Two-stage carotid saphenous vein interposition graft and superficial temporal artery bypass for acute carotid occlusion

Lukas Andereggen, MD,[a,b] **Robert H. Andres, MD,**[a] **Marcel Arnold, MD,**[c] **Andreas Raabe, MD,**[a] **Jürg Schmidli, MD,**[d] and **Michael Reinert, MD,**[a,e] *Bern and Lugano, Switzerland; and Boston, Mass*

The safety and efficacy of bypass surgery to achieve cerebral revascularization for cerebral hypoperfusion are controversial. However, bypass surgery still plays an important role for a select group of patients.
The indication to perform a high-flow or low-flow bypass in cases of acute symptomatic artery occlusion is not defined. Neurologic symptoms in acute symptomatic occlusion are usually blood pressure dependent, and acute blood flow restoration may be considered. This report reviews the case of a patient with an acute carotid occlusion in which a two-stage bypass technique was used to achieve revascularization and discusses the issues influencing the decision-making. (J Vasc Surg Cases 2015;1:161-4.)

The safety and efficacy of bypass surgery to achieve cerebral revascularization for chronic cerebral hypoperfusion are controversial. Several limitations with the key studies have been addressed and debated,[1,2] and cerebral revascularization continues to be implemented for different diseases.[3-5] The indication to perform a high-flow bypass (ie, commonly using either a radial artery or saphenous vein graft) or a low-flow bypass (ie, superficial temporal artery [STA] to middle cerebral artery [MCA]) in cases of acute symptomatic artery occlusion is not defined.

Neurologic symptoms in acute symptomatic occlusion are usually blood pressure dependent, and acute blood flow restoration may be considered in these cases. Cerebral hyperperfusion appears to depend on the assessment technique, with up to 19% of patients carrying the risk of cerebral hemorrhage as the worst complication.[6] This report reviews the case of a patient with an acute carotid occlusion and discusses the issues influencing the decision to perform an STA to MCA bypass or a high-flow anastomosis. The patient consented to publication of this manuscript.

From the Department of Neurosurgery[a] and Department of Neurology,[c] University Hospital Bern, Neurocenter, Inselspital, Bern; the Department of Neurosurgery and F.M. Kirby Neurobiology Center, Boston Children's Hospital, Harvard Medical School, Boston[b]; the Department of Cardiovascular Surgery, Swiss Cardiovascular Center, Inselspital, Bern[d]; and the Department of Neurosurgery, Neurocentro Lugano, Lugano.[e]

Author conflict of interest: none.

Reprint requests: Michael Reinert, MD, Department of Neurosurgery, Ente Ospedaliero Cantonale, Lugano Neurocentro, Via Tesserete 26, 6930 Lugano, Switzerland (e-mail: Michael.Reinert@eoc.ch).

The editors and reviewers of this article have no relevant financial relationships to disclose per the Journal policy that requires reviewers to decline review of any manuscript for which they may have a conflict of interest.
2352-667X

http://dx.doi.org/10.1016/j.jvsc.2015.03.013

CASE REPORT

This 71-year-old man was admitted to Bern University Hospital, Switzerland, with a history of brief transient ischemic attacks in the form of vertigo and sensorimotor hemisyndrome on the right side, sometimes accompanied by motoric aphasia. Recent episodes lasted for only 15 minutes after standing up and walking. Magnetic resonance imaging showed multiple infarction areas in the left hemisphere. Digital subtraction angiography depicted occlusion of the common carotid artery (CCA) on the left side, and delayed filling of the left MCA and left anterior cerebral artery (ACA) was observed (Fig 1, *A*). There were marked pial collaterals from the posterior territory to the left MCA territory (Fig 1, *B*). The stump of the STA was retrogradely filled through scalp collaterals from the right occipital artery to the left posterior auricular artery (Fig 1, *C*). Right-sided extracranial and intracranial vessels were patent, with no occlusion or stenosis and normal flow on the magnetic resonance angiogram (Fig 1, *D*).

The patient's history revealed peripheral arterial occlusive disease, arterial hypertension, dyslipidemia, and nicotine abuse of 80 pack-years. The patient was under statin and antiplatelet treatment. The antihypertensive therapy was withdrawn to improve cerebral perfusion. Antiplatelet therapy was switched from acetylsalicylic acid (Aspirin Cardio, 100 mg; Bayer HealthCare, Whippany, NJ) to clopidogrel (Plavix, 75 mg; Sanofi-Aventis, Bridgewater, NJ).

Because of persistence of the clinical symptoms after slight mobilization and delayed perfusion of the MCA and ACA territories on magnetic resonance imaging (perfusion-weighted images), a revascularization procedure was indicated. After interdisciplinary discussion, it was decided to perform a two-stage revascularization procedure with an option to restore the flow in the CCA, internal carotid artery (ICA), and external carotid artery (ECA) in the first stage by performing an endarterectomy or to bypass the occluded carotid segment with a saphenous vein graft from the CCA to the STA. In a second stage, intracerebral revascularization was then planned using an extracranial-intracranial (ECA-ICA) STA to MCA bypass.

First operation. The left CCA, ICA, and ECA were exposed. The ICA was opened to check for the presence of backflow and to

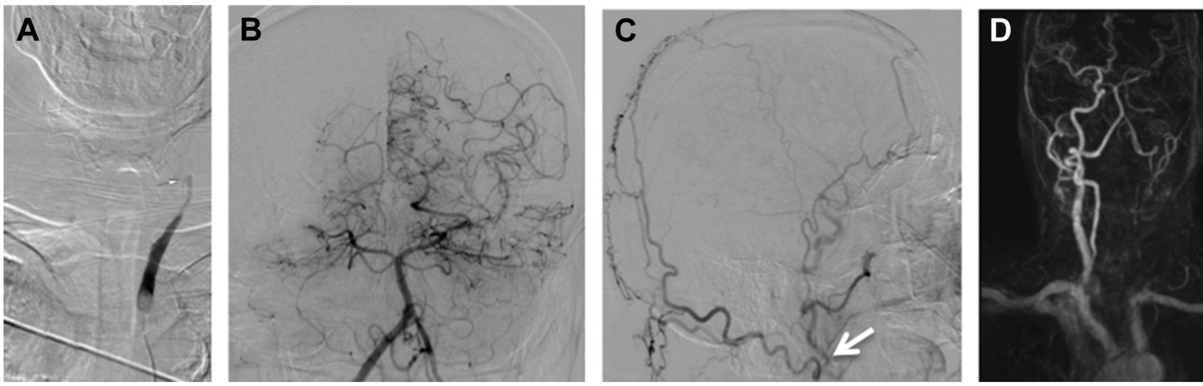

Fig 1. Angiographic findings of acute common carotid artery (CCA) occlusion and collateralization. **A,** Occluded CCA on the left side at the level of carotid bifurcation with stasis in the vessel stump (*arrow*). **B,** Marked pial collaterals from the posterior territory to the left middle cerebral artery (MCA) territory on the left side. **C,** Substantial external-external collaterals mainly from the occipital artery of the right side to the stump of the superficial temporal artery (STA; *arrow*). **D,** Right-sided extracranial and intracranial vessels were patent, showing normal flow on the magnetic resonance angiogram.

investigate whether an endovascular revascularization procedure would be possible. After no backflow, a Fogarty balloon catheter was introduced into the ICA, advanced to the carotid siphon, inflated, and gently retracted. A backflow was established; however, the flow decreased significantly over time, and a revascularization procedure of the extracranial ICA was abandoned to avoid thromboembolic events. The CCA was then incised from distal to proximal and endarterectomized in this segment and remained patent. Meanwhile, the saphenous vein was prepared and harvested from the left side and then attached in an end-to-side fashion to the proximal stump of the CCA and with an end-to-side outflow anastomosis to the STA. The distance from the patent CCA and a suitable MCA branch was slightly less than 30 cm. Postoperatively, no neurologic deficits were recorded; however, our stroke protocol restricts mobilization within the first 3 days.

Second operation. Three days later, the patient underwent a standard ECA-ICA STA to MCA bypass. After navigated frontotemporal craniotomy, the end-to-side bypass of the STA on the MCA was performed. Intraoperative flow was 50 mL/min (Flowprobe; Transonic Inc, Ithaca, NY).

Postoperative computed tomography angiography demonstrated a functional interposition bypass (Fig 2, *A*), and angiography showed the open bypass at the STA segment and the intracranial MCA (Fig 2, *B*). Perfusion maps (using perfusion-weighted imaging) showed normalized perfusion in the MCA and ACA territories compared with the preoperative image (Fig 3). The patient was administered aspirin 300 mg/day postoperatively, and recovery was uneventful. Clinical and radiologic follow-up 1 year later revealed no new neurologic deficits or cerebral infarcts.

DISCUSSION

Two points of interest merit discussion in the present case: (1) performance of the revascularization procedure in two stages with prior CCA-interposition vein graft-STA-MCA bypass to augment flow for the STA; and (2) the question of whether to perform a high-flow or low-flow bypass.

Staged bypass. Literature regarding staged bypass approaches for flow augmentation is limited. In general, cerebral bypass operations are performed in a single session, given the urgency of the indication,[7] or even in cases of bilateral progressive arteriopathies.[8]

A two-stage bypass with a saphenous vein graft interposition was performed on the basis of the following considerations:

1. Both the ICA and ECA were occluded from the carotid bifurcation. Furthermore, the STA was perfused from the contralateral ECA through an anastomosis to the occipital artery and flow was modest, so hemodynamic security was addressed. We thus aimed to first increase the flow of the bypass rather than performing a direct STA-MCA bypass with the risk of bypass obliteration due to thrombus formation in case of low flow.
2. Bypass length required to cover the distance from the CCA to the intracranial anastomosis site was approximately 30 cm; it could not be achieved with a compatible vessel size in this patient. Radial artery graft has the advantage of lower graft flow compromise arising from scalp compression at the side of the anastomosis, yet both arterial and vein bypasses have shown good long-term patency.[9]

Intraoperative endarterectomy of the CCA in addition to the anastomosis was performed. This is a common occurrence in vascular surgery,[10] although not in the context of cerebral bypass.[11]

Because each suture and vascular irregularity represents a potential source for thromboembolic complications,[12,13] staging the bypass so that the patient can tolerate cerebral

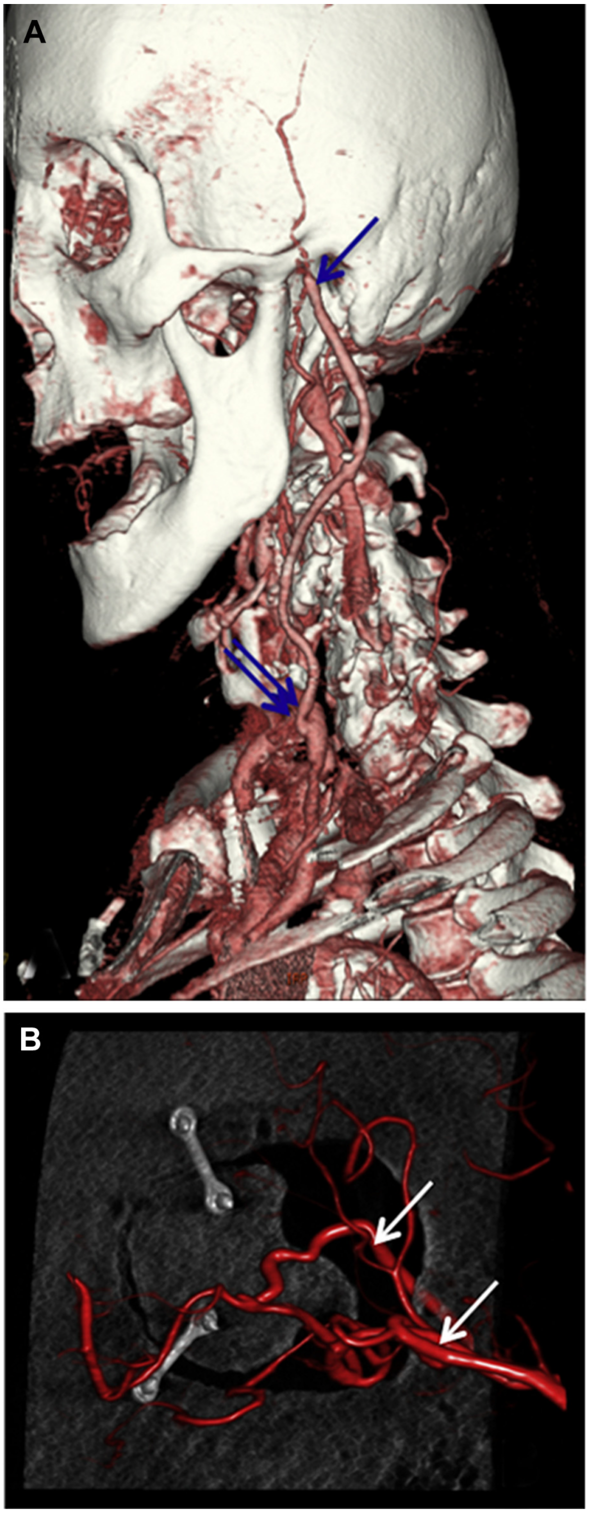

Fig 2. Postoperative images after first and second bypass operation. **A,** Postoperative computed tomography angiography shows open venous bypass anastomosis of the common carotid artery (CCA) on the left side (*double arrow*) to the superficial temporal artery (STA; *arrow*) on the ipsilateral side. **B,** Three-dimensional angiography depicts the anastomosis of the intracranial part bypass.

hypoperfusion in the supine position and under hypertensive therapy might reduce perioperative cerebral ischemic events. Furthermore, a combination of competing blood flows from both the collateral circulation and the STA and impaired cerebral autoregulation may lead to episodic focal decreases in regional cerebral blood flow,[3,14] and staging the bypass might therefore be beneficial. However, performing both operations under a single anesthesia procedure, assuming that no intraoperative complications or other technical considerations arise, might reduce alterations in the perioperative blood pressure and subsequently cerebral flow in a patient in whom cerebral perfusion depends on maintaining relative hypertension.[5,8]

Cerebral hyperperfusion. A well-functioning STA-MCA bypass has the potential to take over a large amount of the territory supplied by the MCA, which can be accomplished a few days after anastomosis.[15] For intracranial bypass, the STA is dissected from surrounding tissue and the end cut, resulting in higher flow measurements in the absence of resistance (free cut flow). It has been shown that intraoperative cut flow index (postbypass flow/free cut flow) measurements and postanastomosis flow assessments are highly predictive for the success rate of the bypass.[16,17]

The risk of cerebral hemorrhage due to hyperperfusion syndrome after standard extracranial-intracranial bypass reported in the literature varies enormously.[18-21] High-flow revascularization using either a radial or vein graft has been associated with a total risk of hemorrhage of 16.6%; flow values in patients suffering this complication were, however, not significantly higher compared with nonhemorrhagic cases, and values were similar to those in the present case.[20]

Limitations. This is a single case using a staged vein interposition graft to perform an STA-MCA bypass on the basis of symptomatic carotid occlusion. Superiority over a direct bypass procedure with regard to the long-term risk of stroke can therefore not be evaluated.

CONCLUSIONS

The two-stage bypass technique to assess revascularization in carotid occlusive disease was feasible and seemed to be successful with regard to flow augmentation in postoperative perfusion maps and clinical outcome.

REFERENCES

1. Sundt TM Jr. Was the international randomized trial of extracranial-intracranial arterial bypass representative of the population at risk? N Engl J Med 1987;316:814-6.
2. Goldring S, Zervas N, Langfitt T. The Extracranial-Intracranial Bypass Study. A report of the committee appointed by the American Association of Neurological Surgeons to examine the study. N Engl J Med 1987;316:817-20.
3. Lee M, Guzman R, Bell-Stephens T, Steinberg GK. Intraoperative blood flow analysis of direct revascularization procedures in patients with moyamoya disease. J Cereb Blood Flow Metab 2011;31:262-74.
4. Guzman R, Lee M, Achrol A, Bell-Stephens T, Kelly M, Do HM, et al. Clinical outcome after 450 revascularization procedures for moyamoya disease. Clinical article. J Neurosurg 2009;111:927-35.

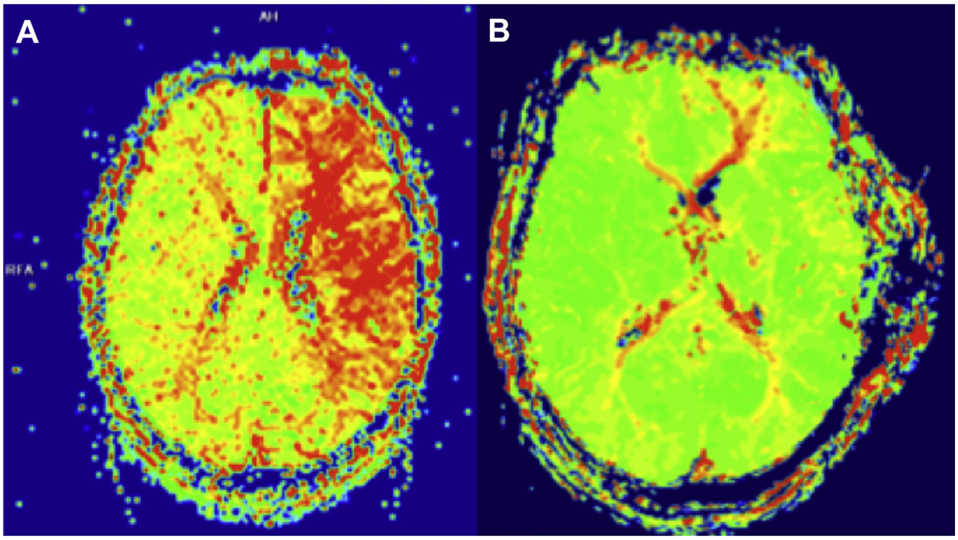

Fig 3. Perioperative perfusion-weighted imaging maps. Preoperative perfusion-weighted images show delayed perfusion in the middle cerebral artery (MCA) and anterior cerebral artery (ACA) territory on the left side **(A)** compared with the postoperative perfusion-weighted imaging sequences **(B)**.

5. Lin N, Aronson JP, Manjila S, Smith ER, Scott RM. Treatment of moyamoya disease in the adult population with pial synangiosis. J Neurosurg 2014;120:612-7.
6. Yamaguchi K, Kawamata T, Kawashima A, Hori T, Okada Y. Incidence and predictive factors of cerebral hyperperfusion after extracranial-intracranial bypass for occlusive cerebrovascular diseases. Neurosurgery 2010;67:1548-54.
7. Kalani MY, Zabramski JM, Hu YC, Spetzler RF. Extracranial-intracranial bypass and vessel occlusion for the treatment of unclippable giant middle cerebral artery aneurysms. Neurosurgery 2013;72:428-36.
8. Scott RM, Smith ER. Moyamoya disease and moyamoya syndrome. N Engl J Med 2009;360:1226-37.
9. Sia SF, Davidson AS, Assaad NN, Stoodley M, Morgan MK. Comparative patency between intracranial arterial pedicle and vein bypass surgery. Neurosurgery 2011;69:308-14.
10. Bennett KM, Scarborough JE, Shortell CK. Predictors of 30-day postoperative stroke or death after carotid endarterectomy using the 2012 carotid endarterectomy-targeted American College of Surgeons National Surgical Quality Improvement Program database. J Vasc Surg 2015;61:103-11.
11. Cutlip DE, Pinto DS. Extracranial carotid disease revascularization. Circulation 2012;126:2636-44.
12. Powers WJ, Clarke WR, Grubb RL Jr, Videen TO, Adams HP Jr, Derdeyn CP. Lower stroke risk with lower blood pressure in hemodynamic cerebral ischemia. Neurology 2014;82:1027-32.
13. Reynolds MR, Derdeyn CP, Grubb RL Jr, Powers WJ, Zipfel GJ. Extracranial-intracranial bypass for ischemic cerebrovascular disease: what have we learned from the Carotid Occlusion Surgery Study? Neurosurg Focus 2014;36:E9.
14. Mukerji N, Cook DJ, Steinberg GK. Is local hypoperfusion the reason for transient neurological deficits after STA-MCA bypass for moyamoya disease? J Neurosurg 2015;122:90-4.
15. Neff KW, Horn P, Dinter D, Vajkoczy P, Schmiedek P, Duber C. Extracranial-intracranial arterial bypass surgery improves total brain blood supply in selected symptomatic patients with unilateral internal carotid artery occlusion and insufficient collateralization. Neuroradiology 2004;46:730-7.
16. Amin-Hanjani S, Du X, Mlinarevich N, Meglio G, Zhao M, Charbel FT. The cut flow index: an intraoperative predictor of the success of extracranial-intracranial bypass for occlusive cerebrovascular disease. Neurosurgery 2005;56(Suppl):75-85.
17. Morton RP, Moore AE, Barber J, Tariq F, Hare K, Ghodke B, et al. Monitoring flow in extracranial-intracranial bypass grafts using duplex ultrasonography: a single-center experience in 80 grafts over 8 years. Neurosurgery 2014;74:62-70.
18. Grubb RL Jr, Powers WJ, Clarke WR, Videen TO, Adams HP Jr, Derdeyn CP. Surgical results of the Carotid Occlusion Surgery Study. J Neurosurg 2013;118:25-33.
19. Stiver SI, Ogilvy CS. Acute hyperperfusion syndrome complicating EC-IC bypass. J Neurol Neurosurg Psychiatry 2002;73:88-9.
20. van Doormaal TP, Klijn CJ, van Doormaal PT, Kappelle LJ, Regli L, Tulleken CA, et al. High-flow extracranial-to-intracranial excimer laser-assisted nonocclusive anastomosis bypass for symptomatic carotid artery occlusion. Neurosurgery 2011;68:1687-94.
21. Powers WJ, Clarke WR, Grubb RL Jr, Videen TO, Adams HP Jr, Derdeyn CP. Extracranial-intracranial bypass surgery for stroke prevention in hemodynamic cerebral ischemia: the Carotid Occlusion Surgery Study randomized trial. JAMA 2011;306:1983-92.

Submitted Jan 12, 2015; accepted Mar 17, 2015.