Preoperative MRA Flow Quantification in CEA Patients
Flow Differences Between Patients Who Develop Cerebral Ischemia and Patients Who Do Not Develop Cerebral Ischemia During Cross-Clamping of the Carotid Artery

D.R. Rutgers, MD; J.D. Blankensteijn, MD, PhD; J. van der Grond, PhD

Background and Purpose — We sought to investigate whether preoperative volume flow in the internal carotid arteries (ICAs), the basilar artery (BA), and the middle cerebral arteries (MCAs) and collateral flow via the circle of Willis differ between patients who do and patients who do not develop cerebral ischemia during clamping of the carotid artery in carotid endarterectomy (CEA).

Methods — Quantitative volume flow in the ICAs, BA, and MCAs and directional flow in the circle of Willis were measured preoperatively with 2-dimensional phase-contrast MR angiography in 86 CEA patients. During the operation, electroencephalographic (EEG) recordings were obtained that were monitored by a clinical neurophysiologist. Reference volume flow values were assessed in 24 control subjects.

Results — In patients with an ICA stenosis without contralateral ICA occlusion (n = 62), of whom 16% developed ischemic EEG changes during clamping, preoperative flow in the clamped ICA was significantly higher in patients with cerebral ischemia than in patients without (mean, 278 versus 160 mL/min; P < 0.05). Flow in the contralateral ICA (156 versus 273 mL/min; P < 0.01), flow in the BA (116 versus 165 mL/min; P < 0.05), and presence of collateral flow via the circle of Willis to the clamped ICA (0% versus 37%; P < 0.05) were significantly lower. MCA flow did not differ significantly between groups. Additionally, in patients with an ICA stenosis and a contralateral ICA occlusion (n = 24), of whom 42% developed cerebral ischemia, preoperative flow in the clamped ICA was significantly higher in patients with cerebral ischemia than in patients without (309 versus 239 mL/min; P < 0.05). BA flow, MCA flow, and presence of willisian collateral flow (0% versus 14%) did not differ significantly between groups.

Conclusions — Preoperative volume flow in the clamped ICA is significantly higher in CEA patients with ischemic EEG changes during clamping than in CEA patients without such changes. The latter patients probably have better developed collateral pathways preoperatively. (Stroke. 2000;31:3021-3028.)

Key Words: carotid artery diseases ■ carotid endarterectomy ■ magnetic resonance angiography

Carotid endarterectomy (CEA) is an important mainstay in the management of patients with a stenosis of the carotid artery. In CEA procedures, patients are often monitored with electroencephalography (EEG) to assess whether cerebral ischemia develops during the necessary cross-clamping of the carotid artery.1-7 The development of ischemic EEG changes likely indicates cerebral hypoperfusion.1,8,9 Preoperative evaluation of flow in the carotid artery that is clamped and of cerebroptal flow in other arteries such as the contralateral carotid artery and the basilar artery (BA) can add to the understanding of pathophysiological differences between patients who develop ischemic EEG changes during clamping and patients who do not. For example, when the carotid artery that is clamped has little flow, its relative contribution to cerebral blood supply is small, and clamping may carry little risk for cerebral ischemia.

The purpose of the present study was to investigate whether preoperative quantitative volume flow in the internal carotid arteries (ICAs), the BA, and the middle cerebral arteries (MCAs), as assessed with magnetic resonance angiography (MRA), differed between CEA patients who did and CEA patients who did not develop ischemic EEG changes during clamping of the carotid artery. In addition, we investigated whether these patients differed with respect to the presence of collateral flow via the circle of Willis.

Subjects and Methods

Patients and Control Subjects
Eighth-six patients with an ICA stenosis in whom a CEA was performed were included in this study. The patients had been referred to the departments of vascular surgery and neurology of our...
hospital between September 1995 and July 1999. Patients with an ICA stenosis only (n=62) had been included between 1995 and 1997, and subsequently patients with an ICA stenosis in combination with an occlusion of the contralateral ICA (n=24) were included between 1997 and 1999. We performed separate analyses for these patient groups because the presence of an occlusion of the contralateral ICA is thought to be associated with a higher risk of ischemic EEG changes during clamping of the carotid artery.3–5 The carotid arteries were investigated with intra-arterial digital subtraction angiography in 77 patients, according to the criteria of the North American Symptomatic Carotid Endarterectomy Trial (NASCET).10

In 9 patients, the carotid arteries were investigated with Doppler ultrasonography.

Clinically, patients were either asymptomatic (n=16) or had suffered from retinal (n=13) or cerebral ischemic symptoms (transient ischemic attack, n=29; stroke, n=28) in the 6 months previous to CEA. Retinal symptoms included transient monocular blindness (n=12) and retinal infarction (n=1). Transient monocular blindness was diagnosed if patients had attacks of loss of vision with sudden onset that lasted a maximum of 24 hours. Retinal infarction was diagnosed when vision loss in 1 eye lasted >24 hours and ophthalmoscopic examination showed infarction of the retina. A hemispheric transient ischemic attack was defined as an attack of dysphasia, unilateral paresis, or unilateral sensory symptoms with sudden onset that lasted a maximum of 24 hours. If symptoms lasted >24 hours, stroke was diagnosed. In patients with an ICA stenosis only, symptoms had occurred ipsilateral to the ICA that was operated on. In patients with an ICA stenosis and a contralateral ICA occlusion, symptoms had occurred either ipsilateral (n=4) or contralateral (n=20) to the operated ICA.

To obtain reference values for the quantitative MRA flow investigations, 24 control subjects were investigated. They were recruited from the departments of neurology and urology, where they were hospitalized for other than intracranial diseases. They had no history of cerebral neurological complaints or atherosclerotic disease. MRI scans did not show cerebral abnormalities in these subjects.

The Human Research Committee of our hospital approved the study protocol. All patients and control subjects gave informed consent to participate in the study.

**Magnetic Resonance Angiography**

The median time interval between the MRA scan and CEA was 2 days (25th percentile, 1 day; 75th percentile, 12 days). Investigations were performed on a 1.5-T whole-body system (ACS-NT 15 model, Philips Medical Systems). On the basis of 2 localizer MRA scans in the coronal and sagittal planes, a 2-dimensional phase-contrast (2D PC) slice was positioned at the level of the skull base to measure flow in the ICAs and the BA (nontriggered; repetition time [TR], 16 ms; echo time [TE], 9 ms; flip angle, 7.5°; slice thickness, 5 mm; field of view, 250×250 mm; matrix size, 256×256; 8 averages; velocity sensitivity, 40 cm/s). The 2D PC scans were positioned on the basis of the MIP reconstruction of the circle of Willis. Two investigators (D.R.R. and J. van der G.) independently evaluated the 2D PC images, along with the source images and MIP reconstructions of the circle of Willis, to assess the presence of retrograde flow in the A1 segments and the presence of posteroanterior flow in the posterior communicating arteries (PCoAs). This was performed blinded to the EEG findings during CEA.

To assess the presence of collateral flow via the circle of Willis, 2 consecutive 2D PC measurements were performed. Several studies have found PC MRA to be a reliable method to investigate collateral flow via the circle of Willis.26–28 One of the 2D PC measurements was phase encoded in the anteroposterior direction and another in the left-right direction (TR, 16 ms; TE, 9.1 ms; flip angle, 7.5°; slice thickness, 13 mm; field of view, 250×250 mm; matrix size, 256×256; 8 averages; velocity sensitivity, 40 cm/s). The 2D PC slices were positioned on the basis of the MIP reconstruction of the circle of Willis. Two investigators (D.R.R. and J. van der G.) independently evaluated the 2D PC images, along with the source images and MIP reconstructions of the circle of Willis, to assess the presence of retrograde flow in the A1 segments and the presence of posteroanterior flow in the posterior communicating arteries (PCoAs). This was performed blinded to the EEG findings during CEA.
C.E.A. Retrograde flow via the A1 segment was considered to indicate the presence of collateral flow via the anterior communicating artery (AcoA). Interobserver differences were reevaluated in a consensus meeting.

**Carotid Endarterectomy and Electroencephalography**

C.E.A. was performed under general anesthesia, which was induced with etomidate/fentanyl and maintained with N2O-O2/propofol. In addition, patients were given a local blockade of the cervical plexus to allow a relatively low level of anesthesia, thereby optimizing EEG monitoring for cerebral ischemia. Silver/silver chloride skin electrodes were positioned according to the International 10-20 System. A clinical neurophysiologist monitored the recordings continuously during the surgical procedure. Both the neurophysiologist and the surgeon who performed the C.E.A. were blinded to the preoperative MRA results. During the operation, the ICA and external carotid artery were clamped after prior intravenous heparinization to test whether cerebral ischemia occurred. Unilateral or bilateral decrease of alpha and beta activity during the first 2 minutes of test clamping, with or without simultaneous increase of theta or delta activity, was considered to indicate development of cerebral ischemia. In these cases, a Javid shunt (Impra Inc) was introduced, after which C.E.A. was performed. In all other patients, C.E.A. was performed without an intraluminal shunt.

**Statistical Analysis**

Differences in age, male/female ratio, degree of ICA stenosis, collateral flow via the circle of Willis, and clinical diagnoses were investigated with Student’s t test, χ2 test with Yates correction for continuity, or Fisher’s exact test. Dunnett’s post hoc analysis after ANOVA was used to compare flow between control subjects and each patient group. Student’s t test was used to compare flow between patients who developed cerebral ischemia and patients who did not. The t tests were preceded by an F test to determine equality of variances. If the F test was statistically significant, the results for unequal variances were used. Logistic regression analysis was applied to identify variables that were related to the presence or absence of cerebral ischemia during clamping of the carotid artery. A P value <0.05 was considered statistically significant.

**Results**

In patients with an ICA stenosis only, of whom 16% (n=10) showed ischemic EEG changes during intraoperative clamping (Table 1), age, male/female ratio, and degree of ICA stenosis did not differ significantly between patients who developed cerebral ischemia and those who did not. In patients with an ICA stenosis and a contralateral ICA occlusion, 42% (n=10) developed cerebral ischemia. These patients were significantly older (P<0.05) than those without cerebral ischemia. Male/female ratio and degree of ICA stenosis did not differ significantly between groups.

Table 2 shows preoperative quantitative volume flow for patients with an ICA stenosis only as well as for control subjects. Patients who developed cerebral ischemia did not differ significantly from control subjects with respect to flow, although they tended to have higher flow in the clamped ICA and lower flow in the contralateral ICA. Patients who did not develop cerebral ischemia had significantly lower flow in the clamped ICA than control subjects (P<0.05), while flow in the contralateral ICA and the BA was significantly higher (P<0.01). MCA flow did not differ between control subjects and patients. In patients with cerebral ischemia, flow in the clamped ICA was significantly higher than in patients without cerebral ischemia (P<0.05), while flow in the contralateral ICA and the BA was significantly lower (P<0.01 and P<0.05, respectively). The statistical significance of flow

### Table 1. Demographic Characteristics and Preoperative Degree of ICA Stenosis in Control Subjects, CEA Patients Who Developed Ischemic EEG Changes, and CEA Patients Who Did Not Develop Ischemic EEG Changes During Clamping of the Carotid Artery

<table>
<thead>
<tr>
<th>Demographic characteristics</th>
<th>ICA Stenosis</th>
<th>ICA Stenosis + Contralateral ICA Occlusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean±SD, y</td>
<td>62±9</td>
<td>63±12</td>
</tr>
<tr>
<td>Male/female, %</td>
<td>79/21</td>
<td>90/10</td>
</tr>
<tr>
<td>Degree of ICA stenosis, %</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clamped side</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mild to moderate*</td>
<td>20</td>
<td>21</td>
</tr>
<tr>
<td>Severe*</td>
<td>80</td>
<td>75</td>
</tr>
<tr>
<td>Near-occlusion</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>Contralateral side</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mild to moderate*</td>
<td>80</td>
<td>...</td>
</tr>
<tr>
<td>Severe*</td>
<td>20</td>
<td>...</td>
</tr>
<tr>
<td>Near-occlusion</td>
<td>0</td>
<td>...</td>
</tr>
<tr>
<td>Occlusion</td>
<td>zzz</td>
<td>100</td>
</tr>
</tbody>
</table>

*Mild to moderate refers to <70% ICA stenosis; severe, ≥70% ICA stenosis.
†P<0.05 vs control subjects.
‡P<0.05 vs patients with an occlusion of the contralateral ICA who did not develop ischemic EEG changes.
differences did not change when patients with a near-occlusion were excluded. There were no significant differences in MCA flow between patient groups.

Table 3 shows preoperative quantitative volume flow for patients with an ICA stenosis and a contralateral ICA occlusion. In patients who developed cerebral ischemia, flow in the clamped ICA was significantly higher than in control subjects \((P<0.01)\). In both patients with cerebral ischemia and patients without, flow in the BA was significantly higher than in control subjects \((P<0.01\) and \(P<0.001\), respectively), while flow in the MCA contralateral to the clamped ICA was significantly lower \((P<0.05\) and \(P<0.01\), respectively). In patients who developed cerebral ischemia, flow in the clamped ICA was significantly higher than in patients who did not develop cerebral ischemia \((P<0.05)\). There was no significant difference in BA flow, although it tended to be higher in patients without cerebral ischemia. MCA flow did not differ significantly between patient groups. No significant flow differences were found between patients who had symptoms ipsilateral to the clamped ICA and patients who had symptoms contralateral to the clamped ICA.

Figure 3 shows typical examples of directional flow investigations in patients with an ICA stenosis only. Table 4 shows the prevalence of collateral flow via the circle of Willis to the side of the clamped ICA. None of the patients with an ICA stenosis only who developed cerebral ischemia had collateral flow via the circle of Willis (Table 4). In those without cerebral ischemia, significantly more patients had collateral flow via the circle of Willis to the side of the clamped ICA compared with patients with cerebral ischemia \((P<0.05)\). In patients with an ICA stenosis and a contralateral ICA occlusion, those with cerebral ischemia did not have collateral flow via the circle of Willis to the side of the clamped ICA. Patients without cerebral ischemia had collateral flow to the side of the clamped ICA more often; however, this was not statistically significant. In both patients with cerebral ischemia and those without, a great majority had collateral flow via the circle of Willis to the side of the ICA occlusion (90% and 86%, respectively). In the pooled data of patients with an ICA stenosis only, those without collateral flow via the circle of Willis had significantly higher flow in the clamped ICA (mean, 190 mL/min; 95% CI, 160 to 220 mL/min) than patients with collateral flow to the side of the clamped ICA (mean, 138 mL/min; 95% CI, 109 to 168; \(P<0.05\)). Flow in the contralateral ICA was significantly lower (mean, 233 mL/min; 95% CI, 199 to 268 versus mean, 

<table>
<thead>
<tr>
<th>Quantitative MRA Flow, mL/min</th>
<th>Control Subjects (n=24)</th>
<th>ICA Stenosis (n=62)</th>
<th>(P^*) (Ischemia vs No Ischemia)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICA, clamped side</td>
<td>212 (188–236)</td>
<td>278 (162–395)†‡</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>ICA, contralateral side</td>
<td>212 (188–236)</td>
<td>156 (60–252)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>BA</td>
<td>106 (93–119)</td>
<td>116 (66–166)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>MCA, ipsilateral to clamped ICA</td>
<td>121 (110–131)</td>
<td>112 (93–130)</td>
<td>NS</td>
</tr>
<tr>
<td>MCA, contralateral to clamped ICA</td>
<td>121 (110–131)</td>
<td>125 (92–158)</td>
<td>NS</td>
</tr>
</tbody>
</table>

Values are mean (95% CI).
*Student’s \(t\) test.
†\(P<0.05\), ‡\(P<0.01\), §\(P<0.001\) vs control subjects (Dunnett’s post hoc analysis after ANOVA).
320 mL/min; 95% CI, 280 to 361; \( P < 0.01 \)), while flow in the BA tended to be lower (mean, 151 mL/min; 95% CI, 131 to 172 versus mean, 181 mL/min; 95% CI, 152 to 210). Figure 4 shows the clinical diagnoses in patients who developed cerebral ischemia and those who did not for both patients with an ICA stenosis only and patients with an ICA stenosis and a contralateral ICA occlusion. In both patient groups there was no significant difference in the distribution of clinical diagnoses between patients with ischemic EEG changes during clamping and patients without such changes.

In a logistic regression analysis of all patients, the development of cerebral ischemia during clamping was significantly related to flow in the clamped ICA (\( P < 0.001 \)). Clinical diagnosis (\( P = 0.24 \)), age (\( P = 0.23 \)), sex (\( P = 0.30 \)), and degree of stenosis of the clamped ICA (\( P = 0.14 \)) or contralateral ICA (\( P = 0.76 \)) were not significantly related.

**Discussion**

The present study relates preoperative MRA volume flow in cerebropetal arteries and MCAs to EEG findings during clamping of the carotid artery in CEA procedures. The most important finding is that patients with an ICA stenosis who developed cerebral ischemia had significantly higher preoperative volume flow in the ICA that was clamped than patients who did not develop cerebral ischemia.

The development of ischemic EEG changes during clamping of the carotid artery is likely caused by reduced cerebral blood supply.\(^1\,\text{8,9}\) Blood flow to the cerebrum predominantly takes place through the ICAs and the BA. In patients who developed cerebral ischemia, the ICA that was clamped had a relatively high volume flow, suggesting that its contribution to cerebral blood supply was relatively large. In patients who did not develop cerebral ischemia, the ICA that was clamped had a relatively small contribution to cerebral blood supply. These findings suggest that clamping of an artery that contributes relatively much to cerebral blood supply causes ischemic EEG changes.

There may be a number of explanations for the relatively high flow in the clamped ICA in patients with cerebral ischemia after clamping versus patients without. One expla-

**Figure 3.** Examples of MRA investigations of the circle of Willis in 3 patients with a right-sided ICA stenosis without contralateral ICA occlusion. Each column of images represents a patient. The top row of images shows MIP of the circle of Willis as investigated with 3-dimensional time of flight MRA. The middle row shows 2D PC images that were phase encoded in the anteroposterior (AP) direction. Blood flowing in the anterior direction is black, and blood flowing in the posterior direction is white. The bottom row shows 2D PC images that were phase encoded in the left-right (LR) direction. Blood flowing to the patient’s right is black, and blood flowing to the patient’s left is white. Left column, No collateral flow via the circle of Willis. Middle column, Collateral flow via the ACoA. Right column, Collateral flow via the PCoA.

<table>
<thead>
<tr>
<th>Collateral Flow Via Circle of Willis to Clamped ICA</th>
<th>ICA Stenosis (n=10)</th>
<th>ICA Stenosis + Contralateral ICA Occlusion (n=14)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Ischemia (n=10)</td>
<td>Ischemia (n=10)</td>
</tr>
<tr>
<td></td>
<td>No Ischemia (n=52)</td>
<td>No ischemia (n=14)</td>
</tr>
<tr>
<td>Via ACoA or PCoA</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Via ACoA only</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Via PCoA only</td>
<td>0</td>
<td>12</td>
</tr>
<tr>
<td>Via ACoA and PCoA</td>
<td>0</td>
<td>6</td>
</tr>
</tbody>
</table>

Values are percentages.

*\( P < 0.05 \) vs patients with ICA stenosis who developed cerebral ischemia.
nation is that it reflects an absence of collateral flow. In patients with an ICA stenosis, the circle of Willis is regarded as the primary collateral pathway, while leptomeningeal anastomoses are recruited when the circle of Willis falls short. The ophthalmic artery is also considered a secondary pathway, but whether it makes a functional contribution to cerebral blood supply has been questioned in a number of studies. When collateral pathways, such as the ACoA and PCoA, are absent or hypoplastic, flow in the stenosed ICA may be relatively high because compensatory collateral flow is lacking. This assumption is supported by our results, since we found that the relatively high flow in the clamped ICA in patients with ischemia was accompanied by absence of collateral flow via the circle of Willis. When flow in the clamped ICA is low, this may indicate that collateralization is present. In such patients, flow in the contralateral ICA and the BA likely is increased because these vessels are important sources of collateral flow. Our findings in patients with an ICA stenosis only are in accordance with this hypothesis. In patients with an ICA stenosis and a contralateral ICA occlusion, the presence of collateral flow via the circle of Willis did not differ between patients with relatively high flow and patients with low flow. Possibly, there were differences in leptomeningeal collateral flow, but this was not investigated in the present study. Another explanation for the relatively high flow in the clamped ICA in patients with ischemia may be that we underestimated the degree of ICA stenosis in patients with low flow. In particular, patients with a near-occlusion may be graded too low if the diameter of the stenosis is compared with the diameter of the artery beyond, since in these patients the diameter of the artery beyond may become very small. Using NASCET criteria, we have anticipated this pitfall as much as possible. Although we cannot exclude it, we assume that we did not underestimate the degree of ICA stenosis in our patients.

Patients who did not develop cerebral ischemia had relatively low flow in the clamped ICA. Despite this, ipsilateral MCA flow was not reduced. Again, this may suggest that they had better-developed collateral flow, through which MCA flow was maintained. It is likely that better-developed collateralization, whether or not in combination with other factors such as better vasoreactive capacity, has contributed to the fact that cerebral ischemia did not occur in these patients during clamping. However, it should be emphasized that we did not measure collateral flow or vasoreactivity at the time of clamping.

Several studies have investigated collateral flow patterns preoperatively in CEA patients who showed cerebral ischemia during clamping of the carotid artery. Schwartz et al studied cerebral angiograms in 30 CEA patients to assess the presence of collateral flow via the circle of Willis to the hemisphere ipsilateral to the clamped artery. Ischemic EEG changes during clamping were present in all patients who did not have collateral flow (n = 15). In patients who did have collateral flow (n = 15), most (14/15) did not develop cerebral ischemia. A similar angiography study was performed by Lopez-Bresnahan et al. In 65 patients who underwent endarterectomies, they found that ischemic EEG changes occurred less frequently in patients who had collateral flow via the ACoA (9/43) compared with patients who did not have such flow (12/24). Wain et al related preoperative cerebral angiograms to intraoperative measurement of somatosensory evoked potentials. Ischemic somatosensory evoked potential changes occurred less frequently in patients with collateral flow via the ACoA (0/51) than in patients without such flow (9/36). In addition, patients without collateral flow via the ACoA had fewer ischemic somatosensory evoked potential changes (1/17) if they had collateral flow via the PCoA than if they had no willisian collateral flow at all (8/19). Using transcranial Doppler sonography, Schneider et al assessed preoperative collateral flow patterns in 47 CEA patients. Ischemic EEG abnormalities during clamping occurred less frequently in patients with good collateralization via the circle of Willis (1/29) than in patients without (6/18). These studies all indicate that collateral flow is better developed in patients who do not develop cerebral ischemia during clamping than in patients who do develop such changes.

MRA is used as an umbrella term for MR methods to visualize vascular flow. Several techniques are available, such as time of flight, PC, black blood, and contrast-enhanced
angiography. When vessels are visualized anatomically, each technique has its advantages and disadvantages. These are related to many factors, such as acquisition time, sensitivity to low flow, artifacts, and the burden for the patient. PC MRA has an additional advantage in that it provides a quick and easy way to assess volume flow. To obtain reliable measurements, a number of problems have to be considered, such as minimizing partial volume effects and choosing an adequate velocity sensitivity value to avoid artifacts. Nevertheless, PC MRA is the most commonly used MR method for clinical volume flow measurement. In the present study we found that quantitative PC MRA in the ICAs and BA was related to the presence of collateral flow via the circle of Willis in patients with an ICA stenosis only. However, it should be emphasized that MRA flow quantification cannot replace conventional angiography in assessing which particular collateral, such as the circle of Willis or leptomeningeal anastomoses, is recruited in the individual patient with carotid artery disease.

To prevent cerebral ischemia during clamping of the carotid artery, CEA may be performed with an intraluminal shunt. The use of a shunt during CEA remains a controversial topic. It may reduce cerebral hypoperfusion but may also carry an additional risk of complications such as embolism and dissection of the carotid artery. When shunting is performed selectively, there are several methods to assess the need for shunt placement. In most methods, patients are monitored intraoperatively through continuous EEG registration, neurological evaluation of the awake patient under regional anesthesia, or carotid stump pressure measurements. To assess preoperatively which patients will be in need of a shunt, collateral flow patterns on conventional cerebral angiograms may be indicative. The present study suggests that MRA flow quantification may provide an alternative method to determine preoperatively which CEA patients are at risk for cerebral ischemia and thus may need a shunt. In particular, this seems useful when MRA is applied to assess the degree of ICA stenosis in the preoperative workup of patients. An additional quantitative MRA flow measurement would take <1 minute of extra scan time in these patients. However, from the present study it cannot be concluded whether such an approach is beneficial in the management of CEA patients.

Our study may have a number of limitations. Although it is known that flow in a stenosed ICA is related to the degree of stenosis, in particular when the ICA is severely obstructed, we did not subdivide our results accordingly. The reason is that ICA flow is also related to the presence of collaterals that contribute to flow downstream of the stenosis. Although we measured the presence of collateral flow via 2 collaterals, namely, the ACoA and the PCoA, we did not investigate other pathways such as leptomeningeal anastomoses. These collaterals may also be important. Nevertheless, it is likely that when the ICA is nearly occluded, its degree of stenosis will be the most important factor that determines ICA flow. We found cerebral ischemia during clamping in 16% of patients with an ICA stenosis only and in 42% of patients with an ICA stenosis and a contralateral ICA occlusion. Although these proportions are in good accordance with a number of previous studies, other studies found higher proportions, ranging from 25% to 38% in patients with an ICA stenosis only and from 59% to 71% in patients with an ICA stenosis and a contralateral ICA occlusion. It is hard to explain these discrepancies, but there may have been differences in composition of study groups, methodology of EEG monitoring, or interpretation of EEG results. In patients with an ICA stenosis and a contralateral ICA occlusion, we found that patients who developed cerebral ischemia were older than those who did not develop cerebral ischemia. This does not confound our observation of higher flow in the clamped ICA in patients with cerebral ischemia, since cerebropetal blood flow is known to decrease with age. Consequently, the significant flow difference we found between patients likely has been underestimated.

In conclusion, PC MRA flow quantification in cerebropetal arteries can provide important hemodynamic information in patients who are scheduled for CEA. Preoperative distribution of flow in the ICAs and BA differs between CEA patients who develop ischemic EEG changes during clamping of the carotid artery and CEA patients who do not develop such changes. The former patients have significantly higher volume flow in the clamped ICA than the latter patients.

Acknowledgment

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