The safety, efficacy, and durability of external carotid endarterectomy

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Results of 21 external carotid endarterectomies (ECEAs) in 19 patients with symptoms were reviewed retrospectively. No patients died or had new strokes referable to ECEA within 30 days of surgery. Overall, six (32%) patients died during an average 41 months of follow-up (range 1 to 134 months). Persistent symptoms followed five (71%) of seven ECEAs performed for cerebrovascular insufficiency or transient ischemic attacks. One patient treated urgently for an evolving stroke failed to improve and died after hospital discharge. Another patient with crescendo transient ischemic attacks had a preoperative deficit that did not resolve for 6 months and had a second stroke 2 years later. Another patient had a stroke after a contralateral carotid reconstruction but recovered. In contrast, only two (14%) of 14 ECEAs performed for monocular amaurosis fugax had persistent symptoms after surgery. Durability of 16 ECEAs was evaluated by arteriography or duplex scanning. Of six ECEAs closed primarily, three (50%) occluded, one has 60% restenosis, and only two (33%) had no restenosis (mean follow-up 36 months). Of 10 ECEAs closed by patch angioplasty, none occluded, 2 had 20% restenosis, and eight (80%) had no restenosis (mean follow-up 47 months). Life-table analysis indicated improvement in ECEA patency and durability with patch angioplasty ($p = 0.011$). From these data, ECEA can be performed with relative safety but is more effective for treatment of monocular amaurosis fugax in patients with a microembolic source at the external carotid origin. Patients with any other indications for ECEA did not benefit consistently from this operation. (J VASC SURG 1992;16:407-13.)

External carotid endarterectomy is an infrequently performed operation that has been used in the treatment of selected patients with retinal or cerebral ischemia and ipsilateral internal carotid artery occlusion.[1-19] The rationale for reconstruction of the external carotid artery is based on observations that its branches form an extensive collateral network reconstituting the ophthalmic and cerebral arteries in the presence of ipsilateral internal carotid artery occlusion.[1-6] Stenotic or ulcerated lesions at the origin of the involved external carotid artery become sources of ischemia or microembolism through these collaterals. Such lesions can thus be corrected surgically by external carotid reconstruction to improve cerebrovascular blood flow and eliminate microembolic sources.

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A number of recent clinical studies, technical articles, and collective reviews have attempted to establish the role of external carotid endarterectomy among the various extracranial carotid revascularization procedures.[7-20] This operation is especially appealing because of the limited number of treatment options usually available for patients with symptoms of chronic internal carotid artery occlusion. However, external carotid reconstruction has not been without controversy concerning its clinical safety, efficacy, and durability.[11-15,19] These issues prompted a review of our clinical experience with patients treated by external carotid endarterectomy.

PATIENTS AND METHODS

Our collective surgical experience with external carotid artery revascularization was reviewed retrospectively. A combined total of 19 patients with symptoms underwent 21 external carotid endarterectomies (two patients underwent bilateral external carotid endarterectomies) during the past 11 years. These 21 external carotid endarterectomies represent approximately 1% of all carotid operations performed by these surgeons. Of these 19 patients, 11 (58%) were men and eight (42%) were women, 16

(84%) were white and three (16%) were black, and average age at the time of operation was 60 years (range 46 to 73 years). Risk factors for arterial occlusive disease included smoking (n = 14; 74%), hypertension (n = 12; 63%), diabetes mellitus (n = 8; 42%), and hyperlipidemia (n = 3; 16%).

Interestingly, 11 patients (58%) underwent previous operations for extremity arterial occlusive disease and four (21%) had abdominal aortic aneurysms. Seven patients (37%) had a history of myocardial infarction and four required coronary artery revascularization. In addition, eight patients (42%) had a past history of stroke ipsilateral to the internal carotid occlusion. Eight patients (42%) also underwent carotid endarterectomies before or during the study period: five were contralateral to and three were ipsilateral and prior to a later external carotid endarterectomy. One patient had bilateral carotid endarterectomies followed 1 and 7 years, respectively, by an external carotid endarterectomy.

All 19 patients had chronic internal carotid artery occlusion and preoperative symptoms referable to disease at the origin of the ipsilateral external carotid artery. Fourteen external carotid endarterectomies (67%) were performed for ipsilateral monocular amaurosis fugax. Seven (33%) were performed for hemispheric cerebrovascular insufficiency: five had ipsilateral transient ischemic attacks (TIAs), one had crescendo TIAs and a preoperative neurologic deficit, and one had an acute stroke in evolution during carotid arteriography for evaluation of amaurosis fugax.

The 21 external carotid endarterectomies were performed in a similar fashion by standard operative techniques. Intraoperative shunting of the external carotid artery was used in only three procedures. A variety of techniques were employed to close the endarterectomy site: saphenous vein patch angioplasty (n = 8), primary arteriotomy closure (without patch angioplasty) (n = 7), internal carotid arterial patch angioplasty (n = 3), and prosthetic patch angioplasty (n = 3) (polytetrafluoroethylene patch, 2, and Dacron patch, 1).

Clinical results of surgical treatment were available for all 19 patients 30 days after surgery and were used to determine the relative safety. The long-term effectiveness of this operation could be evaluated in 16 patients. Of the other three patients, one patient died and two were lost to follow-up after the early postoperative period. Follow-up arteriography or duplex scan was available in 14 patients (16 external carotid endarterectomies) and was used to determine the durability of various external carotid reconstruction techniques. Groups were compared with the Kwikstat Statistical Data Analysis Program (Texasoft, Cedar Hill, Texas).

RESULTS

Operative findings. Lesions found during surgery at the external carotid orifice included one or more of the following: stenotic plaque (n = 17; 81%), plaque ulceration (n = 8; 38%), an internal carotid artery cul-de-sac (n = 8; 38%), and thrombus extending into the external carotid artery (n = 1; 5%). Of the six external carotid endarterectomies performed for TIAs, all had external carotid artery stenosis: three (50%) had stenotic lesions alone, two had plaque ulceration, and one an internal carotid cul-de-sac. Of the 14 external carotid endarterectomies performed for amaurosis fugax and the single patient with amaurosis fugax who had an evolving stroke after arteriography (15 total), nine (60%) had stenosis plus an ulcer or an internal carotid cul-de-sac, four (27%) had an isolated internal carotid cul-de-sac, and only two (13%) had a stenotic lesion alone.

Treatment results. None of these patients had new neurologic deficits referable to external carotid endarterectomy or died less than 30 days after surgery. To date, the average postoperative clinical follow-up of all these patients is 41 months (range 1 to 134 months). Overall, six (32%) of the 19 patients have died during the study follow-up period. Causes of death were acute myocardial infarction (n = 3; 2 to 3 years later), stroke (n = 2; 1 month and 6 years later, respectively), and intraabdominal sepsis (n = 1; 7 months later).

Five (71%) of seven external carotid endarterectomies performed for cerebrovascular insufficiency or TIAs continued to have persistent symptoms after surgery. The one patient treated urgently for a stroke in evolution after carotid arteriography failed to improve neurologically and subsequently died after discharge from the hospital. Another patient had acute crescendo right hemispheric TIAs that progressed to a left-sided neurologic deficit. A right external carotid endarterectomy was performed, but the deficit did not resolve for 6 months after surgery. Right hemispheric TIAs recurred 1 year later and were followed 2 years later by another stroke from which the patient again recovered. This patient died 6 years after surgery, presumably of yet another stroke.

A third patient first underwent a left external carotid endarterectomy for ipsilateral hemispheric and ocular ischemia. This procedure was followed 1 week later by a complex right carotid and innominate

artery reconstruction, and was complicated by a right hemispheric stroke. This patient recovered and subsequently underwent a right external carotid endarterectomy 7 years later for amaurosis fugax. This was the only patient of the five with postoperative cerebral symptoms who had an operable contralateral carotid lesion. The remaining two patients with persistent postoperative ipsilateral TIAs were treated successfully by anticoagulation. None of these patients had postoperative external carotid artery restenosis or occlusion by duplex scan.

In contrast, only two (14%) of 14 external carotid endarterectomies performed for ipsilateral monocular amaurosis fugax resulted in any persistent ipsilateral problems. One patient lost partial vision in the affected eye 6 months later because of temporal arteritis. Another patient was found to have recurrent retinal artery cholesterol-microemboli in the early postoperative period but was clinically symptom free. Neither patient with postoperative ocular disease was found to have external carotid occlusion or restenosis by duplex ultrasonography.

Durability and long-term patency. The durability and long-term patency of external carotid endarterectomy were studied in 14 patients (16 external carotid endarterectomies) by arteriography (four patients) or duplex scan (13 patients). Of six external carotid endarterectomies closed primarily, three (50%) occluded, one has 60% restenosis, and only two have no restenosis during an average 36-month follow-up evaluation (range 1 to 87 months). Of 10 external carotid endarterectomies closed by patch angioplasty, none have occluded, two prosthetic patches (one polytetrafluoroethylene and one Dacron) have minimal 20% restenosis, and the remaining eight (80%) have had no restenosis during an average 47-month follow-up evaluation (range 23 to 87 months). The occurrence of a greater than 50% postoperative restenosis or occlusion in these two groups was statistically different (Fisher exact test, $p = 0.043$). Life-table analysis of postoperative external carotid artery patency is shown in Fig. 1 and indicates greater durability of this operation with patch angioplasty ($p = 0.011$). Because of the small numbers, differences in external carotid patency between the various patch materials could not be determined.

DISCUSSION

Despite recent and widespread clinical awareness of external carotid revascularization, this treatment option has been used infrequently compared with other carotid operations, particularly internal carotid endarterectomy. There are relatively few published studies pertaining to external carotid endarterectomy, and the number of patients reported is actually quite small.[10,12,17] In reported series, external carotid reconstructions constitute 1% to 6% of all carotid artery operations.[9,14,18,19] Given the limited data available about the results of external carotid endarterectomy, it is of concern that so much controversy has been generated as to its morbidity and mortality rates, effectiveness, and long-term durability.[11-15,19]

Several recent studies reported a disproportionately high incidence of postoperative strokes and deaths after external carotid endarterectomy.[11,14] These untoward events appear to be related largely to contralateral cerebral revascularization procedures performed in association with the external carotid endarterectomy and not directly with the external carotid endarterectomy itself.[11,14] One patient in our series had a new contralateral hemispheric stroke in the early postoperative period. This stroke was not referable to the initial external carotid endarterectomy but occurred after a complex contralateral carotid and innominate artery reconstruction requiring an interposition graft.

External carotid revascularization has been shown to increase ipsilateral cerebral blood flow by 15% to 39%,[7] but it is clear that certain patients do not respond favorably to this treatment. Two of our patients with preoperative neurologic deficits (stroke in evolution and crescendo TIAs) did not improve after surgery, and one died in the early postoperative period. Other reports and our experience with these cases suggest that external carotid endarterectomy does not benefit patients with evolving neurologic deficits or provide cerebral protection to those requiring subsequent contralateral carotid reconstructions.[11,14] Although it is now possible to assess objectively the adequacy of collateral circulation in patients with cerebrovascular occlusive disease, no data are currently available that indicate these tests can be used to select patients for external carotid endarterectomy.[21]

A recent collective review of 218 external carotid revascularization procedures (195 external carotid endarterectomies) demonstrated that 83% of patients can be surgically relieved of their symptoms and another 7% will have improvement.[17] The compiled neurologic complication rate was 5% and the postoperative death rate was 3%, most of which followed strokes. It was concluded by these and other authors that external carotid endarterectomy is more likely to benefit patients with specific ipsilateral cerebral or ocular symptoms than those with nonspecific neurologic complaints or strokes.[11-19] Patients with ipsilat-

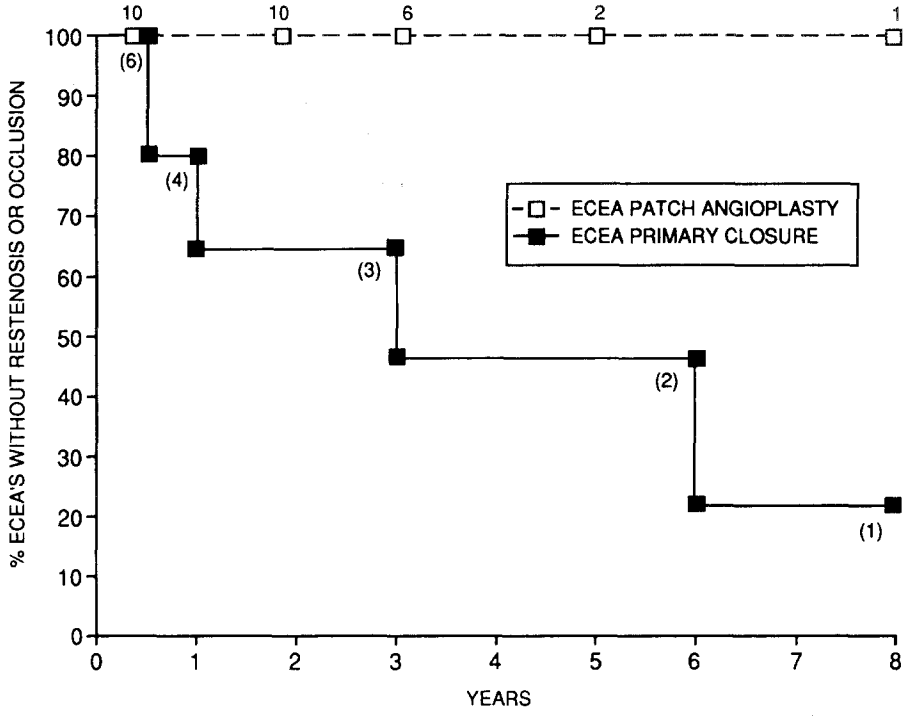

Fig. 1. Life-table analysis of postoperative external carotid endarterectomy *(ECEA)* restenosis compares 10 external carotid arteries reconstructed with patch angioplasty and six with primary arteriotomy closure alone. ECEA was considered restenotic or occluded if the postoperative arterial lumen was reduced by 50% to 100%. Mantel-Haenszel test was used to compare results ($\chi^2, p = 0.011$).

eral TIAs and monocular amaurosis fugax, which result from microemboli through external carotid collaterals, are the best candidates.[11-19]

This observation is especially true of patients with amaurosis fugax.[18] Although not statistically significant, only 14% of our patients with amaurosis fugax had ocular symptoms after surgery compared with 71% with persistent cerebral symptoms among those patients with TIAs. In an effort to explain this difference, it was noted that 87% of our patients with amaurosis fugax had an ulcerated plaque or residual internal carotid artery cul-de-sac that was most likely a microembolic nidus. By comparison, only 50% of our patients with TIAs had such lesions. Prophylactic external carotid endarterectomy in symptom-free patients has been reported; no patients in our series were without symptoms.[14] Because of the few cases reported in the literature, it is unclear how, or even if, symptom-free patients should be selected for this operation.

The long-term prognosis of patients requiring external carotid endarterectomy has not been well established. Although perioperative internal carotid artery occlusions often occur asymptomatically, up to 42% of these patients will have an accompanying stroke, as was found in our series. Of more concern, results from the Joint Study of Extracranial Arterial Occlusion indicated that 19% of patients with an internal carotid artery occlusion die of stroke within 5 years.[22] Despite external carotid revascularization, 32% of patients in our series died during the 4-year follow-up. This observation suggests that deaths from all causes, including stroke, might be higher in this subgroup of patients. These survival data are similar to those reported in patients undergoing various cerebrovascular operations, especially subgroups with a history of stroke or an internal carotid artery occlusion.[14,23-26]

The concept of carotid patch angioplasty in conjunction with endarterectomy has received much attention in the recent surgical literature.[27-30] Although the actual rate of postendarterectomy carotid restenosis remains a matter of debate, several series report that carotid patch angioplasty significantly improves long-term internal carotid artery patency.[27-30] In none of these reports, however, has the difference in patency between primary internal carotid arteriotomy closure and patch angioplasty

been as great as that found associated with external carotid reconstructions in this study. The importance of obliteration of the residual internal carotid artery orifice during external carotid endarterectomy to prevent early occlusion was recently emphasized.[19] Failure to accomplish this aspect of the operation adequately may be the critical technical flaw associated with early occlusion after primary external carotid arteriotomy closure. In our series three of six external and common carotid arteries closed primarily occluded within 3 years and another has an asymptomatic 60% restenosis. None of the reconstructions performed with a patch angioplasty have developed a significant restenosis or occluded during more than 3 years of follow-up evaluation.

External carotid endarterectomy can be performed with an acceptable morbidity and mortality rate but has several important limitations. Patients most likely to benefit from this operation should be selected on the basis of ipsilateral, monocular amaurosis fugax and have a probable source of microemboli at the external carotid orifice. In our series, patients with other indications for external carotid endarterectomy did not consistently benefit from operation. Patch angioplasty closure of the external carotid arteriotomy with obliteration of the internal carotid orifice improves long-term patency and durability. Venous, arterial, and prosthetic patches appear to function equally well in our series to prevent post-external carotid endarterectomy restenosis.

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DISCUSSION

Dr. William H. Edwards, Sr. (Nashville, Tenn.). In your retrospective review, the series probably mirrors most reported series in numbers of men and women, the patients, and their risk factors. It is not unusual for this population to require secondary vascular operations.

The efficacy of external carotid endarterectomy in this report, as in others, leads one to the conclusion that patients with amaurosis are those who are going to obtain the best results. This really should come as no surprise. Embryologically the ophthalmic and external carotid artery develop separately but have rich collaterals. With further development these collaterals diminish and may rarely be seen angiographically. However, with internal carotid artery occlusion, these collaterals are brought back into play and it has been estimated, as you said, that approximately 30% of internal flow into the carotid siphon may result from collaterals when there is internal carotid occlusion.

Operative technique is obviously important. Whether the cause is embolic or stenotic, the technique of endarterectomy and closure is important. The operation, be it internal or external carotid endarterectomy, is now a mature operation that can be performed safely with low morbidity and mortality rates. I think external carotid endarterectomy is neither more risky nor more technically demanding than internal carotid endarterectomy, but as you have shown in your series, it is important to consider patch angioplasty because your long-term follow-up shows that patients who have undergone patch angioplasty have a much greater chance of patency. When measured by angiography or duplex studies, one half of those endarterectomies closed primarily had occluded or a restenosis had occurred as opposed to the 10 that were closed with patch angioplasty, and their patency rate was much greater.

I noticed on one of your angiograms that you did see filling of the siphon by external endarterectomy and its collaterals. Was this demonstrated in any other patients? I think this is an important finding. I feel much more comfortable operating on the external carotid artery if I can demonstrate by angiography that there is filling of the siphon, making me feel that I am going to at least accomplish more for restoring flow.

In the six primary closures of which three were occluded and one had 60% stenosis, how many of these patients had symptoms?

Dr. Daniel S. Rush. With respect to your first question about filling of the carotid siphon through external carotid collaterals, in my own cases in this series we almost invariably saw this arteriographic finding in patients with symptoms. I think this phenomenon has a lot to do with arteriographic technique, that is, whether the angiographer allows time for these collateral vessels to fill and then takes delayed films. It is then that you see collateralization develop with carotid siphon filling.

With regard to your question about primary external carotid arteriotomy closures in the three patients who had occlusion after surgery, you have raised a very interesting point because all three patients remained symptom free. We were surprised, but I think that this is an important clinical observation and one that might actually be expected given internal carotid occlusion and failure of the external carotid reconstruction. With no flow through that portion of the carotid system, distal microembolism becomes unlikely and these patients remain symptom free.

Dr. Daniel F. Fisher, Jr. (Chattanooga, Tenn.). I agree with many of the points you made. I agree strongly that patching these external carotid arteries really is the way to do it rather than trying to close them primarily.

Is there a place to perform this operation in a symptom-free patient to try to increase the head of pressure that you are trying to get in the circle of Willis?

Dr. Rush. We have not operated on any symptom-free patients, but this is an important consideration related to the point I was trying to make in my question after Dr. Perler's presentation yesterday, that is, whether an external carotid endarterectomy in patients with an occluded internal carotid artery might not provide some cerebral protection during a subsequent contralateral internal carotid endarterectomy. Based on two patients in our series in whom external carotid endarterectomy did not offer cerebral protection, I would have to take a negative view of performing this operation in symptom-free patients. There are not enough data, however, to support a conclusion one way or the other.

Dr. James W. Dennis (Jacksonville, Fla.). How would you handle a patient with transient ischemic attacks in the hemisphere that is ipsilateral to an external carotid stenosis but with an internal carotid stenosis in the contralateral side? Do you go ahead and do the internal carotid endarterectomy on the contralateral side, or do you do the external carotid endarterectomy on the ipsilateral side?

Dr. Rush. As a matter of fact, Dr. Dennis, there was one patient in the series who met the particular situation you describe. We performed the external carotid endarterectomy first and then came back and treated the contralateral carotid lesion as if it were an asymptomatic stenosis. Obviously, our approach might be grounds for debate as to the staging of these operations. I suppose that any clinical decision would depend on the internal carotid anatomy, the circle of Willis, and a determination of exactly where the blood flow to the ipsilateral side was coming from. I think perhaps this issue gets back to the question that Dr. Edwards raised and the point he made about visualization of the carotid siphon from external carotid collaterals as an important indicator that there is a "watershed" area of cerebral ischemia.

Dr. G. Melville Williams (Baltimore, Md.). I have had three or four patients who have truly had bright-light amaurosis. Their retinas just go out in bright sunlight so

that they avoid going out at noon to walk and so forth. I believe that this is really worth distinguishing in patients who complain of amaurosis, because I think bright-light amaurosis is probably not an embolic phenomenon but flow related. The retina simply does not have the blood flow to meet the metabolic requirements necessitated by the bright light. I wonder in your series whether you made that distinction, because clearly I think this group of patients would be benefitted best by external carotid endarterectomy.

Dr. Rush. Once we became aware of articles describing the bright-light amaurosis phenomenon a few years ago, we started looking for this symptom and found one patient who had that particular complaint. Our other patients had more typical monocular amaurosis fugax and, in fact, several of them were referred by ophthalmologists with cholesterol emboli in their retinal arteries.

Dr. Lewis Friedlander (New Haven, Conn.). I think this is an area that really deserves a lot more attention when evaluating these patients before surgery. One of the comments I wanted to make was germane to this last gentleman's point that the differences in visual disturbances may not always be embolic phenomena. In fact, we have demonstrated in a number of patients that low-flow phenomena are often involved, and having a technique or set of techniques to distinguish this before surgery may be of benefit beyond just the angiographic appearance. We found in a number of cases both before and during surgery that the occlusion of the external carotid artery or temporary ligation actually improves the ocular and cerebral flow by noninvasive technology. Some of these cases actually may not be the best choice to try to divert further flow from the carotid artery on partial occlusion or even total occlusion, because collaterals have already developed. So preoperative assessment of the collaterals by techniques other than angiography have been found to be helpful. I think that is an important point for these patients as far as the symptoms of amaurosis not necessarily being embolic.

Dr. Rush. I have nothing further to add to these comments.