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Outcome of Carotid Stenting Versus Endarterectomy
A Case-Control Study

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Background and Purpose—To compare perioperative and midterm results of carotid artery stenting (CAS) versus carotid endarterectomy (CEA) in similar cohorts of patients, a retrospectively matched case-control study was performed.

Methods—Three hundred and one case subjects undergoing CAS with cerebral protection and 301 concurrent matched-controls undergoing CEA were examined. Matching was by sex, age (±2 years), symptoms and coronary disease.

Results—The 30-day disabling stroke/death rate was 2.6% in the CAS group versus 1.3% in the CEA group (odds ratio [OR] 2; 95% CI, 0.54 to 9.35; $P=0.4$). CAS patients had a significantly higher risk of periprocedural stroke (7.9% versus 2.3%; OR, 5.2; 95% CI, 1.7 to 18; $P=0.001$) than CEA patients. However, there was a decreasing trend in 30-day neurological event rates for the last 201 CAS matched cases: 5.4% versus 1.9% (OR 2.8; 95% CI, 0.8 to 10.2; $P=0.1$). Fifty percent of CAS disabling strokes occurred during cannulation of epiaortic vessels before placement of cerebral protection. Conditional multivariate analysis revealed CAS as a predictor of 30-day stroke (hazard ratios [HR] 3.9; 95% CI, 1.6 to 9.4; $P=0.002$) but not of 30-day disabling stroke/death (HR 3.6; 95% CI, 0.93 to 13.9; $P=0.06$). Restenosis free intervals at 36 months were 93.6% versus 92.1% for CAS and CEA, respectively, ($P=0.6$).

Conclusions—When comparing CAS with CEA, the risk of any neurological events is still higher, particularly during catheterism and ballooning. The effect of the learning curve related to technical expertise and patient selection may influence the outcome of CAS versus CEA. In the midterm the restenosis rate of CAS compares favorably to CEA. **(*Stroke*. 2006;37:1221-1226.)**

Key Words: angioplasty ■ carotid artery ■ carotid endarterectomy ■ stent

Carotid artery angioplasty and stent placement (CAS) has recently emerged as an alternative to carotid endarterectomy (CEA) for primary and secondary prevention of stroke related to carotid stenosis.[1–3] Although initial outcome studies indicated higher morbidity and mortality rates for CAS than standards considered acceptable for CEA,[4] the development of new stent technologies and the advent of embolic cerebral protection devices (CPD) have improved procedural safety and clinical outcomes.[1,2] Yet, there has been some resistance to widespread use of CAS as an alternative to CEA because of the lack of high-level proof of CAS efficacy in randomized trials.[5–7]

Whereas single-center case series and multicenter registries report low perioperative risks during CAS,[3,8–9] other studies in different populations provide different results.[5,6] The inability to control important confounding variables, such as anatomic characteristics, previous symptoms, comorbidities and risk factors, was one plausible explanation for these different findings. To examine the relationship between CAS and perioperative risk of stroke while controlling medical and anagraphic risk factors in subjects undergoing intervention for primary carotid stenosis, a retrospective matched case-control study was performed. Perioperative mortality and morbidity and midterm outcome in a consecutive series of CAS patients was compared with a concurrent risk-matched group of CEA patients.

Subjects and Methods

Patient Population

A matched case-control study recruited patients who had undergone CAS at a single tertiary hospital and controls from the registry (prospective collected records) of patients who had concurrently undergone CEA in the same hospital. After the first 50 carotid procedures performed in a learning curve phase and when the techniques were standardized and routinely applied, all patient data were systematically collected in separate prospective single-center databases (CAS and CEA Registries) including preprocedural, intraprocedural and follow-up information.

From May 2001 to December 2004, 1225 procedures were performed on 1071 patients with carotid stenosis. A total of 855 CEAs on 732 patients (123 staged, bilateral) and 315 CAS on 284 patients (31 staged, bilateral) were performed for primary stenosis for a total of 1170 interventions and 1016 patients. CAS procedures represented 27% of the patient population treated during that interval, according to plaque morphology, comorbidities and patient preference. The primary crite-

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rion for treatment was either symptomatic (≥60%) or asymptomatic severe (>70%) internal carotid artery (ICA) stenosis. Patients with recurrent carotid stenosis, previous cervical radiation therapy, tracheostomy or ICA stenosis above C2 level were excluded from the present study in both CAS and CEA series, resulting in 303 CAS procedures and 270 CAS patients to be matched.

Recruitment of Cases, Controls and Matching

CAS were presented as a consecutive series of patients from the CAS Registry. For each patient undergoing CAS, a matched-control subject was selected from the list of CEA patients during the same period (years 2001 to 2004). Matching was by sex, age (±2 years), history of cerebrovascular ipsilateral symptoms and presence of coronary artery disease. For 2 CAS subjects a paired-match control was not identified. A total of 301 case-control pairs were enrolled.

Preoperative Evaluation

All patients underwent preoperative duplex ultrasound (US) 1 month before carotid revascularization. A stenosis >70% was diagnosed when the peak systolic velocity (PSV) exceeded 200 cm/s and >90% if PSV exceeded 300 cm/s. Duplex US scanning was performed by experienced vascular surgeons (using ATL HDI 3000 with 12.5 MHz linear probe) who defined site, degree and length of stenosis, plaque characteristics and vessel measurements in order to select proper size of balloon and stent. Duplex velocity criteria were previously validated against angiography as a gold standard using the European Carotid Surgery Trial (ECST) criteria.[10] In all patients undergoing CAS, the presence of ICA stenosis ≥70% was always confirmed by angiography during the stent procedure. Preoperative angiography, cerebral computed tomography (CT) or CT angiography scans were used selectively. All patients scheduled for CAS received full antiplatelet therapy consisting of acetylsalicylic acid (mean dosage of 125 mg/d) and clopidogrel (75 mg/d) for at least 30 days after a 300 mg loading dose, 12 hours before the procedure. For patients undergoing CEA, antiplatelet medication was not interrupted for surgery.

Operative Techniques

CAS was performed by a team of vascular surgeons and interventional radiologists with experience in endovascular procedures, using a standardized protocol. During CAS the patient's neurological status was checked by having the patient squeeze a toy in the appropriate hand and by talking. Transcranial Doppler monitoring (TCD 4040 Pyoneer Eme) was applied when possible (45%).

Intravenous heparin (100 U/kg) was routinely given before selective catheterization of the common carotid artery.

CAS was performed in all cases after proper placement of CPD in the distal ICA under roadmap guidance. Four different protection systems were used: Boston Scientific FilterWire EX (n 219, 73%), EV3 Microvena Trap Filter (n 26, 8.6%), Johnson and Johnson-Cordis Angioguard Filter (n 55, 18.3%) and Accunet Guidant filter (n 1, 0.33%). Predilation was performed when needed at discretion of the operator; 26 procedures required predilation.

After CPD deployment, a self-expandable (Boston Scientific Carotid Wallstent, n 232, 77%), or a nitinol stent (Precise, Cordis, n 64, 21%; Acculink Guidant, n 3, 1%; Exponent Medtronic, n 2 0.66%) was selected (depending on operator preference, lesion characteristics, and commercial availability) and placed across the stenosis. During the dilation phase, atropine (mean dosage, 1 mg IV) was used in 193 procedures at the discretion of the anesthesiologist.

Procedural success for CAS was defined as complete stent deployment with resolution of stenosis or with residual stenosis ≤30% at the completion angiogram (double projection).

CEA was performed under local (127=42%) or general (174=58%) anesthesia at discretion of the anesthesiologist. Monitoring was performed using the same technique as for CAS on the awake patients; under general anesthesia TCD or stump pressure measurements were used to evaluate clamping ischemia. Carotid shunt was used in 68 (23%) procedures.

Systemic heparinization was always used during the procedure at the same dosage as CAS and reversed after declamping of the ICA. Eversion CEA was performed in 239 (79%), patch closure in 42 (14%), and primary closure in 20 (12%) procedures. A single drug was used for platelet antiaggregation for all patients at discharge.

A duplex US of the operated vessel was performed within 1 month, and procedural success for CEA was defined as presence of residual stenosis ≤30%.

Definition of Outcome and Complications

Outcome measures were stroke, death, cardiac events and local complications. Perioperative stroke was defined as any new neurological event persisting >24 hours and occurring within 30 days from the procedure. Strokes were classified as fatal, disabling (symptoms persisting for >1 month) or nondisabling (lasting >24 hours and cleared at 30 days). Transient ischemic attack (TIA) was defined as any new retinal or neurological focal event with complete recovery within 24 hours. Myocardial infarction was diagnosed in the occurrence of a new Q-wave in ≥2 leads or the presence of elevated enzymes (including troponin >0.1 ng/mL). ECG and enzymes were routinely checked in postoperative period. The same team of neurologists and cardiologists evaluated all patients with clear or suspected symptoms regardless of the procedure. Restenosis was defined as carotid stenosis ≥50% after intervention at follow-up Duplex examination (using PSV ≥125 cm/s as threshold).

For CAS, it was recorded when intraprocedural complications occurred during the catheterism phase (phase 1), including the passage of the aortic arch and cannulation of target vessel, the crossing lesion phase (phase 2), including placement of CPD, or the stent-ballooning phase (phase 3), including stent implantation, balloon dilation, and recovery of the protection system. For CEA, perioperative complications were differentiated whether occurring during the procedure (at awakening for general anesthesia patients), within the first 24 hours of surgery or later.

Postprocedure and Follow-Up

For 24 hours after the procedure (either CAS or CEA), the clinical condition of the patient was monitored continuously, and in the case of symptoms or uncertainty the patient was examined by a neurologist and the necessary diagnostic imaging was performed (CT scan or magnetic resonance). Clinical and duplex examination were performed before discharge and at 1, 3, 6 months and every 6 months after procedure in CAS patients. After CEA, patients were evaluated at 1, 6, 12 months and annually thereafter. Patients were instructed to inform the vascular surgeon or general practitioner when any new symptoms occurred after hospital discharge. Restenosis was evaluated at a maximum follow-up of 36 months with reliable standard error (mean 18 months, range 3 to 48 months).

Statistical Analysis

For univariate comparisons of risk factors and preoperative findings between patients with CAS and controls, statistical significance was assessed by 2-tailed χ^2 test with Yates correction or Fisher exact test. For comparisons of outcomes in paired cases and controls χ^2 Mc Nemar corrected was used. Odds ratios (OR) and hazard ratios (HR) with 95% CI were calculated by standard method. Continuous data are expressed as mean±SD. The restenosis rates after procedure were calculated by life table analysis. To control simultaneously for potential confounding variables on the risk of periprocedural neurological complications, multivariate analyses with conditional logistic-regression models were used. Twelve variables, chosen for inclusion in this model, were known cardiovascular risk factors (age, male sex, atrial fibrillation, hypertension, contralateral occlusion, history of ipsilateral symptoms, diabetes, peripheral artery obstructive disease [PAOD], coronary disease, CAS, urgency, hyperlipemia). Significant values were considered with $P<0.05$. Both on treatment and intention-to-treat analysis were performed, although the primary analysis was based on treatment method to obtain more reliable information on outcomes of each specific procedure, this being a nonrandomized study. Statistical package Software SPSS (SPSS Inc) and EPIINFO Software were used for all analyses.

TABLE 1. Patient Characteristics

	Cases (CAS)		Controls (CEA)				
	n=301	%	n=301	%	OR	95% CI	*P* Value
Age (range)	71.6±7.9	(49–90)	71.4±7.6	(49–88)			
Male	216	72	216	72			
Smoke	55	18	72	24	0.7	0.47–1.07	0.1
Hypertension	241	80	229	76	1.26	0.84–1.90	0.2
Diabetes	90	30	82	27	1.14	0.79–1.65	0.5
Hyperlipemia	141	47	150	50	0.89	0.64–1.24	0.5
Coronary disease	131	43	131	43	1	0.72–1.40	1
Peripheral vascular disease	59	20	66	22	0.87	0.57–1.31	0.5
Contralateral carotid occlusion	27	9	17	6	1.65	0.84–3.29	0.1
Atrial fibrillation	8	3	9	3	0.89	0.29–2.63	1

Results

Characteristics of 301 cases and 301 controls are shown in Table 1. Cases and controls were balanced for common cardiovascular risk factors. Table 2 reports the distribution and type of preoperative symptoms. Technical success rate was 100% in the CEA group and 95% in the CAS group attributable to 3 cases of residual stenosis >30% at the end of procedure and 12 conversions to open surgery (4%).

Overall, 10 disabling strokes occurred, 8 (2 fatal) in the CAS group and 2 (fatal) in the CEA group. In the CAS group, 4 disabling strokes were attributable to massive embolization during phase 1; 1 was followed by massive ipsilateral hemorrhage. The remaining 4 disabling CAS strokes occurred during phase 3. CT scan showed 6 ipsilateral, 1 contralateral and 1 posterior ischemic lesion. In the CEA group, of the 2 fatal strokes 1 was attributable to carotid occlusion and the other to respiratory complications after progression of a massive ischemic stroke in a patient with acute symptoms at the time of operation.

In the CAS group, of 16 nondisabling strokes, 1 occurred during the phase of catheterism, 1 during the phase of stenting/ballooning/recovery, 10 within 24 hours and 4 after 24 hours from treatment. In the CEA group, 2 nondisabling strokes occurred within 24 hours after awakening of patients and the other 3 after 24 hours. The majority of TIA (18/19) during CAS occurred in phase 3.

Particulate material was found in retrieved filter in 37% of CAS procedures and in 75% of those with complications. A marked hemodynamic response (bradicardia or hypotension) was recorded in 34% of CAS patients with or without periprocedural stroke, despite the use of atropine.

TABLE 2. Preoperative Symptoms

	Cases (CAS)		Controls (CEA)				
	n=301	%	n=301	%	OR	95% CI	*P* Value
Ipsilateral	77	26	77	26			1
TIA	26		32		0.79	0.44–1.42	0.48
Stroke	51		45		1.16	0.73–1.84	0.57
Contralateral	30	10	37	12	0.79	0.46–1.36	0.4
Vertebrobasilar	11	4	6	2	1.86	0.62–6.22	0.3
Asymptomatic	183	61	181	60	1.03	0.73–1.44	0.9

Perioperative outcome is shown in Table 3. The stroke incidence, statistically significantly higher after CAS than after CEA, markedly decreased in CAS patients over the study period. If we considered as an additional learning-curve phase for the first 100 CAS and excluded the first 100 pairs in outcome analysis, we found that in the last 201 CAS the risk of stroke was not significantly different from that of the corresponding 201 CEA-matched controls. Thirty-day any stroke rates for CAS and CEA were 5.4% versus 1.9%, respectively (P=0.1; Table 4).

Two perioperative cardiac deaths (fatal myocardial infarction) occurred in patients who had undergone CEA; in 1 patient CEA was the primary treatment option, whereas in the other, CEA was performed immediately after a failed attempt at CAS. Comparisons in complications rates between CAS and CEA groups by on-treatment and intention-to-treat analysis are reported in Tables 3 and 4. The incidence of myocardial infarction was similar in both groups.

There were no significant differences in the overall rate of local complications between the 2 groups (Tables 3 and 4). Cranial nerve damage and hematoma requiring revision were more frequent in the CEA group. In the CAS group vascular access complications occurred in 13 patients; 12 were attributable to false aneurysm formation at the femoral puncture site. The other was attributable to median nerve injury after percutaneous brachial access in a patient with severe iliac obstruction.

Multivariate analysis showed that CAS was not an independent predictor of major outcomes (disabling stroke/death), the only significant predictor being urgency for treatment (Table 5). On the contrary, CAS, urgency, diabetes and advanced age were predictors of any stroke (Table 5).

At mean follow-up of 18 months (range 3 to 48 months), there were 4 restenoses in the CAS group and 10 in the CEA group (1.3% versus 3.3%; P=0.2; OR 0.4, 95% CI, 0.1 to 1.4). Restenosis free intervals at 36 months (standard error 5%) were 93.6% versus 92.1% for CAS and CEA, respectively (P=0.6; Figure and Table 6). No new neurological events occurred at the available follow-up.

TABLE 3. Complications at 30 Days in 301 Pairs

	Cases (CAS)		Controls (CEA)				
	n=301	%	n=301	%	*P* Value	OR	95% CI
Disabling stroke/death (on-treatment)	8	2.6	4	1.3	0.4	2.04	0.54–9.35
Disabling stroke/death (intention-to-treat)	9	2.9	3	0.9	0.1	4	0.8–27.2
Any stroke*	24	7.9	7	2.3	0.001	5.2	1.7–18.03
Nondisabling stroke*	16	5.3	5	1.7	0.02	3.7	1.2–13.3
Myocardial infarction (on-treatment)	2	0.6	5	1.6	0.45	0.4	0.04–2.46
Myocardial infarction (intention-to-treat)	3	1	4	1.3	1	0.7	0.13–3.9
TIA*	19	6.3	3	1	0.0004	9.5	2.14–58.9
Local complications*	13	4.3	10	3.3	0.67	1.31	0.52–3.40
Haematoma	12	3.9	2	0.7	0.01	6	1.3–38.8
Nerve lesion	1	0.3	8	2.8	0.04	0.13	0.01–0.97

*Same results for intention-to-treat or on-treatment analyses.

Discussion

We report the first individually matched case-control study on the risk of stroke in CAS patients. There was no evidence of a statistically significant increase in the major risk of treatment regarding disabling stroke and death in CAS patients compared with CEA-matched controls (2.6% versus 1.3%; OR 2; 95% CI, 0.54 to 9.35; $P=0.4$). However, the wide confidence intervals indicate that it is not possible to exclude a difference of one treatment versus the other. On the other hand, any stroke risk favored open treatment (7.9% versus 2.3%; $P=0.001$).

Nevertheless, our complication rate after CAS was similar to that reported in several large case series[3,11–12] and in the recent meta-analysis of randomized controlled trials on CAS and CEA performed by Coward et al,[6] who reported an 8% rate for any stroke and death. Yet, it is possible that our results are affected by the learning curve process and consequently prone to further improvement, as shown by the decreasing trend in stroke rate from the first to the last period of the study. Indeed, despite the statistically significant higher risk of periprocedural stroke for patients with CAS, when we excluded the first 100 pairs of subjects the overall 30-day stroke incidence after CAS markedly decreased and was not significant in comparison to that after CEA (5.4% versus 1.9%; $P=0.1$; Table 4).

Our study included many patients with coronary disease, PAOD, or contralateral occlusion. These subsets have been shown to be at high perioperative risk.[12,13] True rates of neurological complications after CAS are a challenging issue attributable to the available low-evidence literature data in the absence of results from randomized trials.[8–9,11] In the meta-analysis by Coward et al on the early outcomes of CAS,[6] 5 completed or discontinued randomized trials of CAS compared with CEA between 1998 and 2004 were included for a total of 1269 treated patients. The 30-day safety data showed no significant differences between treatments for major outcomes of stroke/death, disabling stroke/death, and any stroke. However, the authors emphasized that the study was not robust enough to rule out any advantage or disadvantage of one treatment over the other.

It is noteworthy that advanced age and symptomatic diseases are associated with risk of stroke after both CEA and CAS.[12,13] Recently, Kastrup et al analyzed the role of patient-related-factors (particularly presenting symptoms) in determining periprocedural neurological complications in 299 patients after CAS. TIA/stroke/death rate was 18.6% in

TABLE 4. Complications at 30 Days in the Last 201 Pairs (excluding learning curve effect)

	Cases (CAS)		Controls (CEA)				
	n=201	%	n=201	%	*P* Value	OR	95% CI
Disabling stroke/death (on-treatment)	6	2.9	2	0.9	0.3	3	0.6–21.4
Disabling stroke/death (intention-to-treat)	5	2.5	3	1.5	0.7	1.7	0.4–8.8
Any stroke*	11	5.4	4	1.9	0.1	2.8	0.8–10.2
Nondisabling stroke*	7	3.4	4	1.9	0.5	1.8	0.5–7.1
Myocardial infarction (on-treatment)	1	0.5	3	1.5	0.6	0.3	0.01–3.6
Myocardial infarction (intention-to-treat)	0	0	4	1.9	...	...	...
TIA*	10	4.9	2	0.9	0.04	5	1.04–33
Local complications*	9	4.4	7	3.4	0.67	1.31	0.52–3.40
Hematoma	9	4.4	1	0.5	0.02	9	1.2–89.8
Nerve lesion	0	0	6	2.9	0.04	...	...

*Same results for intention-to-treat or on-treatment analyses.

TABLE 5. Independent Risk Factors

	Dependent Variables					
	Disabling Stroke/Death			Any Stroke		
Predictors	HR	*P* Value	95% CI	HR	*P* Value	95% CI
CAS	3.6	0.06	0.93–13.9	3.9	0.002	1.6–9.4
Urgency	8.9	0.009	1.71–46.4	4.6	0.03	1.2–18.6
Diabetes	...			2.2	0.045	1.01–4.83
Age	...			1.06	0.02	1.01–1.1

symptomatic versus 3.1% in asymptomatic patients.[12] Our analysis, with all the limitations of nonrandom allocation, attempted to adjust for some potential confounders of perioperative risk, as a number of risk factors were matched in the study design or resulted in similar prevalence (smoking, PAOD, diabetes, hyperlipemia, hypertension, contralateral occlusion, atrial fibrillation) between the 2 treatment groups. Multivariate analysis confirmed the absence of statistical association between CAS and perioperative disabling stroke/death risk. On the other hand, CAS, urgency, diabetes and advanced age were predictors of any stroke. In particular, urgency of treatment for recurrent TIA or acute stroke (6 CAS and 13 CEA) represented an increased risk factor no matter which treatment was used (HR 8.9; $P=0.009$).

We attempted to provide further insight for periprocedural stroke risk during carotid revascularization by analyzing the timing of complications during CAS and CEA. In CAS both catheterization phase and ballooning/stenting phase were at higher risk for neurological complications, as 2/3 of strokes occurred during these time intervals. In particular, 50% of CAS disabling strokes occurred during cannulation of epiaortic vessels, before placement of cerebral protection. Few complications occurred after 24 hours, in line with other CAS experiences suggesting outpatient performance of CAS.[14] It should be noted that particulate material was found in 37% of CAS procedures and in 75% of those with stroke, whereas a marked cardiac rate response was recorded in 34% regardless of the use of atropine. These findings support that embolism was the major hazard of CAS. The non-negligible conversion rate (4%) of the present series may be partly explained by the fact that we preferred to turn down and convert the patient to CEA when cannulation of epiaortic vessel could not be easily performed to decrease the potential embolic risk attributable to repeated attempts of catheterization. In CEA patients, intraprocedural stroke risk was principally attributable to the risk of early carotid thrombosis or possibly to clamping ischemia in acute stroke patients.

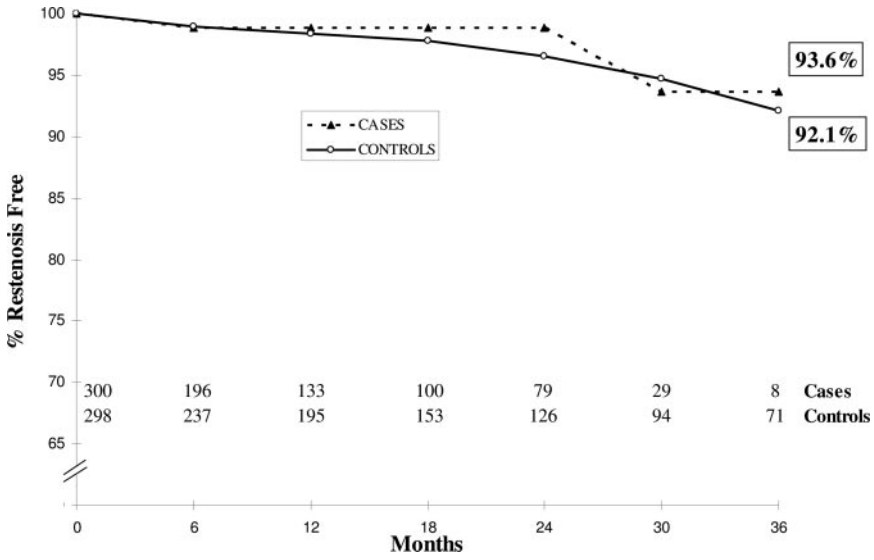
The uncertainty on the duration of the benefit of CAS with respect to traditional surgery[15,16] was partially disproved by our data that showed encouraging midterm results after CAS. At mean follow-up of 18 months, the actuarial risk of restenosis after CAS appeared acceptable and even reduced with respect to CEA (6.4% versus 7.9%). No new strokes occurred.

In the present study an actuarial restenosis risk after CEA of 7.9% may appear excessively high. Our Duplex velocity criteria may have influenced this result. Furthermore, because it is well known that the maximum incidence of intimal hyperplasia occurs within 2 years, it is likely that the maximum incidence of restenosis should have already occurred, and we may expect better results with longer follow-up.

In CEA patients we used eversion technique in the majority of cases (80%), based on the results of a randomized trial showing a reduced restenosis risk at 4 years after eversion compared with standard CEA.[17]

Methodological Aspects

Most previous studies analyzed the frequency of stroke during CAS as compared with the frequency in nonselected CEA series. This study allowed us to compare case and control patients with similar characteristics and thereby adjust some of the potential confounders and increase the precision of the comparison. However, we recognize that there are weaknesses: (1) matching could have reduced the power of the case-control study, decreasing the effective sample size of the patient population; (2) the retrospective analysis and the lack of randomization cannot exclude selection bias.



Restenosis free interval for CAS and CEA patients.

TABLE 6. Restenosis Free Intervals in 301 CAS and 301 CEA Patients

Intervals in Months	No. at Risk	No. Failed	Cumulative Survival Rates, %	Stroke-Free Standard Error
CAS				
Entry	300	3	98.8	0.69
6 mo	196	0	98.8	0.69
12 mo	133	0	98.8	0.69
18 mo	100	0	98.8	0.69
24 mo	79	0	98.8	0.69
30 mo	29	1	93.6	5.10
36 mo	8	0	93.6	5.10
CEA				
Entry	298	3	98.9	0.64
6 mo	237	1	98.4	0.78
12 mo	195	1	97.9	0.96
18 mo	153	2	96.5	1.36
24 mo	126	2	94.7	1.81
30 mo	94	0	94.7	1.81
36 mo	71	1	92.1	3.13

We could not establish risk and benefit of CAS, but we aimed to offer an opportunity to analyze the association between the risk of stroke and CAS in a common population generally undergoing CEA.

In conclusion, when comparing CAS to CEA, the risk of any neurological events is higher, particularly during catheterism and ballooning despite the use of CPD. This risk may be reduced after an appropriate learning curve involving series of patients even larger than those usually required as credentialing.[7] Knowledge of continuous technological progress, advance in technical expertise and patient selection are crucial to reduce the risk of CAS. In the midterm, the restenosis rate of CAS compares favorably with CEA patients. As yet, there is no evidence of efficacy of CAS versus CEA; therefore, it is necessary that randomized trials comparing the 2 treatments continue to recruit patients.

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