The role of indirect extracranial-intracranial bypass in the treatment of symptomatic intracranial atheroocclusive disease

Clinical article

RICARDO J. KOMOTAR, M.D.,† ROBERT M. STARKE, B.A.,† MARC L. OTTEN, M.D.,† MAXWELL B. MERKOW, B.S.,† MATTHEW C. GARRETT, B.S.,† RANDOLPH S. MARSHALL, M.D.,‡ MITCHELL S. V. ELKIND, M.D.,‡ and E. SANDER CONNOLLY JR., M.D.†

Departments of †Neurological Surgery, and ‡Neurology, Columbia University, New York, New York

Object. The optimal treatment of medically refractory intracranial atheroocclusive disease remains unclear. The EC-IC Bypass Study Investigators found that patients with internal carotid and middle cerebral artery (ICA and MCA) occlusion received no benefit from direct superficial temporal artery to MCA bypass, and that patients with ICA occlusion and MCA stenosis may have actually fared worse after surgery, perhaps in part due to flow reversal in critical perforator-bearing segments. Although the results of recent investigations have suggested that direct bypass may be beneficial in a subgroup of patients with hemodynamic failure secondary to unilateral ICA occlusion, similar data do not exist for patients with hemodynamic failure from other intracranial stenoocclusive diseases. Indirect bypass via encephaloduroarteriosynangiosis offers a surgical alternative that may avoid rapid flow reversal while providing additional flow to at-risk, distal vascular territories.

Methods. Twelve patients with medically resistant hemodynamic failure from intracranial atheroocclusive disease underwent indirect vascular bypass. Eight patients had ICA occlusion and coexistent MCA stenosis, 1 patient had tandem ICA stenoses and MCA stenosis, 1 patient had tandem ICA and MCA occlusion, 1 patient had ICA and posterior cerebral artery occlusion and an ischemic hemisphere supplied via a proximal superficial temporal artery branch, and 1 patient had poor donor arteries and severe medical comorbidities that precluded the use of general anesthesia. Patient evaluation included clinical assessment of neurological status, CT scanning, MR imaging, digital subtraction angiography, and transcranial Doppler ultrasonography with CO₂ reactivity, or SPECT with acetazolamide challenge. Patient records were reviewed and patients were interviewed for outcome assessment, including transient ischemic attack (TIA), cerebral infarction, change in cerebral perfusion, graft patency, and functional level according to the modified Rankin scale. Kaplan-Meier cumulative failure curves for the primary end point of cerebral infarction were used to compare these patients to a control group of 81 patients derived from the literature who received medical management for severe symptomatic hemodynamic failure.

Results. Eleven patients underwent encephaloduroarteriosynangiosis and 1 patient received bur holes with dural and arachnoid incisions; the mean length of follow-up was 51.2 ± 40.1 months. Five patients had decreased perfusion on follow-up despite graft patency, and 10 patients suffered new infarctions or TIAs during the follow-up period. Five patients (42%) suffered infarctions within 1 year of surgery. A meta-analysis of 4 studies of patients with symptomatic ICA occlusion and severe hemodynamic failure who underwent medical treatment revealed a new infarction rate of 30% in the first year after entry into the study. There was no significant difference between patients with severe hemodynamic failure who underwent surgery and those in the medically treated control group (log-rank test, p = 0.179).

Conclusions. The authors found that indirect bypass does not promote adequate pial collateral artery development and appears to be of limited utility in patients with symptomatic ICA or MCA stenoocclusive disease and secondary hemodynamic failure. Rates of postoperative TIAs or cerebral infarctions after indirect bypass in this patient population do not differ from previous reports in patients who received medical management only.

(DOI: 10.3171/2008.9.JNS17658)

Key Words • bypass • encephaloduroarteriosynangiosis • hemodynamic failure • internal carotid artery • stenoocclusive disease

Fifteen percent of patients with TIAs or stroke present with atherosclerotic ICA occlusion or stenosis.3,9,11,41 The optimal management of this condition remains controversial, as a large number of these patients will continue to be symptomatic despite maximal medical therapy.3,9,11,41 Therapeutic regimens may be further complicated in those with complete and/or bilateral vessel occlusion. Moreover, stenoocclusive disease of the ICA at or above the C-2 vertebral body is not amenable to carotid endarterectomy.

In the first clinical trial to randomize patients with ICA stenoocclusive disease to medical or surgical therapy, it was found that STA-MCA bypass provided no benefit over medical therapy.9,10 In particular, patients who underwent direct bypass for ICA occlusion or MCA...
Indirect bypass for symptomatic ICA occlusive disease

stenosis appeared to have worse outcomes than those who received medical treatment. At that time, available technology did not allow clinicians to differentiate between ischemic episodes caused by embolic disease and those secondary to hemodynamic failure. Although authors of more recent studies have suggested that patients with symptomatic ICA occlusion and associated severe hemodynamic failure may benefit from direct vessel bypass, there have been few, if any, studies specifically addressing contemporary surgical management in patients in whom direct bypass might be harmful, such as those with MCA stenosis. Direct bypass may be harmful in patients with MCA stenosis in part because flow reversal in critical perforator-bearing segments may lead to either conduit vessel or perforating artery failure secondary to stasis. In contrast to direct bypass, indirect bypass may be performed in patients with extremely small donor vessels and might also augment cerebral perfusion while avoiding flow reversal in highly stenotic segments.

Encephaloduroarteriosynangiosis is a method of indirect bypass and cerebral revascularization that is most commonly used in patients with moyamoya disease. Bur holes with arachnoid and dural incisions have also been used to stimulate neovascularization. In this study, we present our institutional experience using indirect bypass in the treatment of patients with medically refractory atheroocclusive disease.

Methods

Patient Population and Treatment Protocol

From September 1999 to July 2006, 12 patients with symptomatic atheroocclusive disease underwent indirect bypass with EDAS or bur holes at Columbia University Medical Center. All patients consented to have their outcomes reviewed in this institutional review board–approved study. Patient evaluation included clinical assessment of neurological status, CT scanning, MR imaging, digital subtraction angiography, and TCD ultrasonography with CO2 reactivity or SPECT with acetazolamide challenge. Admission, discharge, and follow-up handicap status was graded according to the modified Rankin Scale. Medical therapy had previously failed in all included patients, meaning that they had experienced recurrent TIA or stroke while receiving blood pressure control and antithrombotic or anticoagulation therapy. Patients with active TIs or strokes received medical therapy until they were without new ischemic events for at least 5 weeks before surgery, but all patients were symptomatic within the 3 months prior to surgery. Intractable TIs were defined as > 5 episodes of reversible focal symptoms.

Imaging Studies

Cerebral blood flow was evaluated on SPECT after intravenous administration of 21 mCi of Tc-99m hexamethyl propyleneamine oxime. Patients were assessed for perfusion failure and inadequate collateral circulation following acetazolamide challenge. Cerebrovascular reserve capacity was also evaluated by assessing CO2 partial pressure and TCD ultrasonography measurements of the MCA during normocapnia and hypercapnia (induced by breathing a fixed mixture of 5% CO2 in 95% O2).

All patients had hypoperfusion in the MCA distribution at baseline, and severe hemodynamic failure demonstrated on SPECT with acetazolamide challenge or TCD with CO2 reactivity. The relative change in perfusion between baseline status and the acetazolamide study was calculated for each patient to identify the extent of hemodynamic failure: [(normalized acetazolamide study – normalized baseline study)/baseline study] × 100%. On SPECT a normal hemodynamic status was defined as an increase in perfusion following acetazolamide challenge, mild hemodynamic failure as 0–10% perfusion deficits, and severe hemodynamic failure as a > 10% decrease in perfusion after acetazolamide challenge. Sufficient CO2 reactivity on TCD ultrasonography testing was defined as a 10% increase in MCA blood flow during hypercapnia. Mild hemodynamic failure was defined as a lack of increase in flow during hypercapnia, and severe hemodynamic failure as decreased flow during hypercapnia. Patients were followed up on SPECT scanning and acetazolamide challenge or TCD and CO2 challenge starting 3 months after receiving EDAS.

To compare surgical to nonsurgical treatment, we performed a meta-analysis of medically treated patients with available follow-up information and symptomatic internal cerebral artery atheroocclusive disease with severe hemodynamic failure. Patient history, assessment of severe hemodynamic failure, and outcomes in the literature population are presented in Table 1.

Surgical Planning

The first 9 patients underwent EDAS rather than direct bypass surgery because the EC-IC Bypass Study Investigators demonstrated that patients with ICA occlusion and MCA stenosis fared significantly worse with surgery, perhaps in part due to flow reversal in critical perforator-bearing segments. The patient in Case 10 had a severe MCA occlusion that precluded direct, surgical MCA-STA bypass. The patient in Case 11 had ICA occlusion with retrograde filling from the external carotid through the ophthalmic artery. Because of a hypoplastic P1 segment of the posterior cerebral artery, it was believed that the patient was dependent on this flow and direct bypass might reverse flow supplied by this critical segment. One patient, who had a poor donor STA and multiple medical comorbidities that precluded a lengthy surgery, received only bur holes. None of the patients were candidates for endovascular surgery at the time of EDAS. The patients in Cases 1–4, 6, and 10–12 all had ICA occlusions that precluded endovascular access to the MCAs. The patient in Case 7 received a stent into the ICA just after the common carotid artery bifurcation, and also had lengthy sections of intracranial stenosis that would have required extensive stenting. The patient in Case 8 underwent intracranial stent placement; this patient became symptomatic ipsilateral to the stent and significant stenosis in the region of the stent was also discovered. The region of stenosis was noted to further involve the ICA and MCA.
The patients in Cases 5 and 9 underwent significantly lengthy dissections that precluded endovascular access to the stenotic MCA sections.

**Surgical Treatment**

Encephaloduroarteriosynangiosis was performed in 11 patients using either the STA or occipital artery. Encephaloduroarteriosynangiosis was performed on the symptomatic side in the distribution of perfusion failure as noted on SPECT or TCD ultrasonography. Five patients received bur holes in addition to those necessary for EDAS, as well as dural and arachnoid incisions to stimulate angiogenesis in ischemic regions not perfused by the EDAS bypass. One patient with a poor donor STA and multiple medical comorbidities that precluded a lengthy surgery received only bur holes (Fig. 1).

Intraoperative Doppler ultrasonography was used to demonstrate graft patency. All patients received outpatient follow-up, and follow-up imaging was performed with SPECT, TCD ultrasonography, angiography, CT, or MR imaging as clinically indicated. Postoperatively, medical management was optimized to treat vascular risk factors, and patients received antiplatelet therapy (aspirin or clopidogrel); blood pressure was managed conservatively to avoid relative hypotension and decreased cerebral blood flow in hemodynamically challenged patients.

Briefly, EDAS involves placement of an external carotid artery (most commonly the STA) branch beneath the arachnoid mater in ischemic territories. In certain circumstances, depending on the territory at risk, the occipital artery may also be used. Preoperatively, TCD ultrasonography is used to map the course of the target artery. Intraoperatively, the target artery is dissected completely free, a craniotomy is performed, and the dura mater is opened. The target artery is then sewn to the pia/arachnoid with a 10–0 Prolene suture under microscopic vision after extensive arachnoid dissection. The bone flap is replaced after cutting out the artery’s entry and exit sites. In selected patients, multiple bur holes with arachnoid and dural incisions were made over the region of interest without vessel transplantation.

**Statistical Analysis**

Kaplan-Meier cumulative failure curves for the primary end point of infarction were used to compare our patients to those reported in the literature who received medical treatment for symptomatic ICA occlusion and severe hemodynamic failure (81 cases). The log-rank test was used to assess differences in survival curves and Cox regression was used to assess hazard ratios. Probability values ≤ 0.05 were considered statistically significant.

**Results**

Twelve patients underwent indirect bypass using EDAS and/or bur holes for the treatment of symptomatic ICA/MCA occlusion (Fig. 1). All patients had hypoperfusion on baseline SPECT scans and severe hemodynamic failure following acetazolamide challenge. On angiography all patients had either stenosis (70–99%) or occlusion of at least 1 ICA. Eight patients had unilateral ICA occlusion, 2 patients had bilateral ICA occlusion, and in 2 patients ICA occlusion was caused by a prior dissection. On presurgical angiography studies, no patient was found to have developed significant EC-IC collateral vessels, and no patient had abnormal vasculature suggesting moyamoya disease on arterial phase angiography.

The study cohort included 6 men and 6 women with a mean age of 55.8 ± 13.5 years at the time of surgery (Table 2). The majority of patients had at least 1 known vascular disease risk factor: 8 had a history of hypertension, 5 were smokers, 4 had hypercholesterolemia, 3 had a history of coronary artery disease, and 2 had diabetes mellitus. All patients had a history of TIAs or stroke; 11 had a history of TIAs, and 11 had a history of stroke. Additionally, 6 patients had intractable TIAs and 1 patient experienced 2 strokes.

The mean follow-up period was 51.2 ± 40.1 months (Table 3). Complications developed in 3 patients as a result of surgery: in 1 patient there was delayed wound breakdown and granuloma formation that required surgical removal, 1 patient with bilateral ICA occlusions experienced an infarction contralateral to the surgical site 2 months later, and 1 patient experienced a significant de novo moyamoya disease on arterial phase angiography.
days postoperatively (possibly due to self-discontinuation of antiplatelet/antithrombotic treatment), and in 1 patient who required anticoagulation therapy immediately after surgery a subdural hematoma developed that necessitated evacuation. Postoperatively, 3 patients experienced a decline in functional ability: 2 patients had minor deficits (1 point change on the modified Rankin Scale), and 1 patient developed temporary hemisensory loss and weakness that returned to baseline 2 months later.

Eleven of 12 patients underwent a mean of 1.6 (range 1–3) postoperative SPECT scans with acetazolamide challenge or TCD with CO₂ challenge starting 3 months
after EDAS (range 3–39 months). On delayed follow-up, 2 patients had increased perfusion to previously hypoperfused areas, 5 experienced further decrements in perfusion, and in 4 patients there was no change. Three of the 5 patients demonstrated decreased perfusion on follow-up SPECT in comparison with previous imaging results despite clear evidence of continued graft patency on follow-up angiography.

Nine patients had TIAs or infarctions after discharge from the hospital: 7 patients had TIAs, 4 patients had strokes, and 6 patients had new infarctions on follow-up imaging (4 symptomatic), 5 of which were ipsilateral to the EDAS procedure. Including the patient in whom an infarction developed contralateral to the EDAS procedure 2 days postoperatively due to possible self-discontinuation of antiplatelet/antithrombotic treatment, 7 patients had infarction. Of the 6 patients with previously intractable TIAs, 1 patient had 2 TIAs followed by bilateral infarction, 1 had an ipsilateral infarction and continued to have TIAs until 26 months postoperatively, 1 continued to have intractable TIAs until 13 months postoperatively, 2 had ipsilateral infarction, and 1 had no further TIAs.

Five (42%) of 12 patients had infarctions within a year of surgery (Fig. 2). One patient was lost to follow-up after 4 months. Assuming the worst case scenario that treatment failed in this patient, the rate of infarction within 1 year of surgery is estimated at 50%. Based on available information in medically treated patients, the rate of infarction in the first year may be estimated at 30% (Fig. 3). The median time to infarction was 14 months in patients who underwent surgery versus 36 months in those who received medical treatment only. There was no significant difference between cumulative infarction curves between the 2 groups (p = 0.179). Patients who underwent surgery did not have a significantly increased risk of infarction (hazard ratio 1.72, 95% CI 0.76–3.91; p = 0.192).

Of the patients with infarctions, 5 were ipsilateral and 2 contralateral to the EDAS site. Considering that it takes time to develop collateral vessels after EDAS, we examined the rates of infarction starting 6 months postoperatively. Of the patients with available follow-up who remained infarction-free for the first 6 months, subsequent infarctions developed in 2 of 8. Importantly, there were no significant differences between patients with infarctions starting 6 months postoperatively and medically treated patients reported in the literature (p = 0.179).

From the time of admission to the last follow-up, 3 patients had improvement in functional ability, and 1 patient had improvement before experiencing a decline in health due to medical comorbidities 4 years postoperatively. Five patients had no change in functional ability from their baseline status, and 3 patients had a decrease in functional abilities due to cerebral ischemia or complications of treatment after surgery. There was no difference in the incidence of infarction/TIA, perfusion changes, or functional outcome in patients with dissection and MCA stenosis compared to the rest of the cohort.

<table>
<thead>
<tr>
<th>TABLE 2: Summary of patient characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Characteristic</td>
</tr>
<tr>
<td>age (yrs)</td>
</tr>
<tr>
<td>female</td>
</tr>
<tr>
<td>medical hx</td>
</tr>
<tr>
<td>hypertension</td>
</tr>
<tr>
<td>hypercholesterolemia</td>
</tr>
<tr>
<td>diabetes mellitus</td>
</tr>
<tr>
<td>coronary heart disease</td>
</tr>
<tr>
<td>myocardial infarction</td>
</tr>
<tr>
<td>smoking</td>
</tr>
<tr>
<td>presentation</td>
</tr>
<tr>
<td>weakness/hemiparesis</td>
</tr>
<tr>
<td>dysarthria</td>
</tr>
<tr>
<td>sensory loss</td>
</tr>
<tr>
<td>limb shaking</td>
</tr>
<tr>
<td>visual defect</td>
</tr>
<tr>
<td>seizure</td>
</tr>
<tr>
<td>syncope</td>
</tr>
<tr>
<td>hx of TIA</td>
</tr>
<tr>
<td>hx of intractable TIAs</td>
</tr>
<tr>
<td>hx of stroke</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>TABLE 3: Summary of outcomes in 12 patients*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Outcome</td>
</tr>
<tr>
<td>in-hospital complications</td>
</tr>
<tr>
<td>medical</td>
</tr>
<tr>
<td>surgical</td>
</tr>
<tr>
<td>perfusion</td>
</tr>
<tr>
<td>increased</td>
</tr>
<tr>
<td>decreased</td>
</tr>
<tr>
<td>no change</td>
</tr>
<tr>
<td>FU complications</td>
</tr>
<tr>
<td>TIA</td>
</tr>
<tr>
<td>infarction</td>
</tr>
<tr>
<td>infarct ipsilateral to op</td>
</tr>
<tr>
<td>infarct w/in 1 yr of op</td>
</tr>
<tr>
<td>infarct 6 months to end of FU</td>
</tr>
<tr>
<td>functional outcome‡</td>
</tr>
<tr>
<td>improved independence</td>
</tr>
<tr>
<td>decreased independence</td>
</tr>
<tr>
<td>no change</td>
</tr>
</tbody>
</table>

* One patient did not undergo follow-up imaging.
† Percent out of 8 patients without infarction and with available follow-up.
‡ According to the modified Rankin Scale.
**Indirect bypass for symptomatic ICA occlusive disease**

In the first randomized clinical trial, it was found that direct STA-MCA bypass provided no benefit over medical therapy.\(^9,10\) More specifically patients who underwent direct bypass for symptomatic ICA occlusion and MCA stenosis had a 1.2- (95% CI 1.00–1.59) and 1.6-fold (95% CI 1.08–2.35) increased risk of infarction, respectively, compared to those who received medical treatment.\(^9,10\)

Since the EC-IC Bypass Trial, however, improved technology has allowed the identification of a subset of patients with ICA stenoocclusive disease and hemodynamic failure. Although more recent studies have shown that direct bypass may be beneficial in this patient population,\(^2,5,6,13,15,22,30,33,34,42,46\) there have been no studies in patients with intracranial atheroocclusive disease and proven perfusion failure who may not tolerate direct bypass. Indirect bypass might in theory avoid rapid flow reversal while slowly providing additional flow to distal vascular beds at risk. Furthermore, in contrast to direct bypass, indirect bypass may be safer, generally less complicated, and augment a greater region of cerebral perfusion.

Encephaloduroarteriosynangiosis is an indirect bypass method that is commonly used in the treatment of patients with symptomatic moyamoya disease secondary to documented hemodynamic failure.\(^17,18,23,28,31,36,37,44\) The authors of some studies have demonstrated that in this population, EDAS is effective in decreasing TIAs and maintaining cognitive function,\(^25,26,28,29\) and it has been reported to successfully induce angiogenesis and collateral growth,\(^12\) even in older patients.\(^20,39\)

Twelve patients at our institution with medically refractory atheroocclusive disease and perfusion failure demonstrated on preoperative imaging underwent indirect bypass via EDAS and/or bur holes in an attempt to promote angiogenesis and collateral blood flow development. Eight patients had MCA stenosis with or without ICA stenoocclusive disease. Three patients had hypoplastic circle of Willis portions with concomitant intracranial stenoocclusive disease and were deemed to be dependent on diminished ICA blood flow to maintain critical perforator-bearing segment patency. Lastly, 1 patient with serious medical comorbidities that precluded prolonged surgery received 3 bur holes bilaterally with dural and arachnoid incisions.

Despite documented initial graft patency, collateral growth failed to develop in our patients and the majority of individuals continued to have hypoperfusion with subsequent TIAs and/or cerebral infarction. Patients with atherosclerotic disease may have impaired angiogenesis as a result of reduced endothelial repair capacity. It is known, for example, that endothelial progenitor cells, characterized as (KDR+)/CD133+ cells,\(^4\) are reduced in patients with atherosclerotic risk factors and cardiovascular disease.\(^16\)

The rate of infarction among our patients within 1 year of surgery was high (42%) and the overall functional outcome was poor. The actual 1-year rate of infarction may have been as high as 50%, because 1 patient was lost to follow-up at 4 months. The rate of infarction we report does not appear to differ significantly from that in prior studies of conventional medical therapy for severe ICA stenoocclusive disease. In the Warfarin–Aspirin Symptomatic Intracranial Disease Study, the rates of ischemic stroke due to all causes in patients treated with aspirin were 15% in the first year and 20% at the end of the second year; patients who received warfarin therapy had rates of 15 and 18%, for the first and second years, respectively.\(^8\) In 20 studies of patients with TIAs and stroke in the presence of ICA occlusion, the overall annual risk of death and recurrent stroke was 5.5%, and in 11 studies with available information, the overall annual risk of ipsilateral stroke was 2.1%.\(^21\) Authors of studies in patients with impaired cerebral perfusion demonstrated a worse prognosis, with a 12.5% annual risk of stroke and a 9.5% risk of ipsilateral stroke.\(^21\) In their meta-analysis of 3 studies, Klijn et al.\(^21\) found that patients with symptomatic ICA occlusion and severely impaired hemodynamic measurements had even worse outcomes, with a 41.4% overall annual risk of stroke and 31% overall annual risk of ipsilateral stroke.

To compare outcomes in patients who received surgical versus medical treatment, we performed a meta-analysis of medically treated patients with symptomatic ICA occlusion and severe hemodynamic failure. There was no significant difference in the rates of infarction among our surgically treated patients and those reported in the literature who underwent medical treatment only: medically treated patients had an infarction rate of 30% in the first year versus 42% in the surgically treated patients in our study (Fig. 3).\(^14,20,45,47\) This finding should be interpreted cautiously, however, as it may be due to inadequate power, selection bias, or differences in patient characteristics across studies. Patients included in this report may have had more severe clinical symptoms, as 11 of 12 patients
had had infarctions prior to surgery. In previous studies the time to infarction was measured from the time of surgery in patients who received surgical intervention compared to the time from enrollment in the study for cases reported in the literature.

In patients with moyamoya disease it takes weeks to months to form collateral circulation after EDAS. In the present study, 5 patients had infarctions (3 ipsilateral and 2 contralateral to the surgical site) between 3 days and 5 months after the procedure. In the best case scenario it takes 6 months to form collateral vessels; therefore, all patients who had infarctions within 6 months of surgery are assumed to follow the natural disease course. However, in 2 of 8 patients, infarctions occurred after the first 6 months of follow-up. There was still no significant difference between patients with infarctions starting 6 months after EDAS and those in the medically treated group in the literature. Although this finding is subject to the previously reported limitations, we are unable to reject the null hypothesis.

Within the bounds of the limitations we have described, our findings do not provide evidence that indirect bypass is superior to medical therapy in patients with atheroocclusive disease and secondary hemodynamic failure. Although EDAS may stimulate the formation of collateral vessels over time, it appears that these patients require immediate augmentation of cerebral perfusion, as supported by their higher rate of early and repeated cerebrovascular accidents or TIs. In addition, CO2 reactivity may slowly improve after indirect revascularization, possibly accounting for the high rates of continued cerebral ischemia during the 1st postoperative year and the significantly lower complication rates thereafter. Although follow-up imaging failed to reveal collateral development, it is possible that these studies were performed too early. In contrast to the patients with moyamoya, in whom collateral vessel growth is demonstrated within a few weeks to months after EDAS, patients in our cohort exhibited lesser degrees of growth. Preoperative imaging did not reveal significant development of EC-IC collateral vessels in any of our patients. The development of these collateral vessels is a common finding in patients with moyamoya disease.

Appropriate patient selection for the operative management of atherosclerotic ICA stenoocclusive disease requires distinguishing hemodynamic failure from thromboembolic pathological entities. Although bypass surgery is intended to provide collateral circulation and compensate for hemodynamic insufficiency, this technique offers little benefit in cases of thromboembolic origin, and our results do not support the use of indirect bypass in this patient population. Direct bypass, however, may be indicated in carefully selected individuals. Our data also support the need for further studies into the possible benefits of endovascular revascularization, as well as the development of new medical therapies for patients with MCA stenosis.

**Conclusions**

Indirect bypass with EDAS and/or bur holes does not appear to be beneficial in patients with medically
Indirect bypass for symptomatic ICA occlusive disease

refractory symptomatic intracranial atheroocclusive disease and secondary hemodynamic failure. Patients who undergo this procedure remain at a high risk of cerebral ischemia and demonstrate poor collateral development in the first postoperative year.

Acknowledgments

The authors thank the Department of Nuclear Medicine and Radiology at Columbia University for their expertise in imaging, and Nancy Heim for her artistic assistance.

References

32. Rankin J: Cerebral vascular accidents in patients over the age

Please include this information when citing this paper: published online January 30, 2009; DOI: 10.3171/2008.9.JNS17658.
Address correspondence to: Ricardo J. Komotar, M.D., Department of Neurosurgery, Columbia University, 710 West 168th Street, Room 431, New York, New York 10032. email: rjk2103@columbia.edu.