Conservative management of residual and recurrent lesions after carotid endarterectomy: Long-term results

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Purpose: **To document the natural history of residual and recurrent carotid stenoses that are initially treated without surgery, and to identify risk factors for recurrent stenosis.**
Methods: **Review of data from a prospective carotid database with clinical and duplex follow-up. Analysis of rate of restenosis and rate of late reoperation by life table. Risk factor analysis by χ^2 and LEE-DESU statistics.**
Results: **Three hundred forty-eight patients were available for follow-up, with 12 residual lesions (3.7%) and 22 recurrent lesions (6.6%). Rate of recurrent stenosis by life table analysis was 8.7% and 13% at 3 and 5 years. Restenosis was associated with smoking (p = 0.04) and contralateral progression. Only 21% of patients were underwent an operation within 5 years (p = 0.007) of restenosis developing, but eventually 10 of 22 patients required reoperation at long-term follow-up, eight for symptoms and two for progressive proximal stenoses. The late stroke rate was increased in patients who had residual or recurrent lesions compared with those who had normal duplex study results (18% vs 6%; p = 0.16) and was related to the ipsilateral artery.**
Conclusions: **Recurrent lesions that remain asymptomatic can be managed without operation with likelihood of success in the near term (5 years). However, these patients are at increased risk of late stroke, and almost half will eventually require operation. Therefore, in good-risk patients operation for asymptomatic restenoses should be considered.**
(J Vasc Surg 1997;26:963-72.)

Carotid endarterectomy has been established as the gold standard for patients with severe (>70%) stenoses of the carotid bifurcation. Many studies have documented that this operation can be performed with minimal morbidity and mortality risks. Despite its success, residual stenoses are found in a small number of patients after endarterectomy, and recurrent lesions develop in a subset of patients on long-term follow-up. The proper management of these lesions is subject to debate. Several authors documented an increased risk with secondary operations for carotid stenosis,[1,2] whereas others have reported series with complication rates that are not significantly different from those of primary endarterectomy.[3-6] The indications for reoperation vary between surgeons; some advocate routine operation for severe stenosis, whereas others, including ourselves,[7-10] have adopted a more conservative approach because of the low incidence of symptomatic recurrence even with severe lesions. In most operative series, one third to one half of operated lesions are asymptomatic. There are no long-term prospective data on the results of managing asymptomatic recurrent stenosis expectantly. The purpose of this review was to document the late outcome of patients with recurrent and residual stenosis initially managed without operation, with particular attention to the rate of reoperation, the late stroke rate, and the morbidity of secondary carotid reconstruction.

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Presented at the Eleventh Annual Meeting of the Eastern Vascular Society, Atlantic City, N.J., May 2–4, 1997.
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0741-5214/97/$5.00 + 0 **24/6/85268**

MATERIALS AND METHODS

We reviewed all carotid endarterectomy procedures performed by a single surgeon that had been entered prospectively into a carotid database. Patients were prospectively followed-up with reference to development of new neurologic symptoms as well as evidence of recurrent stenosis by duplex ultrasound. Patients who had both clinical and duplex ultrasound follow-up were selected for further analysis. Residual or recurrent stenoses were identified on

Table I. Patient characteristics

Indicator	*Study group (n = 348)*	*Excluded patients (n = 95)*	*p*
Patient age (yr)	68.1 ± 8.5	68.1 ± 8.2	NS
Male	53.2%	64.5%	0.05*
Coronary artery disease	51.9%	43.4%	0.19
Hypertension	74.3%	72.0%	0.68
Diabetes mellitus	31.7%	29.6%	0.72
Smoking	43.4%	64.1%	0.001*
Asymptomatic	38.9%	30.7%	0.115
Bilateral disease at time of surgery	39.1%	50.0%	0.45
Postoperative cerebrovascular accident	2.0%	9.7%	<0.001
Late stroke-free rate (3 yr)	94.6%	91.9%	0.95
Late stroke-free rate (5 yr)	92.7%	92.7%	

*Statistically significant.

Table II. Residual lesions (n = 13)

Location	*50% to 74%*	*75% to 99%*	*Occluded*	*Total*
Common carotid artery	1	0	1	2
Internal carotid artery	1	7	3	11
Total	2	7	4	13

the basis of elevations in peak systolic velocity using criteria validated in our laboratory. The incidence of residual and recurrent lesions was determined in the study group. Lesions that caused >50% diameter stenosis by duplex ultrasound were defined as significant and were further divided into moderate (50% to 74%), severe (75% to 99%), and occluded groups. Based on our previous report,[7] residual lesions are defined as those that appeared on the first postoperative study performed within 12 months of endarterectomy. Recurrent stenoses are defined as those that appeared subsequent to a normal duplex examination or more than 12 months after the initial examination. The risk of restenosis for the study group was calculated at 3 and 5 years by life table methods. Patients who had asymptomatic residual or recurrent lesions were initially followed-up without operation on the basis of our prior published experience.[7] In patients who had residual and recurrent lesions, the number of late reoperations and the rate of reoperation was determined. Finally, the late stroke rate of patients with residual and recurrent lesions was compared with that of patients without such lesions, and the complications associated with secondary operation were documented. For this final analysis, we reviewed the results of all secondary operations performed during the review period, including those in patients whose primary operation was performed elsewhere.

RESULTS

A total of 443 patients was entered into the database between 1982 and 1996. The overall perioperative stroke and death rate was 2.3%. Long-term clinical and duplex follow-up was available in 348 cases, which form the basis for the remainder of this report. Duplex follow-up was not available in 95 patients. Many of these patients underwent operation in the early experience before an established ultrasound protocol existed. When compared with the study group, patients without follow-up were more likely to smoke ($p = 0.001$) and had a higher perioperative stroke rate ($p < 0.001$). The data are shown in Table I. Clinical follow-up was available in 81 of these patients, and the late stroke rate did not differ from that of the study group. There were 13 residual lesions identified in the study group (3.7%), including five postoperative occlusions. A listing of these lesions and their location and severity is presented in Table II. Duplex evidence of recurrent stenosis was seen in 22 patients (6.6%). Ten patients (2.9%) had evidence of a stenosis of 50% to 79%. Severe lesions developed in 12 patients (3.7%), including one late occlusion. The risk of restenosis >50% developing after primary endarterectomy was 8.7% at 3 years and 13% at 5 years by life table analysis and is presented in Fig. 1. We evaluated the potential effect of gender, smoking history, and patch closure on the development of recurrent stenosis, and only smoking history was significant ($p < 0.04$; Table III). The development of restenosis was associated with progression of disease in the contralateral carotid artery ($p = 0.007$; LEE-DESU). In patients who did not have contralateral disease progression, the risk of developing a recurrent stenosis was 6% at 3 years and 12.77% at 5 years, compared with 25.3% at both 3 and 5 years in patients who had contralateral progression (Fig. 2).

The initial management of residual and recurrent lesions was nonoperative in all cases. Our initial strategy of nonoperative management was successful in most cases; only 14.2% of recurrent lesions were operated on within 3 years of their development, with 20.6% eventually operated on by 5 years of follow-up. These data are presented graphically in Fig. 3. One patient underwent secondary operation for a severe asymptomatic ulcerated residual lesion in the common carotid artery. The remainder of resid-

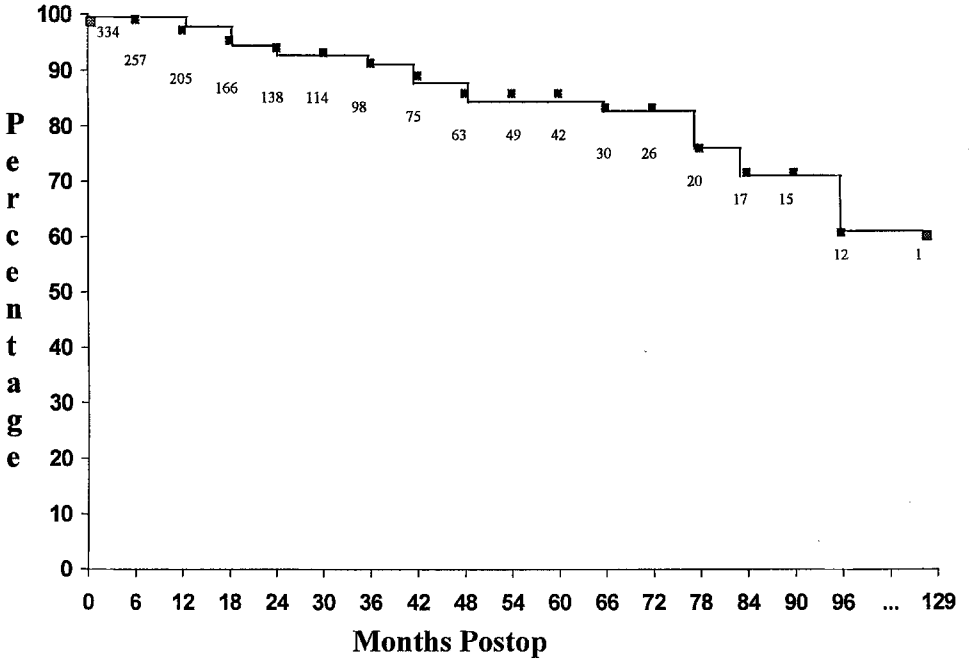

Fig. 1. Restenosis-free rate in 334 patients who underwent long-term Doppler evaluation after carotid endarterectomy, excluding postoperative occlusion and residual stenosis, reveals restenosis rates of 8.7% and 13% at 3 and 5 years, respectively.

ual lesions were followed-up. Reoperation for recurrent stenosis was more frequent. Eventually, 10 of the 22 arteries with recurrent lesions came to reoperation (47.8%). Indications for reoperation in patients with recurrent lesions included symptoms in eight patients, whereas two patients were asymptomatic at operation. These patients both had progressive asymptomatic common carotid artery stenoses outside the original endarterectomy site.

Neurologic symptoms developed during follow-up in four of the 13 patients who had residual disease (one transient and three permanent) and in nine of the 22 patients who had recurrent lesions (six transient and three permanent). Development of neurologic symptoms with restenosis was not related to the patients' neurologic status before the initial endarterectomy. With one exception, all neurologic symptoms were ipsilateral to the residual or recurrent lesion. The late stroke rate was increased in patients who had residual or recurrent lesions compared with those who had no significant ipsilateral disease after carotid endarterectomy (18% vs 6% at 5 years), although this did not reach statistical significance ($p = 0.16$ by LEE-DESU).

To determine the complication rate after carotid reoperation, we examined our entire experience with secondary carotid operations. During this time period, a total of 15 operations were performed for recurrent carotid stenosis, including five in patients whose primary procedure was performed elsewhere. There were a total of three neurologic deficits (20%); two were permanent, and one resolved within 30 days. All three of these lesions were consistent with a watershed infarction on computed tomographic examination, and patency of the carotid artery was documented by angiogram. In two of these patients perioperative hypotension was documented, whereas the third patient was having crescendo transient ischemic attacks and could not be shunted. There were two patients who had evidence of cranial nerve dysfunction after reoperation (12.5%). In both cases, this dysfunction involved dysphagia and eventually resolved.

Table III. Correlation between potential risk factors and recurrent stenosis

Factors	*Rate of restenosis*	*p*
Male gender	5.9% (11 of 155)	0.59
Female gender	7.4% (12 of 150)	
Primary closure	6.6% (5 of 76)	0.98
Patch closure	6.6% (18 of 271)	
Smoker	10.4% (14 of 134)	0.04
Nonsmoker	4.5% (8 of 178)	

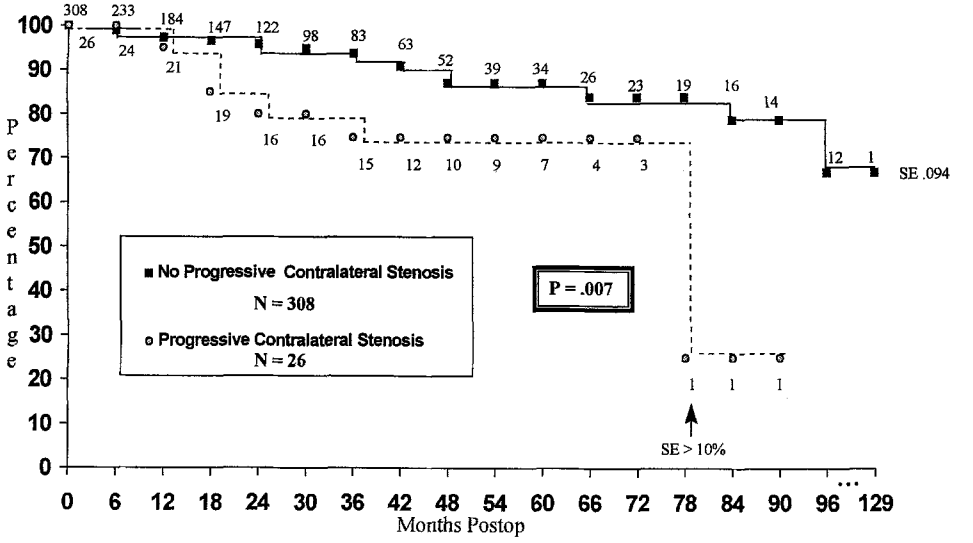

Fig. 2. Restenosis-free rate after carotid endarterectomy in 308 patients without progressive contralateral disease compared with 26 patients with contralateral progressive disease during follow-up is statistically significant (6% at 3 years vs 25.3% at 3 and 5 years; $p = 0.007$).

DISCUSSION

Carotid endarterectomy has been established as the gold standard for treatment of severe bifurcation stenosis on the basis of the results of prospective randomized trials.[11-14] Although safe and highly successful, this operation is clearly associated with a small but real incidence of residual or recurrent stenosis. The incidence of such lesions has been estimated between 6% and 18% by various authors and depends on whether stenosis is detected by angiography or duplex scanning and whether routine follow-up of asymptomatic patients after CEA is done.[6-10,15-18] Those who report the highest incidence of abnormalities base their conclusions on disturbances in Doppler flow velocity,[16] about half of which normalize over time. Reoperation for ipsilateral symptoms associated with recurrent stenosis after carotid endarterectomy is considerably less frequent than asymptomatic stenosis; the incidence approximates 1% to 2%. In our own series of 348 patients, there were eight reoperations for symptoms (2.3%). This has led most clinicians to the conclusion that the majority of recurrent stenoses will remain asymptomatic, particularly if they are the result of neointimal hyperplasia. In 1992,[7] we reported our initial observations on recurrent and residual stenoses and suggested that the majority of lesions could be followed-up for prolonged periods without operation, a position supported by Sumner and others.[8-10] Other investigators have been more liberal in their surgical indications and advocate reoperation for severe lesions (>80% diameter reduction) even when asymptomatic.[4-6,19] This issue is of some importance because the risk of reoperation has been reported by some, but not others, to be increased over that of primary operation. In 1985, Das et al.[1] from the Cleveland Clinic reported on 65 carotid reoperations with a combined stroke and death rate of 4.6% and an incidence of cranial nerve deficits of 9.2%. Shortly thereafter, Piepgras et al.[2] from the Mayo Clinic reported 57 reoperations in 51 patients with an operative complication rate of 10.5% (1986), which was a fourfold increase over their complication rate for primary operation. In a more recent survey by the Southern Association for Vascular Surgery, Rosenthal et al.[20] identified 31 patients who underwent secondary reoperation without stroke or death, but with a 10% rate of peripheral nerve injury. Contemporary series from New England Medical Center,[4,21] Dallas,[6] and New York[5] have reported morbidity rates that approach those of primary operation. Most recently, the issue has been further complicated by assertions that recurrent stenosis is an indication for endovascular therapy. Initial reports with angioplasty alone have been disappointing, with complication rates as high as 33%, but more recent reports of angioplasty with stent placement are more encouraging.[22-24]

There is a lack of objective data on the long-term follow-up of patients with recurrent carotid stenoses. Such data are important to allow informed decisions as to when intervention for recurrent stenosis is indi-

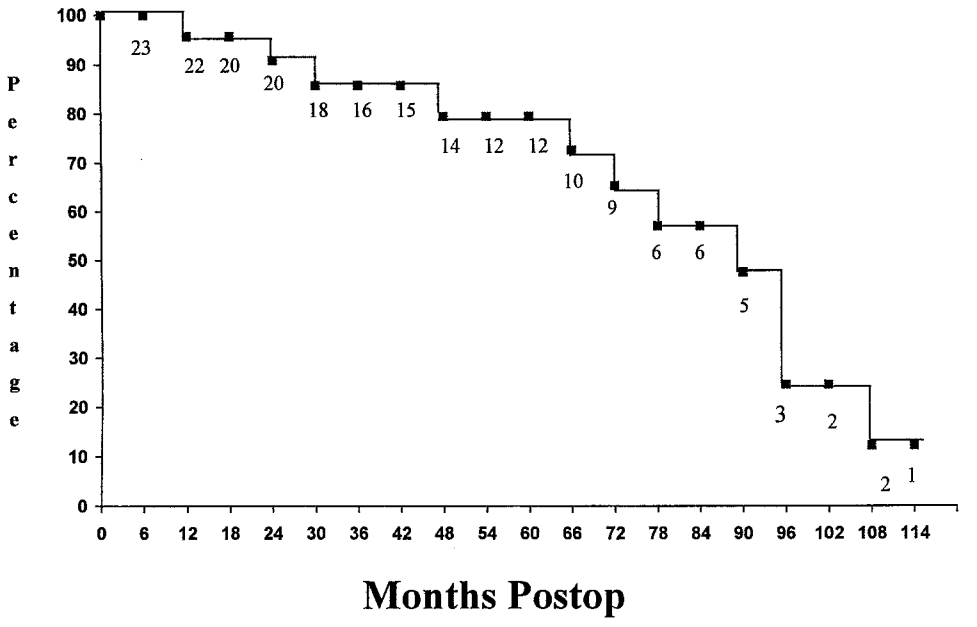

Fig. 3. Interval to reoperation in 23 patients with recurrent stenosis reveals 21% reoperation rate at 5 years. Although the numbers of patients followed-up more than 5 years is small, the slope of the curve suggests progressive incidence of reoperation after prolonged follow-up. Eventually, 48% of our patients required reoperation.

cated, as well as to provide a benchmark for operation and endovascular therapy. For many years our initial approach to residual and recurrent lesions has been nonoperative, and this was reinforced by our earlier review.[7] The purpose of this report was to provide prolonged follow-up of a cohort with significant lesions that were managed conservatively by a single surgeon. We specifically wished to address the frequency of such lesions, the rate of late reoperation and its complications, and the incidence of late symptoms in patients with residual or recurrent disease. Our study group included 78.3% of our total population (347 of 443). It is important to remember that of the 95 patients excluded, 81 had clinical follow-up but were excluded because of the lack of duplex data. This is an acknowledged problem of retrospective reviews over long periods of time and always raises questions concerning the accuracy of our conclusions. In an effort to answer these concerns, we compared the groups with and without follow-up. These comparisons merit some comment. Male gender and smoking were more common in patients who did not have follow-up. The association between smoking and restenosis suggests that the incidence of restenosis may be understated by our study population. Perioperative stroke was seen in 10% of patients without follow-up, most of whom underwent operation before 1988. This probably reflects more residual lesions in the group without follow-up. However, if patients after 1987 are compared, there is no difference in perioperative stroke rate between patients who were excluded for lack of duplex follow-up and those in the study group. This suggests that our data on residual lesions reflect contemporary results. These issues are of only secondary importance to the focus of this paper, which is on the long-term results of nonoperative treatment in patients who have proven lesions. In this regard, the long-term stroke rate in the 81 patients who were excluded for lack of duplex examination but had clinical follow-up is important. There was no difference in the late stroke rate in patients with and without duplex follow-up ($p = 0.95$ by life table). This provides assurance that the major conclusion of our studies are valid. We believe that these analyses suggest that the basic conclusions of our study were not altered by the lack of complete follow-up in all patients.

The incidence of residual lesions after carotid endarterectomy was 3.7%, which is not significantly different from that in our earlier report. These lesions were found at both the proximal and distal ends of the endarterectomy. In an attempt to decrease the incidence of residual proximal disease, we have more recently extended our endarterectomy more proximally in the common carotid artery, even to the point of a subclavian-to-carotid bypass graft in one

instance. The problem of proximal carotid plaque has been noted by several authors.[25,26] Although we have not used the modification suggested by Archie[26] to address this problem, it appears to have merit. As recently reported, we do not routinely use completion imaging after endarterectomy.[27] Our own data suggest that the occurrence of residual lesions was associated primarily with long or difficult endarterectomy procedures and is unlikely to be affected by routine imaging.[27] Several authors suggest that routine completion imaging would reduce the incidence of both residual and recurrent disease.[28,29] We are unable to determine whether routine imaging would have further reduced our overall incidence of recurrent disease, which was seen in 6.6% of patients and compares with that reported in the literature, but we believe that this is the best argument available for completion imaging.

Factors that have been suggested to be associated with an increased incidence of recurrent stenosis include smoking, female gender, and primary closure.[7,30,31] We did not find any association with gender or primary closure, although our high incidence of patch closure (77.5%) may have precluded detecting such a relationship. The lack of gender effect that has been reported by others is probably a result of the very frequent use of patch closure, which has been advocated in women. A history of smoking was associated with an increased incidence of recurrent disease. Along with the strong association between recurrent stenosis and the progression of contralateral disease ($p < 0.007$), this supports the premise that uncontrolled progressive atherosclerosis is a common denominator in patients who have recurrent lesions. One other report, by Cantelmo et al.,[32] noted an increased incidence of restenosis in patients who had bilateral disease, which supports our observations. Our data showed a progressive risk of restenosis over time, with many patients requiring reoperation after being observed for recurrent lesions after follow-up of more than 5 years. These data emphasize the importance of late follow-up in patients with recurrent disease and point to the role played by progressive atherosclerosis rather than intimal hyperplasia in the development of symptomatic lesions.

Our policy of watchful waiting appeared justified during the early follow-up of our patients; only one residual lesion required operation, and 79% of recurrent lesions could be observed for 5 years after development (life table analysis). This finding was consistent with our earlier published observations. However, symptoms eventually developed in 39% of our patients on prolonged follow-up, and 47% (11 of 23) ultimately required reoperation. These data are the first to document the extended natural history of recurrent stenosis in a cohort of patients in whom recurrent stenosis is identified by prospective routine screening. Our data indicate that more than one third of these lesions will eventually become symptomatic. This high incidence of late symptoms from recurrent stenosis is also consistent with the hypothesis that most of these lesions that become symptomatic represent recurrent atherosclerosis rather than intimal hyperplasia. The increased late stroke risk in this group of patients suggests that we may have been too conservative in recommending reoperation to these patients.

Our data bear some relevance to the current debate over the appropriateness of carotid angioplasty and stenting in patients in whom recurrent stenosis develops. The low reoperation rate at 3 and 5 years again emphasizes that most lesions, particularly early ones, will remain asymptomatic in the near term. We believe that early intervention in asymptomatic patients should not be undertaken lightly, particularly in high-risk patients who may have a limited life expectancy. This is particularly true when an investigational technique with unknown durability is used. Our personal results with recurrent operation are sobering, although the absolute number of operations is not large. We believe that these results must be interpreted in light of our strict criterion for reoperation, where 13 of 16 lesions were symptomatic (including one patient with crescendo transient ischemic attacks). In our patients, postoperative neurologic complications were related to hypoperfusion and not occlusion of the endarterectomy site. Results of larger contemporary series reported by others[4,5,6,20,21] indicate that reoperation can be undertaken with considerably less risk than was reported in the mid-1980s and than was seen in our own series. Future evaluation of treatment for recurrent stenosis should compare angioplasty with carotid reoperation as currently performed, rather than reference series reported a decade ago. Furthermore, because of the early benign course of this condition, any evaluation of therapeutic intervention for recurrent carotid stenosis should include a control group of patients who do not undergo operation.

What, then, can one conclude about the appropriate treatment of recurrent carotid disease? Clearly, the efforts to prevent this problem must continue to be emphasized. These include extensive endarterectomy, liberal use of patch closure, and aggressive risk factor modification to retard the progression of ath-

erosclerosis. Performance of a technically flawless operation also appears to influence the incidence of late recurrence, and any methods that serve that end, including completion imaging, are appropriate. As indicated in our prior publication, we believe that periodic duplex follow-up to detect recurrent stenoses is appropriate—currently, patients who have normal study results are followed-up every 2 years. Those in whom abnormalities develop are followed-up every 6 months. If recurrent lesions occur, one must remember that the majority of these lesions will remain asymptomatic for 5 years or more. Therefore, routine repair of such lesions does not appear to be justified. Asymptomatic recurrent stenoses in patients who have multiple medical comorbidities or a limited life expectancy can be observed, as most will remain asymptomatic. Similarly, asymptomatic recurrences that would present particular technical challenges (i.e., high lesions, multiple recurrences) need not be repaired unless symptoms supervene. However, our initial reluctance to reoperate on any asymptomatic lesion appears unjustified, particularly in view of the increased late stroke risk in these patients and the fact that operation was ultimately required in almost half of our patient group. We now consider reoperation for asymptomatic recurrent stenoses for good-risk patients who have a significant life expectancy. This should receive particular consideration if recurrence is in the proximal common carotid artery. The role of endovascular therapy in this disease remains to be defined by controlled trials.

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Submitted May 2, 1997; accepted Aug. 5, 1997.

DISCUSSION

Dr. Robert W. Hobson II (Newark, N.J.). Dr. Ricotta has reviewed data on 348 carotid endarterectomy procedures performed between 1982 and 1996. I would caution you that although the data are studied with great care, the number of recurrent lesions is relatively small. There were 13 residual lesions (an incidence of 3.7%) and 23 restenotic lesions (an incidence of 6.6%). In addition, the definition in the manuscript of residual lesion included those patients with stenoses identified up to 1 year after operation. Is it possible that some of the residual lesions actually were early myointimal hyperplastic lesions rather than residual lesions? And if that's possible, how would it have affected your analysis?

Secondly, a residual lesion may be of significance in terms of subsequent development of stroke. Consequently, I would like your opinion with regard to its identification during the operation. What are your recommendations on the use of imaging techniques or intraoperative angiography to identify a residual lesion, thereby allowing intraoperative correction rather than delayed management?

Another area of interest was the restenotic lesion. They are of great interest to us, and your data regarding gender and patching were useful. I would be interested in your comments on recommendations for use of carotid patches. What patches are you currently using? Three quarters of your patients were patched, and yet you report a substantial number of restenoses. What was the distribution of restenoses in the patched group versus those closed primarily?

One of the most interesting aspects of this paper was the progression of contralateral disease identified in the group with restenosis. This observation has not been well documented in the literature. What are your thoughts as to the etiologic mechanism? How are ipsilateral restenoses and atherosclerotic progression in a contralateral artery related?

Finally, because the incidence of cranial nerve palsies after operations for restenosis remain higher than reported for initial procedures, is carotid angioplasty and stenting an option in managing these patients?

I enjoyed this paper. It presents a number of important issues that will stimulate additional study.

Dr. John J. Ricotta. I agree with your first point about early recurrence versus restenosis. Because this was a long time period and we didn't have a routine duplex protocol in the early part of the study, we had to make some arbitrary distinctions. It is certainly possible that some of these residual lesions may have been early recurrences. That is part of the reason that when I looked at the late stroke rate I tried to group those two lesions together. It had to be the patient's first postoperative duplex scan that was abnormal. Now, unfortunately, there were a couple of patients who didn't undergo their first postoperative duplex scan until 6 to 8 months after the operation. Because they had a lesion, I didn't know where to put them, so I made this definition.

I think your point about identifying these lesions during the operation is extremely important. Now we don't perform routine imaging. I can tell you, I went back and looked at the operation notes on my patients who had these internal lesions because I was very concerned about this. These were all people who had very high lesions. My approach has been that if I have done the best I can do, I'm not sure I want to see an angiogram because it can't be improved. I mean, many of these stenoses were up above the hypoglossal nerve, and further repair would be difficult. I have a couple of patients in whom I have performed saphenous vein interposition and have had a stenosis distally. And I think that that's a technical problem. We're a little bit comforted by the fact that they haven't been symptomatic, but certainly it's disturbing to have these lesions.

In the common carotid lesion, I don't think you're going to do anything with completion imaging because it's going to be below where you're going to do your angiogram. However, the data that Dr. Bandyk mentioned relating residual disease and the development of late recurrence is concerning to me. It may be that that's probably the best reason to do some sort of a completion study, whether it's a duplex scan or an angiogram. If you can reduce your rate of recurrence stenoses by performing duplex surveillance in the operating room, that may be useful. Our rate is about what everybody else's rate is, but certainly at 6.6% there is room for improvement.

We currently use external jugular vein preferentially. I will use Hemashield. There was some polytetrafluoroethylene used in this study. We could not correlate patch material with rate of restenosis, but it wasn't a prospective, randomized study. And as you know, Dr. AbuRahma has done a study like that and reported it here. There was a lower incidence of restenosis in the patched group. I think Dr. Green and several other people have reported that there is a lower incidence of restenosis in patching. This data set would not answer that question because my practice progressed in a way where most of my unpatched patients were early in my practice and most of my patched patients were later, and I hope I learned how to do an endarterectomy better as I got more mature. So I can't make any comments about my own data.

I think that myointimal hyperplasia is less important than progressive atherosclerosis, and I think our data with contralateral progression confirmed my prejudice that the people who were coming back, at least for symptomatic recurrences, were people who had very, very aggressive atherosclerosis. I think we've got to work on keeping these people from smoking, we've got to work on their lipids and that sort of business, and I think we don't pay enough attention to that.

I have sent one patient for angioplasty with stenting, and it is a patient with a recurrent carotid stenosis. It's a second-time recurrence that is very high and very severe. I thought that this patient might benefit. I think that if endovascular procedures are going to be useful it will be in this type of patient. My concern is that they not be routinely recommended in asymptomatic patients with recurrence. I think that we need to know what the rate of complications are both for surgery and for endovascular procedures before we recommend them as a routine procedure in asymptomatic patients because so many of these people with recurrent stenosis will in fact remain asymptomatic for a number of years.

Dr. Luis A. Queral (Baltimore, Md.). In spite of the presented large series, the number of recurrent carotid stenoses is quite small. This would explain why the rest of us see relatively small numbers of these patients. Consequently, it's hard to decide what to do with the patients. However, when I note the high incidence of cranial nerve palsies and an 18% stroke rate when operating on these patients, an alternative endovascular approach has to be considered. This new approach would encompass a small surgical cutdown in the cervical common carotid artery and treatment of the offending lesion with minimal dissection.

I am troubled by the small number of these patients and find it difficult to determine whether managing the recurrences using a standard surgical technique or by using an endovascular approach is optimal. I would like your comments on this matter, because it appears to me that this issue will not be resolved unless a multicenter prospective cooperative study can be instituted.

Dr. Ricotta. I would agree with you, Dr. Queral. I think that's the only way that it's going to be accomplished. The largest series that I could find in the literature on this was Dr. Clagett's series of 70 patients. So there is nobody who had a big series of patients who has reported on it.

I think that the questions are exactly as you outlined them. Is endovascular therapy better than surgery? And I would add a third arm, and that is observing the patients who are asymptomatic. So I think ultimately, if we're going to decide truly the right way to take care of these patients, that we need to compare the two forms of intervention in symptomatic patients, and then in asymptomatic patients compare those two forms of intervention with observation.

The interest that we had here was that we were committed to not operating on these patients. I wanted to see what would happen. There is not a lot in the literature to tell us what the natural history of these lesions is if you are committed to observe them for long periods of time. Most of the series, even though I'm sure there are patients that have been observed, haven't reported separately on those patients. Maybe this is something that the Registry Committee in this Society can do. If we got the collective experience in a prospective fashion with recurrent stenoses of 500 people that perform vascular surgery, or even got half of those people, to randomize their patients into various treatment options, we may be able to add something to a literature that has no series of a hundred patients in it.

Dr. Paul Gagne (Chesapeake, Va.). I have a quick question for Dr. Ricotta. Many of the series on recurrent stenosis evaluate all stenotic lesions >50%. I was wondering whether you got a sense in this study whether the stenotic lesions that were more midrange, 50% to 79%, say, behaved differently than lesions that were more high-grade?

Dr. Ricotta. I think in our first report, we found that none of the moderate lesions led to symptoms. Now, subsequently we've had a couple of patients who started out with a moderate lesion and eventually had symptoms. There are only two or three of them, and I have to be honest with you, I can't tell you whether they had progressed when the symptoms developed or not. My prejudice is that that's probably what was happening, that they had a moderate lesion, we followed them up for a prolonged period of time, and they showed some progression, and it would fit with the thesis of atherosclerosis.

The real question to me is when you have an 80% lesion, do you watch it or do you intervene? I'm not convinced that my policy of watching is always the right thing to do, so I'm more inclined now to intervene in some way, whether it's endovascular or operative.

Dr. Richard M. Green (Rochester, N.Y.). I enjoyed your paper, and it sounds to me that you started out with a very conservative approach and have moved to an aggressive approach, which I would agree with. We have been struck over the past few years that an inordinate number of these cases are neither atherosclerotic or intimal hyperplastic, but they are this florid thrombotic process that occurs in the endarterectomized bulb. For that reason, we have

been very aggressive in treating these patients, not with conventional reendarterectomy, but with exclusion bypass grafting. By approaching the carotid artery very low and very high through a lateral approach, the original operative site is not disturbed, and the number of emboli and cranial nerve palsies have been reduced. So I would caution that (1) I think recurrent carotid disease is significant; (2) a number of the lesions are gross thrombus, and one should not treat them with endovascular procedures; and (3) an aggressive approach is warranted.

Dr. Enrico Ascher (Brooklyn, N.Y.). I very much enjoyed your presentation. I have a couple of questions that maybe you can clarify for me. Where is the exact site of the restenosis? Is it the bulb? Is it distal internal carotid artery? Is it in the proximal internal carotid artery? Because that may have some clinical implications. If it's in the common carotid artery, it may not be as bad to just watch them, even with severe stenosis. We really don't know what the natural history of these lesions is. Certainly, in the bulb we may have some clinical implications in terms of using a patch or not. I have noticed that those that I have seen in terms of restenosis have been right at the bulb, and these patients have had patches before. So I'm not so sure the patch is as protective as we think.

My second question relates to residual problems. Is that a residual plaque, or this is a stenosis repair that was performed by poor technique? Maybe the natural history of those lesions is quite different, and I wonder whether you can shed some light on these questions.

Dr. Ricotta. First, Dr. Green, I agree with you. We've started to do some interposition grafting the last couple that I've done. The one problem is that it is very difficult to use a shunt in those patients, at least in my experience, when you do that. In fact, the patient who had the deficit had crescendo transient ischemic attacks, and we tried to figure out a way to put a shunt in and we couldn't. We used an interposition graft in him, and we put the shunt in once we'd gotten the distal anastomosis done through the vein graft, but it was a problem. So I think that's a very good technique, and I've used it. I think it simplifies the operation.

Dr. Ascher, the stenoses were widely distributed. I thought I was going to find them all in one place, and I didn't. There is no question that there are some patients who had a distal stenosis where either I didn't sew it in correctly or there was a clamp problem distally. But there is no question that about a third of the patients had a stenosis at the distal end point. Now, some of those patients were not patched, and I think that patch helps there. There were some bifurcation stenoses, and I think that this has to do with what Dr. Green is talking about, and it may be that a big patch may be a problem because it may collect thrombus and may stimulate hyperplasia. There were a few people with proximal common carotid lesions.

Now, I would suggest to you that a proximal common carotid lesion is a big problem. I think Drs. Green and Ouriel reported this at the national meetings about 6 or 7 years ago, in which they followed-up a number of these patients and they had a very high incidence of recurrent stenoses. So if I see a proximal lesion that I think is atherosclerotic, I'm very aggressive about going back to get that. And I wouldn't leave the proximal lesions alone. I would probably fix the middle lesions. What you do with the distal lesions, I think, is sometimes very difficult because in our situation we've already gone usually as high as we can go without subluxation of the mandible and that sort of business, and I am very hesitant in an asymptomatic patient to go back and reoperate on those. Performing balloon angioplasty in those patients and putting a stent in may be correct.