The natural history of the external carotid artery after carotid endarterectomy: Implications for management

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Purpose: **Most surgeons perform some type of endarterectomy of the external carotid artery (ECA) routinely during standard carotid endarterectomy (CEA). This approach has been shown to result in a small percentage of ECA occlusions, the clinical significance of which remains poorly understood. We have modified our approach to the management of the ECA during standard CEA by averting any attempt at external CEA. To evaluate the natural history of the untreated ECA after CEA, we reviewed the preoperative, postoperative, and follow-up duplex scans obtained from 232 CEAs over the past 4 years.**

Methods: **Preoperative and postoperative carotid artery duplex examinations with specific evaluation of the extent of ECA stenosis were available for review on 114 CEAs performed between January 1991 and July 1994. All CEAs were performed for internal carotid artery stenosis greater than 75% as determined by duplex scanning, which was confirmed by either contrast arteriography or magnetic resonance angiography.**

Results: **Seventy-three (64.0%) procedures were performed for symptomatic lesions, whereas 41 (36.0%) were performed for asymptomatic stenosis. There were no perioperative strokes or transient ischemic attacks in this group, and there was one postoperative death (0.9%). Short- and intermediate-term follow-up demonstrated insignificant changes in ECA diameter after operation, with no cases of ECA occlusion and only five cases progressing to greater than 75% on the 1-year follow up duplex examination.**

Conclusion: **We conclude from these data that averting external CEA during standard CEA does not result in significant progression of ECA stenosis or occlusion. (J Vasc Surg 1996;23:582-6.)**

Carotid artery endarterectomy (CEA) has proven to be a safe, effective, and durable procedure for reducing the incidence of stroke in both symptom-free patients and patients with symptoms of atherosclerotic occlusive disease of the carotid bifurcation.[1,2] The techniques used to perform the procedure are not standardized, and the type of anesthesia, intraoperative neurologic monitoring, requirements for shunting, and method of closure (primary versus patch) often are based on individual surgeon's preference. In addition to these technical variables, the method of removing plaque at the external carotid artery (ECA) similarly has not been subject to a uniform approach, with simple transection of the plaque at the orifice, blind endarterectomy into the lumen of the artery, or eversion endarterectomy[3-5] all being potential options for completing the endarterectomy in this area. The question of which is the optimal technique has not been answered in part because the natural history of the external carotid artery after routine CEA has not been well described. Because of this and our own inclination to simplify the procedure by transection of the plaque flush with the orifice of the ECA, we determined the impact of this technique on the patency of the ECA by analyzing our experience in a series of CEAs by reviewing the preoperative, postoperative, and follow-up duplex scans.

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MATERIAL AND METHODS

From January 1991 to July 1994, 232 CEAs were performed at Maimonides Medical Center. Preopera-

tive and postoperative carotid artery duplex examinations that specifically evaluated the degree of ECA stenosis were available for review on 114 CEAs performed during this period. These 114 CEAs were performed in 104 patients (44 women, 70 men). The patients' ages ranged from 45 to 91 years, with a mean age of 72. Sixty percent of the patients had a history of hypertension and smoking, and approximately 40% had diabetes. All CEAs were performed for an internal carotid artery (ICA) stenosis of 75% or greater as determined by duplex scanning, which was confirmed by either contrast arteriography or magnetic resonance angiography. Seventy-three (64%) CEAs were performed for symptomatic lesions, whereas 41 (36%) were performed for asymptomatic lesions. Patient follow-up typically was within 2 weeks after operation and then at 3- to 6-month intervals thereafter.

Duplex criteria

All patients were initially evaluated with duplex ultrasonography of the common carotid artery, ICA, and ECA. Standard criteria were used in the diagnosis of ICA disease. Moderate stenosis was believed to be severe if the peak systolic velocity was 130 cm/sec or greater and severe stenosis if the end diastolic velocity of 140 cm/sec or greater. The velocity findings were corroborated by B-mode imaging. The final interpretation was corrected on the basis of the B-mode image for cases of high velocity as a result of carotid tortuosity, kinking, or compensation for contralateral occlusion or severe stenosis and low velocities caused by long plaques or suspected low cardiac output.

The interpretation of ECA disease was based on cross-sectional and longitudinal B-mode ultrasound imaging, as well as Doppler peak systolic velocity measurements. Our estimation of ECA stenosis was validated by comparison with magnetic resonance angiography on the basis of 60 bifurcations studied. The correlation for stenoses either greater or less than 60% diameter reduction was 94% and the Kappa statistic 0.76. ECA peak systolic velocities less than 150 cm/sec or greater than 250 cm/sec were consistently associated with stenoses less than 50% or greater than 60% respectively. The respective negative and positive predictive values were 95% and 87%.

Carotid endarterectomy technique

Our technique of performing the endarterectomy involves the preferential use of regional anesthesia and bilateral hemispheric electroencephalographic monitoring. The vessels are dissected, isolated, and controlled. Systemic anticoagulation is obtained with intravenous administration of heparin and documented with activated clotting time measurement. The ICA is clamped first followed by the ECA, and usually the superior thyroid branch, and finally the common carotid artery. ICA back pressure is measured by releasing the ICA clamp with a 23-gauge needle, connected to a pressure transducer, inserted in the bulb. Nonpulsatile pressure tracings were systolic pressures below 40 mm Hg or electroencephalographic changes suggestive of cerebral ischemia (prolongation of frequency or reduction of amplitude) precipitate shunting. The endarterectomy at the orifice of the ECA is sharply transected flush with its ostia with no attempt to blindly endartectomize more distally into the ECA. Patency is assessed by backbleeding. Primary closure of the arteriotomy is usually performed unless the distal ICA is particularly diminutive or plication was required. In these instances, a patch closure will be performed. Intraoperative duplex evaluation or completion angiography is not used. The patients are monitored in the recovery room overnight and, barring any complication, discharged the following day.

RESULTS

No patient experienced a postoperative neurologic event (transient ischemic attack or stroke). One patient died as a result of a myocardial infarction, for a mortality rate of 0.9%.

Preoperative duplex evaluation of the ECA demonstrated less than 50% stenosis in 89 cases (78%), 50% to 74% stenosis in 16 cases (14%), and 75% to 99% stenosis in 9 cases (8%). No ECA occlusions were noted. Comparison of degree of ECA and ICA stenosis demonstrated no correlation between the two, with only 8% of the cases showing greater than 75% stenosis of both the ICA and ECA.

Early postoperative (less than 1 month) follow-up was available in all cases. Less than 50% stenosis of the ECA was found in 93 cases (82%), 50% to 74% stenosis was observed in 11 cases (10%), and 75% to 99% stenosis was noted in 9 cases (8%). No case was noted to have ECA occlusion after operation. The distribution of stenoses did not significantly differ between the preoperative and early postoperative studies: 63% of the cases remained unchanged (percent stenosis within 10% of preoperative value), 25% of the cases actually demonstrated improvement in the degree of stenosis, and 12% were noted to show increased stenosis.

More extended follow-up with a mean of 20 months and a range from 4 to 36 months was available in 90 cases. Stenosis of the ECA less than 50% was demonstrated in 81% of the cases, 50% to 74% stenosis

was noted in 10% of the cases, and 75% to 99% stenosis was found in 9% of the cases. No ECA occlusion was noted at extended follow-up.

Five of the 89 cases (5.6%) with follow-up ranging from 13 to 34 months progressed from stenosis of less than 55% to severe stenosis of 75% to 99%. In none of these cases was restenosis noted. None of these patients had diabetes; however, two continued to smoke and had elevated lipid levels, and three had hypertension.

DISCUSSION

This study reports our results of the fate of the ECA after a series of CEAs performed over a 4-year period by use of a sparing technique of removing the plaque flush at the orifice of the ECA thereby reducing the complexity of the procedure. This simplification of technique was motivated not only by the obvious benefit of minimizing the number of technical maneuvers required to perform the procedure, but also for our concern of an inadequate ECA endarterectomy being a nidus for embolizing thrombus into the ICA. Moore et al.[6] reported three cases of an intimal flap within the ECA after CEA that subsequently thrombosed retrograde and then embolized anterograde into the ICA. Others have reported similar post-CEA neurologic complications resulting from ICA embolization after ECA thrombosis caused by intimal flap defects remaining within the ECA[7] or as a result of ECA thrombosis after everting ECA endarterectomy.[8] Still others have reported asymptomatic post-CEA ECA stenoses or occlusions ranging from 5% to 16%,[9-12] but most have been unclear on the technique for managing the plaque at the ECA, nor have they specifically compared the degree of postoperative stenosis to preoperative values. Although our series cannot be considered large, it nonetheless is comparable to previous reports in the literature and is notable for 0% postoperative occlusion of the ECA. Even in the presence of significant severe preoperative ECA stenosis, postoperative occlusion did not occur despite intentionally leaving plaque within the ECA distal to its origin from the carotid bulb. Whether one therefore can draw the conclusion that an incomplete or imperfect endarterectomy of the ECA actually may predispose to postoperative complications is debatable, however, our results suggest at least that the sparing ECA endarterectomy technique does not appear to cause ECA thrombosis and remnant disease is relatively unimportant. Finally, the progression of disease in the ECA observed in 12% of the cases in this study did not have an adverse impact on either the patency of the ECA or the clinical outcome of the patient. Progression of disease within the ECA to severe (>75%) stenosis over intermediate follow-up did not lead to occlusion, and clinical sequelae suggesting even severe disease of the ECA is relatively benign.

Another finding of our study was the lack of correlation of disease within the ICA and ECA, that is, significant disease within one vessel was not associated with a similar finding in the other. Severe stenosis (>75%) of both the ICA and the ECA was observed in 8% of the cases; and, in most of the cases (approximately 80%), the ECA was relatively spared of the disease with stenoses less than 50%. Moreover, progression of disease within the ECA after CEA did not lead to restenosis of the ICA, suggesting the independence of disease within these two vessels. However, one caveat must be noted: progression of disease from stenosis to ECA occlusion may be associated with neurologic symptoms resulting from ICA embolism as has already been noted.[6,8,13]

Our study is the first to attempt to compare preoperative and postoperative duplex evaluation of the ECA and to quantitatively assess changes during an intermediate follow-up period. Earlier reports have noted the incidence of postoperative ECA stenosis or occlusion but have not specifically addressed the natural history of stenoses observed before CEA. Our findings suggest that ECA stenoses are relatively stable and that only a minority will progress to severe stenosis. Those that do progress to severe stenosis do not appear to confer additional risk of neurologic complication. Finally, the relatively simple technique of transection of the plaque at the orifice of the ECA does not result in increased incidence of ECA occlusion or of perioperative neurologic complications.

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DISCUSSION

Dr. Robert W. Hobson II (Newark, N.J.). I have always admired Dr. Ascer's track record of innovative inquiries concerning topics of surgical dogma. I suppose most of us were trained to do a modification of an eversion external CEA as part of our routine operation and never really considered questioning it. Dr. Ascer's group, however, asks about its importance and significance in the overall conduct of CEA. His review of the literature demonstrates only anecdotal evidence of neurologic complications directly related to the external CEA. He further concludes that simple division of the plaque at the ECA orifice is sufficient, and "thereby reduc[es] the complexity of the procedure."

From 1991 to 1994, 232 CEAs were performed by Dr. Ascer's service; however, only 114 of the cases (49%) were reviewed on the basis of the availability of preoperative and postoperative duplex scanning.

What were the results on the basis of the other 116 cases if they were included in the analysis? In addition, for example, although you had a meritoriously low stroke and death rate in the first 30 postoperative days of 0.9%, and none of those complications were related to the ECA lesion, did this hold true for the entire sample of 232 operations?

Second, you report detailed duplex data on the degree of ECA stenosis before operation and during a postoperative follow-up of 12 to 20 months. Although you presented peak systolic velocity data for stenoses of less than 50% and those greater than 75%, can you tell us more about your analysis and your method of determining the degree of stenosis? What are the sensitivity, specificity, and positive and negative predictive values of these determinations? It has been our impression that the measurement of ECA stenosis was more difficult as a result of its smaller transverse diameter, as compared with ICA lesions.

Also, during the follow-up you indicated that these ECA plaques had no influence on restenosis rates of the common carotid artery or ICA, but I did not see precise data on the restenosis rate. What was your restenosis rate for the common carotid artery and ICA in these cases and in the entire group of 232 endarterectomies? It may be necessary to monitor this group for a longer period of time before concluding that the ECA lesion has no influence on restenosis rate.

Third, in your report you suggest that "external carotid thrombosis and remnant disease is of no importance." Under different circumstances, what would you advise us to do if there was an ICA occlusion and a high-grade ECA stenosis in a patient with symptoms? I assume you'd recommend external CEA. Why not perform the procedure at the initial operation?

In my opinion, your study should be extended for longer follow-up in a larger number of cases before general adoption of this technical modification in standard CEA. Defining natural history of the ECA lesion may also ultimately require you to consider randomizing your technical suggestion against the standard procedure. In the meantime, your presentation has reemphasized for me the importance of periodically reassessing our standard operative techniques, in this case CEA.

Dr. Mark Gennaro. The reason that we monitored only 114 of the cases rather than all 234 is that our duplex follow-up was the best in those patients. The analysis is a little more complex. We actually had the benefit of having Dr. Sergio Salles-Cunha review our duplex findings and correlate them with the magnetic resonance angiography (MRA) findings on our patients. And using a cap analysis analogous to what was done for the ICA stenosis, he came up with a cavi of about 0.77. It was performed in exactly the same manner, in that he would measure actually the ECA diameter on the MRA and compare it with the duplex findings. For the duplex findings he used both B-mode and a cross-sectional longitudinal evaluation of the ECA diameter with the use of spectral analysis to assist in determining the degree of the stenosis.

In 114 cases one patient had an obvious restenosis that ultimately ended up undergoing reoperation. We had no other patients who demonstrated significant ICA stenosis.

I believe that patients who have an ICA occlusion, and who have symptoms, do have indications for ECA reconstruction, but I think that's a different entity than what

we're describing here. And certainly, although our results are sort of intermediate follow-up, the bottom line is that if there is no ECA occlusion with this technique, and I believe the ECA occlusion causes neurologic sequelae after operation. Indeed, longer term follow-up of these patients with this technique does not demonstrate that occurrence, that it is a valid technique to perform during the CEA.

Dr. Linda M. Harris (Buffalo, N.Y.). From what I understand, you did not actually do anything to the ECA itself; however, you say that in 25% of your patients there was a reduction in the amount of stenosis. How did you get there?

Dr. Gennaro. Regardless of whether there's some orificial stenosis that we relieve with the transection of the plaque or whether it's an increase in pressure, we're not entirely sure. But, in fact, when comparing preoperative and postoperative ECA diameters on the same patient, those are the values that we got.

So, again, whether it's some disease at the orifice of the ECA that was removed by our transection or whether somehow there's an increase in pressure that dilates the vessel somewhat, those are only two potential reasons why it may have increased, but I can't give you the exact reason.

Dr. Jeffrey P. Carpenter (Philadelphia, Pa.). Is it possible that the altered flow dynamics at the bifurcation after the endarterectomy, by favoring flow through the ICA and not through the ECA, caused the lowering of your ECA velocities and that the actual determination of a lesser stenosis after operation is artifactual?

Dr. Gennaro. That's a possibility.

Dr. John J. Ricotta (Buffalo, N.Y.). When you have minor stenosis, your technique makes sense, but if you have a high-grade stenosis, it might be harder to try to transect that high-grade calcified plaque than to just take it out. What is the rationale for leaving a high-grade stenosis in there? Do you believe it's easier? If somebody has amaurosis fugax and a 90% ICA stenosis and an 80% ECA stenosis, are you recommending that we leave the ECA stenosis alone?

Dr. Gennaro. I assume that the amaurosis fugax would be a result of the internal lesion, and certainly relieving that obstruction should benefit the patient. On the basis of this study, the natural history of leaving even a high-grade stenosis is not a virulent or malignant thing to do. As long as backbleeding demonstrates patency, until further follow-up proves that these patients will have occlusion, and those are the patients who will have the problem, it's not an invalid technique.

Dr. Gary A. Fantini (New York, N.Y.). I'm somewhat uncomfortable with the concept of leaving significant disease behind in the ECA. In the setting of ICA occlusion, either after endarterectomy or in the absence of endarterectomy, the ECA can supply important hemispheric flow. It can provide important collateral vessels to the eye, as evidenced by the occurrence of amaurosis fugax via ECA pathways, as well as a conduit for hemispheric transient ischemic attack and stroke. Over a long period of time, would a larger percentage of your patients with development of recurrent stenosis of the ICA have development of symptoms if they still have a high-degree stenosis or occlusion of the ECA?

Dr. Gennaro. If they had recurrent stenosis of the ICA after operation, then if an external CEA had been performed, it might provide enough of a collateral pathway to maintain the patient without any symptoms. That certainly can happen. I believe that the degree of restenosis is very small. In our case it's probably less than what's reported in the literature.

Dr. Fantini. Patients with very high degrees of restenosis of the ICA tend to be symptom free. I wonder if a large enough series of patients with recurrent carotid artery stenosis could be pooled to permit correlation of symptoms with the status of the ECA circulation, to determine whether the patients who have development of symptoms are patients in whom the ECA has occluded or is severely stenotic.