Clinical follow-up rather than duplex surveillance after carotid endarterectomy

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Purpose: The value of duplex surveillance and the significance of contralateral carotid disease after endarterectomy have been assessed.

Methods: Three hundred five patients were observed prospectively after carotid endarterectomy for a median time of 36 months (range, 6 to 96 months), with duplex surveillance performed at 1 day; 1 week; 3, 6, 9, and 12 months; and then each year after endarterectomy.

Results: Thirty patients (10%) had ipsilateral symptoms (13 strokes, 17 transient ischemic attacks [TIAs]) at a median time of 6 months (range, 0 to 60 months). Life table analysis demonstrated that ipsilateral stroke was equally common for patients who had ≥50% restenosis (3% at 36 months) and those who did not (6% at 36 months, $p > 0.5$). Twenty-three patients (8%) developed symptoms (stroke 5, TIA 14) attributable to the contralateral carotid artery at a median time of 9 months (range, 0 to 36 months) after endarterectomy. By life table analysis, 40% of patients with 70% to 99%, 6% with 50% to 69%, 1% with <50% contralateral internal carotid stenosis, and 5% with contralateral carotid occlusion at the time of endarterectomy had a contralateral TIA in the 36 months after endarterectomy ($p < 0.01$). However, contralateral stroke was not significantly more common for patients with severe contralateral internal carotid stenosis demonstrated at the time of endarterectomy (<50% stenosis, 0%; 50% to 69%, 3%; 70% to 99%, 7%; occlusion, 6% stroke rate at 36 months). Seven of the 32 patients who developed progression of contralateral disease had a TIA, compared with 11 of 227 patients who did not develop progression of contralateral disease ($p < 0.01$). None of the 12 patients who progressed from a <70% to a 70% to 99% contralateral stenosis had a stroke.

Conclusions: After carotid endarterectomy restenosis is rarely associated with symptoms; contralateral stroke is rare and is not associated with progressive internal carotid artery disease suitable for endarterectomy. This study has shown no benefit from long-term duplex surveillance after carotid endarterectomy. Selective clinical follow-up of patients who have high-grade contralateral stenoses would appear more appropriate. (J Vasc Surg 1997;25:55-63.)

Duplex surveillance after carotid endarterectomy has become a common practice.[1] Color-coded duplex imaging accurately visualizes the carotid bifurcation and allows both development of restenosis in the operated carotid artery and progression of disease in the contralateral carotid artery to be assessed.[1,2] However, although restenosis is a common finding in surveillance series, it is rarely associated with the development of symptoms.[3,4]

If surveillance is not warranted simply to demonstrate restenosis, it may have a role in monitoring the development of disease in the contralateral internal carotid artery.[5] This study assesses the results of an 8-year program of duplex surveillance after carotid endarterectomy to define which patients might benefit from this type of follow-up.

PATIENTS AND METHODS

Patients were assessed before surgery by both a vascular surgeon and a neurologist. Investigations included color-coded duplex imaging, aortic arch angiography, and brain computed tomography. There was good agreement between the severity of carotid stenosis defined on preoperative angio-

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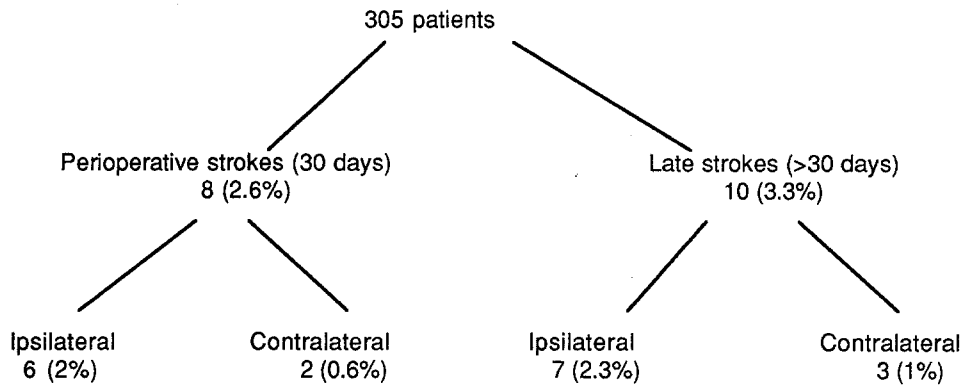


Fig. 1. Stroke after carotid endarterectomy, with reference to side and time after carotid endarterectomy.

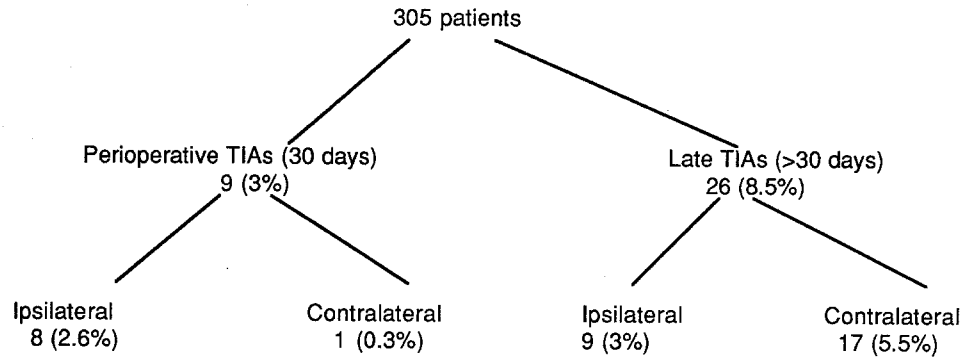


Fig. 2. TIA after carotid endarterectomy, with reference to side and time after carotid endarterectomy.

graphic and duplex scans. Carotid endarterectomy was performed as previously described with selective shunting when stump pressure was less than 50 mm Hg, and selective 5 mm Dacron patch repair for small internal carotid arteries.[6] All operations were performed or supervised by the senior author.

Since 1987 extensive duplex surveillance has been performed after carotid endarterectomy. If any neurologic symptoms developed during the first 24 hours, an urgent duplex study was undertaken and operative intervention planned.[7] Otherwise, the patient was routinely scanned at 1 day; 1 week; 3, 6, 9, and 12 months; and then each year.[8] Patients were seen at the follow-up clinic at the same time and were questioned with respect to neurologic symptoms. Patients were followed-up for a median of 36 months (range, 6 to 96 months).

Progression of contralateral internal carotid artery disease was assessed by duplex using Doppler frequency shift and B-mode imaging (Ultramark 9, HDI, Advanced Technology Laboratories, Bothell, Wash.; and Acuson 128, Mountain View, Calif.) to define the degree of stenosis. Significant progression of disease was defined as progression to >50% stenoses for stenoses originally <50%, progression to >70% for stenoses originally 50% to 70%, and occlusion for stenoses originally >70%. Significant restenosis of the operated carotid was defined as ≥50% stenosis.

In the event of new neurologic symptoms developing (stroke or transient ischemic attack [TIA]), patients were reassessed by a neurologist in light of their duplex surveillance findings and a decision made regarding contralateral carotid endarterectomy. In general, patients were advised to undergo contralateral carotid endarterectomy in the event of a TIA in association with a related 70% to 99% carotid stenosis. Asymptomatic carotid stenoses were treated conservatively. Data were stored on Dbase 4 (Borland, Scotts Valley) and statistical analysis carried out on Statview (Abacus Concepts, Berkeley).

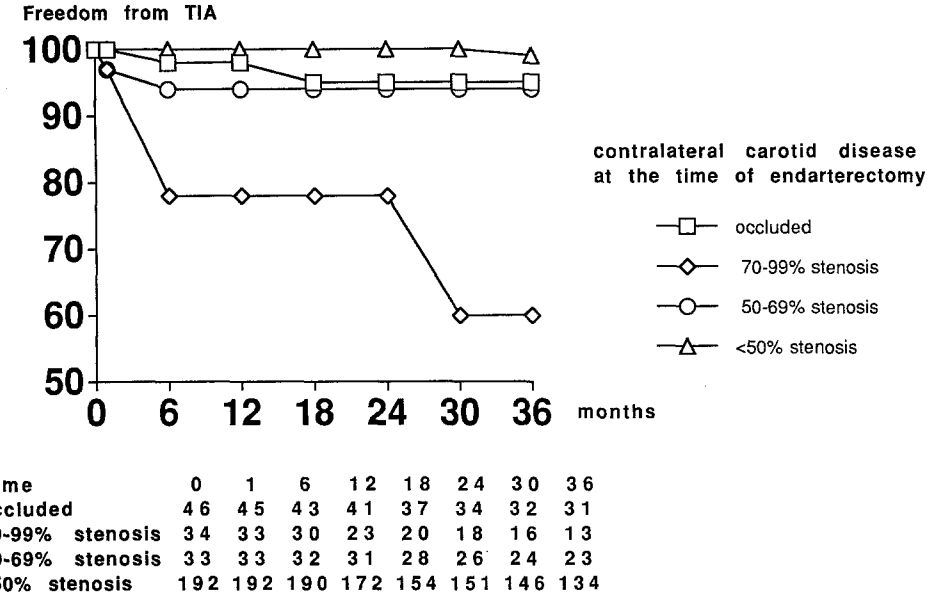


Fig. 3. Contralateral TIA in relation to contralateral internal carotid disease demonstrated at the time of endarterectomy. The y axis has been shortened to concentrate on the region in which TIAs occurred.

RESULTS

Stroke after carotid endarterectomy. Three hundred five patients underwent consecutive carotid endarterectomy procedures and were eligible for duplex surveillance. There were eight (2.6%) perioperative strokes (within 30 days of operation); six (2%) were fatal, and two (0.6%) were in the territory supplied by the contralateral carotid artery (Fig. 1). One of the these patients had a 99% contralateral internal carotid stenosis and the other a 50% stenosis.

Ten patients (3.3%) had a stroke after 30 days; seven (2.3%) were ipsilateral (one associated with a 60% restenosis at 8 months) and three (1%) were contralateral, two associated with contralateral internal carotid occlusion (at 24 and 36 months) and one associated with a 90% internal carotid stenosis (at 24 months).

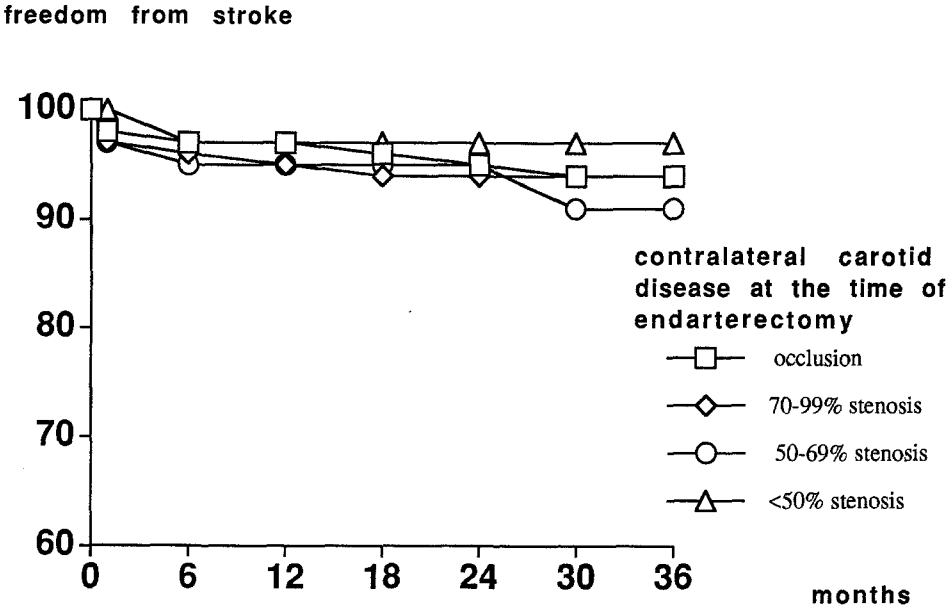
TIAs after carotid endarterectomy. Nine patients (3%) had a perioperative TIA, eight (2.6%) ipsilateral (1 associated with a 65% residual stenosis) and one (0.3%) contralateral associated with a 90% internal carotid stenosis (Fig. 2). Twenty-six patients (8.5%) had a TIA after 30 days, nine (3%) ipsilateral (2 associated with restenosis) and 17 contralateral (11 associated with 70% to 99% internal carotid stenosis).

Effect of contralateral internal carotid disease at the time of endarterectomy on later development of contralateral symptoms. Preoperative color-coded duplex imaging revealed disease of the internal carotid artery contralateral to that operated on in 113 (37%) patients. Thirty-three patients had 50% to 70% stenoses, 34 patients had 70% to 99% stenoses, and 46 had occluded contralateral internal carotid arteries. The risk of contralateral TIA developing is clearly related to the degree of internal carotid stenosis measured before ipsilateral carotid endarter-

Table I. Features of internal carotid disease in patients who developed contralateral symptoms after ipsilateral carotid endarterectomy

	Developed symptoms		*Not develop symptoms*
	TIA	*stroke*	
Contralateral IC disease at time of ipsilateral CEA (n = 305):			
70% to 99% contralateral IC stenosis	12	2	20
90% to 99% contralateral IC stenosis	7	2	16
<70% contralateral IC stenosis	4	1	220
Occluded contralateral IC	2	2	42
Progression of IC disease (n = 32):			
Progression from <50% to 50% to 69%	1	0	13
Occlusion	3	1	2
Progression from <70% to 70% to 99% stenosis	3	0	9

IC, Internal carotid artery; *CEA,* carotid endarterectomy.



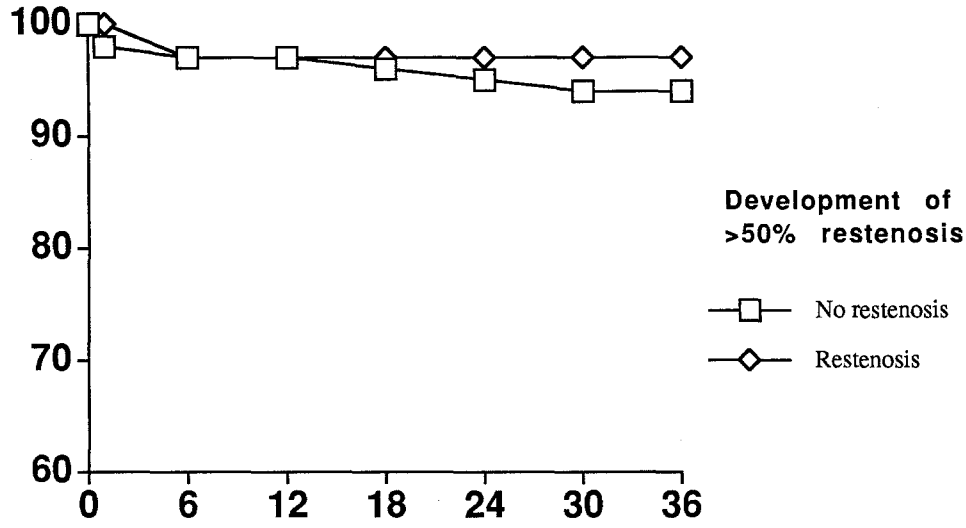
Numbers at risk								
Time	0	1	6	12	18	24	30	36
occluded	46	45	44	42	38	36	33	31
70-99% stenosis	34	33	30	29	26	24	22	20
50-69% stenosis	33	33	31	31	29	27	25	23
<50% stenosis	192	192	190	172	154	151	146	136

Fig. 4. Contralateral stroke in relation to contralateral internal carotid disease demonstrated at the time of endarterectomy. The y axis has been shortened to concentrate on the region in which strokes occurred.

ectomy (Fig. 3; Table I). However, only two of the 305 patients (0.7%) had a stroke without warning associated with a 70% to 99% contralateral internal carotid stenosis, and one of these occurred on the day of operation. There was no association between contralateral stroke and degree of internal carotid stenosis demonstrated at the time of ipsilateral carotid endarterectomy (Fig. 4). Most of the patients who developed symptoms (12 of 23) had TIAs in association with a 70% to 99% stenosis and therefore underwent contralateral endarterectomy.

Effect of progression of contralateral internal carotid disease on the development of contralateral symptoms. Significant progression of contralateral internal carotid disease occurred in 32 patients (10%) at a rate of around 3% per year. Twelve of the 225 patients (5%) with a <70% contralateral internal carotid stenosis at the time of endarterectomy progressed to >70% stenosis. Three of these patients developed symptoms, TIA in all cases (Table 1). Seven of the patients who had progression of contralateral disease (occlusion in three) had a contralateral TIA, compared with 11 patients who did not have progression of contralateral carotid disease ($\chi^2 = 12.8$, df = 1, $p < 0.01$). The three patients who progressed to 70% to 99% stenosis and had a TIA underwent contralateral carotid endarterectomy (Table 1). Two of these patients had a 60% contralateral internal carotid stenosis at the time of ipsilateral endarterectomy.

Ipsilateral symptoms and development of restenosis during follow-up. Four (stroke, 1; TIA, 3) of the 30 patients who developed ipsilateral symptoms had recurrent internal carotid stenosis ≥50% (1 residual, 3 restenoses). Thirty-two of the 36 patients (89%) who developed restenosis remained symptom-free. Life table analysis showed ipsilateral stroke (Fig. 5) to be equally common for patients who developed



Numbers at risk								
Time	0	1	6	12	18	24	30	36
restenosis	36	36	36	35	32	31	29	28
No restenosis	269	260	255	231	203	189	176	159

Fig. 5. Ipsilateral stroke in relation to the development of ≥50% restenosis. The y axis has been shortened to concentrate on the region in which strokes occurred.

restenosis (3% at 36 months) and those who did not (6% at 36 months, $p > 0.5$). Similarly, TIAs were equally common for both groups (Fig. 6).

DISCUSSION

Restenosis is common after carotid endarterectomy. Published restenosis rates vary between 1.5% and 20%[9,10]; however, studies that use duplex surveillance usually report in excess of 10%.[11] Symptomatic restenosis is rare. Mattos et al.[12] in follow-up of 409 endarterectomies, identified 10 symptomatic restenoses (2.4%) and reported an identical ipsilateral stroke-free period in patients without recurrent stenosis. Others have found rates of symptomatic restenosis of 1% to 4%.[13,14]

The overall rate of restenosis in this study was 11.8%, with symptomatic restenosis occurring in 1.3%. Ipsilateral stroke was more common in patients who did not have restenosis (6%) than in those who had restenosis (3%). Thus in keeping with other publications, this study has failed to show any association between the development of restenosis and ipsilateral symptoms.

Duplex surveillance also allows assessment of progression of contralateral carotid artery disease. We identified progression from <70% to 70% to 99% contralateral carotid stenosis in 12 patients, at a rate of around 1.5% per year. Three of these patients developed symptoms; however, this was a TIA in all cases. Therefore, careful clinical follow-up in these patients alone would have alerted the clinician to obtain further carotid duplex studies and then consider contralateral carotid endarterectomy.

Other investigators have similarly found duplex surveillance of the contralateral carotid artery after carotid endarterectomy to be of little potential benefit. Naylor et al.[15] performed duplex surveillance on 151 patients and identified progression to >70% contralateral stenosis in 10 (7%). Three of these patients had symptoms; however, as in our study this was TIA in all cases. Satiani and colleagues[16] studied 127 patients after carotid endarterectomy. Five patients were identified to develop progression of contralateral carotid stenosis to >80%. None of these patients had a stroke, but three had TIAs and could

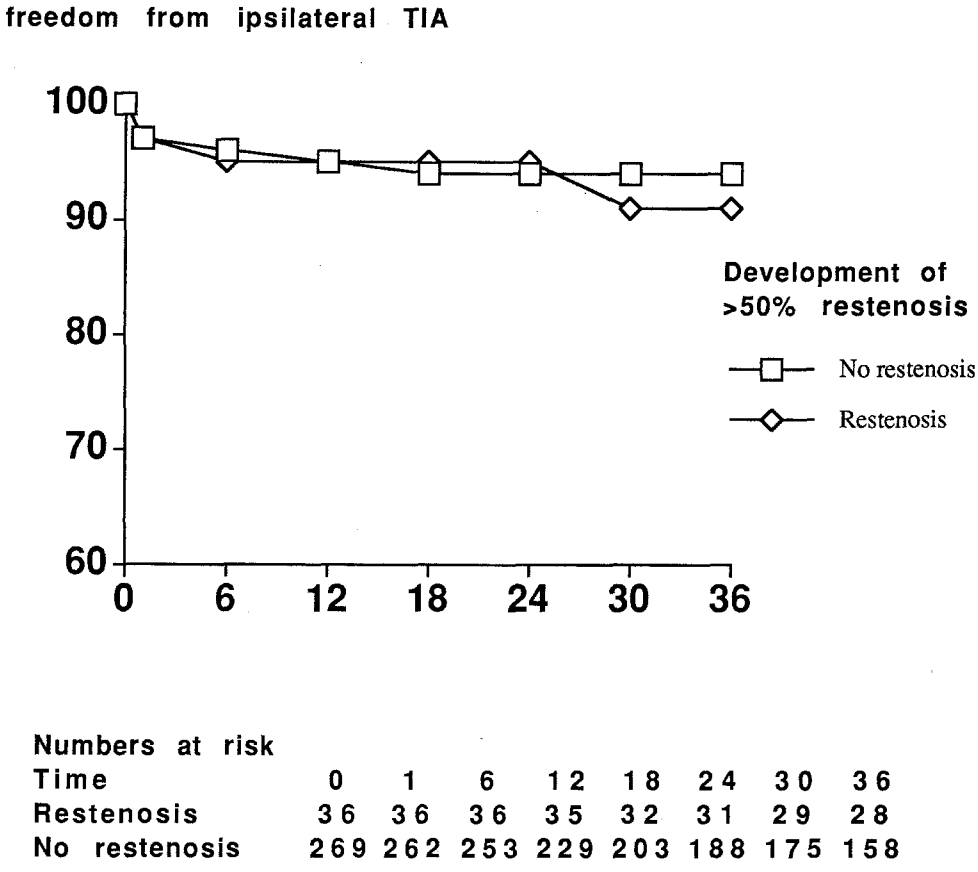


Fig. 6. Ipsilateral TIA in relation to the development of ≥50% or greater restenosis. The y axis has been shortened to concentrate on the region in which TIAs occurred.

be considered for contralateral carotid endarterectomy as symptomatic patients without having suffered a permanent neurologic loss.

The cost of long-term duplex surveillance is considerable. The price of a carotid duplex study has been estimated as £75.[17] Assuming an average of four duplex scans for around 100 patients, this equates to £30,000 per year. With both ipsilateral restenosis and contralateral progression of internal carotid artery disease having been demonstrated not to predict the development of stroke, the benefits of such outgoings are questionable. Selective clinical follow-up of patients with preexisting contralateral artery disease after endarterectomy, with duplex imaging obtained if symptoms develop, would be more appropriate. If follow-up had been restricted to patients with 50% to 99% contralateral stenosis, then this would have reduced follow-up numbers by 78% to 67 while still identifying eight of the nine patients who required endarterectomy.

A number of previous studies have examined the issue of performing contralateral endarterectomy for asymptomatic disease. Podore et al.[18] studied 67 patients over 5 years and found that symptoms principally developed in patients with preexisting contralateral disease and suggested that staged endarterectomy should be performed for patients with contralateral stenosis if the surgeon's perioperative stroke rate is less than 3%.[18] Schroeder et al.,[19] while also finding an association between preexisting contralateral disease and the subsequent development of contralateral symptoms, did not believe that this was sufficient to recommend prophylactic endarterectomy.[19] Others have suggested prophylactic surgery for severe asymptomatic stenoses if the surgical perioperative morbidity rate is low.[20-22] Calligaro et al.[23] studied the outcome of 58 patients who underwent prophylactic contralateral endarterectomy and reported a high incidence of hypertensive or hypotensive problems and a 3.4% perioperative stroke rate for the prophylactic procedure.

This study confirms that it is principally patients with preexisting severe internal carotid artery disease in whom contralateral TIA develop. Sixteen of the 18

patients who suffered a TIA had ≥60% contralateral internal carotid stenosis at the time of their endarterectomy. However, at this stage the patients were reassessed by a neurologist and the majority underwent carotid endarterectomy without having suffered any permanent neurologic deficit. It has not been our practise to operate on asymptomatic carotid stenoses. Despite this, only five patients (1.6%) had contralateral strokes at a median of 36 months follow-up. Two of these patients had a stroke on the day of their ipsilateral carotid endarterectomy, two had occluded contralateral internal carotid arteries, and one patient had a stroke during follow-up associated with a 70% to 99% stenosis. Thus only one of the 33 patients (3%) with 70% to 99% stenosis under surveillance had a stroke after 30 days. Given that the combined 30-day stroke-mortality rate in this study was 3%, it would not appear appropriate to offer prophylactic contralateral endarterectomy to this group.

Carotid endarterectomy performed for asymptomatic disease remains controversial. Preliminary results from the Asymptomatic Carotid Atherosclerosis Study have suggested a small benefit from carotid endarterectomy in asymptomatic patients with 60% to 99% internal carotid stenosis; however, after a median follow-up of 30 months the estimated reduction in stroke rate was only 1% per year.[24,25] Also, it is unclear how applicable these results are to contralateral carotid disease, because a contralateral carotid stenosis in a patient who has previously undergone carotid endarterectomy for a symptomatic stenosis may behave differently from an incidentally detected asymptomatic carotid stenosis.[26,27]

CONCLUSION

Progression of contralateral internal carotid artery disease is usually a slow process, thus symptoms principally develop in patients with severe preexisting contralateral disease. Restenosis is rarely associated with ipsilateral symptoms. On the basis of this study, there appears to be little patient benefit from long-term duplex surveillance after carotid endarterectomy. Selective clinical follow-up of patients with contralateral stenoses, with consideration of endarterectomy if symptoms develop, would appear appropriate.

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DISCUSSION

Dr. Thomas F. O'Donnell (Boston, Mass.). I will try to frame my comments in an SVS rather than SRS manner—most of those attending from the United Kingdom know the difference.

This paper addresses an increasingly important concern for health care systems in which resources are limited—the role of postoperative noninvasive surveillance. This expenditure is particularly relevant after carotid endarterectomy, which, once again, is the most frequently performed major vascular procedure. On the basis of a follow-up of 305 patients over a median time of 36 months, you have heard the authors conclude that there is no benefit from postoperative duplex surveillance. What is the evidence for this global conclusion?

For the operated side, of 320 patients in whom subsequent ipsilateral neurologic symptoms developed, four had stenoses >50%. Only the one stroke patient who had not had an antecedent TIA (about 0.3%) would have had a stroke prevented by duplex surveillance. This finding confirms an earlier 1992 paper authored by Bill Mackey from our group, as well as another study by Mattos and Sumner, both of which showed that duplex surveillance of the operated side was not cost-effective in stroke prevention as a result of the low incidence after carotid endarterectomy of patients who went directly to stroke without antecedent TIA.

Today's paper, however, is one of the few to examine the value of duplex surveillance for the nonoperated contralateral side. The authors found no relationship of stroke to the initial degree of stenosis or to stenosis progression. By contrast, the progression of stenosis was correlated with the development of TIAs. These symptomatic patients, however, would undergo intervention anyway without the need for detection by duplex. On closer examination of the manuscript, however, the authors failed to examine stroke risk with stratification by degrees of stenosis. In addition, the authors used χ^2 rather than life table analysis. The life table statistical method does not differentiate small changes. If life table analysis were used, one would see that there was a 6% incidence of progression in the 70% to 99% group, which is a figure identical to that observed in the ACAS study subgroup of carotid arteries contralateral to a carotid endarterectomy. Would the authors comment on the subgroup's higher risk and the possible implications if analyzed with stratification by degree of stenosis.

We recently presented a paper that specifically addressed the problem of the contralateral side. A similar number of patients were studied, but we arrived at different conclusions. Certainly a more cost-effective maneuver in any surveillance or screening test is to restrict its use to a narrower segment of the population who may be at a greater risk. Our study showed that the clinical factor of age greater than 65, combined with an initial degree of contralateral stenosis in the 50% to 74% range, increased the probability of progression to significant stenosis, that is, >75%, to nearly 49% over 5 years.

I have several questions for the authors. You did not perform any cost-benefit analysis; do you plan to do this? You did not look at the effect of demographic risk factors.

In addition, I have some simple questions on your duplex methods, which I found extremely sketchy in the manuscript, but which is certainly paramount to the interpretation of the paper. What criteria were used? You state spectral scan and B-mode morphologic criteria. Could you be more specific?

Also, two different instruments were used in this study. Were they used sequentially or contemporaneously? What were the peak systolic velocities and end diastolic velocities used? Were these criteria validated in your laboratory versus an arteriogram, as they have been in most ACAS labs? Did you observe any interinstrument variability, as suggested by several authors?

I enjoyed the manuscript, but would urge the authors not to throw the baby out with the bath water. Risk factor stratification with an initial degree of stenosis and other

factors will allow one to selectively conduct surveillance as hypothesized by the authors in the conclusion to their manuscript.

Dr. Jonathan Golledge. The same highly trained vascular technologists used either an Acuson or ATL color duplex scanner sequentially. We graded the severity of stenosis with peak systolic frequency criteria as follows: 50% stenosis, 3.8 KHz; 70% stenosis, 70 KHz; 80% stenosis, 9.0 KHz; 90% stenosis 12.5 KHz; and 99% stenosis, 16.0 KHz. We determined the presence of 1% to 15% and 16% to 49% stenoses by the presence of spectral broadening. Where arteries were seen to have acute angulation to the probe, velocity measurements were recorded and stenoses graded according to Strandness criteria.

The life table analysis method was used to assess any association between stroke or TIA and severity of contralateral carotid artery disease or the development of restenosis because it is the most appropriate statistical method. During follow-up some of the patients died of unrelated causes or were lost to follow-up, and the life table method takes this into account. A simple χ^2 analysis on part of the data is statistically unsound. A total of 34 patients had a 70% to 90% contralateral carotid artery stenosis at the time of carotid endarterectomy, and one of these patients had a stroke during follow-up, a stroke rate of 1.5% per year, a risk no higher than for the patients who had 50% to 69% stenoses in this study.

We did not perform a cost-benefit analysis because, as duplex surveillance failed to demonstrate anything predictive of stroke without warning, then clearly unnecessary duplex surveillance implies unnecessary cost. None of the 12 patients who had progression from <70% stenosis to 70% to 99% stenosis had a stroke, and three had TIAs. Instead of duplex, we emphasize the need for careful clinical follow-up.

In terms of the baby and the bath water and what to throw out, we need to be specific. "The baby" we perceive to be the need for intensive clinical follow-up for TIAs. "The bath water" is the unnecessary duplex surveillance, and can go.