Durability of carotid endarterectomy for treatment of symptomatic and asymptomatic stenoses

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Purpose: **Although many studies have well established that carotid endarterectomy (CEA) is beneficial in selected patients with severe carotid disease, only a few large studies have focused on the durability of the surgical procedure. Carotid artery angioplasty and stenting (CAS) has recently been proposed as a potential alternative to CEA. We analyzed the incidence of late occlusion and recurrent stenosis after CEA.**

Methods: **Over 13 years 1000 patients underwent 1150 CEA procedures to treat symptomatic and asymptomatic high-grade carotid stenosis. CEA procedures involving either traditional CEA with patching (n = 302) or eversion CEA (n = 848) were all performed by the same surgeon, with patients under deep general anesthesia and cerebral protection involving continuous electroencephalographic monitoring for selective shunting. All patients underwent postoperative duplex ultrasound scanning and clinical follow-up at 1, 6, and 12 months, and yearly thereafter. New neurologic events, late occlusions, and recurrent stenoses 50% or greater were recorded. Complete follow-up (mean, 6.2 years; range, 6-156 months) was obtained in 95% of patients (949 of 1000), for an overall average of 95% of procedures (1092 of 1150). Survival analysis was performed with the Kaplan-Meier life table method.**

Results. **Perioperative (30-day) mortality rate was 0.3% (3 of 1000), and stroke rate was 0.9% (11 of 1150), with a combined mortality and stroke rate of 1.2%. The incidence of late occlusion and recurrent stenosis 70% or greater was 0.6% and 0.5%, respectively, with a combined occlusion and restenosis rate of 1.1%. Kaplan-Meier analysis showed that the rate of freedom from occlusion, restenosis 70% or greater, and combined occlusion and restenosis 70% or greater at 12 years was 99,4%, 99.5%, and 98.8%, respectively. Occlusion and restenosis developed asymptomatically.**

Conclusions: **CEA is a low-risk procedure for treating severe symptomatic and asymptomatic carotid disease, with excellent long-term durability. Proponents of CAS should bear this in mind before considering CAS as a routine alternative to CEA. (J Vasc Surg 2004;40:270-8.)**

To date carotid endarterectomy (CEA) is the only revascularization procedure with proved efficacy in reducing the risk for stroke in patients with symptomatic and asymptomatic high-grade extracranial internal carotid artery (ICA) stenosis.[1-3] Although perioperative stroke rates of 6% in patients with symptomatic disease and 3% in patients with asymptomatic disease are still considered acceptable,[4] CEA is now being performed at many single-institution centers with much lower risk, and a perioperative stroke rate of 2% or less in symptomatic disease and less than 1% in asymptomatic disease can be expected as a rule.[5-15] Moreover, the reported incidence of recurrent ICA stenosis after primary CEA varies considerably between centers,[16-22] but the overall risk for recurrent stenosis seems low; only 1% to 8% are symptomatic and require a repeat operation.[20-22]

Carotid angioplasty and stenting (CAS) has recently emerged as a useful and potentially less invasive alternative to CEA.[23-29] Despite the still limited prospective controlled data comparing the relative risks and benefits of CAS with respect to CEA,[30,31] extensive worldwide experience has been collected outside of randomized trials to support the conviction that CAS can be performed successfully in selected patients once sufficient technical expertise has been acquired. However, despite several thousand CAS procedures having been reported to date, there is still grave concern as to the long-term durability of CAS.

Inasmuch as there are few long-term studies in the literature that evaluated the durability of CEA in a sufficiently large number of patients, and because the results of such studies would be essential to enable comparison of the durability of CAS, the purpose of the present analysis was to determine the occlusion and restenosis rates after CEA performed at a single institution in a relatively large sample with a relatively long follow-up.

METHODS

Over 13 years, 1150 consecutive CEA procedures were performed by the same surgeon (E.B.) in 1000 patients, and entered prospectively into a computerized registry. Many of these CEA procedures joined 2 different prospective randomized trials that evaluated the early and late outcome of traditional CEA with routine patch angioplasty versus eversion CEA.[32,33] One hundred fifty patients underwent bilateral CEA: bilateral eversion CEA in 44 patients; bilateral CEA with patching in 10 patients; and bilateral mixed CEA, that is, eversion CEA on one side and

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Competition of interest: none.

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CEA with patching on the contralateral side, in 96 patients. Bilateral CEA was planned at admission in 109 patients (72.6%), with a mean of 7 ± 2 weeks between procedures, and in 41 patients (27.4%) indications for contralateral CEA developed at a mean of 23 ± 7 months after the first procedure. Data for patients scheduled for CEA with concomitant coronary artery bypass grafting and for patients with associated supra-aortic trunk lesions requiring concurrent surgery were excluded from the analysis.

Patient demographic data were collected, including any history of diabetes mellitus, cigarette smoking, hypertension and coronary artery disease, other clinical variables, indications for surgery, details of the operation, and hospital length of stay.

The ICA lesion was diagnosed at preoperative traditional angiography during the earlier part of this experience (used with decreasing frequency over the years), whereas duplex ultrasound (US) scanning was the only preoperative ICA imaging study performed in most patients from mid-1998 and after, combined in selected patients with either magnetic resonance angiography, computed tomography (CT) angiography, or traditional arteriography. The radiologist's estimate of any carotid bulb or ICA stenosis from the final angiography report was recorded with the North American Symptomatic Carotid Endarterectomy Trial (NASCET) method.[1] If no arteriography was performed, stenosis was estimated on the basis of findings at preoperative duplex US scanning, performed in our vascular laboratory. The velocity criteria used to categorize the degree of stenosis revealed a satisfactory correlation with the angiographic findings when the degree of stenosis was calculated as the percentage of diameter reduction in compliance with the NASCET method.[1] These criteria have been published.[34,35]

Clinical findings at presentation were always classified by the consultant neurologist as transient ischemic attack (temporary hemispheric symptoms lasting no more than 24 hours, with complete recovery), amaurosis fugax (transient monocular visual loss), or stroke (neurologic deficit persisting for more than 24 hours, regardless of the mechanism and related to either cerebral hemisphere). Patients with nonhemispheric symptoms, such as episodes of dizziness or vertigo, were included in the asymptomatic group.

Preoperative cerebral CT was performed in all patients with symptomatic disease. Preoperative and postoperative cranial nerve assessment was performed in all patients by a neurologist and an otolaryngologist. Vocal cord movements were assessed with direct fiberoptic laryngoscopy in patients with symptoms or signs of vagus nerve injury.[36] Preoperative patient preparation was standardized. To reduce the incidence of neck hematomas, antiplatelet therapy (aspirin or dipyridamole, and ticlopidine or clopidogrel in the final period) was suspended at least 1 week before surgery, and was not resumed until the patient was discharged from the hospital.

All CEA procedures involved either traditional CEA with patching (n = 302) or eversion CEA (n = 848). The technical details of both procedures have been described.[32,37] Polytetrafluoroethylene (Gore-Tex; W. L. Gore) was the patch material used in all 302 CEAs with patching. In the first period of the study all patients underwent CEA with routine patching, and in 1992 we started to perform eversion CEA. Over time, and as we gained experience with the technique, almost all patients underwent eversion CEA. From 1998 onward we performed only eversion CEA to treat primary carotid occlusive disease.

All CEA procedures were performed with patients under deep general anesthesia and cerebral protection involving continuous perioperative electroencephalographic (EEG) monitoring for selective shunting. All perioperative EEGs were visually analyzed by a neurologist with extensive experience in interpretation of studies during sleep, either natural or induced with hypnotic or anesthetic agents. Shunting criteria were based exclusively on EEG changes consistent with cerebral ischemia. Completion imaging studies were not performed.

Patients were usually monitored in the recovery room for 2 hours, until blood pressure and neurologic status were considered acceptable, before being transferred to a regular nursing unit that specializes in vascular care, and were monitored for 12 to 24 hours after surgery. All patients with severe headache were observed for hyperperfusion syndrome, and hypertension was treated aggressively. Most patients were discharged 48 to 72 hours after CEA.

Surveillance protocol. After discharge, visiting nurses monitored the patients' blood pressure and neurologic status. Clinical evaluation and duplex US scans were performed systematically by a consultant neurologist and 2 experienced technologists in all surviving patients, at 1, 6, and 12 months, and once every year thereafter, to assess any residual ICA stenosis, angulation, recurrent ICA disease, or occlusion, with an Acuson Sequoia 512 ultrasound system. A 5.0-MHz linear scan transducer was used with a 5-mm axial extension of the sample volume and a consistent 60-degree angle of insonation. Residual stenosis was recorded as present when lumen reduction 30% or greater was noted at the proximal or distal end of the endarterectomized site at 30-day follow-up. Residual angulation was defined as a kink greater than 60 degrees detected just beyond the distal end of the patch or endarterectomized zone on the 30-day duplex US scan. Recurrent ICA disease was diagnosed only if the abnormality became apparent subsequent to a normal duplex US scan or more than 12 months after the initial examination. Peak systolic velocity greater than 140 cm/s, with spectral broadening throughout systole, and increased diastolic velocity were consistent with stenosis 50% or greater, and peak systolic velocity greater than 210 cm/s with end-diastolic velocity 110 to 140 cm/s was consistent with stenosis 70% or greater. Stenosis 70% or greater identified at duplex US scanning was confirmed with either carotid CT angiography or traditional arteriography. The study end points were perioperative stroke and death, restenosis 70% or greater, and late occlusion.

All patients were evaluated postoperatively by a consultant neurologist. Minor stroke was defined as minimal and

Table I. Baseline characteristics

	n	%
Parients	1000	
Procedures	1150	
Age (y)		
Mean ± SD	72 ± 6.18	
Range	31-93	
Male gender	679	67.9
Risk factors		
Hypertension	607	60.7
Hyperlipidemia	439	43.9
Current smoking or history of smoking	717	71.7
Diabetes	323	32.3
Coronary artery disease	445	44.5
Peripheral vascular disease/AAA	529	52.9
Symptomatic disease	771	67.0
Transient ischemic attack	476	61.8
Amaurosis fugax	156	20.2
Stroke	139	18.0
Asymptomatic disease	379	33.0
Contralateral carotid occlusion	135	13.5
CEA with patching	302	26.2
Eversion CEA	848	73.8
Perioperative EEG changes	243	21.1
Shunting	163	14.2

AAA, Abdominal aortic aneurysm; *CEA,* carotid endarterectomy; *EEG,* electroencephalogram.

stabilized focal neurologic deficit of acute onset, persisting for more than 24 hours but not leading to disability or significant impairment in activities of daily living. Major stroke was defined as a deficit lasting more than 30 days and inducing a change in lifestyle. Cerebral CT or magnetic resonance was performed in all patients with a new neurologic deficit after CEA. Other perioperative complications and events observed during follow-up were recorded in accordance with the guidelines of the Ad Hoc Committee on Reporting Standards for Cerebrovascular Disease, Society for Vascular Surgery/North American Chapter of the International Society for Cardiovascular Surgery.[38]

Statistical analysis. All values are expressed as mean ± SD. Cumulative life table analyses (Kaplan-Meier method) were performed to assess the incidence of occlusion and restenosis 50% or greater over time, and stroke-free and late survival rates. Statistical significance was inferred at $P < .05$. Cox proportional hazards multivariate analysis was used to determine which factors could influence long-term outcome. Inasmuch as each perioperative and late outcome was correlated with the surgical procedure, and because patients who underwent bilateral CEA were exposed to twice the risk for stroke or carotid occlusion or restenosis, several data items were analyzed vis-à-vis surgical procedures rather than patients.

RESULTS

Clinical features and several perioperative findings are shown in Table I for all patients. Of the 1000 patients who underwent CEA, 679 were men (772 CEA procedures) and 321 were women (378 CEA procedures), with mean age 72 ± 6 years. Of note, 135 patients had contralateral ICA occlusion, 475 had coronary artery disease, and 323 had diabetes. CEA procedures were performed to treat symptomatic severe ICA lesions in 771 patients (67%) Eversion CEA was performed in more than 70% of procedures.

Perioperative death and stroke rates. The 30-day mortality rate was 0.3% (3 of 1000), and the stroke rate was 0.9% (11 of 1150), with a combined mortality and stroke rate of 1.2%. There were 3 perioperative deaths: 2 due to myocardial infarction, and 1 due to stroke. The only fatal perioperative stroke occurred in a patient with symptomatic disease undergoing CEA with patching to treat a severe ulcerated left ICA lesion. EEG monitoring had been uneventful throughout the operation, and shunting was unnecessary. The patient awoke from anesthesia with no neurologic symptoms, but subsequently experienced progressive right-sided hemiparesis, and died on postoperative day 10 of recurrent progressive stroke. None of the other 10 patients with perioperative stroke (6 CEA procedures with patching, 4 eversion CEAs) was shunted, because EEG monitoring was uneventful throughout the operation. All awoke from anesthesia with no apparent neurologic deficit or stroke. In all cases stroke occurred within the first 24 hours after surgery, while the patient was in the recovery room. Duplex US scans immediately confirmed ICA occlusion in the patients who had undergone CEA with patching, and demonstrated ICA patency in patients who had undergone eversion CEA. In the CEA with patching group, 4 patients underwent repeat operation consisting of thrombectomy and new patch plasty, with some improvement in neurologic status in only 1 patient and no improvement in the others. The remaining 2 strokes involved the hemisphere contralateral to the side operated on. One of these was ipsilateral to an occluded ICA. Among the 4 strokes in the eversion CEA group, 2 were major and 2 were minor. Both major strokes occurred in patients (1 with a shunt) with a mildly diseased contralateral ICA, and were probably embolic from the aortic arch or from the heart, because cerebral CT scans demonstrated a cortical infarction in the territory of the middle cerebral artery. Both minor strokes were most likely hemodynamic, as suggested by findings on the CT images. One developed in the hemisphere contralateral to the revascularized ICA and ipsilateral to an occluded ICA.

No hyperperfusion syndrome was observed in any patient. Overall, 31 angulations and 21 residual stenoses were detected on the 30-day duplex US scans of the operated vessels (Table II).

Other important surgical morbidity included an overall 5% incidence of nerve injury (58 of 1150). Injuries involved the cranial nerves in 4.5% of cases (48 of 1150) and the cervical nerves in 0.5% (10 of 1150). There were 25 hypoglossal nerve injuries, 12 recurrent laryngeal nerve injuries, 7 superior laryngeal nerve injuries, 4 marginal mandibular nerve injuries, 7 greater auricular nerve injuries, and 3 transverse cervical nerve injuries. All nerve dysfunctions were transient, and all but 4 recurrent laryngeal nerves

Table II. Early (30-day) and late results

	Total		*CEA with patching*		*Eversion CEA*	
	n	*%*	*n*	*%*	*n*	*%*
Early results (30 d)	N = 1150		n = 302		n = 848	
Stroke	11	0.9	7	0.6	4	0.3
Death	3	0.3	3	1.2	0	
Nerve injury	58	5	12	3.9	46	5.4
Residual angulation	31	2.7	31	2.7	0	
Residual stenosis	21	1.8	15	1.3	6	0.5
Late results (mean, 6.2 y; range, 6-156 mo)	n = 1089		n = 264		n = 825	
All restenoses ≥ 50%	12	1.1	11	4.1	1	0.1
Restenoses ≥ 70%	6	0.5	5	1.9	1	0.1
Occlusions	7	0.6	6	2.2	1	0.1
Restenoses ≥ 70% + occlusions	13	1.1	11	4.1	2	0.2
Stroke	4	0.3	3	1.1	1	0.1
Death	84	8.8	31	15	53	7.1

recovered completely within 6 months of CEA. Two patients regained full function within 12 months, whereas the other 2 did not recover until 31 and 37 months, respectively.

Long-term results. Complete follow-up (mean, 6.2 years; range, 6-156 months) was obtained in 95% of patients (949 of 1000), for an overall of 95% of CEA procedures (1092 of 1150). After excluding the 3 perioperative deaths, 997 patients were alive 30 days after CEA, but 48 were lost to follow-up.

Overall, there were 7 ICA occlusions (0.6%), in 6 patched arteries and 1 everted artery (Table II). All occlusions involved ICAs that were not shunted, and occurred without symptoms within the first postoperative year, all in arteries apparently patent on the first 2 duplex US scans but demonstrating residual angulation on the 30-day duplex US scan. Freedom from all occlusions at 1, 3, 5, 7, 10, and 12 years was 99.4%, 99.4%, 99.4%, 99.4%, 99.4%, and 99.4%, respectively (Fig 1).

Overall, there were 12 recurrent stenoses (1.1%) 50% or greater, 11 of which were detected in patched arteries. Of the 12 restenoses, 6 (0.5%) were 70% or greater, for a combined occlusion and restenoses 70% or greater rate of 1.1% (Table II). All recurrent stenoses involved ICAs that were not shunted, and occurred without symptoms after the first postoperative year. All but 1 of the restenoses remained stable on subsequent duplex US scans, and all but 1 were treated conservatively. In 1 patched artery restenosis 70% or greater progressed rapidly and became severe enough (>90%) to necessitate a second operation 19 months after the first operation. Kaplan-Meier analysis showed that freedom from all restenoses 50% or greater at 1, 3, 5, 7, 10, and 12 years was 100%, 99.4%, 98.9%, 98.9%, 98.9%, and 98.9%, respectively (Fig 2). When the outcomes were stratified into patched versus everted arteries the freedom from restenoses 50% or greater at 1, 3, 5, 7, 10, and 12 years was 100%, 97.8%, 95.8%, 95.8%, 95.8%, and 95.8% versus 100%, 99.9%, 99.9%, 99.9%, 99.9%, and 99.9%, respectively. Freedom from restenoses 70% or greater at 1, 3, 5, 7, 10, and 12 years was 100%, 99.5%, 99.5%, 99.5%, 99.5%, and 99.5%, respectively (Fig 2). When the outcomes were stratified into patched versus everted arteries the freedom from restenoses 70% or greater at 1, 3, 5, 7, 10, and 12 years was 100%, 98.2%, 98.2%, 98.2%, 98.2%, and 98.2% versus 100%, 99.9%, 99.9%, 99.9%, 99.9%, and 99.9%, respectively. Freedom from combined occlusion and restenosis 70% or greater at 1, 3, 5, 7, 10, and 12 years was 99.4%, 98.8%, 98.8%, 98.8%, 98.8%, and 98.8%, respectively (Fig 1). When the outcomes were stratified into patched versus everted arteries, freedom from combined occlusion and restenosis 70% or greater at 1, 3, 5, 7, 10, and 12 years was 97.9%, 96.1%, 96.1%, 96.1%, 96.1%, and 96.1% versus 99.9%, 99.8%, 99.8%, 99.8%, 99.8%, and 99.8%, respectively.

Overall, there were 4 late strokes. One was cardioembolic (fatal stroke in a patient with atrial fibrillation of recent onset), and 3 were lacunar. Kaplan-Meier analysis showed that freedom from all strokes at 1, 3, 5, 7, 10, and 12 years was 100%, 99.8%, 99.6%, 99.6%, 99.6%, and 99.6%, respectively (Fig 3).

Eighty-four late deaths occurred in the series as a whole. The cause of late death was primarily cardiac related (n = 38; Table III). Freedom from death at 1, 3, 5, 7, 10, and 12 years was 99.9%, 96.1%, 92.8%, 91.3%, 87.2%, and 79.3%, respectively (Fig 4).

At multivariate analysis the occurrence of late occlusion was significantly associated with patch closure ($P = .04$) and the presence of angulation at the 30-day control duplex US scan ($P < .0001$), whereas severe recurrent stenosis was associated more strongly with patch closure ($P = .002$). Age, gender, symptomatic disease, diabetes mellitus, carotid artery diameter at surgery, coronary artery disease, serum triglyceride or cholesterol concentrations, and hypertension were not significantly associated with the occurrence of late occlusion or recurrent stenosis.

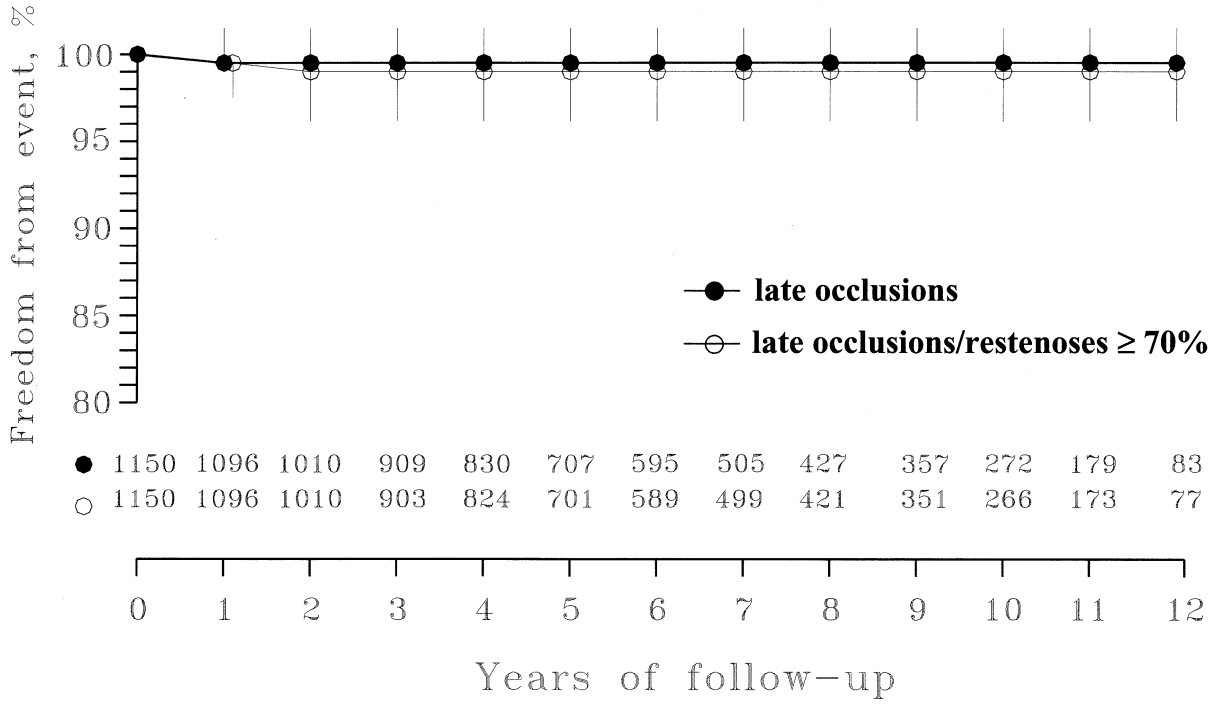

Fig 1. Kaplan-Meier curves show freedom from late occlusion *(solid circle)* and combined occlusion and restenosis ≥70% *(open circle)*. Numbers at bottom of graphic correspond to number of patients at risk at the beginning of each time interval. Vertical bars at each time point represent standard error for the interval.

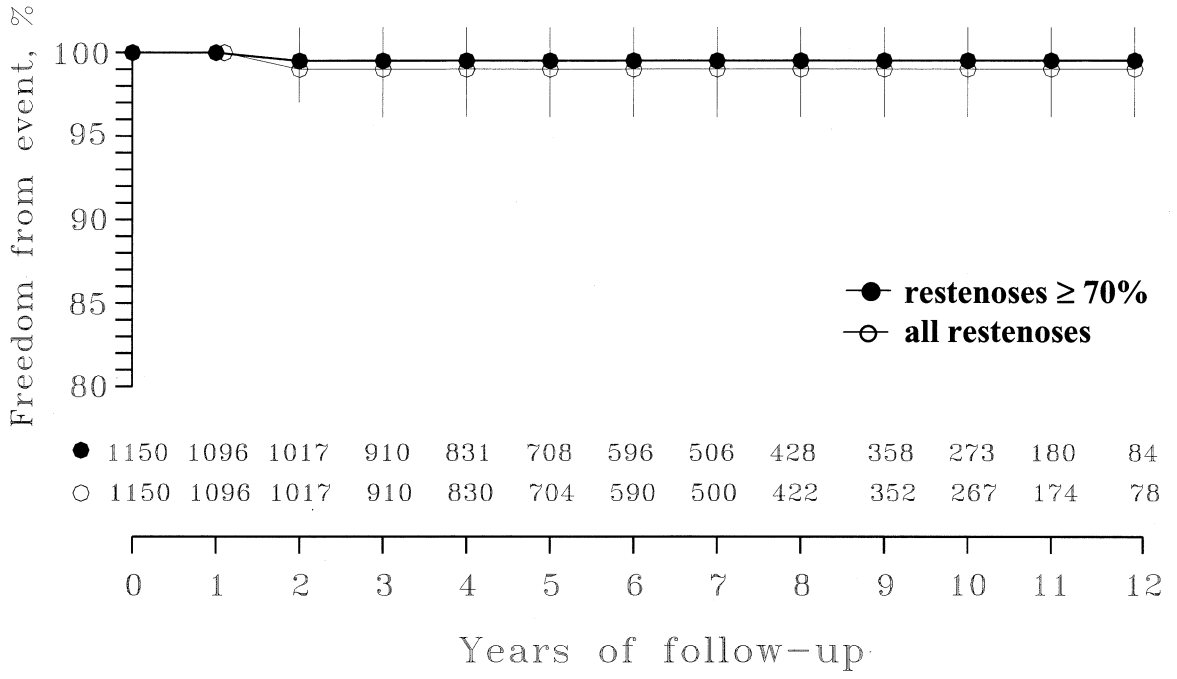

Fig 2. Kaplan-Meier curves show freedom from all recurrent stenoses *(open circle)* and restenosis 70% or greater *(solid circle)*. Numbers at bottom of graphic correspond to number of patients at risk at the beginning of each time interval. Vertical bars at each time point represent standard error for the interval.

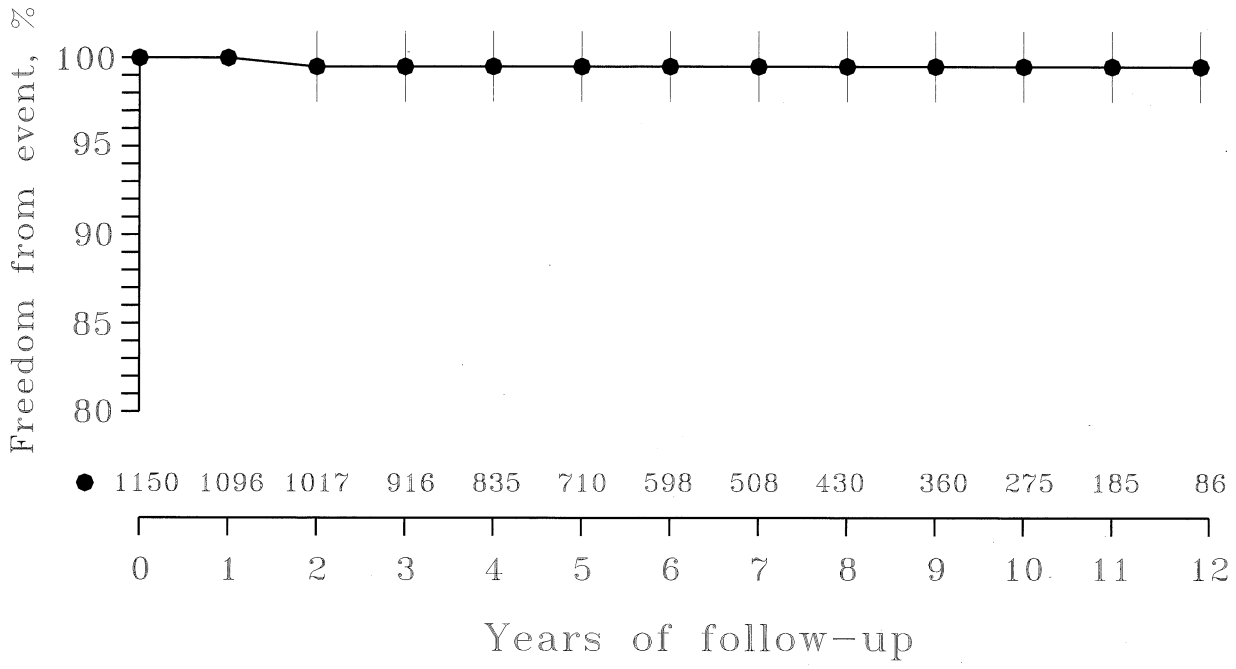

Fig 3. Kaplan-Meier curve of risk of late strokes. The numbers at the bottom of the graphic correspond to the number of patients at risk at the beginning of each time interval. The vertical bars at each time point represent the standard error for the interval.

Table III. Causes of late death

Cause	*n*	*%*
Myocardial infarction	38	45.2
Rupture of abdominal aortic aneurysm	1	1.2
Stroke	1	1.2
Pancreatitis	4	4.7
Cancer	18	21.4
Renal failure	7	8.3
Suicide	1	1.2
Car accident	5	6
Trauma	3	3.6
Unknown	6	7.1

DISCUSSION

The benefit of CEA for stroke prevention in selected patients with symptomatic and asymptomatic disease with severe carotid lesions has recently been established in controlled randomized clinical trials,[1-3] with acceptably low perioperative stroke and death rates.[4] The incidence of CEA-related stroke and death must be 6% or less in patients with symptoms and less than 3% in patients without symptoms for the benefit to hold true over 5 years. Many single-institution reports anecdotally claim a much lower incidence of stroke and death in patients with symptomatic and asymptomatic severe carotid lesions, however.

The results of the present study of 1150 CEA procedures (perioperative mortality rate, 0.3%; stroke rate, 0.9%; combined perioperative mortality and stroke rate, 1.2%) correlate well with recently published large series (≥500 CEA procedures) that report 30-day major stroke and death rates ranging from 0.9% to 4%[5,13] and recurrent stenosis rates of 0.7% to 13.6%[5,8] over an average of 3.3 years (Table IV).

Inasmuch as the incidence of late occlusion and restenosis after CEA can be interpreted as a measure of the durability of CEA, our combined occlusion and restenosis rate of 1.1% over an average of 6.2 years (the second longest follow-up to date of CEA used to treat ICA lesions, after the report from Ecker et al[15] of 975 patients and 1000 CEAs, with average follow-up of 7.1 years) demonstrates that CEA is a durable procedure.

It is surprising that no occlusions resulted from progression of restenosis. All occlusions developed in patched ICAs with residual angulation on 30-day duplex US scans, and at multivariate analysis it was concluded that residual angulation and patch closure are significantly associated with late occlusion ($P < .0001$ and $P = .04$, respectively). The reason that residual angulation disappears in the first few years after surgery (no residual angulation was detected on late duplex US scans) in most cases, while progressing asymptomatically toward occlusion in a few others, is hard to say, and might warrant further speculation. However, inasmuch as we observed angulation in all patched arteries that primarily thrombosed and required exploration (the ICA was occluded and acutely angulated at the end of the distal angle of the patch closure), we might suppose that the

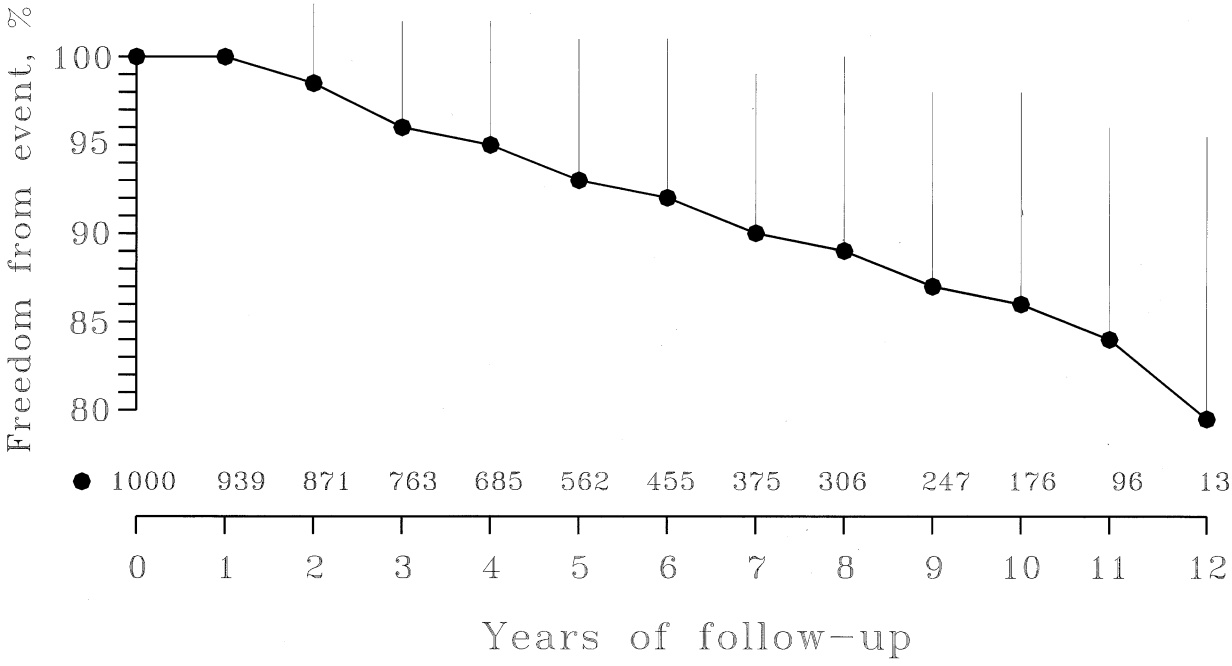

Fig 4. Kaplan-Meier curve of late survival rates. Numbers at bottom of graphic correspond to number of patients at risk at the beginning of each time interval. Vertical bars at each time point represent standard error for the interval.

patch made the artery stiff, and if a little hemodynamically insignificant elongation remains, sometimes increased by the natural weakness of the endarterectomized arterial wall, modest angulation might occur just beyond the distal end of the patch. When the clamps are removed and blood flow is restored, the systolic thrust may drive the stiff patched segment forward against the weakened and angulated patch-free segment beyond, thus accounting for delayed thrombosis in a few cases. Similar dynamics could also account for late occlusion in these vessels. The one occlusion in an everted vessel is also difficult to explain, inasmuch as the ICA seemed normal on previous duplex US scans.

Recurrent carotid stenosis after CEA remains a therapeutic conundrum. Reports of its incidence vary widely, and review of the literature is challenging because of differences in the duration of follow-up, criteria used to determine and define recurrent stenosis, and methods used to close the arteriotomy. The time it takes for recurrent lesions to develop also varies considerably, depending on the different pathologic features of the lesions and their related morbidity. Early restenosis (within 24 months) is usually a consequence of myointimal hyperplasia, whereas stenosis occurring later is generally associated with atherosclerosis. In this series all restenoses 70% or greater occurred within 24 months after surgery, and all but 1 remained stable at subsequent control duplex US scans. Although the purpose of this study was not to compare the results of 2 techniques of closure of the arteriotomy, the rates of occlusion, total restenosis, restenosis 70% or greater, and combined occlusion and restenosis 70% or greater were higher after patching CEA than after eversion CEA (2.2% vs 0.1%, 4.1% vs 0.1%, 1.8% vs 0.1%, 4.1% vs 0.2%, respectively). Moreover, multivariate analysis demonstrated that patch closure is significantly associated with severe recurrent stenosis ($P = .002$). These data are consistent with those of other investigators, who found restenosis rates clearly lower after eversion CEA (range, 0.2%-2.8%) than after patch CEA (range, 0.7%-5.8%) or primary closure (range, 1%-11%; Table IV).

Any extensive review of early and late results in patients who undergo CAS is bound to be methodologically flawed, because the procedure is constantly evolving from a technical standpoint, new stents are rapidly being developed, and a variety of distal protection devices are being introduced. However, in a recent meta-analysis of 2537 CAS procedures performed without cerebral protection and 896 CAS procedures performed with cerebral protection, Kastrup et al[39] found a significantly higher combined 30-day stroke and death rate in patients without cerebral protection (5.5%; 140 of 2537) compared with those with cerebral protection (1.8%; 16 of 896; $P < .001$). This was mainly due to fewer minor and major strokes in patients with cerebral protection (0.5% vs 3.7% [$P < .001$] and 0.3% vs 1.1% [$P < .05$], respectively), whereas the death rate was much the same (~0.8%).

There is still reason for concern about the long-term durability of the CAS procedure. Data regarding restenosis are only available in a few studies, most of which had a relatively short follow-up. Moreover, recurrence was reported with weighting each procedure equally, regardless

Table IV. Large series of carotid endarterectomies: early and late results

Year	*Author*	*Study type*	*Mean follow-up (mo)*	*No. of CEA*	*Symptomatic (%)*	*Death/stroke (% at 30 days)*	*Percent restenosis criterion*	*Method of closure*	*Occlusion plus restenosis (%)*
1987	Hertzer et al[5]	PS	21	917	49	4	>50	Patch 47%	2.6
								Eversion 0%	—
								Primary 53%	11
1990	Rosenthal et al[6]	RS	38	1000	82	2.3	>50	Patch 75%	3.1
								Eversion 0%	—
								Primary 25%	4.0
1996	Plestis et al[7]	RS	61	1006	67	2	>75	Patch 100%	4.1
								Eversion 0%	—
								Primary 0%	—
1997	Lawhorne et al[8]	RS	24	500	71	1	>80	Patch 86%	0.7
								Eversion 0%	—
								Primary 14%	—
1998	Shah et al[9]	RS	18	2723	36	2.3	NR	Patch 3%	—
								Eversion 82%	0.3
								Primary 15%	1
2000	Radak et al[10]	PS	56	2806	96	2.1	>50	Patch 24%	3.1
								Eversion 76%	1.1
								Primary 0%	—
2000	Cao et al[11]	PR	33	1353	59	2.6	>50	Patch 19%	1.5
								Eversion 50%	2.8
								Primary 31%	7.9
2000	Archie[12]	RS	55	1360	62	2.1	>50	Patch 99.6%	2.1
								Eversion 0%	—
								Primary 0.4%	—
2001	Scavee et al[13]	RS	49	600	46	0.9	>50	Patch 100%	5.8
								Eversion 0%	—
								Primary 0%	—
2002	Trisal et al[14]	RS	NR	1648	NR	NR	>70	Patch 62%	3.8
								Eversion 0%	—
								Primary 38%	5.8
2003	Ecker et al[15]	PS	82	1000	59	1.9	>70	Patch 100%	1.0
								Eversion 0%	—
								Primary 0%	—
2004	Present series	RS	74	1150	67	1.2	>70	Patch 26%	4.1
								Eversion 74%	0.2
								Primary 0%	—

PR, Prospective randomized; *PS,* prospective series; *RS,* retrospective series; *NR,* not reported.

of duration of follow-up. Criado et al[28] found a restenosis rate of 3.5% in 135 CAS procedures with a mean follow-up of 16 months; Becquemin et al[40] reported a restenosis rate of 7.5% in 107 CAS procedures with a mean follow-up of 15 months; and more recently McKevitt et al[41] reported a restenosis rate of 15.5% after 333 CAS procedures with a mean follow-up of 12 months. In a series of 217 CAS procedures with a mean neurologic follow-up of 13 months, Christiaans et al[42] found that restenosis after CAS was common (21%), located mainly in the central body of the stent, related to loss of proximal stent apposition alone, and symptomatic in 7% of patients. With longer follow-up and using life table analysis, Lal et al[43] demonstrated recurrent stenotic lesions (ranging from 40% to 99%, none associated with neurologic events) in a significant number of patients (22 of 122, 18%; mean follow-up, 18 months), and the severe restenosis (≥80%) rate at 5 years was 6.4%; yet only 4 of the original 122 patients could be evaluated at 60 months.

While the 30-day mortality and stroke rates for CAS in experienced centers can be acceptable, especially since the introduction of cerebral protection devices, available follow-up data suggest that the durability of CAS remains uncertain.

In conclusion, the results of the present study demonstrate that CEA is a safe, effective, and durable procedure and that it can be performed with excellent results in patients with symptomatic and asymptomatic severe ICA lesions. Although use of cerebral protection devices has reduced the thromboembolic complications (minor and major stroke) with CAS, making the periprocedural risk rates after CAS resulting from single-institution and industry-sponsored trials comparable with perioperative risk rates after CEA emerging from many excellent large, institutional series, the durability of the less invasive procedure may be poor.

To validate the effectiveness of CAS as an alternative to CEA, properly performed randomized trials will have to take into account the rapid improvements in the field of endovascular technology, and adequate follow-up of several years to assess the incidence of recurrent stenosis and the long-term functional outcome of the procedure.

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