Outcome of carotid stent-assisted angioplasty versus open surgical repair of recurrent carotid stenosis

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Purpose: **We compared outcome and durability of carotid stent-assisted angioplasty (CAS) with open surgical repair (ie, repeat carotid endarterectomy [CEA]) to treat recurrent carotid stenosis (RCS).**

Methods: **A retrospective review of anatomic and neurologic outcomes was carried out after 27 repeat CEA procedures (1993-2002) and 52 CAS procedures (1997-2002) performed to treat high-grade internal carotid artery (ICA) RCS after CEA. The incidence of intervention because of symptomatic RCS was similar (repeat CEA, 63%; CAS, 60%), but the interval from primary CEA to repeat intervention was greater ($P < .05$) in the repeat CEA group (83 ± 15 months) compared with the CAS group (50 ± 8 months). In the CAS group, 17 of 52 arteries (33%) were judged not to be surgical candidates because of surgically inaccessible high lesions (n = 8), medical comorbid conditions (n = 4), neck irradiation (n = 3), or previous surgery with cranial nerve deficit or stroke (n = 2). Three patients who underwent repeat CEA had lesions not appropriate for treatment with CAS.**

Results: **Overall 30-day morbidity was similar after CAS (12%; death due to ipsilateral intracranial hemorrhage, 1; nondisabling stroke, 1; reversible neurologic deficits or transient ischemic attack, 2; access site complication, 2) and repeat CEA (11%; no death; nondisabling stroke, 1; reversible cranial nerve injury, 1; cervical hematoma, 1). Combined stroke and death rate was 3.7% for repeat CEA and 5.7% for CAS ($P > .1$). All duplex ultrasound scans obtained within 3 months after CEA and CAS demonstrated patent ICA and velocity spectra of less than 50% stenosis. During follow-up, no repeat CEA (mean, 39 months) or CAS (mean, 26 months) repair demonstrated ICA occlusion, but two patients (8%) who underwent repeat CEA and 4 patients (8%) who underwent CAS required balloon or stent angioplasty because of 80% RCS. At last follow-up, no patient had ipsilateral stroke and all ICA remain patent. At duplex scanning, stenosis-free (<50% diameter reduction) ICA patency at 36 months was 75% after repeat CEA and 57% after CAS ($P = .26$, log-rank test).**

Conclusions: **Carotid angioplasty for treatment of high-grade stenotic ICA after CEA resulted in similar anatomic and neurologic outcomes compared with open surgical repair. Most lesions are amenable to endovascular therapy, and CAS enabled treatment in patients judged not to be suitable surgical candidates. Duplex scanning surveillance after repeat CEA or CAS is recommended, because stenosis can recur after either secondary procedure. (J Vasc Surg 2003;38:432-8.)**

Management of recurrent carotid stenosis (RCS) after surgical endarterectomy (CEA) remains controversial. There is no consensus regarding criteria as to threshold stenosis severity at which to intervene in asymptomatic lesions, and the dilemma of surgical repair with patch angioplasty versus carotid resection has been further complicated by reports of comparable results with endovascular management.[1-7] Providing consul to patients with asymptomatic RCS is cloaked in clinical uncertainty, in part because of the relatively benign natural history of these lesions and the morbidity associated with repeat CEA. RCS is associated with lower risk for embolic complications, that is, incidence of neurologic symptoms, than is primary atherosclerotic carotid bifurcation stenosis, and when followed by serial duplex scanning, regression of myointimal lesions has been documented in more than 10% of patients.[3,8] Because surgical intervention for RCS is perceived to be associated with higher risk for perioperative stroke and cranial nerve deficit, compared with primary CEA, it is not surprising that stent-assisted angioplasty (CAS) has been promoted as a less invasive, alternative treatment, especially in patients with radiation-scarred necks or high internal carotid artery (ICA) recurrent stenosis.[9] Thus, when weighing the decision as to whether to intervene in RCS, the vascular surgeon must carefully consider the risks versus potential clinical benefits of open versus endovascular repair in each patient.

After CEA, RCS develops as a continuum, with early recurrent stenosis the result of technically inadequate repair or myointimal hyperplasia, whereas later lesions have a more typical appearance of atherosclerosis. RCS that develops during the first year after CEA may involve an aggressive proliferative process, but the lesion is smooth, and

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progression to ICA occlusion is more likely than thromboembolism. Carotid angioplasty has been used principally for treatment of myointimal recurrent stenosis that has developed within 3 years after CEA, and treatment of these lesions is associated with high technical success, as well as periprocedural complication rates comparable to those with primary and repeat CEA.[4,5,7,10,11] On the basis of these favorable clinical reports, our vascular surgery division, working in conjunction with an interventional neuroradiologist (A.E.), selected patients with RCS for CAS versus repeat CEA, based on anatomic and patient factors, or for inclusion in a multicenter clinical trial on the safety and efficacy of CAS. This report details a comparative analysis of outcome after operative or endovascular management of RCS, with the goals of providing clinical guidelines for intervention and determining whether CAS is applicable in most RCS lesions encountered in a referral vascular surgery practice.

METHODS

Records for patients who underwent treatment of RCS were retrieved from a vascular registry that detailed symptoms at presentation, atherosclerotic risk factors, procedural details of carotid stenosis repair, and duplex ultrasound scanning results. Inpatient and outpatient clinic charts were then carefully reviewed for both intraprocedural and postprocedural complications and 30-day neurologic morbidity and mortality. After discharge, patient follow-up was accomplished by review of outpatient and vascular laboratory records, with patient interview at clinic visit or via telephone when indicated. Survival data were supplemented with the Social Security Administration data base system if patient follow-up was not complete.

In all patients who underwent CAS, cerebral angiograms were obtained before stenting, and these were reviewed for degree of stenosis (percent diameter reduction), severity of contralateral disease, and presence of intracranial cerebral artery occlusive disease. After intervention with CAS or repeat CEA, all patients were followed up with duplex ultrasound scanning of the extracranial carotid arteries, as well as clinical evaluation, for development of neurologic symptoms, including stroke, transient ischemic attack (TIA), and amaurosis fugax. Patient demographic data, including age, gender, atherosclerotic risk factors, and medical comorbid conditions, and procedure outcome, including patency, intervention to treat recurrent stenosis, occlusion, or neurologic event, and survival, were compared between the two treatment groups. Mean follow-up for patients with repeat CEA was 38 months (range, 3-112 months), and for patients with CAS was 22 months (range, 3-60 months).

Patients. Over 10 years (1993-2002), 27 patients (19 men, 8 women) with symptomatic RCS (17 of 27; 63%) or asymptomatic RCS (11 of 27; 41%) underwent repeat CEA. During a concurrent 6 years (1997-2002), 50 patients (35 men, 15 women) with symptomatic RCS (31 of 52; 60%) or asymptomatic RCS (21 of 52; 40%) underwent CAS. In only 3 of 77 patients (4%) was the original CEA procedure performed by vascular surgery faculty; the remaining patients were referred for evaluation and management of RCS. In general, intervention for symptomatic RCS was recommended for all lesions with greater than 50% diameter reduction. For patients with asymptomatic RCS, high-grade stenosis (>80%) at duplex scanning (end-diastolic velocity >145 cm/s at site of stenosis) and angiography was the clinical threshold for intervention. Five of 27 patients (18%) with repeat CEA and 14 of 50 patients (28%) with CAS had occlusion of the ICA contralateral to RCS.

Table I. Type and size of stents used in 52 carotid stent-assisted angioplasty procedures to treat recurrent carotid stenosis

Stent type	*No. used*	*Size (mm)*
Self-expanding		
Wallstent	27	8 × 20 to 10 × 42
Precise*	12	9 × 30 to 10 × 40
SMART	6	10 × 30 to 10 × 40
Acculink†	3	10 × 30
Balloon-expandable	4	5 × 16 to 7 × 31

*Angioguard filter used during two procedures.
†AccuNet distal protection device used during stent deployment procedure.

The method of vessel closure (primary closure, patch angioplasty, unknown) at the original CEA was similar in the repeat CEA (63%; 37%, 0%) and CAS (57%, 37%, 6%) treatment groups, but the interval from CEA to repeat intervention was longer ($P < .05$) in the repeat CEA group (83 ± 15 months) compared with the CAS group (50 ± 8 months).

Seventeen of 52 patients (33%) who underwent CAS, all with symptomatic RCS, were judged by consultant vascular surgeons not to be surgical candidates, because of surgically inaccessible high lesions (n = 8), comorbidity (n = 4), neck irradiation (n = 3), or recent CEA with cranial nerve deficit or scarred neck (n = 1) or perioperative stroke (n = 1). Three patients who underwent repeat CEA had lesions judged not appropriate for CAS, because of lumen thrombus in RCS and perceived high risk for embolization (n = 2) or inability to safely stent the RCS because of vessel tortuosity (n = 1).

Procedures to treat RCS. All CAS procedures were performed in the angiographic suite with biplane imaging, under the direction of an interventional neuroradiologist (A.E.) experienced in cerebrovascular interventions. Before CAS, patients were given platelet inhibitors (aspirin, 325 mg/d; clopidogrel, 300 mg bolus, then 75 mg/d). Angioplasty procedures were performed with local anesthetic at the femoral or brachial (1 patient) puncture site, intravenous sedation, and monitored anesthetic techniques. Before selective catheterization, patients underwent systemic heparinization (activated clotting time >250 s). Self-expanding stents were preferentially used (Table I), as was predilation of the RCS lesion with a 3 or 4 mm balloon. Balloon-expandable stents were used in select patients only

Table II. Demographics, associated medical conditions, and presenting symptoms in 77 patients treated for 79 recurrent carotid stenoses

	CAS (n = 50)		*Repeat CEA (n = 27)*		
	n	*%*	*n*	*%*	P
Age (y)					
Mean	70.2 ± 8		66.5 ± 11		.001
Range	62–84		38–79		
Men	35	70	19	70	NS
Medical comorbid conditions					
Hypertension	44	88	24	88	NS
Tobacco abuse	33	66	19	70	NS
Hyperlipidemia	35	70	16	59	NS
Peripheral artery disease	24	48	12	44	NS
Coronary artery disease	31	62	13	48	NS
Diabetes mellitus	8	16	8	30	.11
Presenting symptoms	52 arteries		27 arteries		
Asymptomatic	21	40	10	37	NS
Symptomatic	31	60	17	63	NS
TIA or amaurosis fugax	19	37	4	15	.02
Stroke	7	13	10	37	.02
Both	5	10	3	11	

CAS, Carotid stent-assisted angioplasty; *CEA,* carotid endarterectomy; *TIA,* transient ischemic attack; *NS,* not significant.

for high lesions amenable to placement above the mandible, where stent protection was achieved. Completion angiography with extracranial and intracranial imaging was performed in all patients in the CAS group, and more recently color duplex scanning has been used to monitor adequacy of stent patency and deployment. Abciximab (ReoPro; Eli Lilly, Indianapolis, Ind), an antagonist of glycoprotein IIb/IIIa platelet receptors, was used selectively (3 patients) to treat suspected intrastent thrombus formation or in patients believed to be at increased risk for CAS-related thromboemboli.

Patients in the repeat CEA group underwent open surgical repair performed by one of three board-certified vascular surgeons (D.F.B., M.R.B., B.L.J.) using general anesthesia, and either routine shunting or electroencephalographic monitoring during carotid occlusion. All patients received aspirin (325 mg/d) alone or in addition to clopidogrel (75 mg/d) before the procedure. Repair technique consisted of vein (n = 21) or prosthetic (n = 2) patch angioplasty (23 of 27 procedures [85%]), with endarterectomy of atherosclerotic plaques (n = 17) when present. Carotid resection with autogenous vein bypass grafting was performed in 3 patients, and 1 patient underwent primary closure of the ICA after repeat CEA. Completion intraoperative duplex scanning was performed in all patients in the repeat CEA group, and no intraoperative revisions were required.

After intervention, patients in the CAS group were given clopidogrel bisulfate (Plavix; 75 mg/d orally) for 30 days, and aspirin therapy has been continued indefinitely in all patients.

Duplex ultrasound scanning surveillance. After intervention for RCS, patients underwent serial color duplex ultrasound scanning in a laboratory accredited by the Intersocietal Commission for Accreditation of Vascular Laboratories, beginning at 1 month after the procedure, then every 6 months. Duplex ultrasound scanning criteria for recurrent stenosis with greater than 50% diameter reduction involving the repeat CEA site or stented extracranial carotid artery segment were peak systolic velocity greater than 125 cm/s and internal carotid artery–common carotid artery ratio greater than 2. Duplex scanning criteria for greater than 75% stenosis were peak systolic velocity greater than 300 cm/s and end-diastolic velocity greater than 125 cm/s at the site of maximum stenosis, in conjunction with internal carotid artery–common carotid artery ratio greater than 4 and high-grade lumen reduction with poststenotic turbulence at color Doppler scanning. If end-diastolic velocity was greater than 145 cm/s, recurrent stenosis was classified as greater than 80% diameter reduction. Criteria for an occluded angioplasty site or surgical repair were a clearly visualized vessel without color Doppler flow scanning and velocity spectra of antegrade flow. Duplex scanning surveillance was accomplished in all patients at 1 month, and thereafter in all surviving patients in the repeat CEA group and 85% of patients in the CAS group. All patients in the CAS group underwent duplex surveillance scanning at least twice.

Statistical analysis. Analysis of categorical variables between the two treatment groups was performed with χ^2 analysis, and continuous variables were compared with either a two-sided *t* test or the Wilcoxon rank-sum test, as appropriate. The Kaplan-Meier method was used to estimate patient survival, and intervention-free and stenosis-free patency after CAS or repeat CEA. Life-table patency rates were compared with the log-rank test. $P < .05$ was considered significant.

Table III. Interval from CEA to treatment of RCS with CAS or repeat CEA

	CAS (n = 52)	*Repeat CEA (n = 27)*	P
Early (≤36 mo) RCS	33	10	.03
Late (>36 mo) RCS	19	17	
Mean time to intervention (mo)	50 ± 8	83 ± 15	<.05
Median time to intervention (mo)	24	48	

CEA, Carotid endarterectomy; *RCS*, recurrent carotid stenosis; *CAS*, carotid stent-assisted angioplasty.

RESULTS

Patient demographic data. Patients selected for CAS were older than patients who underwent repeat CEA to treat RCS, but the incidence of atherosclerotic risk factors and other medical comorbid conditions were similar in the two treatment groups (Table II). Most patients treated for RCS had symptoms (CAS, 60%; repeat CEA, 63%), and stroke was more common in the repeat CEA group. All asymptomatic RCS lesions demonstrated greater than 80% diameter reduction at both duplex scanning (end-diastolic velocity >145 cm/s) and angiography.

The frequency of early (<3 years) RCS, that is, myointimal lesions, treated with CAS was greater ($P = .03$, χ^2 test) than in the repeat CEA group, with mean time from primary CEA to intervention of 50 ± 8 months for patients with CAS and 83 ± 15 months patients with repeat CEA (Table III). Overall, most RCS lesions selected for intervention were symptomatic (59%; 49 of 77) and treated less than 3 years (54%; 43 of 79) after primary CEA. Nine patients in the CAS group were enrolled in ongoing clinical trials studying the safety and efficacy of carotid stenting with (Carotid Revascularization and Endarterectomy vs Stent Trial [CREST] Lead-In, 3; Acculink for Revascularization of Carotids in High-Risk Patients [ARCHER], 5) and without (Stenting and Angioplasty with Protection in Patients at High Risk for Endarterectomy [SAPPHIRE], 1) cerebral protection device usage.

Periprocedural (30-day) outcome. Mortality, neurologic morbidity, and overall morbidity from treating RCS with CAS or repeat CEA were similar (Table IV). In the repeat CEA group no patient died or sustained a myocardial infarction within 30 days. One patient had a minor ipsilateral stroke, with a cortical infarct demonstrated at magnetic resonance imaging (MRI) after repeat CEA. This complication was thought to be caused by an allergic reaction to dextran-40, manifested as hypotension during the procedure. No permanent neurologic deficit was present in this patient at discharge. No ipsilateral TIA or contralateral stroke occurred in the repeat CEA group. In 1 patient transient hypoglossal nerve paresis developed, with resolution of tongue deviation by 1 month; and 1 patient required operative evacuation of a cervical hematoma. Overall morbidity after 27 repeat CEA procedures was 11% (3 patients), and neurologic or stroke morbidity was 3% (1 patient).

Table IV. Thirty-day perioperative and neurologic outcome after 52 CAS and 27 repeat CEA for recurrent carotid stenosis

	CAS		*Repeat CEA*		
	n	*%*	*n*	*%*	P
Death	1	2	0		NS
Bleeding	2	4	1	4	NS
Myocardial infarction	0		0		NS
All ipsilateral strokes	2	4	1	4	NS
Major*	1	2	0		NS
Minor	1	2	1	4	NS
Contralateral stroke	1	2	0		NS
Ipsilateral TIA	2	6	0		NS
Intracranial embolus	1	2	0		NS
Cranial nerve injury					
Transient			1		
Permanent			0		

CAS, Carotid stent-assisted angioplasty; *CEA*, carotid endarterectomy; *TIA*, transient ischemic attack; *NS*, not significant.
*Patient died of intracerebral hemorrhage.

CAS angioplasty was successful in all patients selected for endovascular treatment. Three patients were deemed unsatisfactory for CAS on the basis of previous cerebral angiographic findings, and were treated surgically. In 1 patient an embolus to the middle cerebral artery developed 4 hours after the CAS procedure, and was treated with intracerebral thrombolyis, with no neurologic sequelae or brain abnormality at MRI. One patient (2%) died of ipsilateral intracranial hemorrhage on the second postprocedure day, after successful combined CAS–intracranial balloon angioplasty to treat symptomatic RCS and middle cerebral stenosis. This complication was thought related to the intracranial procedure and not to stenting of the extracranial carotid lesion. An additional patient in the CAS group was seen 1 week after discharge, with ipsilateral arm and leg weakness and slurred speech. Duplex scans and cerebral angiograms demonstrated a patent carotid stent without thrombus or residual stenosis, and no intracerebral abnormality. This event was classified as a minor stroke, inasmuch as all neurologic deficits resolved within 3 days. TIA occurred in 2 patients on the day after CAS; resolution was within hours, and brain MRI verified no abnormality. A contralateral hemispheric lacunar stroke developed in 1 patient in the CAS group, believed due to hypertension. Total neurologic morbidity (stroke or TIA) attributable to the CAS procedure was 10% (5 patients), and all ipsilateral neurologic symptoms developed more than 24 hours after the CAS procedure. The 30-day combined stroke and death rate after CAS was 5.7% (3 of 52 procedures).

Catheter sheath access complications developed in 2 patients in the CAS group: a brachial artery psuedoaneurysm requiring surgical repair and retroperitoneal hematoma requiring no blood transfusion. No patient in the CAS group had a periprocedural myocardial infarction.

Duplex ultrasound scanning surveillance. No patient had stent or ICA occlusion during mean follow-up of

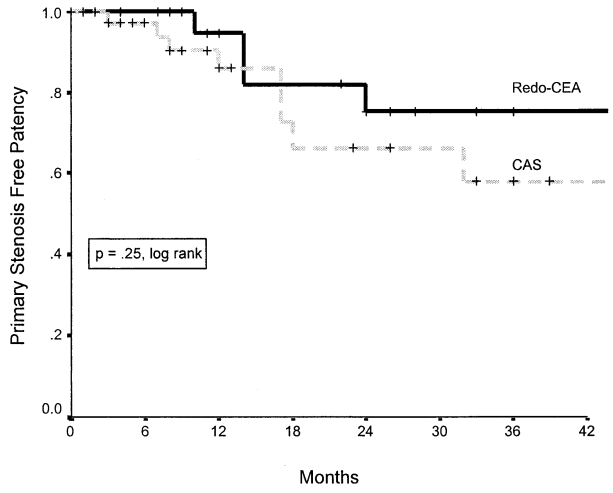

Fig 1. Stenosis-free (<50% diameter reduction) primary patency after carotid stent-assisted angioplasty (*CAS*; n = 51) or repeat carotid endarterectomy (*Redo-CEA*; n = 27).

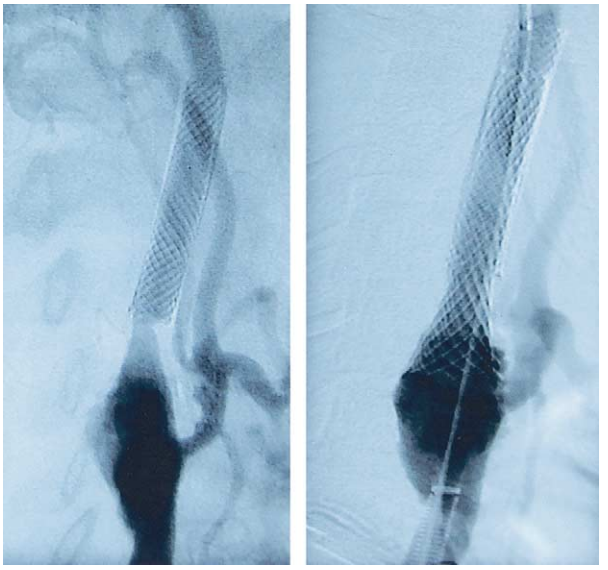

Fig 2. Left, Angiogram demonstrates 60% recurrent stenosis proximal to an internal carotid artery stent that developed 9 months after carotid angioplasty and was associated with recurrent amaurosis fugax. **Right,** Treatment with a second stent corrected the recurrent stenosis.

34 months. Duplex scan–detected stenosis-free (<50% diameter reduction) patency for CAS (66%) was similar to that for repeat CEA procedures (74%) at 24 months ($P >$ .1, log-rank test; Fig 1).

After repeat CEA, greater than 50% asymptomatic recurrent stenosis developed at five repair sites (18%). In 2 patients the lesions progressed to greater than 75% diameter reduction at 10 months and 3 years, respectively, after repeat CEA, and these patients underwent successful, uncomplicated CAS. In the remaining 3 patients with 50% to 74% diameter reduction, repeat CEA site stenosis developed at 8, 18, and 24 months, respectively; these lesions have not progressed in severity, and all have remained asymptomatic.

After CAS, eight stent-angioplasty sites (16%) demonstrated intrastent (n = 4) or adjacent carotid artery (n = 4) greater than 50% diameter reduction stenosis at duplex scanning surveillance. In 1 patient in the CAS group a motor TIA occurred at 9 months, and duplex ultrasound scans and cerebral angiograms demonstrated 60% diameter reduction recurrent stenosis proximal to the ICA stent (Fig 2). A second stent was placed proximal across the origin of the ICA, and the patient has remained asymptomatic thereafter. In 3 patients the lesions progressed without neurologic symptoms to greater than 80% diameter reduction stenosis, and repeat interventions were performed at 5, 11, and 12 months, respectively, after CAS. High-grade (>80%) stenosis was confirmed at angiography, and repeat intervention with balloon dilation (n = 1) or stent-angioplasty (n = 3) was successful and without neurologic complication. In 4 patients with duplex scanning–detected stent stenosis of 50% to 74%, the lesions have remained asymptomatic, with regression to less than 50% stenosis in 1 patient.

Late clinical outcome. Freedom from repeat intervention and stroke rates after CAS and repeat CEA were identical (89%) by life-table analysis at 36 months (Fig 3). No patient had ipsilateral stroke attributable to the repeat CEA or CAS site during mean follow-up of 39 and 26 months, respectively. The incidence of recurrent ipsilateral neurologic symptoms due to recurrent stenosis or treatment of asymptomatic greater than 80% recurrent stenosis was similar in the CAS group (4 of 51 procedures [8%]) and repeat CEA group (2 of 27 procedures [7%]). Patient survival at 36 months after intervention for RCS was equivalent in the repeat CEA (92%) and CAS (92%) groups (Fig 4). In the repeat CEA group 2 patients died of coronary artery occlusive disease, and in the CAS group 1 patient died of coronary artery occlusive disease, 3 patients died of cancer, and 1 patient died of undetermined cause.

DISCUSSION

Management of RCS with CAS was associated with neurologic and anatomic outcome similar to that after open surgical repair with patch angioplasty or carotid artery resection. Accounting for differences in patient factors, including frequency of myointimal lesions, the procedure-related stroke and death rate was similar for CAS (6%) and repeat CEA (3%), as was overall 30-day morbidity, at 12% and 11%, respectively. These mortality and neurologic morbidity rates were similar to our results with primary CEA (1.7%),[10] as well as recent reports of CAS (2.4%-4.3%)[5,7,9,11] and repeat CEA (0%-5%)[1,3,4,6,12-15] outcome for RCS. We have not limited the use of CAS to treatment of early myointimal lesions. In fact, one third of lesions treated with CAS developed more than 3 years after the primary CEA procedure and had duplex scanning and angiographic features of recurrent carotid bifurcation ath-

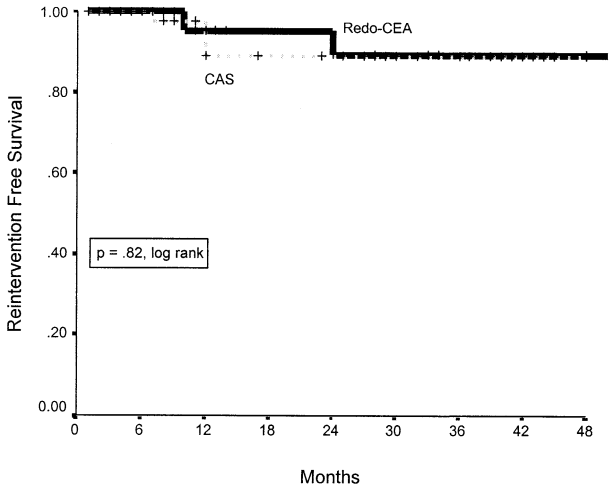

Fig 3. Freedom from repeat intervention for recurrent stenosis after treatment for recurrent carotid stenosis with carotid stent-assisted angioplasty (*CAS*; n = 51) or repeat carotid endarterectomy (*Redo-CEA*; n = 27).

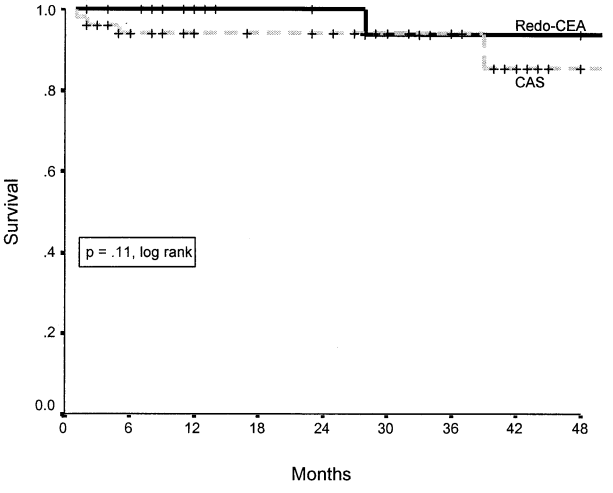

Fig 4. Survival of 77 patients after treatment of recurrent carotid stenosis with carotid stent-assisted angioplasty (*CAS*; n=50) or surgical repair (*redo-CEA*; n = 27).

erosclerosis. In addition, approximately half of the CAS group could be considered at high risk because of contralateral ICA occlusion (n = 14) or judged not to be surgical candidates (n = 17). Of note, a distal protection or "antiembolic" device was used in 6 patients in the CAS group enrolled in the National Institutes of Health–sponsored CREST (n = 3) or Guidant-sponsored ARCHER (n = 5) clinical trials. Despite these differences in patients treated, equivalent results were obtained with CAS and repeat CEA. We believe surgical repair should continue to be the preferred option, especially in patients at good risk with extensive RCS involving both the common and internal carotid artery segments, and for RCS associated with mural thrombus, aneurysm dilation, or ICA tortousity. Repeat carotid surgery has a proved track record, and provides long-term freedom from stroke and recurrent stenosis.[14,15]

In our practice, selection of patients for CAS was based primarily on the presence of aortic arch and carotid anatomy appropriate for CAS. However, some patients in the CAS group were referred from other surgeons specifically for endovascular management, because they would not consent to repeat CEA, and 9 patients were enrolled in clinical trails (CREST, ARCHER, SAPPHIRE) as "high-risk" patients on the basis of an RCS lesion. A significant number of patients, 17 of 50 (34%) in the CAS group were judged not to be suitable surgical candidates, most commonly because of a surgically inaccessible distal ICA lesion, advanced cardiorespiratory disease, or RCS in an irradiated or scarred neck. Thus, having the capability to perform CAS increased the number of patients who could undergo intervention for RCS. The ability to treat the spectrum of patients with RCS is important for a tertiary vascular surgery practice.

Most patients with RCS have anatomy appropriate for endovascular management. We averted use of CAS on lesions with angiographic features of irregular, mural thrombus or when significant ICA tortousity precluded "safe" stent deployment. Despite careful patient selection, three CAS procedures were complicated by development of stent thrombus or distal embolization requiring cerebrovascular rescue with catheter-directed thrombolysis or administration of abciximab. Having the skills to deal with stenting complications is critical if procedure-related morbidity is to be minimized and be comparable to surgical repair.

Our experience in evaluating and treating patients with RCS indicates the natural history of this lesion, including early (<1 year) myointimal stenosis, was associated with low incidence of disabling stroke or progression to ICA occlusion. After treatment with CAS or repeat CEA, no stent angioplasty or carotid repair site became occluded, and no ipsilateral stroke occurred. Only 1 patient had recurrent symptoms (amaurosis fugax after CAS), due to 50% to 74% diameter reduction recurrent stenosis proximal to the stent. We found duplex scanning surveillance to be worthwhile, inasmuch as greater than 50% recurrent stenosis developed in 16% of stented segments and 18% of carotid repairs. Progression to high-grade greater than 75% diameter reduction stenosis was observed in half of our patients, all without development of neurologic symptoms, and led to endovascular repeat intervention in 6 patients, 2 after repeat CEA and 4 after CAS. The durability of CAS in treating RCS, as measured by duplex scanning monitored stenosis-free patency, was comparable to repeat CEA, 66% versus 74%, respectively, at 2 years. While duplex scanning–detected recurrent stenosis rate of 40% to 50% at 2 to 3 years after CAS has been reported, it appears the recurrent stenosis rate leading to endovascular or surgical intervention is in the range of 10% to 15%.[4,5,16] These results support routine use of duplex scanning surveillance after both CAS and repeat CEA procedures. We recommend

angiography and consideration of endovascular repair with balloon dilation or stent-assisted angioplasty in patients when a duplex scan demonstrates greater than 80% diameter reduction stenosis (end-diastolic volume >145 cm/s) at the RCS repair site.

The effectiveness of CAS in treating extracranial carotid occlusive disease has been confirmed at multiple institutions, and has led to prospective multicenter clinical trials to verify equipoise of CAS with CEA. Because of the inherent increased risk for embolic complications associated with CAS, it is appropriate that efficacy of protection devices also be studied.[17] Antiembolic devices provide cerebral protection only while deployed in the distal ICA. Risk for lesion or device embolization during lesion traversal or device removal and immediately after CAS will still be present. Careful patient preparation before CAS, appropriate catheter techniques in performing cerebrovascular interventions, and verification of technically adequate stent deployment may be equally important in reducing neurologic complications. The principal benefit of using distal protection devices in treating RCS may be the increased number of patients who would be candidates for treatment of lesions thought to be at "higher" risk for embolization. At present our experience demonstrates that carotid angioplasty provides equivalent protection against stroke and ICA occlusion compared with open surgical procedures in treatment of both early and late RCS. Ideally, in the future carotid angioplasty will be compared with repeat carotid surgery in a controlled, randomized, multicenter clinical trial.

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