

# Stroke

JOURNAL OF THE AMERICAN HEART ASSOCIATION

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*Stroke* 1988;19:1328-1334

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1524-4628

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# Importance of Cerebral Collateral Pathways During Carotid Endarterectomy

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**Before surgery, we evaluated major intracranial collateral pathways using transcranial Doppler ultrasonography (TCD) in 50 patients who then underwent carotid endarterectomy with concurrent multimodality cerebral monitoring. Patients were grouped with respect to collateral pathways demonstrated preoperatively by TCD: Group 1, good collateralization with an anterior and/or a posterior communicating artery ipsilateral to the operative carotid lesion (29 patients, 58%); Group 2, collateral pathways present but impeded by other proximal stenoses (nine patients, 18%); and Group 3, no collateralization identified (nine patients, 18%). Three patients (6%) could not be classified. TCD identified major collateral pathways with a sensitivity of 89% and a specificity of 80% when compared with arteriography. During carotid endarterectomy mean middle cerebral artery velocity, pulsatility index, and stump pressure were higher and the decrease in middle cerebral artery velocity with extracranial carotid artery cross clamping was significantly less among Group 1 patients than among Group 2 and 3 patients ( $p < 0.05$  for both groups). Group 1 patients required fewer intraoperative carotid artery shunts and developed fewer ischemic electroencephalographic abnormalities than did patients in Groups 2 and 3 ( $p < 0.05$  for both groups). TCD assessment of cerebral collateralization helps predict hemodynamic consequences of cross clamping during carotid endarterectomy. (*Stroke* 1988;19:1328–1334)**

**C**erebrovascular collateral blood supply plays an essential role in cerebral perfusion. In patients undergoing carotid endarterectomy the correlation of preoperative assessment of collateral potential with cerebral perfusion during extracranial carotid artery cross clamping enhances our understanding of intracerebral hemodynamics.

Transcranial Doppler ultrasonography (TCD) can directly insonate the intracranial arteries to evaluate collateral perfusion around the circle of Willis.<sup>1–3</sup> In addition, TCD provides continuous noninvasive monitoring of middle cerebral artery (MCA) blood velocity (MCAV) and immediately detects changes in cerebral perfusion during carotid artery manipulation and cross clamping.<sup>4–6</sup> The purposes of our study were to correlate prospectively the

cerebral collateral patterns identified preoperatively by TCD with the cerebral perfusion during interruption of common carotid artery (CCA) blood flow by cross clamping and to compare TCD with electroencephalography (EEG) and carotid artery back pressure in assessing cerebral ischemia during carotid endarterectomy.

## Subjects and Methods

### Patients

Between August 1986 and February 1988, 350 patients with carotid artery stenosis were evaluated using TCD in the Scripps Clinic and Research Foundation Vascular Laboratory; 54 patients subsequently underwent carotid endarterectomy. Their clinical histories and neurologic deficits at presentation were tabulated. We excluded four of the 54 patients from the study because of our inability to obtain signals through their transtemporal windows. The mean  $\pm$  SD age of the 50 patients studied was  $65.0 \pm 5.0$  years.

### Preoperative Evaluation

Duplex scanning of the extracranial cerebral arteries was performed before TCD evaluation in all 50

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Received April 7, 1988; accepted June 15, 1988.

TABLE 1. Noninvasive Identification of Cerebral Collateral Pathways

Duplex scanning	n	Transcranial Doppler ultrasonography		
		Compression of ipsilateral CCA	ACoA	PCoA
No ipsilateral CCA disease, <80% ICA stenosis	20	Insonate ipsilateral MCA, compress ipsilateral CCA: MCAV <10 cm/sec, no collateralization; 10–20 cm/sec, impeded collateral pathways; >20 cm/sec, developed collateral pathways	Insonate ipsilateral MCA, compress ipsilateral CCA: reversal of blood flow, ACoA present; no reversal, no ACoA (if result negative, perform on contralateral side) Insonate ipsilateral MCA, compress contralateral CCA: alteration in blood flow, ACoA present; no alteration, no ACoA	Insonate ipsilateral PCA, compress ipsilateral CCA: increased blood flow, PCoA present; no change, no PCoA Insonate ipsilateral PCoA: confirm with ipsilateral CCA compression (increased blood flow) and bilateral vertebral artery compression (decreased blood flow)
Ipsilateral CCA disease, ≥80% ICA stenosis; no contralateral CCA disease; <80% ICA stenosis	7	Not performed (proceed to identification of specific collateral pathways)	Insonate contralateral ACA, compress contralateral CCA: reversal of blood flow, ACoA present; no reversal, no ACoA Insonate ipsilateral MCA, compress contralateral CCA: alteration in blood flow, ACoA present; no alteration, no ACoA	Insonate ipsilateral PCoA: confirm with bilateral vertebral artery compression (decreased blood flow) Insonate ipsilateral PCA: hyperdynamic blood flow indirect evidence of PCoA
Bilateral CCA disease, ≥80% ICA stenosis	7	Not performed (proceed to identification of specific collateral pathways)	Insonate either ACA, no CCA compressions: continuously reversed blood flow in proximal ipsilateral ACA and/or hyperdynamic blood flow in contralateral ACA, indirect evidence of ACoA	Insonate ipsilateral PCoA: confirm with bilateral vertebral artery compression (decreased blood flow) Insonate ipsilateral PCA: hyperdynamic blood flow indirect evidence of PCoA

Ipsilateral, contralateral to operative carotid lesion. CCA, common carotid artery; ACoA, anterior communicating artery; PCoA, posterior communicating artery; MCA, middle cerebral artery; MCAV, middle cerebral artery blood flow velocity.

patients. TCD studies were performed using a 2-MHz pulsed-wave, range-gated device (TC2-64, Carolina Medical Electronics, Inc., King, North Carolina). Spectral analysis of each intracranial signal was performed with on-line 64-point fast Fourier transformation; results were displayed as velocity (centimeters per second). The 1-hour pre-operative TCD examination included bilateral quantification of MCAV and pulsatility index (PI), evaluation of blood flow in the anterior communicating artery (ACoA) and posterior communicating artery (PCoA), marking of the transtemporal window ipsilateral to the intended surgical procedure so that it could be identified expeditiously in the operating theater, and evaluation of stenosis of the carotid siphon and MCA. The window was located in the temporal region, slightly superior to the zygomatic arch and anterior to the ear. Flow toward the transducer was displayed as upwardly deflected waves. The criteria for identification of various intracranial arteries included the depth of the signal from the probe, the angle and direction of the probe, the direction of blood flow (toward or away from the probe), the characteristic pattern of the signal, and the response to compressions of the proximal CCA and vertebral artery. Insonation of the MCA has been detailed.<sup>1</sup>

Major collateral pathways were identified using a series of specific maneuvers outlined in Table 1. Manual occlusion of the proximal CCA for several cardiac cycles just superior to the clavicle produces changes in MCAV and waveform that emulate the expected result of extracranial carotid artery cross clamping. Severe internal carotid artery (ICA) stenosis (i.e., >80%) and/or any evidence of CCA disease contraindicate the use of this maneuver; it was performed in 20 patients (40%). Because extracranial arterial compressions were necessary to delineate collateral pathways by TCD, duplex scanning of the carotid arteries was always performed to evaluate the carotid arteries before compressions were undertaken, and TCD maneuvers therefore depended on the pathways of carotid disease present (Table 1). Mean MCAV of >20 cm/sec was usually associated with specific collateral pathways (ACoA and/or ipsilateral PCoA) to that hemisphere, which were detectable by TCD.

The ACoA was not usually insonated directly but was identified in its capacity as a collateral pathway between the two cerebral hemispheres. The anterior cerebral artery (ACA) was located at a depth of 65–75 mm with a slight anterior angulation of the probe and usually with blood flowing away from the probe (downward deflection of the signal). The

interruption of ACA blood flow by compression of the ipsilateral proximal CCA caused a reversal of blood flow in the ipsilateral proximal ACA when an ACoA was present. This maneuver diverted blood from the contralateral hemisphere through the ACoA toward the side of the compression. A significant contribution of blood flow to the MCA territory through an ACoA was also detectable by insonating the MCA in question and simultaneously compressing the contralateral proximal CCA. MCAV changed when collateral blood flow was supplied through an ACoA. This maneuver was particularly useful when ipsilateral proximal CCA compression was contraindicated.

The presence of a PCoA could be demonstrated directly by insonation or indirectly by the use of extracranial artery compressions. When the PCoA was insonated directly, it was located at a depth of 60–75 mm and was usually represented by an upwardly deflected signal. After identification of the ICA bifurcation into the MCA and ACA, the probe was directed slightly posterior and inferior to locate the PCoA. Compression of the CCA caused an increase in PCoA blood flow while that in the ACA, carotid siphon, and MCA decreased. Simultaneous bilateral compression of the vertebral arteries was routinely employed to further confirm the origin of the signals in question. Vertebral artery compression was performed immediately inferior to the mastoid process with an extended index finger and caused a concurrent decrease in PCoA blood flow.<sup>7</sup> The PCoA could also be located by insonating the ipsilateral posterior cerebral artery (PCA) (posterior probe angulation, blood flow toward the probe, depth 65–80 mm) and tracking the proximal PCA along its course toward the PCoA. Hyperdynamic PCA signals were usually associated with major collateral blood flow through a PCoA toward the circle of Willis. Indirect evidence of blood flow through a PCoA could also be obtained by insonating the PCA and compressing the ipsilateral CCA; an increase in PCA blood flow was observed with CCA compression due to increased blood flow through the PCoA.

Before endarterectomy, patients were grouped with respect to collateral pathways present on preoperative noninvasive evaluation. Group 1 had well-developed major collateral pathways, such as an ACoA and/or PCoA ipsilateral to the operative carotid lesion. In Group 2, collateral pathways were present but were impeded by proximal stenoses of >80% or by occlusion. When both an ACoA and a PCoA were present and blood flow in one was impeded, the patient was included in Group 1, but if blood flow in both were impeded the patient was included in Group 2. Group 3 comprised those patients in whom no collateral pathways could be identified by preoperative TCD evaluation. In the identification of collateral pathways using TCD, significant technical challenges were encountered when carotid artery compressions were contraindi-

cated, when the transtemporal window was inadequate for insonation of the multiple vessels, or when diffuse intracranial artery occlusive disease was present.

For each MCA signal, the peak systolic, mean, and end-diastolic blood velocities were recorded and the PI was calculated as<sup>8</sup>

$$PI = (MCAV_{\text{systolic}} - MCAV_{\text{diastolic}}) / MCAV_{\text{mean}}$$

Formal contrast arch and carotid arteriography was performed. Cerebral arteriograms were adequate for the evaluation of intracerebral collateral pathways in 42 patients (84%). Arteriographic evidence of major collateral pathways was assessed by a neuroradiologist who was unaware of the results of the preoperative TCD evaluation.

#### *Intraoperative Evaluation*

Continuous noninvasive cerebral monitoring was performed during carotid endarterectomy using both TCD and an EEG compressed spectral array. The Doppler apparatus was used with an on-line videocassette recorder for intraoperative monitoring of ipsilateral MCAV. After induction of anesthesia, the TCD probe was secured over the transtemporal window with an elastic bandage and holder. Consistent insonation of the MCA required careful and continued attention. An automated compressed EEG with continuous spectral assay (Lifescan, Neurometrics, San Diego, California) was monitored during surgery through scalp electrodes. ICA back pressure or stump pressure (SP) was measured immediately after and distal to the placement of CCA and external carotid artery cross clamps. Indications for selective intraoperative carotid artery shunt placement included SP of <50 mm Hg, neurologic deficit, or contralateral carotid artery occlusion. Indications for shunt placement were not influenced by TCD data during the course of our study.

#### *Postoperative Evaluation*

We performed follow-up studies using TCD 3 days after endarterectomy. The neurologic status of each patient was evaluated, and changes in MCAV and PI were noted. Because the postoperative evaluation was performed only once, data are not available to assess long-term hemodynamic changes that may have occurred as a result of endarterectomy.

#### *Data Analysis*

The CLINFO PLUS program (General Clinical Research Center of Scripps Clinic) was used to analyze the data. Analysis of variance was used to compare specific values among the three groups.  $\chi^2$  analysis was used to compare the incidence of shunt placement and EEG abnormalities between Groups 1 and 2. Student's two-tailed *t* test was used to compare MCAV and PI before and after endarterectomy.<sup>9</sup> Variance is reported as  $\pm$  SD.

**TABLE 2. Results of Preoperative Noninvasive Evaluation of Collateral Pathways in 50 Patients by Transcranial Doppler Ultrasonography**

Group	Collateral pathways	n	%
1	Anterior and/or ipsilateral posterior communicating artery	29	58
2	Present but impeded by proximal stenosis	9	18
3	None identified	9	18
—	Could not classify	3	6
Total		50	100

## Results

### Preoperative Evaluation

Good collateralization with an ACoA and/or a PCoA ipsilateral to the operative carotid lesion was identified in 29 patients (58%) (Group 1) (Table 2). In another nine patients (18%), collateral pathways were demonstrated but blood flow was found to be impeded by other significant proximal stenoses, such as an ACoA with a contralateral carotid artery occlusion (Group 2). In nine patients (18%) no major collateral pathways could be demonstrated (Group 3). Three patients (6%) could not be classified due to technical difficulties.

TCD had a sensitivity of 89% and a specificity of 80% for the identification of patients with major collateral pathways present on arteriography (Table 3). TCD had a specificity of >90% for the demonstration of ACAs, but there was a high false-negative rate, giving a sensitivity of 52%. Conversely, the sensitivity of TCD for the detection of PCoAs ipsilateral to an operative carotid lesion was 83%, while the specificity was somewhat lower (60%). When intracranial stenosis was diagnosed by TCD, it was confirmed by arteriography in all patients, and the sensitivity of TCD was 80%.

Patients with various collateral pathways were compared with respect to preoperative MCAV and PI, degree of ICA stenosis, and indications for surgery (Table 4). Although mean MCAV tended to be higher and PI lower in Group 1, the differences between groups were not significant. There was also no difference between groups with respect to severity of ICA stenosis or indications for endarterectomy.

**TABLE 3. Identification of Cerebral Collateral Pathways in 50 Patients: Correlation of Transcranial Doppler Ultrasonography With Formal Contrast Biplane Arteriography**

Finding	Sensitivity (%)	Specificity (%)
Any collaterals	89.3	80.0
Anterior communicating artery	52.4	91.7
Ipsilateral posterior communicating artery	83.3	60.0
Intracranial disease	80.0	100.0

### Intraoperative Evaluation

The adequacy of the collateral circulation during cross clamping was evaluated using TCD, EEG, and other parameters (Figure 1). Mean MCAV during cross clamping was higher in Group 1 ( $30.2 \pm 9.8$ , range 15–50 cm/sec) than in Group 2 ( $14.1 \pm 8.2$ , range 0–22 cm/sec;  $p < 0.05$ ) or Group 3 ( $4.7 \pm 7.0$ , range 0–21 cm/sec;  $p < 0.01$ ). PI during cross clamping was also markedly reduced in Group 3 ( $p < 0.05$ ). The percentage decrease in mean MCAV with cross clamping was  $29.7 \pm 22.0\%$  in Group 1, but there was a  $63.6 \pm 31.0\%$  decrease in MCAV in Group 2 and a  $80.2 \pm 19.8\%$  drop in Group 3 ( $p < 0.05$  vs. Group 1). SP was higher in Group 1 ( $64.1 \pm 14.7$  mm Hg) than in Groups 1 and 2 ( $41.6 \pm 9.9$  and  $32.7 \pm 16.7$  mm Hg, respectively;  $p < 0.05$ ). Intraoperative carotid artery shunts were placed in <15% of the Group 1 patients whereas more than three quarters of the Group 2 and 3 patients required shunt placement ( $p < 0.05$ ). Ischemic EEG abnormalities developed during cross clamping much more frequently in Groups 2 and 3 (33.3%) than in Group 1 (3.4%,  $p < 0.05$ ).

EEG abnormalities developed in seven patients. In six of these seven, collateral pathways were inadequate by preoperative TCD and MCAV was low (i.e.,  $\leq 18$  cm/sec) during cross clamping. One patient developed a mild contralateral EEG abnormality during the long cross clamp period despite good collateral pathways and an adequate MCAV. Although no patient developed a postoperative neurologic deficit, EEG changes occurred predominantly in patients shown to be at risk for ischemia by both preoperative and intraoperative TCD findings.

**TABLE 4. Preoperative Evaluation of Collateral Pathways in 47 of 50 Patients Before Carotid Endarterectomy**

Group	N	MCAV (cm/sec)	Pulsatility index	Stenosis				Indications for surgery					
				<80%		>80%		None		TIA		Stroke	
				n	%	n	%	n	%	n	%	n	%
1	29	$52.9 \pm 14.4$	$0.79 \pm 0.20$	11	38	18	62	7	24	18	62	4	14
2	9	$49.8 \pm 18.1$	$0.89 \pm 0.22$	2	22	7	78	5	56	3	33	1	11
3	9	$40.4 \pm 25.2$	$1.03 \pm 0.43$	4	44	5	56	2	22	5	56	2	22

Group 1, anterior and/or posterior communicating artery present; Group 2, collateral pathway present but impeded by proximal stenosis; Group 3, no major collateral pathways present. Three patients could not be classified due to technical difficulties. Values for MCAV and pulsatility index are mean  $\pm$  SD. MCAV, middle cerebral artery blood flow velocity; TIA, transient ischemic attack.

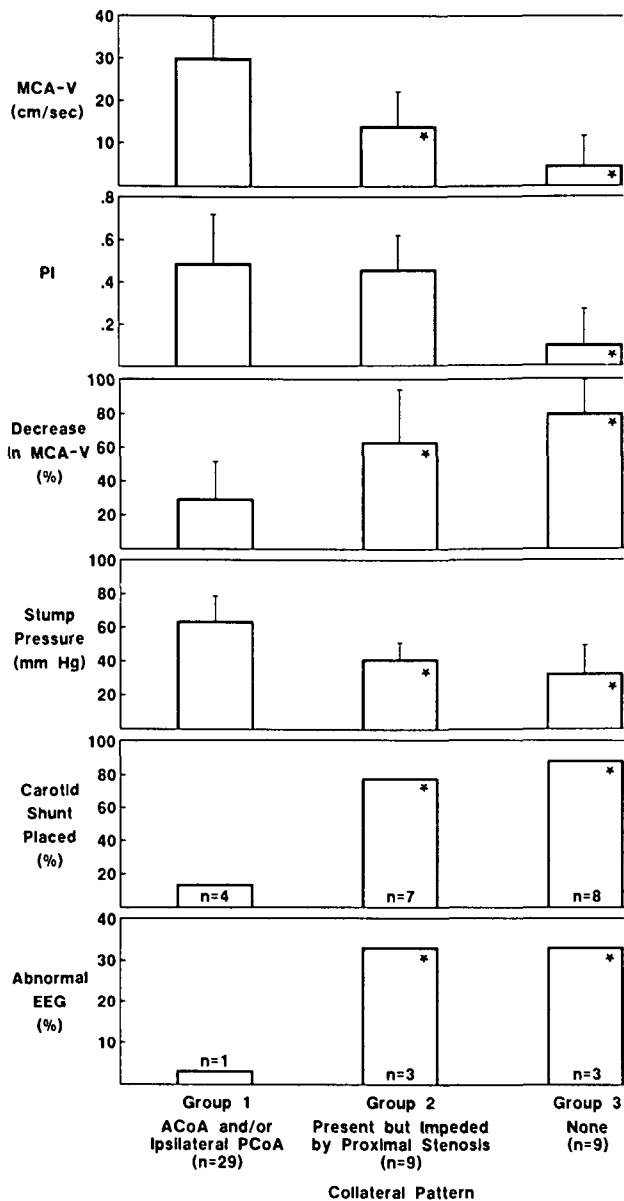


FIGURE 1. Collateral circulation during carotid artery cross clamping with respect to collateral patterns identified in 47 patients by preoperative noninvasive evaluation. Group 1 (patients with well-developed collateral pathways to hemisphere ipsilateral to carotid endarterectomy) had higher mean middle cerebral artery blood flow velocity (MCA-V) and pulsatility index (PI) and smaller decrease in mean MCA-V with clamp placement than Groups 2 and 3 (patients with inadequate collateral pathways). Group 1 patients had higher stump pressures, required fewer carotid shunts, and developed fewer ischemic electroencephalographic (EEG) abnormalities than patients in Groups 2 and 3. \* $p < 0.05$ . Error bars, +1 SD.

#### Postoperative Evaluation

The immediate effect of carotid endarterectomy was demonstrated by an increase in mean MCAV and PI from  $49.3 \pm 17.7$  cm/sec and  $0.86 \pm 0.27$  before to  $61.8 \pm 19.1$  cm/sec and  $1.05 \pm 0.27$  3 days after surgery ( $p < 0.05$  for both). Figure 2 compares

changes in MCAV and PI in each group. MCAV and PI increased following surgery; the largest increases occurred in Group 3.

#### Discussion

Using a series of stepwise maneuvers as part of the routine preoperative TCD examination (Table 1), we identified specific intracranial collateral pathways. Patients were grouped using a simple system (Table 2) classifying collateral circulation in the cerebral hemisphere ipsilateral to the intended carotid endarterectomy. Although differences between groups were not significant in the preoperative evaluation (Table 4), there were some striking differences in cerebral perfusion when the collateral circulation was challenged during cross clamping (Figure 1). Group 1 patients had higher mean MCAV and PI during cross clamping and a smaller percent decrease in mean MCAV with clamp placement than patients in Groups 2 and 3. In addition, Group 1 patients had a higher SP, necessitating placement of fewer intraoperative shunts, and developed fewer EEG abnormalities with cross clamping than patients in either Group 2 or 3. Following endarterectomy, Group 3 patients had the largest increases in both mean MCAV and PI (Figure 2).

Although TCD was compared with arteriography in the identification of collateral pathways, it is not clear which technique provides more clinically useful information regarding hemodynamically significant intracerebral collateral pathways. Arteriography is the current clinical standard for the delineation of intracranial vascular anatomy, but it may not be the best method of investigating the physiologic collateral potential around the circle of Willis. Unless need increases blood flow in a specific major collateral vessel, the vessel may not be seen on routine arteriography. Because TCD relies on extracranial arterial compressions to direct blood through major collateral vessels, TCD provides relevant hemodynamic information. The sensitivity and specificity of TCD in the identification of major collateral pathways present on arteriography was excellent (Table 3). Identification was facilitated by the performance of test compressions of the ipsilateral CCA, which identifies collateral perfusion around the circle of Willis in the hemisphere distal to the operative carotid lesion. In studying the ACoAs, a positive result by TCD can be achieved only when collateral blood flow is actually observed through an ACoA (Table 1). The ability of arteriography to identify small ACoAs, which may be hemodynamically inadequate to function as major collateral pathways during either extracranial carotid compression or cross clamping, may explain the high false-negative rate with TCD in the identification of ACoAs. Conversely, the size of PCoAs appears to be a function of the need for collateral blood flow. PCoAs are more likely to occur in patients with severe extracranial occlusive disease.<sup>10</sup> Therefore, unless progressive disease (such as a severe carotid artery

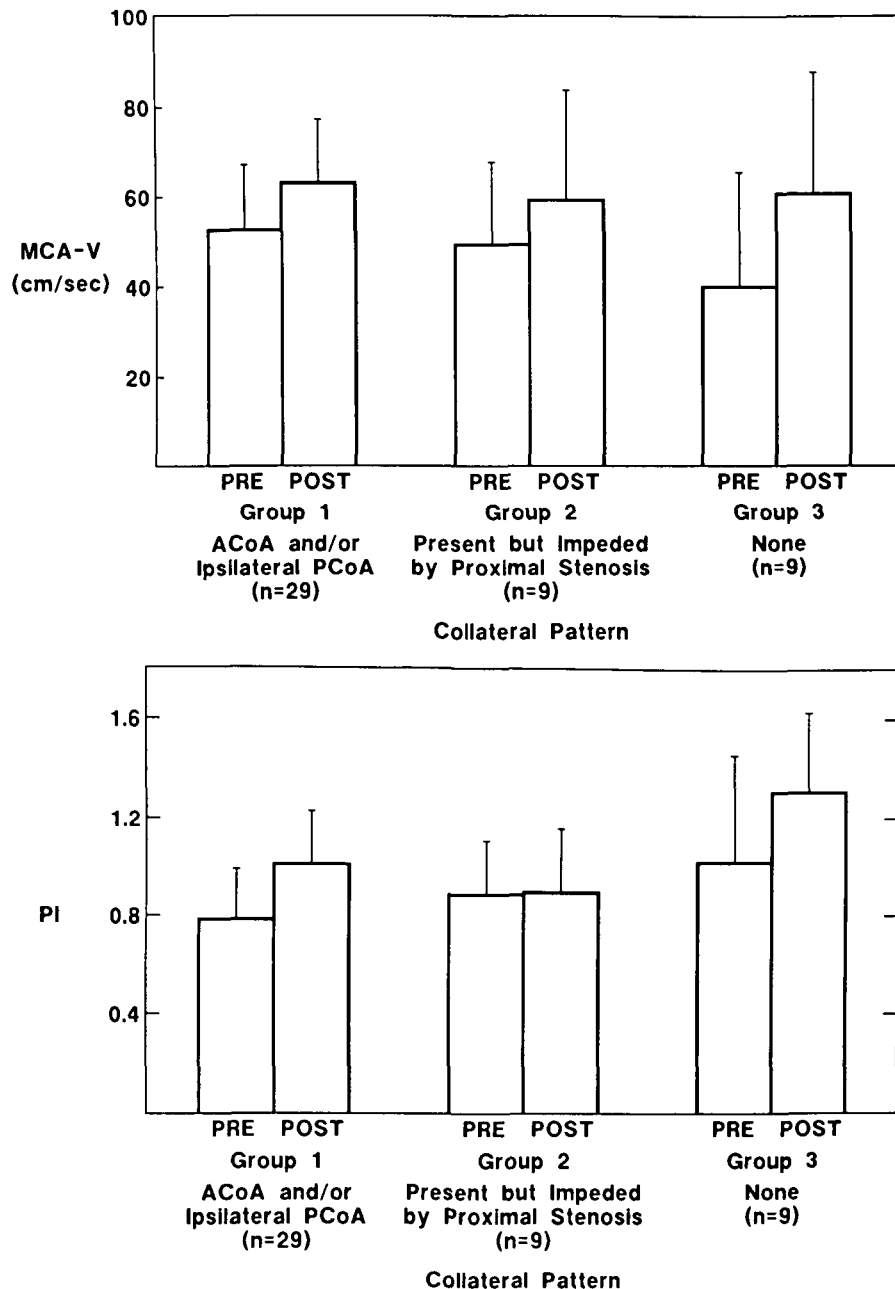


FIGURE 2. Effects of carotid endarterectomy 3 days after surgery with respect to patterns of collateral circulation. Top: Comparison of pre-operative and postoperative mean middle cerebral artery blood flow velocity (MCA-V). Increase in each group following surgery; largest increase in Group 3 (patients in whom no collateral pathways could be identified before surgery). Bottom: Group 3 also sustained largest increase in pulsatility index (PI) following endarterectomy. Error bars, +1 SD.

stenosis) requires the continued use of a PCoA, it may be difficult to identify the vessel by arteriography. There was a high false-positive rate with TCD (specificity of 60%) for the identification of PCoAs, perhaps due in part to the inability of arteriography to localize PCoAs that are not being actively recruited although they may be patent and may be available to function as major collateral pathways should the need arise.

Several previous studies have demonstrated that TCD can identify major collateral pathways around the circle of Willis.<sup>2,3,10-13</sup> In a study of 77 patients with extracranial cerebrovascular disease, both the sensitivity and specificity of TCD in the identification of major intracranial collaterals (i.e., ACoA and PCoA) were >85% compared with arteriography.<sup>3</sup>

A recent study with angiographic correlation in 39 patients demonstrated a TCD sensitivity of 94% for the ACoA and 88% for the PCoA.<sup>13</sup> Although manual compressions of the CCA have been suggested in the delineation of collateral vessels,<sup>2,12</sup> we routinely compressed both the anterior and posterior cerebral circulations in an outlined, stepwise manner, which led to the complete evaluation of 94% of our 50 patients. We assessed the ability of TCD to provide clinically useful, hemodynamically accurate data regarding cerebral collateral circulation potential further as patients proceeded to carotid endarterectomy and intraoperative multimodality cerebral monitoring.

We assessed cerebral perfusion during cross clamping using EEG, TCD, and SP. Although SP is

commonly used,<sup>14</sup> EEG has been used extensively in some centers over the past decade.<sup>15,16</sup> More recently, initial evaluations of the use of TCD in continuous intraoperative cerebral monitoring have been promising.<sup>4-6</sup> We have previously shown a direct correlation between SP and mean MCAV during cross clamping,<sup>11</sup> which has also been observed by others.<sup>12</sup> TCD has the advantage of providing continuous information on the current circulatory status of the brain, whereas SP provides only a single, initial measurement. EEG has not been extensively compared with TCD during carotid surgery. Our data are the most detailed comparison available between these two modalities.

Six of the seven patients with ischemic EEG abnormalities had very low MCAV ( $\leq 18$  cm/sec) during cross clamping. With continued comparison of TCD and EEG in more patients the ischemic threshold for MCAV may become more apparent. Each method of cerebral evaluation, although based on differing physical principles, demonstrated significant differences in the three groups during cross clamping.

TCD permits noninvasive evaluation of the integrity of cerebral collateralization, which correlates with the subsequent hemodynamic consequences of CCA cross clamping. There are numerous potential applications of TCD in the identification of cerebral collateral pathways as an indicator of risk for cerebral ischemia in the setting of proposed therapeutic interventions and with certain patterns of extracranial cerebrovascular disease.

#### Acknowledgments

The authors thank Susie May and the Department of Academic Affairs of Scripps Clinic and Research Foundation for technical assistance.

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KEY WORDS • collateral circulation • endarterectomy • ultrasonics