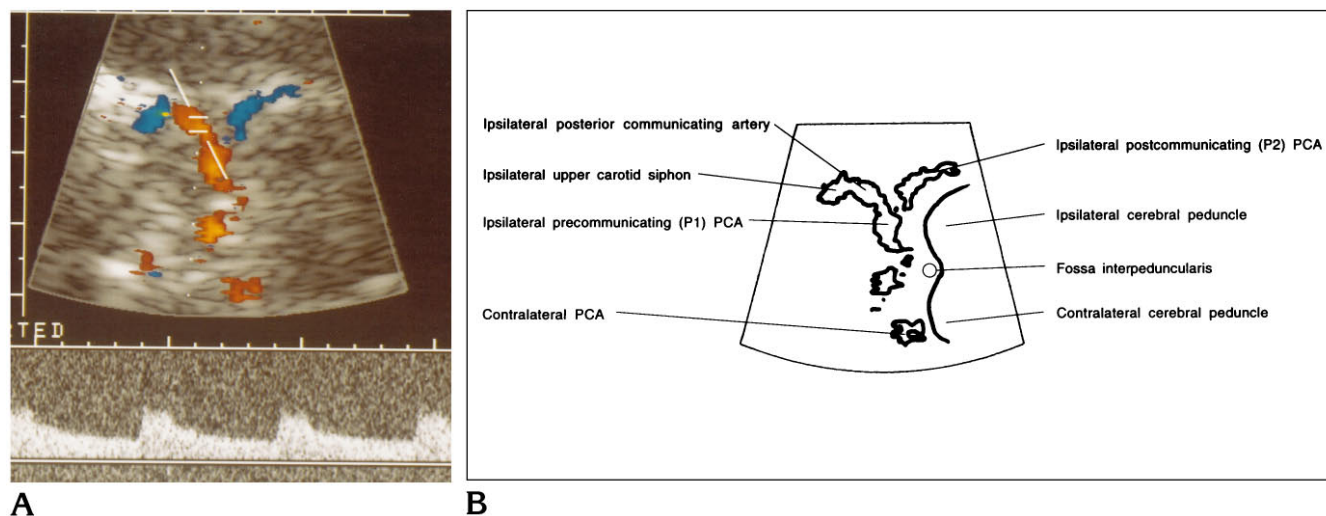


Fig 4. A, Cross flow through the PCoA to the P1 segment of the PCA is shown on TCCD obtained by using transtemporal insonation in the axial plane in a patient with an occluded basilar artery. The PCoA and the P1 segment of the PCA are depicted in red, indicating collateral flow from the ICA through the PCoA to the P1 segment. Doppler spectra obtained from the PCoA confirm the flow direction observed by means of color Doppler imaging. Corresponding diagram is shown in B.

TABLE 1: Transcranial color-coded duplex sonographic and angiographic evaluation of cross flow through the anterior communicating artery in 70% to 99% stenoses and occlusions of the carotid arteries*

Cross Flow through Anterior Communicating Artery at Angiography	Findings at Transcranial Color-Coded Duplex Sonography		
	Reversed Flow in Anterior Cerebral Artery on Obstructed Side, n (%)	Decreased Velocity in Middle Cerebral Artery during Compression of Common Carotid Artery on Unobstructed Side, n† (%)	Cross Flow through Anterior Communicating Artery, n (%)
Present (n = 66)	58 (88)	7 (11)‡	65 (98)
Absent (n = 51)	0 (0)	0 (0)§	0 (0)

* One hundred seventeen patients with 103 unilateral and 14 bilateral stenoses and occlusions.

† Common carotid artery compression was performed only if the anterior cerebral artery was missed by sonography.

‡ One patient did not tolerate discomfort resulting from pressure on his neck.

§ In seven patients, the middle cerebral artery velocity remained unchanged during compression of the common carotid artery.

(3%) because three MCAs and one ACA were occluded at angiography. The rationale for excluding all patients with occluded MCAs and ACAs was that it is not possible to decide whether such a patient would have had a collateral flow through the ACoA to the MCA. Therefore, 120 patients (34 women, 86 men; mean age $\pm$ SD, 59 ± 15 years) remained for the final analysis. Of these, 117 patients had 81 stenoses (63 extracranial, 12 in the siphon, six in the terminal segment) and 52 occlusions of the ICA (45 extracranial, two in the siphon, five in the terminal segment). The stenoses and occlusions of the ICA were unilateral in 101, unilateral and tandem in two, and bilateral in 14 patients. The remaining three patients had occlusions of the basilar artery that extended from

the middle third to the origin of the superior cerebellar arteries.

The results of TCCD and angiographic evaluation of cross flow through the ACoA and PCoA in patients with stenoses and occlusions of the ICA are summarized in Tables 1 and 2, respectively. Digital compression of the common carotid artery was performed in 14 patients (12%) with unilateral stenoses and occlusions of the ICA and missed ipsilateral ACAs. In seven of eight patients with ACoA cross flow, MCA velocity decreased, one patient did not tolerate this maneuver. In six patients without ACoA cross flow, MCA velocity remained unchanged during carotid compression. Four patients with elevated peak systolic velocities in the P1 segment of the PCA had no functional PCoA, but

TABLE 2: Transcranial color-coded duplex sonographic and angiographic evaluation of cross flow through the posterior communicating artery in 70% to 99% stenoses and occlusions of the carotid arteries*

Cross Flow through Posterior Communicating Artery at Angiography	Findings at Transcranial Color-Coded Duplex Sonography		
	Posterior Communicating Artery Identified, n (%)	Increased Velocity in Posterior Cerebral Artery, n† (%)	Cross Flow through Posterior Communicating Artery, n (%)
Present (n = 69)	43 (62)	53 (77)	58 (84)
Absent (n = 62)	0 (0)	4 (6)	4 (6)

* One hundred seventeen patients with 103 unilateral and 14 bilateral stenoses and occlusions.

† Peak systolic velocities in the precommunicating segment of the posterior cerebral artery > 2 SD above the mean values of age- and sex-matched control subjects.

TABLE 3: Accuracy of transcranial color-coded duplex sonographic evaluation of collateral flow through the anterior and posterior communicating arteries in 70% to 99% stenoses and occlusions of the carotid arteries*

Collateral Flow	Sensitivity, %	Specificity, %	Positive Predictive Value, %	Negative Predictive Value, %
Anterior communicating artery	98	100	100	98
Posterior communicating artery	84	94	94	84

* One hundred seventeen patients with 103 unilateral and 14 bilateral stenoses and occlusions.

leptomeningeal anastomoses supplied by this PCA were seen at angiography. TCCD identified all three functional PCoAs in patients with occluded basilar arteries.

The calculations for sensitivity, specificity, and positive and negative predictive values of TCCD evaluation of cross flow through the circle of Willis in 117 patients with obstructive carotid artery disease were performed with the assumption that the total number of potentially functional ACoAs was 117 (103 ACoAs from 103 patients with unilateral and 14 ACoAs from 14 patients with bilateral ICA stenoses and occlusions). The corresponding calculations for the PCoA were performed with the assumption that the total number of potentially functional PCoAs was 131 (103 PCoAs from 103 patients with unilateral and 28 PCoAs from 14 patients with bilateral ICA stenoses and occlusions). The results are given in Table 3.

Age- and sex-matched reference peak systolic velocities in the P1 segment of the PCA $\pm$ 2 SD obtained from 158 control subjects were 71 ± 23 and 67 ± 24 cm/s for men and women aged 20 to 39 years, 66 ± 24 and 62 ± 20 cm/s for men and women aged 40 to 59 years, and 61 ± 25 and 55 ± 16 cm/s for men and women aged 60 to 79 years.

Discussion

Conventional transcranial Doppler sonography is of established value in assessing collat-

eral flow through the circle of Willis in patients with severe stenosis or occlusion of the carotid arteries (6, 12–15, 17). This technique identifies the cerebral arteries in a blind fashion using the depth of the sample volume, the spatial relationship to the ACA-MCA bifurcation, velocities, and the response to ipsilateral carotid compressions as diagnostic criteria (17). TCCD, however, identifies cerebral arteries according to their anatomic location in the color-coded B-mode image and, if necessary, also uses the above criteria (8) (Fig 1). Therefore, TCCD may increase diagnostic confidence in transcranial sonographic evaluation of cross flow through the circle of Willis.

In the present study, the sensitivity, specificity, and positive and negative predictive values for TCCD detection of collateral flow through the ACoA were 98% to 100% in patients with adequate temporal windows. These figures are identical to those reported in a TCCD study that examined patients with unilateral stenoses of more than 69% reduction in vessel diameter and occlusions of the ICA, and are in the range of an MR angiographic study with a reported sensitivity of 95% (18, 19). However, the present TCCD results are superior to some transcranial Doppler sonographic studies reporting sensitivities of 52% to 95% and specificities of 85% to 100% (6, 13–15). In this study, 88% of functional ACoAs were identified using detection of reversed flow direction in the ipsilateral ACA (Fig 2). Therefore, manual compressions of the common ca-

rotid artery that are often needed in transcranial Doppler sonography were necessary in only 12% of our patients (6, 12–15). Although complications from common carotid artery compression are rare, they include cardiac arrhythmias, carotid thrombosis, transient ischemic attacks, strokes, and death (20–23). Moreover, false-positive carotid compressions may result from asystole (24). We assume that the ability of TCCD to evaluate reliably the functional state of the ACoA without the use of a potentially harmful test represents an important advance of transcranial sonography.

Sensitivity, specificity, and positive and negative predictive values for TCCD detection of collateral flow through the PCoA ranged from 84% to 94% in patients with occlusive carotid artery disease and adequate sonographic windows. These results are similar to those reported in one TCCD study and in several transcranial Doppler sonographic reports giving sensitivities of 83% to 88% and specificities of 60% to 95%, but are surpassed by an MR angiographic study reporting a sensitivity of 97% (6, 12–15, 18, 19). Direct sonographic detection of a functional PCoA was just possible in 62% of cases (Fig 3). This is caused by the fairly perpendicular course of the PCoA to the ultrasound beam that impedes detection (25). Using the indirect criterion of pathologically increased velocity in the P1 segment of the PCA, 77% of all functional PCoAs were identified. This velocity increase results from the enhanced perfusion territory of the PCA that also supplies the MCA. The 23% false-normal velocity in the P1 segment of the PCA was probably caused by the stringency of the diagnostic criterion. However, the use of less severe criteria would have increased the number of false-positive PCoAs, which was already four (7%) in this series. In addition, hypoplasia as a cause of low velocity in the P1 segment of the PCA is not rare (1, 7). Therefore, in patients with a functional PCoA and a hypoplastic P1 segment, the velocity in the P1 segment may just increase to false-normal levels. Four patients with increased velocity in the P1 segment and false-positive PCoAs showed leptomeningeal anastomoses supplied by this PCA at angiography. The latter four patients illustrate the limitations of this hemodynamic criterion, because other conditions may infrequently lead to increased velocity in the P1 segment, such as leptomeningeal collaterals, stenoses, vasospasm, and arteriovenous mal-

formations. Simultaneous bilateral compression of the vertebral arteries for identification of a functional PCoA was not performed, because it often causes discomfort to the patients, is technically difficult, and may be unreliable (18).

Three functional PCoAs with cross flow from the ICA to the P1 segment of the PCA in patients with occluded basilar arteries were correctly assessed by TCCD (Fig 4). Nevertheless, the small number of patients prevents general conclusions about the reliability of TCCD for evaluation of this pattern of cross flow through the PCoA.

For the present study, cerebral angiography was used as the standard of reference, although we were aware that this technique may distort cerebral hemodynamics owing to the injection of a significant volume of contrast material or an increased pressure in the injected vessel (26). Transcranial Doppler sonography is of established value in assessing the pattern of collateral circulation across the circle of Willis (17). In a preliminary study of 78 patients with unilateral stenoses of more than 69% reduction in vessel diameter and occlusions of the ICA, we found that the results of TCCD evaluation of cross flow through the ACoA using flow direction in the ipsilateral ACA were identical to those obtained with angiography in all cases (18). Moreover, TCCD evaluation of cross flow through the PCoA in cases with identified PCoA gave the same results as angiography (18). We concluded that cerebral angiography is a reliable method for evaluating cross flow through the circle of Willis.

A limitation of TCCD is the quality of the acoustic window that prevented bilateral trans-temporal insonation in 7% of cases. With advancing age, and especially in women, the temporal window becomes smaller or disappears and may prevent insonation in up to 30% of cases (17, 27, 28). Another shortcoming is that the reliability of TCCD findings depends on the knowledge, skill, and experience of the sonographer.

The present results indicate that TCCD both increases diagnostic confidence in sonographic assessment of collateral flow across the ACoA and decreases the number of potentially harmful compression tests. Therefore, it is suggested that TCCD replace conventional transcranial Doppler sonography in the evaluation of cross flow through the circle of Willis.

We conclude that in patients with adequate

sonographic windows, TCCD is a valuable method for noninvasive evaluation of collateral flow across the ACoA. However, TCCD evaluation of cross flow through the PCoA is less reliable, because hemodynamic criteria that may cause false-positive or false-negative results have to be used.

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