Early carotid endarterectomy after acute stroke

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Purpose: **Carotid endarterectomy (CEA) after acute stroke is generally delayed 6 to 8 weeks because of fear of stroke progression. This delay can result in an interval stroke rate of 9% to 15%. We analyzed our results with CEA performed within 1 to 4 weeks of stroke.**
Methods: **Records for all patients undergoing CEA after stroke between 1980 and 2001 were analyzed. Perioperative evaluation included carotid duplex scanning or angiography, and head computed tomography or magnetic resonance imaging. All patients with nonworsening neurologic status, additional brain territory at risk for recurrent stroke, and severe ipsilateral carotid stenosis underwent CEA. Patients were grouped according to time of CEA after stroke: group 1, first week; group 2, second week; group 3, third week; group 4, fourth week. Statistical analysis was performed with the χ^2 test, logistic regression, and analysis of variance.**
Results: **Two hundred twenty-eight patients underwent CEA within 1 to 4 weeks of stroke. Perioperative permanent neurologic deficits occurred in 2.8% of patients in group 1 (72 procedures), 3.4% of patients in group 2 (59 procedures), 3.4% of patients in group 3 (29 procedures), and 2.6% of patients in group 4 (78 procedures). There was no relationship between location or size of preoperative infarct and time of surgery. Only preoperative infarct size correlated with probability of neurologic deficit after CEA ($P < .05$).**
Conclusion: **Incidence of postoperative stroke exacerbation is similar at all intervals. The results are within acceptable limits for treatment of symptomatic carotid stenosis. CEA may be performed within 1 month of stroke with similar results at all intervals during this period. (J Vasc Surg 2004;39:148-54.)**

Although the rationale for treatment of carotid stenosis and minor completed stroke is supported by data from well-controlled prospective trials, the time of carotid endarterectomy (CEA) after acute stroke remains controversial.[1,2] In previous reports, a delay of 6 to 8 weeks between onset of stroke and surgery was recommended to reduce the risk for conversion of a bland infarct to a hemorrhagic infarct.[3,4] Recommendations to delay carotid surgery 6 weeks after stroke were reinforced by the conclusions of the Joint Study of Extracranial Arterial Occlusion[3] and supported by a retrospective study by Giordano et al[4] that demonstrated increased rate of postoperative stroke exacerbation in patients undergoing CEA within 5 weeks of stroke as compared with CEA at later intervals. The dismal outcome in earlier studies was often a result of poor patient selection, inasmuch as these patients often had a profound neurologic deficit in the setting of acute carotid artery occlusion.[5,6]

In several studies the interval between stroke and treatment was shortened to less than 4 weeks.[7-10] In addition, several studies have shown the efficacy of carotid surgery in patients with recent stroke and internal carotid artery occlusion.[11,12] Many of these studies recommend a waiting period in patients with areas of infarction on preoperative brain imaging studies.

We reviewed the results of CEA performed within 1 month of acute stroke to identify factors and surgical practices that might have a positive effect on patient outcome.

METHODS

Records for all patients undergoing CEA after ipsilateral acute stroke between 1980 and 2001 were reviewed. Data had been prospectively collected in a computerized database. Analysis of data for patients who underwent surgery within 1 month of stroke was performed. Patient demographic data, including history of diabetes mellitus, cigarette smoking, hypertension, and coronary artery disease, were identified. When available, preoperative brain computed tomography (CT) scans and magnetic resonance (MR) images were reviewed, and infarct size and lesion location were tabulated according to criteria of the North American Symptomatic Carotid Endarterectomy Trial (NASCET). Preoperative assessment included clinical examination, brain imaging studies, and carotid artery duplex scanning, supplemented with biplanar contrast-enhanced or MR angiography of extracranial carotid and vertebral arteries with intracerebral arterial runoff. In all patients the neurologic deficit at clinical examination had been present for longer than 24 hours or an infarct referable for clinical examination was present on preoperative brain imaging studies. Patients with carotid arteries ipsilateral to the side of clinical infarct with stenosis greater than 50% underwent

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Competition of interest: none.
Presented at the Fifty-Sixth Annual Meeting of The Society for Vascular Surgery, Boston, Mass, June 9-12, 2002.
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Published online Nov 10, 2003.
0741-5214/2004/$30.00 + 0

doi:10.1016/j.jvs.2003.08.003

CEA. In general, patients underwent CEA once neurologic symptoms had reached plateau. The interval from time of stroke to performance of CEA was dictated by the time to referral by the neurologist or physician to the Institute for Vascular Health and Disease at Albany Medical College. In a small subset of patients, operation was performed for stroke in evolution, in which case the interval between stroke and surgery was less than 24 hours. In all patients in this series there was residual cerebral territory at risk for further stroke; that is, there were no complete middle cerebral artery distribution strokes. Patients with profound neurologic deficit, as manifested by dense hemiparesis suggestive of large area middle cerebral artery infarction with obtundation, did not undergo surgical treatment.

CEA was performed with either general anesthesia or deep and superficial cervical plexus blockade, depending on surgeon preference. CEA technique involved either standard longitudinal or eversion endarterectomy, as described.[13] Shunts were placed routinely in patients under general anesthesia and selectively because of neurologic deterioration in patients under cervical blockade anesthesia.

Neurologic deficits occurring after CEA were classified as transient if there was complete clinical resolution within 24 hours and no infarct on repeat brain CT scans or MR images. Permanent deficits included exacerbation of previous clinical deficit lasting longer than 24 hours, with or without demonstrable extension of the preoperative infarct or development of a new ipsilateral cerebral infarct. Postoperative neurologic status was evaluated by the vascular surgical service in all patients, with supplemental evaluation by a neurologist in most patients. In the last 2 years of the study all patients were evaluated preoperatively and postoperatively by a consulting neurologist in addition to the vascular surgical service. Patients were seen postoperatively within 1 month after hospital discharge, at 3 months, and at 6-month intervals thereafter.

Patients were separated into four groups according to interval between stroke onset and performance of CEA: group 1, less than 1 week; group 2, 1 to 2 weeks; group 3, 2 to 3 weeks; group 4, 3 to 4 weeks.

A retrospective analysis was performed to explore the relationship between permanent and transient neurologic deficits occurring after CEA and potential influential variables related to patient demographic data, extent of disease, and diagnostic parameters. Dichotomized outcome variables determined at follow-up included presence or absence of a permanent neurologic deficit, or presence or absence of either a temporary or permanent neurologic deficit. Variables considered potentially associated with these outcomes were diabetes, smoking history, coronary artery disease, hypertension, stroke in evolution, and CT or MRI classification variables before surgery. CT or MRI variables considered included presence of a detectable lesion, lesion size in centimeters, and lesion location (NASCET).[8,14] Binary logistic regression was used to individually assess the association of these potential predictor variables with both criteria of outcome success. Only univariate analysis was performed; the number of adverse events was too small to justify multivariate analysis. The low incidence of neurologic deficit in these patients also has an effect in that type II error is difficult to avoid, so negative findings with regard to potentially influential factors should be treated with caution.

In addition, subanalysis of the differences between groups with respect to demographic criteria and preoperative brain imaging study data, as well as operative parameters such as type of anesthesia, shunt use, and type of endarterectomy, was performed. Beginning in 1999, preoperative and postoperative National Institutes of Health Stroke Scale scores (NIHSSS) were determined. The potential influence of these variables on development of postoperative neurologic deficit was also evaluated. Differences between groups in continuous variables such as patient age, size of infarct, and NIHSSS were examined with analysis of variance (ANOVA). Comparisons between groups in categorical variables were assessed with the χ^2 test or Fisher exact test, when appropriate. Analysis was performed with MINITAB Statistical Software (Minitab, State College, Pa).

RESULTS

Over 22 years, 5845 CEA procedures were performed. The indications for operation were asymptomatic stenosis (>70%) in 3632 procedures (62%), transient ischemic attack in 1104 procedures (19%), amaurosis fugax in 556 procedures (10%), and minor stroke in 553 procedures (9%). Within this group, 238 CEA procedures were performed in 228 patients within 1 month of ipsilateral hemispheric stroke. Although 10 patients ultimately underwent bilateral CEA, no neurologic deficits or deaths were recorded in this group, and subsequent analysis of risk is on a per patient basis rather than a per procedure basis. The study population included 138 men (60%) and 90 women (40%), with mean age 69 years (range, 36-94 years). Perioperative risk factors included diabetes mellitus (n = 57, 25%), cigarette smoking (n = 78, 34%), hypertension (n = 104, 65%), and coronary artery disease (n = 62, 37%). Twelve patients underwent CEA within 24 hours to treat stroke in evolution. The odds ratio (OR) for neurologic deficit after CEA did not achieve statistical significance for any of these variables (Table I), although the number of neurologic deficits was small.

Preoperative brain imaging studies were available for review in 72% (164/228) of patients undergoing CEA prior to acute stroke. Lesions found on preoperative CT and MRI studies were classified according to the criteria utilized in NASCET as previously mentioned. There were 57 (35%) cortical, 27 (16%) small internal borderzone infarcts and 21 (13%) deep infarcts in the vascular territory of perforating arteries. In addition there were 59 (36%) preoperative brain imaging studies in which there was no infarct visualized in spite of acute fixed neurologic deficits. There was no statistically significant association found between NASCET lesion criteria or location of lesion and the development of perioperative neurologic deficit (Table II).

Table I. Association of demographic variables with neurologic deficit in 228 patients

Potential risk factors	*Patients with data*	*Patients with risk factor*		*Permanent neurologic deficit*					*Permanent or temporary neurologic deficit*				
		n	*%*	*n*	*%*	*OR*	*CI*	P	*n*	*%*	*OR*	*CI*	P
All patients	228	228	100	7	3.1				13	5.7			
Diabetes mellitus	228	57	25	0	0				0	0			
Smoking history	228	78	34	3	3.8	1.2	0.3, 5.5	.82	5	6.4	1	0.3, 3.1	.99
Coronary artery disease	167	62	37	0	0				2	3.2	0.5	0.1, 2.4	.36
Hypertension	161	104	65	3	2.9	1.7	0.2, 16.5	.66	5	4.8	0.9	0.2, 4	.91
Age (OR per year of age; mean ± SD)	228	69.2 ± 10.1		69.3 ± 9.7		1	0.9, 1.1	.99	68.6 ± 9.3		1	0.9, 1.1	.81

OR, Odds ratio; *CI*, 95% confidence interval.
Odds ratio not computed when there were fewer than three cases. No risk factors were found to have significant influence (although it should be noted that because of small numbers this lack of association may be a type II error.

Table II. Association of preoperative risk factors with neurologic deficit in 228 patients

	Patients with data	*Patients with risk factor (%)*		*Permanent neurologic deficit*					*Permanent or temporary neurologic deficit*				
		n	*%*	*n*	*%*	*OR*	*CI*	P	*n*	*%*	*OR*	*CI*	P
All patients	228	228	100	7	3.1				13	5.7			
Stroke in evolution	228	12	5	0	0				2	16.7	3.9	0.8, 20	.10
Location (NASCET criteria)													
Borderzone	164	27	16	0	0				1	3.7			
Cortical	164	57	35	4	2.4	8.2	0.9, 75.4	.06	5	8.8	2	0.6, 7.3	.28
Deep	164	21	13	1	0.6	1.8	0.2, 17	.60	2	9.5	1.9	0.4, 9.4	.46
None	164	59	36	0	0				2	3.4	0.43	0.1, 2.1	.28
Size (OR per cm; mean ± SD)	163	1.17 ± 1.42		2.7 ± 1.76		1.7	1.1, 2.8	.03	1.95 ± 1.7		1.4	1, 2	.09
Stenosis (OR per degree)	198	84.7 ± 13.6		82.1 ± 10.7		1	0.9, 1	.59	81.9 ± 12.1		1	1, 1	.42
Pre-NIHSSS	34	5 ± 3.1							6 ± 0		1.1	0.7, 1.7	.64
Post-NIHSSS	34	4.6 ± 3.2							11 ± 7.1		1.8	0.9, 3.6	.11

OR, Odds ratio; *CI*, 95% confidence interval; *NASCET*, North American Symptomatic Carotid Endarterectomy Trial; *NIHSSS*, National Institutes of Health Stroke Scale score.
Odds ratio not computed when there were fewer than three cases. Mean not computed when there were fewer than two cases. Only size was found to have significant influence (although it should be noted that because of small numbers this lack of association may be a type II error).

Table III. Location of preoperative infarct

Interval between stroke onset and CEA (wk)	*Cortical infarct*		*Borderzone infarct*		*Deep infarct*		*No infarct*		*Infarct size (cm; mean ± SD)*	*Patients with data*	*Patients with no data*
	n	*%*	*n*	*%*	*n*	*%*	*n*	*%*			
0-1	21	38	10	17	7	12	18	33	1.4 ± 1.6	56	13
1-2	14	39	4	11	6	17	12	33	1.3 ± 1.3	36	21
2-3	4	21	3	16	3	16	9	47	0.7 ± 0.9	19	9
3-4	18	34	10	19	5	9	20	38	1.0 ± 1.4	53	21

CEA, Carotid endarterectomy.

However, increasing lesion size on preoperative CT scan or MRI increased the odds of permanent neurologic deficit by 1.7 (95% confidence interval [CI], 1.1-2.8) per centimeter of diameter.

With respect to interval between onset of stroke and performance of CEA, there were no differences in infarct location ($P = .89$, χ^2; Table III) or size ($P = .11$, ANOVA). Of 228 patients with acute stroke, CEA was performed in 69 patients within 1 week (group 1), in 57 patients within 1 to 2 weeks (group 2), in 28 patients within 2 to 3 weeks (group 3), and in 74 patients within 3 to 4 weeks (group 4).

Table IV. Association of operative factors with neurologic deficit in 228 patients

Operative factors	*Patients with data*	*Patients with risk factor*		*Permanent neurological deficit*					*Permanent or temporary neuro. deficit*				
		n	*%*	*n*	*%*	*OR*	*CI*	P	*n*	*%*	*OR*	*CI*	P
All patients	228	228	100	7	3.1				13	5.7			
Eversion technique	228	156	68	2	0.9	0.2	0, 1	.05	4	2.6	0.2	0.1, 0.7	.01
Use of shunt	213	22	10	1	0.5	0.9	0.1, 7.5	.90	3	13.6	1.6	0.4, 6.2	.48
General anesthesia (relative to local)	222	25	11	0	0				0	0			
Emergency	225	34	15	0	0				2	5.9	1.1	0.2, 5.1	.92
Use of patch	228	3	1	0	0				1	33.3	6.2	0.6, 63.8	.13
Weeks between stroke and surgery (OR per week; mean ± SD)	228	2.47 ± 1.23		2.4 ± 1.3		1	0.5, 1.8	.92	2.5 ± 1.2		1	0.6, 1.6	.97

Odds ratio not computed when fewer than three cases. Mean not computed when fewer than two cases. Only eversion technique was found to have significant influence (although it should be noted that because of small numbers, lack of association may be a type II error).

Table V. Operative parameters

	Total		*Interval (wk)*								
			0-1		*1-2*		*2-3*		*3-4*		
	n	*%*	*n*	*%*	*n*	*%*	*n*	*%*	*n*	*%*	P
Procedures	228		69		57		28		74		
Eversion CEA	156	68	56	81	41	72	20	71	39	53	.003
Standard CEA	72	31	13	19	16	28	8	29	35	47	
Anesthesia: block	201	88	60	87	49	86	23	82	69	93	.367
Anesthesia: general	27	12	9	13	8	14	5	18	5	7	
Elective shunt	22	9	10	14	6	11	2	7	4	5	.548
Emergent shunt	15	7	5	7	5	9	1	4	4	5	
No shunt	19		54		46		25		66	0	

CEA, Carotid endarterectomy.

Operative factors potentially associated with presence of neurologic deficit are shown in Table IV, and distribution of operative parameters across time intervals are shown in Table V. Only use of the eversion technique was found to have significant influence on neurologic deficit, because patients undergoing eversion endarterectomy had less postoperative neurologic deficit compared with patients undergoing standard CEA (Table IV, last row). The number of weeks between occurrence of stroke and surgery did not influence risk (Table IV), inasmuch as there was no significant difference in neurologic outcome of operative procedure regardless of how long after stroke the surgery was performed. There was also no detected influence on neurologic deficit according to type of anesthesia, or use of a patch or shunt (Table IV).

Two hundred one procedures (88%) were performed with regional block anesthesia, and the remaining 27 procedures were performed with the patient under general anesthesia. Intraluminal shunts were placed in 15 awake patients (7% of the total population of patients) selectively because of neurologic deterioration. Shunts were placed routinely in 11 patients who received block anesthesia and in 11 patients under general anesthesia. Operative parameters separated according to interval between onset of stroke and operation are shown in Table V. There were no differences in CEA technique, type of anesthesia, or use of selective or routine shunting between the four intervals. There was a significant association of eversion CEA technique and interval group, with eversion CEA more likely to be performed early after stroke (Table V).

Operative mortality in the entire cohort was 1.3% (3 of 228 patients). Cause of death was cardiac-related in one patient, who also had a postoperative permanent neurologic deficit (Table VI), and in the other two patients was related to postoperative permanent neurologic deficits. Nonfatal permanent neurologic deficits occurred in an additional four patients, for a stroke rate of 3.1% (7 of 228 patients). Five of these seven patients had associated post-

Table VI. Fatal and nonfatal complications

	Time between stroke onset and time of surgery (wk)									
	0-1		*1-2*		*2-3*		*3-4*		*Total*	
	n	*%*	*n*	*%*	*n*	*%*	*n*	*%*	*n*	*%*
Number of procedures	72		59		29		78		238	
Mortality	0		2	3.4	1	3.4	0		3	1.3
Cardiac	0		1*		0		0		1	0.4
Permanent neurologic deficit	0		1		1		0		2	0.8
Morbidity										
Permanent neurologic deficit	2	2.8	0		0		2	2.6	4	1.7
Transient neurologic deficit	3	4.2	0		0		3	3.8	6	2.5
Occlusion	1	1.4	0		0		2	2.6	3	1.3
Wound hematoma	1	1.4	2	3.4	1	3.4	0		4	1.6
Cardiac	0		1	1.7	0		1	1.3	2	0.8
Nerve injury	0		0		0		1	1.3	1	0.4
Seizure	0		0		0		1	1.3	1	0.4
Total permanent neurologic deficit (fatal and nonfatal)	2	2.8	2	3.4	1	3.4	2	2.6	7	2.9

*Also had permanent neurologic deficit.

Table VII. NIH stroke scale score (mean ± SD)

	Interval between stroke onset and surgery (wk)			
	0-1	*1-2*	*2-3*	*3-4*
Number of patients	21	9	3	1
NIHSSS				
Preoperative	6.38 ± 2.8*	3.67 ± 1.7	0	1
Postoperative	5.9 ± 3.2*	3.44 ± 1.67	0	3
	Depth and location of infarct			
	Cortical	*Borderzone*	*Deep*	*None*
Number of patients	19	8	2	3
NIHSSS				
Preoperative	5.4 ± 3.2	5.0 ± 3.0	4.0 ± 1.4	4.3 ± 3.8
Postoperative	5.3 ± 3.5	3.6 ± 2.0	4.0 ± 1.4	4.3 ± 3.8

NIHSSS, National Institutes of Health Stroke Scale score.
*$P < .05$.

operative CEA thrombosis, with extension of the preoperative infarct on brain imaging studies. In all cases, CEA thrombosis was associated with development of a permanent neurologic deficit. All five patients were returned to the operating room, and emergent reconstruction procedures included carotid artery bypass in four patients and thrombectomy in one patient. Of the remaining two patients, one had hemorrhagic conversion of the preoperative infarct and the other had a new ipsilateral cerebral infarct most likely related to embolization during CEA.

Transient deficits resolving within 24 hours postoperatively occurred in 2.5% of all procedures (6 of 238). There were no new infarcts or exacerbation of previous infarcts in these patients. In addition, there were no postoperative neurologic deficits in patients with stroke in evolution undergoing CEA within 24 hours. Fatal and nonfatal complications according to interval are shown in Table VI. There was no significant difference between incidence of postoperative neurologic deficit between intervals. Mean length of stay was 7 days in groups 1 and 2, 9 days in group 3, and 8 days in group 4.

NIHSSS, compiled for 34 patients in the series, are shown in Table VII. Patients undergoing CEA within 1 week of stroke had significantly greater scores both preoperatively and postoperatively compared with patients who underwent CEA at any other interval ($P < .05$). There were no significant differences between distribution and location of infarcts and NIHSSS before and after CEA. There was, however, a significant relationship between preoperative size of infarct and NIHSSS, determined with linear regres-

sion: NIHSSS = 3.2 + 0.85 × size of preoperative infarct in centimeters ($P < .01$). The coefficient of determination (r^2) was 0.2.

DISCUSSION

The rationale for performing CEA after acute stroke is to preserve neurologic function and prevent stroke exacerbation. Although this was the goal of surgeons performing early CEAs in the late 1950s, the results of surgery after acute stroke were attended by an unacceptably high rate of neurologic complications.[5,6,15] In patients with completed stroke treated by Rob,[5] operative mortality was 21%, with improvement in only 47% of patients at hospital discharge. Similar dismal results were reported by the Joint Study of Extracranial Arterial Occlusive Disease[3] and by Wylie et al.[6] Most of the patients in these series underwent treatment of acute carotid artery thrombosis in the setting of profound preoperative neurologic deficits. Kusunoki et al[16] performed CEA in 40 patients with stroke after carotid thrombosis, with a 15% mortality rate and 17.5% stroke rate. Although these results were not substantially better than in previous studies, these authors were able to identify characteristics of neurologic status that were associated with poor outcome, and suggested that improved results might be obtained in patients at reasonable medical risk with mild, stable deficits.[17]

The emphasis of most recent reports dealing with the time of CEA after acute stroke has been on defining a population of patients who might benefit from surgery within 4 to 6 weeks after stroke onset. Gasecki et al[8] showed that CEA could be performed within 1 month after nondisabling stroke, with mortality and morbidity comparable to that after delayed endarterectomy. Results of the present study show that in patients in whom neurologic symptoms have plateaued after acute stroke, there is no difference in incidence of postoperative neurologic deficit after CEA performed at 1 to 4 weeks after stroke onset. In the present study only 12 CEA procedures were performed to treat stroke in evolution, whereas patients with profound neurologic deficit and obtundation did not undergo CEA. Although 315 patients underwent CEA more than 4 weeks after stroke onset, and are not included in this survey, most of these CEA procedures were performed early in our experience. Stroke-related mortality in these patients was no different from that in other patients with symptoms(9 of 315, 2.8%; $P = .9$, χ^2 test). We do not have complete data for those patients who had strokes or worsening symptoms during the intervening period between initial stroke and CEA. Thus it is impossible to make a valid comparison between the study group and patients in whom CEA was performed more than 4 weeks after stroke.

Selection of an appropriate patient population would seem to be important to obtaining a good result. Dosick et al[17] showed the safety of CEA within 2 weeks of stroke in patients with no infarct on preoperative CT scans. In an earlier report from Ricotta et al,[18] patients with and without CT evidence of infarction had vastly different outcomes after CEA. Patients with an infarct had a 40% incidence of stroke, whereas those with no infarct had a 6% stroke rate. However, there was a heterogeneous group of patients in this study with transient ischemia, fluctuating deficit, and completed stroke; therefore it is difficult to determine which patients with completed stroke had postoperative neurologic decline. In a previous study of patients undergoing CEA within 6 months of stroke, we found no correlation between time of surgery, extent of preoperative infarct, and subsequent development of postoperative neurologic dysfunction.[19] In the present study, although size of infarct affected risk for neurologic deficit, we found that outcome of CEA performed at any time within 1 month of stroke was comparable in patients with similar infarct size and location at presentation.

Although depth and location of preoperative infarct did not correlate with postoperative neurologic deficit, size of infarct did. Risk for permanent neurologic deficit after CEA increased by a factor of 1.73 with each 1-cm increase in preoperative diameter of infarct. This finding differs from most previous reports in which there was no correlation with presence of infarction preoperatively and subsequent risk for stroke.[8,20,21] However, none of the previous reports specifically compared risk for stroke with initial infarct size.

Results of the present study suggest that patients with larger infarcts may be at increased risk for postoperative permanent neurologic deficits. It remains unclear whether waiting longer than 4 weeks will reduce this risk; the present study examined surgical treatment of patients within 1 month of stroke. We have not delayed surgery in these patients once neurologic status has plateaued or evaluated an algorithm for delaying CEA on the basis of size of preoperative infarct; therefore a randomized prospective study will be necessary.

During the last 7 years of the study, there was also a shift in type of CEA technique, from standard longitudinal to eversion endarterectomy starting in the early to mid-1990s. In parallel with the increase in use of the eversion technique, there has been a significant increase in use of routinely placed intraluminal shunts during CEA ($P < .05$). There were fewer postoperative neurologic deficits, either permanent or temporary, with the eversion technique compared with standard endarterectomy: 2.5% (4 of 158) and 11.2% (9 of 80), respectively ($P < .01$). In addition, there were also significantly fewer permanent neurologic deficits after eversion versus standard endarterectomy: 1.3% (2 of 158) and 6.2% (5 of 80), respectively ($P < .05$). Within the group of patients with post-CEA thrombosis, incidence of thrombosis was greater after standard endarterectomy (4 of 80) compared with eversion endarterectomy (1 of 158) ($P < .05$). In the present study, postoperative neurologic deficits in five of seven patients were secondary to thrombosis of the endarterectomy site, and may be directly related to technical errors. Ballotta et al[20] and Riles et al[22] reiterated that the predominant cause of perioperative stroke is technical and can be avoided by improving the details of the procedure.

Also in the latter part of the present study, we inserted intraluminal shunts in more routinely, regardless of whether the procedure was performed with the patient under awake anesthesia with regional block or under general anesthesia. It is therefore difficult to advocate for or against shunt placement. The predominant use of intraluminal shunts during CEA by our group was not a reaction to increased postoperative neurologic deficits with selective shunting, but a concerted effort to minimize cerebral ischemia, extension of infarct, and reperfusion injury during CEA. This change in the paradigm of treatment of acute stroke was instituted by all vascular surgeons in our group. In other studies, Piotrowski et al[9] inserted shunts either prophylactically or after low stump pressure in more than 85% of patients in whom CEA was performed within 6 weeks of stroke. Rosenthal et al[23] demonstrated the unreliability of stump pressure measurement and electroencephalographic monitoring in patients undergoing early CEA after acute stroke. Routine shunting by that group reduced the incidence of postoperative stroke from 38% to 0%. Although increased use of routine shunting in the latter part of our study was not significantly associated with substantially fewer postoperative neurologic deficits, the incidence of neurologic deficit is so small that type II error cannot be ruled out.

Toward the latter part of our study we performed CEA in patients with larger preoperative cerebral infarcts. More of these procedures within the last 2 years of the study were performed less than 1 week from onset of acute stroke. The delay between performance of CEA and onset of stroke was determined primarily by time to referral to the vascular surgeon. Appropriate stroke severity designation and risk stratification has enabled us to treat many of these patients with increasing radiologic and clinical severity, as assessed by CT and MRI studies and NIHSSS, at earlier intervals. Size of preoperative infarct also correlates with stroke score, making this an effective tool to enable stratification of perioperative risk with surgical and or radiologic intervention. Although there was a trend toward improvement in postoperative NIHSSS, it was not statistically significant.

In conclusion, CEA may be performed as early as 1 week after acute stroke, with acceptable results in patients with mild to moderate preoperative neurologic deficits. Use of the eversion endarterectomy technique and routine shunting may contribute to improved patient outcome. Risk for permanent postoperative neurologic deficit may be predicted on the basis of infarct size on preoperative imaging studies.

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Submitted Jun 17, 2002; accepted Aug 4, 2003.