Contralateral carotid artery occlusion is not a contraindication to carotid endarterectomy even if shunts are not routinely used

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Objective: **Although controversial, carotid artery stenting (CAS) has been proposed as being safer than carotid endarterectomy (CEA) for patients with a contralateral internal carotid occlusion (CCO). Arguably, with a CCO, CAS should be even safer than CEA if a shunt is not used. Accordingly, we reviewed our experience with 2183 CEAs performed routinely without a shunt to evaluate the risk of CEA performed in a subset of 147 patients with a CCO.**
Methods: **Between 1988 and 2011, 147 CEAs (111 men [75%], 36 women [25%]) were routinely performed without a shunt despite CCO. Of these patients, 76% were asymptomatic. CEAs were performed by seven surgeons using standard techniques (not eversion), with patients under general anesthesia and blood pressure maintained at >130 mm Hg. All patients received heparin (7500 U), and protamine reversal was routine. Median cross-clamp time was 20 minutes (range, 14-40 minutes).**
Results: **Three neurologic events occurred ≤30 days (2.0%). One transient ischemic attack (TIA) occurred immediately, and one occurred on the first postoperative day due to occlusion of the endarterectomy site. One patient sustained an immediate stroke and died of a large computed tomography-documented atheroembolic shower.**
Conclusions: **Our data demonstrate the safety of CEA in the presence of a CCO, even when performed without a shunt. It is unlikely that the stroke or delayed TIA could be attributed to nonshunting or CCO. Even if so, the stroke and death rates would be lower than those previously reported for patients undergoing CEA in the presence of a CCO. This may be due to short cross-clamp times, careful technique, general anesthesia, and blood pressure support. Given these low adverse event rates, our experience refutes the assumption that patients with a CCO are at such a high risk for CEA that the only alternative is CAS. (J Vasc Surg 2013;58:935-40.)**

The optimal intervention for severe ipsilateral carotid stenosis with a contralateral carotid occlusion (CCO) remains controversial. Revascularization of patients in the presence of a CCO has been considered by some to be high risk and associated with stroke rates as high as 5% to 10%.[1,2] Such high stroke risks for carotid endarterectomy (CEA) in the presence of CCO have been used to justify carotid artery stenting (CAS) as an alternative to CEA.[3] This bias against CEA is especially prevalent in the cardiology literature.[4] Others, however, have suggested that the CEA risk in this setting is exaggerated and that CCO may actually afford stroke protection during CEA.[5]

Adding to the confusion about the safety of CEA in the presence of CCO is the controversy about whether shunts should be used during CEA. Accordingly, we reviewed our experience with 2183 CEAs performed routinely without a shunt to evaluate the risk of CEA performed in a subset of 147 patients with a CCO. Unique to this experience is that our group routinely performs CEA on all-comers without a shunt, even in the presence of a CCO. As such, it is conceivable that the results of our nonshunt technique would provide a strong argument for the risks or safety of CEA in the presence of a CCO.

METHODS

Design. This is a nonrandomized but prospective experience that includes 147 consecutive CEAs routinely performed without a shunt despite CCO. The study period was from May 1988 through November 2011. During the same interval, 2036 CEAs were routinely performed without a shunt but with a patent, although usually stenosed, contralateral internal carotid artery. Demographic and surgical data were obtained on all patients at the time of surgery and maintained in an AtriumMed electronic database (Atrium Medical Corp, Hudson, NH).

CEAs were performed by seven surgeons with various experience ranging from junior partner to high-volume operator. The procedures were performed at two local hospitals. Indications for surgery were consistent with the Society for Vascular Surgery (SVS) Updated Guidelines for the Management of Extracranial Carotid Disease.[6]

Determination of the degree of ipsilateral stenosis and confirmation of CCO was obtained by duplex ultrasound

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imaging on all patients and angiography, computed tomography (CT) scan, or magnetic resonance angiography (MRA) selectively, using the North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria.[7,8] Our duplex criteria for determining the degree of ipsilateral stenosis have been previously reported.[9] Evaluation of the completeness of the circle of Willis was not performed because this would have required preoperative angiography, MRA, high-resolution CT angiography, or transcranial Doppler imaging in all patients. We believe that the information gained would not have warranted the increased expense or risk, especially if an invasive diagnostic technique was used. Further, we are not convinced that this information would change our procedure.

The database and medical record review were used to determine the development of any new neurologic deficit or death ≤30 days of surgery. A new deficit was defined as a change in motor, sensory, or cognitive performance. A neurologist evaluated all patients who sustained a new neurologic event, and relevant treatment was initiated. Permanent craniofacial nerve injury, infection, and return to the operating room for bleeding were also evaluated. Myocardial infarction (MI), clinically evident or based on enzyme results, was not studied for the purpose of this report.

Surgical technique. Although CEA was performed by seven surgeons and multiple anesthesiologists and nurse anesthetists, an essentially uniform technique for CEA was used throughout the 23-year period of this study. Aspirin therapy was continued before CEA, but clopidogrel was discontinued for at least 5 days before surgery unless emergent surgery was required. All patients underwent general anesthesia using a variety of anesthetic agents. Halothane was used in the early procedures; sevoflurane and desflurane have been used during the last 15 years. Induction agents have included pentobarbital or propofol. Normocapnia was preserved.

Systolic blood pressure was maintained at >130 mm Hg during cross-clamp, using pharmacotherapy (phenylephrine) as necessary. We have no evidence to support any specific blood pressure, but we assumed that the patient should be at least normotensive and possibly mildly hypertensive. However, by requesting that the blood pressure always remain at >130 mm Hg, we have avoided periods of hypotension, which may, or may not, be significant. We do not adjust the blood pressure to higher levels for patients who come to the operating room hypertensive but rather allow them to remain at that level unless they have pressures >200 mm Hg, in which case it is lowered pharmacologically with medications selected by the anesthesiologist.

A standardized dose of heparin (7500 U) was used in all patients, regardless of weight. No method to determine cerebral perfusion was used. Control of the internal carotid artery (ICA) was achieved using a Kartchner clamp to maximize distal exposure. Other vessels were occluded using vessel loops or DeBakey clamps, if necessary. A long arteriotomy was standard procedure to fully visualize the end point in the ICA. Accordingly, tacking sutures were seldom required.

Our technique includes meticulous removal of debris from the vessel wall, back bleeding of the ICA, and antegrade perfusion of the external carotid artery upon completion of the arteriotomy closure to avoid ICA embolization. The only change in technique occurred after 2001. Before 2001, Dacron patches (DuPont, Wilmington, Del) were only used when the ICA measured <3 mm or with redo procedures. Since 2001, patches have been used routinely.

Full protamine reversal was used in all patients. Postoperative blood tests to evaluate for myocardial ischemia were not routinely performed.

Statistical methods. Statistical analysis was performed using SPSS 20 software (SPSS Inc, Chicago, Ill). Categoric data are expressed as numbers and percentages and continuous variables as mean with standard deviation. Possible significant differences were analyzed between the group means with the χ^2 test and Fisher exact test. A multiple analysis of variance was used to see the main and interaction effects of categoric variables on multiple dependent interval variables. $P < .05$ was defined as statistically significant. However, because of the small number of events (see below), no statistically significant variables could be determined.

RESULTS

A total of 147 procedures were performed on 111 men (75%) and 36 women (25%). Patients were a mean age of 72 years (range, 46-90 years); 29 patients (20%) were aged >80 years. CEA was performed for asymptomatic critical stenosis in 112 patients (76%) and for symptomatic stenosis in 35 (24%). Preoperative symptoms included transient ischemic attack (TIA) or amaurosis fugax in 22 patients (15%) and cerebrovascular accident (CVA) in 13 (9%). Median ipsilateral stenosis was 90% (range, 45%-99%). The patient who underwent a CEA with a 45% stenosis was symptomatic from a de novo carotid aneurysm. Abnormal unilateral vertebral artery flow was documented in 11 patients: retrograde in five (3.4%) and absent in six (4.1%).

Risk factors were recorded prospectively. However, data suitable for SVS classification were available for only 91 patients. Of those, 16 (18%) were diabetic, 77 (85%) were hypertensive, 78 (86%) were diagnosed with hyperlipidemia, 61 (67%) had a history of smoking, 66 (73%) were taking antiplatelets, and 63 (69%) were taking statins. All patients were prescribed antiplatelets and statins postoperatively, unless contraindicated. Coronary artery disease was present in 48 (53%) and chronic kidney disease in 12 (13%).

All procedures were performed in one of two local hospitals, with 115 (76%) operations in one hospital and 32 (24%) in the other. Surgeons who did >20 CEAs yearly performed 128 procedures (87%), and five surgeons with less experience performed 19 procedures (13%).

General anesthesia was used routinely, and shunts were never used. A patch was used in 82 procedures (66%), of

which 5 (3%) were redo procedures. Cross-clamp times (CCTs) averaged 20.4 minutes (standard deviation, 5.4 minutes; range, 14-40 minutes). CCT was significantly longer for less-experienced surgeons ($P < .001$) and when patches were used ($P < .005$), but there were no associated neurologic events. Additionally, because of the infrequent occurrence of any morbidity or prolonged CCTs, the effect of longer CCTs cannot be determined.

There were three neurologic events, no MI, and one death. One patient sustained bihemispheric embolic infarcts and died of the profound cerebral injury. As previously reported,[9] we presume this was related to proximal intraplaque hemorrhage and debris that showered after removal of the proximal clamp, which had inadvertently been applied in an area of severe plaque. The CT scan performed immediately after the event demonstrated multiple embolic infarcts throughout both cerebral cortices consistent with an embolic shower and not watershed ischemia that might have arisen from nonshunting anoxia.

The second patient who sustained an event awoke with expressive aphasia that resolved after 18 hours. The endarterectomy site remained patent by duplex imaging, and early CT revealed no evidence of infarct. The CCT was 15 minutes. Conceivably, an MRI could have shown an infarct; however, by strict definition, a cerebral symptom that resolves ≤24 hours in the presence of a negative CT scan is a TIA and not a stroke.

A TIA occurred in the third patient on postoperative day 1, manifested as slurred speech and a slight facial droop that resolved in 20 minutes. The duplex scan was equivocal, so an arch aortogram and cerebral angiogram were performed. This demonstrated bilateral ICA occlusions. On the nonpatched endarterectomy side, there was an abrupt occlusion of the proximal ICA 1 cm from its origin, without reconstitution of the ICA. The vertebral and basilar arteries were widely patent. Despite bilateral ICA occlusion, the patient remained asymptomatic, and no further intervention was performed. A CT scan was not performed, and it is conceivable that it might have shown an infarct. Thus, although clinically this event was a TIA, one could suggest that it could have been classified as a "minor" stroke if such a CT finding were present.

Accordingly, in the best-case scenario, the adverse events were two TIAs (1.4%), one CVA (0.7%), and one death (0.7%). However, if the last patient did indeed sustain a CVA, the results would be one TIA (0.7%) and two CVAs (1.4%). Regardless, the total central neurologic event rate was 2.0%. Three cranial nerve injuries (2.0%) were documented at 30 days: two injuries to the marginal mandibular branch of the facial and one temporary hypoglossal. No patient required a return to the operating room for bleeding or infection during the 30-day postoperative period.

DISCUSSION

Although controversial, some have suggested that CEA in the presence of a CCO is associated with an increase in adverse neurologic outcomes.[10,11] Accordingly, supporters of CAS have proposed that it is a safer alternative than CEA, ostensibly because procedural cerebral ischemic time would be shorter.[4,12,13] However, there are limited data supporting the role of CAS in the presence of CCO. The Stenting with Angioplasty and Protection in Patients at High Risk for Endarterectomy (SAPPHIRE) study was designed to evaluate the efficacy of CAS in a high-risk patient population. Patients required only one high-risk variable for inclusion, one of which was CCO. Of those, only 36 of the 167 patients (23.6%) in the CAS and 42 of the 167 patients (25.3%) in the CEA groups had a CCO. The investigators reported noninferiority, but the incidence of primary end points was exceptionally high for the CAS and CEA groups, at 12.2% vs 21.1%, respectively.[14] Further, adverse event rates for the subset of patients with CCO were not reported.

The ACCULINK for Revascularization of Carotids in High-Risk Patients (ARCHeR) trial also addressed the high-risk patient.[15] CCO was present in 96 of the 581 patients (16.5%) enrolled. To qualify for inclusion, patients had to have at least one other risk factor, including recent coronary artery bypass grafting, two or more diseased coronary arteries, unstable angina, or MI ≤30 days and in need of revascularization. The 30-day stroke and death rate was 6.8% for the total population and was 11.6% for symptomatic patients and 5.4% for asymptomatic patients, which remains well above the acceptable standards for revascularization.[15] Again, however, event rates for patients with a CCO were not reported.

The Carotid RX Acculink/Accunet Post-approval Trial to Uncover Unanticipated or Rare Events (CAPTURE) registry included 288 patients (8.2%) with CCO of the 3500 enrolled.[16] It also had a high primatry end point of 6.3% and has been criticized because it was not a randomized controlled trial. CCO was not independently associated with stroke in that study,[16] suggesting that CAS with CCO also had high event rates. The 6.3% that was reported remains above the stroke rate reported in our study population.

Another high-risk CAS registry was the Boston Scientific EPI: A Carotid Stenting Trial for High-risk Surgical Patients (BEACH) trial.[17] The primary end point was composite morbidity and mortality. Of the 480 patients enrolled, 87 (18%) met inclusion criteria based on CCO. The composite 1-year morbidity and mortality rates were 8.9%. The event rate in the setting of a CCO was 1.1% (n = 5).

In a recent evaluation of real-world data from the Carotid Artery Revascularization and Endarterectomy (CARE) registry, the primary composite end point of death, nonfatal MI, and nonfatal stroke from CAS was 2.1% in patients with CCO and 2.6% in patients without CCO.[4] This would suggest that CAS in the presence of CCO is noninferior to CAS in patients without a CCO. The authors admitted that they did not directly compare CEA with CAS in patients with CCO, yet they stated that, "CEA is known to be associated with increased risk in the presence of CCO."[4]

However, our data and many recent studies challenge the assumption that CEA is unsafe in the presence of a CCO. A literature review in 2004 by Rockman[18] identified 338 CEAs with CCO and found the presence of a CCO did not significantly increase the risk of perioperative stroke and late outcomes. Dalainas et al[13] studied a similar number of CEAs with CCO and also found no significant difference in perioperative cardiac or neurologic events. The da Silva et al[1] evaluation of 700 patients undergoing CEA, of which 108 (15.4%) had a CCO, found no significant difference in stroke or death rates. Flanigan et al[19] investigated 207 SAPPHIRE-eligible high-risk patients vs 235 normal-risk patients and found an overall postoperative stroke rate of 1.4%, with no statistical differences between groups. Mozes et al[20] evaluated 323 SAPPHIRE eligible high-risk patients, of which 66 patients had CCO and also found no differences in stroke or death rate.

In our series, the adverse neurologic event rate for CEA in the setting of a CCO was 2.0% (n = 3). We believe only one neurologic event (0.7%) was directly related to the CCO. That patient awoke with expressive aphasia that resolved after 18 hours. The endarterectomy site remained patent by duplex imaging, and early CT revealed no evidence of infarct. Of interest, this was the only patient with an adverse outcome who had a preoperative TIA. Even if this patient were found to have suffered a stroke by an MRI (which was not performed), this event would have been classified at worst as a minor stroke and would not have affected the overall neurologic complication rate, which we have defined by combining TIA and stroke.

The CCO was not responsible for the patient who died of a stroke. That neurologic complication was related to atherosclerotic debris that embolized on declamping. The only other event, a TIA on postoperative day 1, was also not related to performing the CEA in the presence of a CCO. Rather, it was due to occlusion of the endarterectomy site. Regardless of whether these other neurologic events were, or were not, related to CCO, the overall stroke and death rate of 2.0% is in keeping with previous reports and is well within the recommended standards of a 3% perioperative stroke and death rate.[6,21,22]

To further highlight the safety of CEA in the setting of CCO, consider that our experience included routine nonshunting in all patients. Many find this technique is counterintuitive for the preservation of cerebral perfusion, especially in patients with a CCO. As such, the necessity for shunting continues to polarize the vascular community. Proponents for shunting argue that it is a safe and reliable method for providing cerebral protection[23,24] while allowing an unhurried approach.[15,16] However, shunts are not necessarily benign and have been associated with dislodgement of embolic material, air embolization, creation of distal flaps, and shunt occlusion. Outcomes with a shunt are also operator-dependent. Surgeons who routinely place shunts have better results than those who place shunts selectively.[25] Further, there are no reliable indicators to select patients who should receive a shunt.[26] Multiple studies, including a Cochrane Review, have found no effect of shunting vs nonshunting on stroke or death after CEA with CCO.[5,17,18,26,27] Some have hypothesized that with most CCOs, the cerebral circulation develops adequate collateral circulation to maintain perfusion to the brain.[5]

Despite these studies, it has been suggested that low adverse event rates with routine nonshunting can only be achieved by experienced surgeons who can perform an expeditious CEA. After 2001, we used patches exclusively, yet despite an increase in CCTs, no patient sustained a new event. This is in keeping with the Rockman et al[28] finding that patching was associated with a significant reduction in perioperative stroke compared with primary closure. The three adverse events in our patients occurred with primary closures.

Further, studies have found no correlation between CCTs and adverse neurologic complications, even with average CCTs >45 minutes.[29,30] In 1984, Littooy et al[31] reported significantly increased neurologic events only if CCTs >60 minutes. Fortunately, perioperative management and surgical techniques have improved considerably since their report. Our median CCT was 20 minutes (range, 14-40 minutes). Despite a significant difference between the surgeons' CCTs, no correlation was found between CCTs and neurologic events ($P = .40$). Further, our five less-experienced surgeons performed 13% of the CEAs with CCO, with no adverse neurologic events despite CCTs that averaged 26 minutes. Accordingly, we believe our results are reproducible, provided the surgeon has at least had formal training and experience with CEA.

Our results have evoked strong emotional reactions regarding nonshunting, with some alluding to "pure luck" that our results are good. On the basis of their experience with CEA under local anesthesia, some surgeons may agree that shunts might not always be necessary but find it impossible to believe that shunts are not required with a CCO. However, we routinely use general anesthesia with pharmacologic blood pressure support. Further, although for the purpose of this article we have chosen to report only our experience with CEA in the presence of CCO, our group has routinely not shunted 2036 "all-comers" with patent contralateral ICAs, with a current stroke rate of 1.18%.

Some limitations of our study warrant consideration. All data were nonrandomized observational data collected prospectively but analyzed retrospectively. Although demographic and risk factor data were collected prospectively, some information was missing. It could be argued that our patient sample does not represent the general population that requires CEA. However, the patients in this series are similar to the CCO sample reported in NASCET.[10] Most were men aged >65 years, with severe ipsilateral stenosis. Most had a history of tobacco use and were hypertensive. Of interest, the current sample had nearly double the rates of coronary artery disease and hyperlipidemia, which likely reflects the historical timing of the studies and evolving risk factor modification and diagnostics for coronary disease. Further, the patients

with CCO mirrored our overall CEA patient population. More important, because our combined neurologic event and death rate was so small, it is extremely unlikely that the missing risk factors could have played any role in the outcome and validity of our conclusions.

Another limitation could be that preoperative and postoperative neurologic examinations by neurologists were only requested postoperatively when the operating surgeon noted a new event. Our protocol is to evaluate the patient at discharge and at the 1-week, 1-month, and 6-month visits. Postoperative duplex evaluation of the operated-on carotid artery is also performed at 1 month and 6 months and then every 6 months or once yearly, depending on the ultrasound findings. It is possible that subtle neurologic events might not have been recorded. However, our group took great pains to evaluate our patients for even minor defects because of our interest in our nonshunting technique and the awareness that this technique could evoke controversy. For example, we classified one patient who did not have a CCO as having sustained a stroke when her carotid artery occluded after surgery. Her only neurologic finding was persistent micrographia and an inability to accurately place letters in the down column of a crossword puzzle.

Our study also does not answer the question of whether CEA can be performed safely under local anesthesia with CCO. Several large randomized clinical trials and the General Anesthetic vs Local Anesthetic for carotid surgery (GALA) trial did not find significantly different event rates between general and local anesthesia.[2,32] Our technique involved a general anesthetic for all patients. We do believe that the ultimate choice of anesthetic agent should be at the discretion of the surgeon, but we think that general anesthesia may confer advantages in patients undergoing CEA with CCO. For example, blood pressure can be easily controlled pharmacologically and oxygenation well maintained throughout. Regardless, because none of our procedures was performed under local anesthesia, we cannot make any determinations about CEA without a shunt using anything other than general anesthesia.

In addition, we could find no evidence that CCT influenced outcome, so we cannot assume that very prolonged CCTs are safe. Nevertheless, in our overall experience with CEA, we have had a few CCTs that were >40 minutes, without neurologic deficits.

We also have not evaluated the effect of our procedure on postoperative MI, because enzymatic tests for MI were not routinely performed. However, recent evaluation of the Carotid Revascularization Endarterectomy Versus Stenting Trial (CREST) data suggest that although enzymatic-defined MI can result in decreased long-term survival, the increased risk of stroke with CAS also results in increased long-term mortality.[33]

CONCLUSIONS

We have presented the largest series of CEA with routine nonshunting in the setting of CCO. It is unique in that we never shunt, as opposed to other studies where nonshunting was based on predetermined criteria. Our data suggest that the presence of a CCO does not place the patient at increased risk for post-CEA neurologic complications, provided that the procedure is performed under general anesthesia. More importantly, our experience refutes the assumption that patients with CCO are at such a high-risk for CEA that the only alternative is CAS.

AUTHOR CONTRIBUTIONS

Conception and design: RS
Analysis and interpretation: JC, RS, DS, DN, ML
Data collection: JC
Writing the article: RS, JC,
Critical revision of the article: RS, JC, DS, ML, DN
Final approval of the article: RS, JC
Statistical analysis: JC
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RS and JC contributed equally to this article and share co-first authorship.

REFERENCES

1. da Silva AF, McCollum P, Szymanska T, de Cossart L. Prospective study of carotid endarterectomy and contralateral carotid occlusion. Br J Surg 1996;83:1370-2.
2. Menyhei G, Bjorck M, Beiles B, Halbakken E, Jensen LP, Lees T, et al. Outcome following carotid endarterectomy: lessons learned from a large international vascular registry. Eur J Vasc Endovasc Surg 2011;41:735-40.
3. Shawl F, Kadro W, Domanski MJ, Lapetina FL, Iqbal AA, Dougherty KG, et al. Safety and efficacy of elective carotid artery stenting in high-risk patients. J Am Coll Cardiol 2000;35:1721-8.
4. Mercado N, Cohen DJ, Spertus JA, Chan PS, House J, Kennedy K, et al. Carotid artery stenting of a contralateral occlusion and in-hospital outcomes: results from the CARE (Carotid Artery Revascularization and Endarterectomy) Registry. JACC Cardiovasc Interv 2013;6:59-64.
5. Bydon A, Thomas AJ, Seyfried D, Malik G. Carotid endarterectomy in patients with contralateral internal carotid artery occlusion without intraoperative shunting. Surg Neurol 2002;57:325-30; discussion: 331-2.
6. Ricotta JJ, Aburahma A, Ascher E, Eskandari M, Faries P, Lal BK. Updated Society for Vascular Surgery guidelines for management of extracranial carotid disease: executive summary. J Vasc Surg 2011;54:832-6.
7. Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. North American Symptomatic Carotid Endarterectomy Trial Collaborators. N Engl J Med 1991;325:445-53.
8. Moneta GL, Edwards JM, Chitwood RW, Taylor LM Jr, Lee RW, Cummings CA, et al. Correlation of North American Symptomatic Carotid Endarterectomy Trial (NASCET) angiographic definition of 70% to 99% internal carotid artery stenosis with duplex scanning. J Vasc Surg 1993;17:152-7; discussion: 157-9.
9. Samson RH, Showalter DP, Yunis JP. Routine carotid endarterectomy without a shunt, even in the presence of a contralateral occlusion. Cardiovasc Surg 1998;6:475-84.
10. Gasecki AP, Eliasziw M, Ferguson GG, Hachinski V, Barnett HJ. Long-term prognosis and effect of endarterectomy in patients with symptomatic severe carotid stenosis and contralateral carotid stenosis or occlusion: results from NASCET. North American Symptomatic Carotid Endarterectomy Trial (NASCET) Group. J Neurosurg 1995;83:778-82.
11. Maatz W, Kohler J, Botsios S, John V, Walterbusch G. Risk of stroke for carotid endarterectomy patients with contralateral carotid occlusion. Ann Vasc Surg 2008;22:45-51.
12. Crawley F, Clifton A, Buckenham T, Loosemore T, Taylor RS, Brown MM. Comparison of hemodynamic cerebral ischemia and

microembolic signals detected during carotid endarterectomy and carotid angioplasty. Stroke 1997;28:2460-4.
13. Dalainas I, Nano G, Bianchi P, Casana R, Malacrida G, Tealdi DG. Carotid endarterectomy in patients with contralateral carotid artery occlusion. Ann Vasc Surg 2007;21:16-22.
14. Yadav JS, Wholey MH, Kuntz RE, Fayad P, Katzen BT, Mishkel GJ, et al. Protected carotid-artery stenting versus endarterectomy in high-risk patients. N Engl J Med 2004;351:1493-501.
15. Gray WA, Hopkins LN, Yadav S, Davis T, Wholey M, Atkinson R, et al. Protected carotid stenting in high-surgical-risk patients: the ARCHeR results. J Vasc Surg 2006;44:258-68.
16. Gray WA, Yadav JS, Verta P, Scicli A, Fairman R, Wholey M, et al. The CAPTURE registry: predictors of outcomes in carotid artery stenting with embolic protection for high surgical risk patients in the early post-approval setting. Catheter Cardiovasc Interv 2007;70:1025-33.
17. Iyer SS, White CJ, Hopkins LN, Katzen BT, Safian R, Wholey MH, et al. Carotid artery revascularization in high-surgical-risk patients using the Carotid WALLSTENT and FilterWire EX/EZ: 1-year outcomes in the BEACH Pivotal Group. J Am Coll Cardiol 2008;51:427-34.
18. Rockman C. Carotid endarterectomy in patients with contralateral carotid occlusion. Semin Vasc Surg 2004;17:224-9.
19. Flanigan DP, Flanigan ME, Dorne AL, Harward TR, Razavi MK, Ballard JL. Long-term results of 442 consecutive, standardized carotid endarterectomy procedures in standard-risk and high-risk patients. J Vasc Surg 2007;46:876-82.
20. Mozes G, Sullivan TM, Torres-Russotto DR, Bower TC, Hoskin TL, Sampaio SM, et al. Carotid endarterectomy in SAPPHIRE-eligible high-risk patients: implications for selecting patients for carotid angioplasty and stenting. J Vasc Surg 2004;39:958-65; discussion: 965-6.
21. Ballotta E, Saladini M, Gruppo M, Mazzalai F, Da Giau G, Baracchini C. Predictors of electroencephalographic changes needing shunting during carotid endarterectomy. Ann Vasc Surg 2010;24:1045-52.
22. Ballotta E, Da Giau G. Selective shunting with eversion carotid endarterectomy. J Vasc Surg 2003;38:1045-50.
23. Ballotta E, Da Giau G, Baracchini C. Carotid endarterectomy contralateral to carotid artery occlusion: analysis from a randomized study. Langenbecks Arch Surg 2002;387:216-21.
24. Bellosta R, Luzzani L, Carugati C, Talarico M, Sarcina A. Routine shunting is a safe and reliable method of cerebral protection during carotid endarterectomy. Ann Vasc Surg 2006;20:482-7.
25. Goodney PP, Wallaert JB, Scali ST, Stone DH, Patel V, Shaw P, et al. Impact of practice patterns in shunt use during carotid endarterectomy with contralateral carotid occlusion. J Vasc Surg 2012;55:61-71.e1.
26. Rerkasem K, Rothwell PM. Routine or selective carotid artery shunting for carotid endarterectomy (and different methods of monitoring in selective shunting); (Online). Cochrane Database Syst Rev 2009: CD000190.
27. Aburahma AF, Mousa AY, Stone PA. Shunting during carotid endarterectomy. J Vasc Surg 2011;54:1502-10.
28. Rockman CB, Halm EA, Wang JJ, Chassin MR, Tuhrim S, Formisano P, et al. Primary closure of the carotid artery is associated with poorer outcomes during carotid endarterectomy. J Vasc Surg 2005;42:870-7.
29. Mocco J, Wilson DA, Komotar RJ, Zurica J, Mack WJ, Halazun HJ, et al. Predictors of neurocognitive decline after carotid endarterectomy. Neurosurg 2006;58:844-50; discussion: 850.
30. Frawley JE, Hicks RG, Woodforth IJ. Risk factors for peri-operative stroke complicating carotid endarterectomy: selective analysis of a prospective audit of 1000 consecutive operations. Aust N Z J Surg 2000;70:52-6.
31. Littooy FN, Halstuk KS, Mamdani M, Calandra D, Hayes AC, Baker WH. Factors influencing morbidity of carotid endarterectomy without a shunt. Am Surg 1984;50:350-3.
32. Lewis SC, Warlow CP, Bodenham AR, Colam B, Rothwell PM, Torgerson D, et al. General anaesthesia versus local anaesthesia for carotid surgery (GALA): a multicentre, randomised controlled trial. Lancet 2008;372:2132-42.
33. Hill MD, Brooks W, Mackey A, Clark WM, Meschia JF, Morrish WF, et al. Stroke after carotid stenting and endarterectomy in the Carotid Revascularization Endarterectomy versus Stenting Trial (CREST). Circulation 2012;126:3054-61.

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INVITED COMMENTARY

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In this retrospective study, Samson and colleagues present excellent results (2% neurologic event rate, 0.7% mortality, and no clinically apparent myocardial infarctions) in 147 consecutive contralateral carotid occlusion patients, who underwent carotid endarterectomy (CEA) without shunting. They attribute their success to the use of general anesthesia with careful maintenance of systolic blood pressure above 130 mm Hg. Their results add fuel to the controversies over neurologic risk in contralateral occlusion patients and over the role of shunting.

While provocative, the study is not definitive. It suffers from the weaknesses inherent in retrospective analyses. The credibility of the outcome data suffers from the absence of independent routine pre- and post-CEA neurologic assessment and of a set protocol for post-CEA brain imaging. It is almost certain that there were more perioperative strokes than they report, though these events were clinically silent or subtle. Furthermore, given the authors' use of phenylephrine-induced hypertension as a means of cerebral protection, their failure to routinely monitor postoperative cardiac enzymes represents a missed opportunity to confirm the safety of their protocol. Theoretically, at least, the routine use of vasopressors will add to the hemodynamic stress of the surgery, thereby raising the risk of perioperative cardiac morbidity.

Despite these shortcomings, this report confirms four facts: (1) Well-trained surgeons, working with detail-oriented anesthesiologists and adhering to well-defined management protocols, can achieve excellent results with CEA even in putatively high-risk patients. (2) CEA patients with contralateral occlusion are not necessarily at high neurologic risk. (3) The argument that contralateral occlusion represents an indication for carotid stenting is specious. (4) Contralateral occlusion is not an absolute indication for shunt placement during CEA.

The report does not establish the superiority of the authors' practices, but it does establish their management protocol as one acceptable approach to the management of CEA patients with contralateral occlusion. Those who achieve excellent results in this patient population with carotid stenting or with CEA with shunting are not likely to be converted by these data, but now have another standard against which to measure their outcomes. Controversy over the risk associated with contralateral occlusion and the role of shunting will continue. In the face of ongoing controversy, surgeons will continue to adopt and report those practices that provide optimal results for their patients.