Factors associated with stroke or death after carotid endarterectomy in Northern New England

Philip P. Goodney, MD, Donald S. Likosky, PhD, and **Jack L. Cronenwett, MD, for the Vascular Study Group of Northern New England,** *Lebanon, NH*

Objective: **This study investigated risk factors for stroke or death after carotid endarterectomy (CEA) among hospitals of varying type and size participating in a regional quality improvement effort.**

Methods: **We reviewed 2714 patients undergoing 3092 primary CEAs (excluding combined procedures or redo CEA) at 11 hospitals in Northern New England from January 2003 through December 2007. Hospitals varied in size (25 to 615 beds) and comprised community and teaching hospitals. Fifty surgeons reported results to the database. Trained research personnel prospectively collected >70 demographic and clinical variables for each patient. Multivariate logistic regression models were used to generate odds ratios (ORs) and prediction models for the 30-day postoperative stroke or death rate.**

Results: **Across 3092 CEAs, there were 38 minor strokes, 14 major strokes, and eight deaths (5 stroke-related) ≤30 days of the index procedure (30-day stroke or death rate, 1.8%). In multivariate analyses, emergency CEA (OR, 7.0; 95% confidence interval [CI], 1.8-26.9; $P = .004$), contralateral internal carotid artery occlusion (OR, 2.8; 95% CI, 1.3-6.2; $P = .009$), preoperative ipsilateral cortical stroke (OR, 2.4; 95% CI, 1.1-5.1; $P = .02$), congestive heart failure (OR, 1.6; 95% CI, 1.1-2.4, $P = .03$), and age >70 (OR, 1.3; 95% CI, 0.8-2.3; $P = .315$) were associated with postoperative stroke or death. Preoperative antiplatelet therapy was protective (OR, 0.4; 95% CI, 0.2-0.9; $P = .02$). Risk of stroke or death varied from <1% in patients with no risk factors to nearly 5% with patients with ≥3 risk factors. Our risk prediction model had excellent correlation with observed results ($r = 0.96$) and reasonable discriminative ability (area under receiver operating characteristic curve, 0.71). Risks varied from <1% in asymptomatic patients with no risk factors to nearly 4% in patients with contralateral internal carotid artery occlusion (OR, 3.2; 95% CI, 1.3-8.1; $P = .01$) and age >70 (OR, 2.9; 95% CI, 1.0-4.9, $P = .05$). Two hospitals performed significantly better than expected. These differences were not attributable to surgeon or hospital volume.**

Conclusion: **Surgeons can "risk-stratify" preoperative patients by considering the variables (emergency procedure, contralateral internal carotid artery occlusion, preoperative ipsilateral cortical stroke, congestive heart failure, and age), reducing risk with antiplatelet agents, and informing patients more precisely about their risk of stroke or death after CEA. Risk prediction models can also be used to compare risk-adjusted outcomes between centers, identify best practices, and hopefully, improve overall results. (J Vasc Surg 2008;48:1139-45.)**

Several groups have reported risk factors associated with stroke or death after carotid endarterectomy (CEA), with the intent of improving preoperative assessment and patient selection.[1-6] Across these studies, the most consistent risk factor found to predict stroke or death after CEA has been preoperative neurologic symptoms (Table I).[1-3,5-8] Other variables associated with increased operative risk have included emergency operation,[1] renal failure,[5] and diabetes.[3] Interestingly, preoperative antiplatelet medications were found to be protective in only one of these models,[6] despite level 1 evidence that such therapy reduces stroke risk associated with CEA.[9-12] This is likely because most of the cited studies used administrative data, without access to clinic-level data such as use of antiplatelet agents.[3]

Another role for risk prediction models is to allow comparison of risk-adjusted outcomes among different centers. An important element in surgical quality improvement is to establish benchmarks for performance.[13-15] Benchmarks allow surgeons and hospitals to measure their performance relative to others, which can lead to adoption of best practices.[16] Most outcome benchmarks require risk adjustment, to account for potential differences in patient populations that could influence outcomes.[17] Accordingly, multivariate risk prediction models are developed to account for differences in patient characteristics when outcomes are compared.[18,19]

We have developed a regional quality improvement program in vascular surgery involving 50 surgeons in 11 centers in our region. This group has prospectively collected both detailed patient demographic, operative, and clinical outcome data for CEA since 2003.[20] We hypothesized that a clinic-level database such as this might disclose new patient-level characteristics that were associated with stroke or death after CEA based on contemporary practice in a variety of hospital types.

From the Section of Vascular Surgery Dartmouth-Hitchcock Medical Center.

This project was funded in part with federal funds from The Department of Health and Human Services Centers for Medicare & Medicaid Services (CMS) under Cooperative Agreement Award number 18-C-91674/1/01 awarded to the Trustees of Dartmouth College.

Competition of interest: none.

Presented at the Twenty-First Annual Meeting of the Eastern Vascular Society, Baltimore, Md, Sep 27-29, 2007

Correspondence: Philip P. Goodney, MD, Section of Vascular Surgery, Dartmouth-Hitchcock Medical Center, Lebanon, NH 03765 (e-mail: philip.goodney@hitchcock.org).

0741-5214/$34.00

doi:10.1016/j.jvs.2008.05.013

Table I. Previous studies analyzing risk of stroke or death after carotid endarterectomy

First author (year)	*Patients, No.*	*Study type*	*Risk factors*
Musser (1994)[1]	562	Single-center, retrospective series	AF, emergency operation, PVCs, intra-op hypotension, ESRD
Goldstein (1998)[7]	1160	Multicenter, retrospective administrative database	Female, age >75, CHF
Rothwell (1999)[2]	2060	Multicenter, prospective trial	Symptomatic status, DM, recent MI
Plaque characteristics			
Frawley (2000)[8]	1000	Single-center retrospective series	Female gender
Kresowik (2001)[6]	10,561	Retrospective chart review of Medicare beneficiaries	Aspirin/ticlopidine use, heparin use, patch angioplasty
Tu (2003)[3]	6038	Regional retrospective database	Symptomatic status, AF, contralateral occlusion, CHF, DM
Nicolaides (2005)[5]	1115	Prospective clinical trial	Symptomatic status, degree of stenosis, creatinine

AF, Arial fibrillation; *CHF,* congestive heart failure; *DM,* diabetes mellitus; *ESRD,* end-stage renal disease; *MI,* myocardial infarction; *PVC,* premature ventricular contractions.

METHODS

Subjects and databases. This report uses data collected as part of the Vascular Study Group of Northern New England (VSGNNE), a regional cooperative quality improvement initiative developed in 2002 to study regional outcomes in vascular surgery. Further details on this database have been published previously.[20]

For this analysis, we included only patients who underwent primary CEA (excluding redo operations or those combined with coronary bypass grafting). The analysis included all patients who underwent CEA by participating surgeons in the 11 study hospitals from January 1, 2003, through December 31, 2007. Trained nurses or clinical data abstractors entered data prospectively on >70 clinical and demographic variables. Research analysts were blinded to patient, surgeon, and hospital identity.

The VSGNNE data were validated using discharge claims data from each participating institution. These audits aimed to ensure not only complete inclusion of all CEAs performed in study hospitals but also to confirm our main outcome measure, 30-day postoperative stroke or death. Specifically, we evaluated *International Classification of Diseases, 9th Revision* (ICD9) codes for CEA (38.12), postoperative iatrogenic stroke (997.02), and discharge status (alive, dead).

The validation analyses found initially that 92% of CEAs that had been performed by participating surgeons during the specified time interval at all centers had been entered into the VSGNNE database. Data from the remaining 8% of patients were then retrieved from hospital charts. Thus, this data set represents 100% of CEAs performed by VSGNNE members during the specified time period. An audit of cases with administrative codes for postoperative iatrogenic stroke revealed that these patients were properly recorded in VSGNNE. In addition, three strokes were reported to VSGNNE that had not been coded in claims data. No postoperative strokes captured in claims data had been "missed" by the VSGNNE data-reporting mechanism.

We obtained discharge follow-up status for 100% of patients and 30-day follow-up status for 89% of patients. The 11% of patients for whom long-term follow-up was not available underwent discharge abstract and Social Security Death Index audit. This audit was performed to ensure that no deaths had been reported using claims data but had not been reported to the VSG. This audit failed to reveal any additional deaths that were not reported in the VSG database.

Outcome and variable definitions. We selected 30-day postoperative stroke or death as our main outcome measure. Major stroke was defined as disability causing non-independent living status; minor stroke was defined as nondisabling. The determination of major or minor stroke was made according to these definitions by the nurse clinician or trained research assistant responsible for entering the data into the database at each participating institution. Operative volume categories were defined using cut points guided by prior studies,[21-23] modified to create tertiles of approximately equal size from our data set. Our volume analyses were repeated with quartiles and quintiles, and at several different volume cut points, without any changes in findings.

Preoperative medication usage was defined as administration ≤36 hours before surgery, confirmed by examination of the patient record by the surgeon or research personnel reporting the data. Urgent surgery was defined as surgery required ≤24 hours of admission, and emergency surgery was defined as surgery required ≤6 hours of admission. Symptomatic status was subdivided by symptom type: ocular (visual loss or blurring), cortical (transient ischemic attack [TIA], stroke), vertebrobasilar (ataxia, bilateral motor deficits), and miscellaneous (global or other symptoms).

Risk model construction. To predict 30-day postoperative stroke or death, we initially performed univariate comparisons between our main outcome measures and many patient-level variables. We selected appropriate patient preoperative variables for comparison based on previous publications.[1,3,24-26] Although our major interest in developing this model was to examine preoperative patient risk, we also examined the effect of intraoperative factors such as shunt or patch usage because our goal was to

Table II. Univariate analysis of 30-day stroke or death

Variable	*Stroke/death rate, %*		P
	If not present	*If present*	
Patient characteristics			
Urgent operation	1.7	3.5	.022[a]
Emergency operation	1.7	13.0	.0001[a]
Age >70	1.6	2.0	.378
Age >80	1.8	1.6	.799
Female sex	2.1	1.4	.161
Smoking (prior or current)	1.7	1.8	.989
Diabetes	1.8	1.7	.914
Hypertension	1.4	1.9	.772
β-Blockers	1.8	1.8	.955
Coronary disease	1.7	1.9	.87
Congestive heart failure	1.6	3.5	.045[a]
Degree of stenosis			
Ipsilateral <60%	1.7	5.7	.029[a]
Ipsilateral <70%	1.8	1.5	.789
Ipsilateral <80%	1.8	1.4	.44
Ipsilateral <90%	1.6	1.8	.713
Contralateral ICA occlusion	1.6	4.0	.014[a]
Symptom status			
Ipsilateral symptoms	1.4	2.8	.014[a]
Cortical			
TIA	1.7	2.2	.395
Stroke	1.6	3.5	.007[a]
Ocular	1.7	1.3	.885
Vertebrobasilar	1.7	1.4	.975
Contralateral symptoms (all)	1.7	1.6	.775
Pre-op medication regimen			
Anti-platelet agent			
ASA only	3.1	1.5	.013[a]
Clopidogrel only	1.9	1.0	.141
ASA + clopidogrel	3.3	1.5	.04[a]
Statin use	2.3	1.5	.146
β-Blocker use	1.4	1.7	.526
Operative characteristics			
General anesthesia	0.8	1.8	.268
Routine shunting	1.8	1.6	.671
Shunting for neurologic changes only	1.6	3.2	.088
Eversion endarterectomy	1.7	2.2	.434
Patch angioplasty	2.4	1.5	.096
Dextran use	1.7	2.8	.274
Intravenous antihypertensive use	1.7	2.5	.573
Completion study	1.2	2.6	.014[a]

ASA, acetylsalicylic acid; *TIA,* transient ischemic attack.
[a]Statistically significant ($P < .05$).

develop the most robust risk prediction model available. Variables tested in univariate analysis are reported in Table II. Univariate patient-level predictors that were significant at $P < .10$ were then entered into a multivariate model using backwards stepwise multivariate logistic regression, which was used to generate odds ratios (ORs) and 95% confidence intervals (CIs) for 30-day postoperative stroke or death. The Institutional Review Board at Dartmouth Medical School reviewed and approved our study protocol.

We used the resulting model to calculate an expected stroke/death rate for each patient, based on that particular patient's characteristics. By this method, we were able to risk-adjust outcomes for each hospital by calculating observed (O) to expected (E) stroke or death rates for the patients treated at that hospital. Although 11 participating hospitals contributed CEA data to the registry, we eliminated any center that did not perform at least 50 CEAs during the study period when comparing centers, leaving eight centers available for comparison of O/E outcomes at the "center" level. Kruskal-Wallis comparisons determined significance between O/E rates across centers. All analyses were performed using Excel (Microsoft Corp, Redmond, Wash) and Stata software (StataCorp, College Station, Texas). All tests of significance were performed at the 0.05 level.

Table III. Patient and operative characteristics

Characteristic	*Value*
Total carotid endarterectomies, No.	3092
Average age, years	69
Male	59%
Smoker (current or prior)	79%
History of hypertension	86%
History of diabetes	30%
History of coronary disease	35%
Pre-op β-blocker use	82%
Pre-op antiplatelet agent use	86%
Pre-op statin use	67%
Symptomatic carotid disease (any symptom)[a]	44%
Ocular symptoms, ipsilateral	16%
Ocular symptoms, contralateral	2.5%
Cortical symptoms, ipsilateral	
Transient ischemic attack	22%
Stroke	11%
Cortical symptoms, contralateral	
Transient ischemic attack	1.2%
Stroke	1.7%
Vertebrobasilar symptoms	
Transient ischemic attack	2.3%
Stroke	0.2%
Nonspecific symptoms	5.6%
Operative details	
Urgent/emergency procedure	10%
General anesthesia	92%
Eversion endarterectomy	15%
Shunt	46%
Patch angioplasty	76%

[a]Not additive across symptom categories, as some patients had more than one category of symptoms.

RESULTS

Overall, we studied 2714 patients undergoing 3092 primary CEAs. Most patients were men, with a history of smoking and hypertension, and nearly half were symptomatic (Table III). Most procedures were performed electively, under general anesthesia. Eversion endarterectomy was seldom used, and shunts and patches were commonly used.

Across these 3092 CEAs, we found 38 minor strokes, 14 major strokes, and eight deaths (5 stroke related) ≤30 days of the index procedure, for a 30-day stroke/death rate of 1.8%). Five of the 52 strokes (10%) and two of the eight deaths (25%) were reported after discharge but <30 days.

Table IV. Multivariate analysis of factors associated with 30-day stroke or death after carotid endarterectomy[a]

Variable	*OR*	*95% CI*	P
Age >70 years	1.3	0.8-2.3	0.315
Contralateral ICA occlusion	2.8	1.3-6.2	0.009
Antiplatelet agent use	0.4	0.2-0.9	0.02
Congestive heart failure	1.6	1.1-2.4	0.03
Emergency procedure[b]	7.0	1.8-26.9	0.004
Pre-op ipsilateral cortical symptoms	2.4	1.1-5.1	0.02

CI, Confidence interval; *ICA*, internal carotid artery; *OR*, odds ratio.
[a]Area under receiver operating characteristic curve, 0.71.
[b]A procedure ≤6 hours of admission.

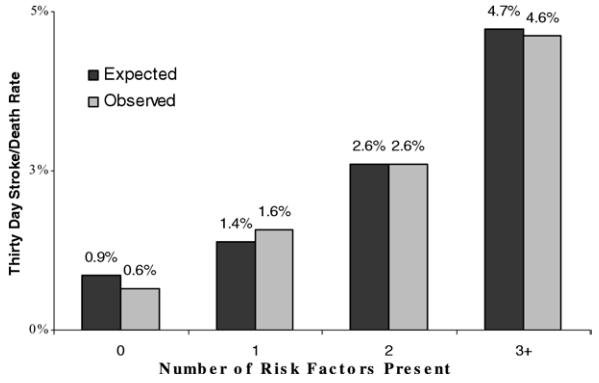

Fig 1. Comparison of observed *(gray bars)* and expected *(black bars)* 30-day stroke/death rate. Risk factors include emergency procedure, preoperative ipsilateral stroke, age >70, lack of antiplatelet agent, contralateral internal carotid artery occlusion, and congestive heart failure.

Significant univariate predictors of ipsilateral stroke/death are reported in Table II. Emergency operation, contralateral internal carotid artery (ICA) occlusion, ipsilateral cortical symptoms (both TIA and stroke), congestive heart failure, performance of a completion study, and ICA stenosis <60% all were associated with an increased risk of 30-day stroke or death. Of the patients with ICA stenosis <60%, 36 of 53 (68%) were symptomatic.

Preoperative use of antiplatelet medications decreased the risk of 30-day stroke or death by nearly 50% in univariate analysis. Operative variables such as shunt use, arteriotomy closure method, dextran infusion, and use of intravenous antihypertensive drugs after surgery were not predictive of stroke or death at 30 days.

By multivariate analysis, emergency procedure, contralateral ICA occlusion, preoperative ipsilateral cortical stroke, congestive heart failure, and age >70 years were associated with a significantly higher risk of stroke or death, whereas preoperative acetylsalicylic acid (ASA) or clopidogrel use was protective (Table IV). We examined the impact and additive effects of the number of risk factors present (Fig 1). In patients with none to one of these risk factors, the risk of stroke or death was <1%; however, when patients had three or more risk factors, the risk of stroke or death increased to nearly 5%. When expected stroke or death rates were compared with the observed stroke or death rates, excellent correlation was observed ($r = 0.96$). The model was found to have reasonable discriminative ability (area under receiver operating characteristic [AUROC] curve, 0.71).

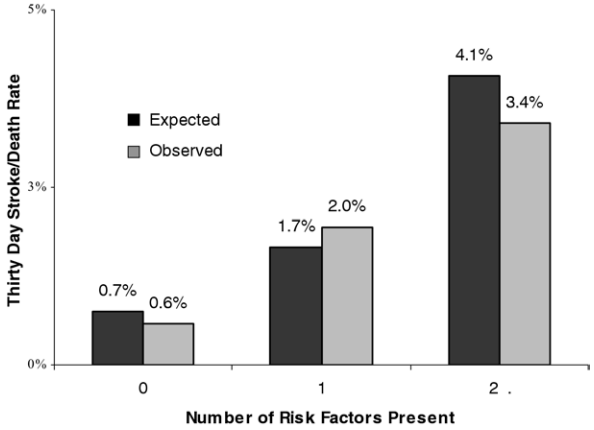

Fig 2. Comparison of observed *(gray bars)* and expected (black bars) 30-day stroke/death rate, in asymptomatic patients. Risk factors include age >70 and contralateral internal carotid artery occlusion.

We repeated our analysis in the subgroup of asymptomatic patients. Multivariate analysis showed that contralateral ICA occlusion (OR, 3.7; 95% CI, 1.5-9.4; $P = .006$) and age >70 years (OR, 2.9; 95% CI, 1.0-4.9; $P = .05$) were associated with an increased risk of 30-day stroke or death. The predicted patient risk of stroke or death in asymptomatic patients varied from <1% in patients with no risk factors to nearly 4% in patients aged >70 years with a contralateral ICA occlusion (Fig 2). This model also had reasonable discriminative ability (AUROC curve, 0.69), but very few patients (88 of 1973) were within the highest risk category, limiting the generalizability of these findings in higher-risk asymptomatic patients.

To benchmark performance across hospitals, we calculated expected 30-day stroke/death rates for each patient using our prediction model and compared observed and expected stroke/death rates by hospital (Table V). Across eight hospitals, the expected stroke/death rate ranged from 1.5% to 2.0%, whereas observed results varied from 0% to >4%. Two hospitals had O/E ratios >1, indicating a higher observed 30-day stroke/death rate than expected. The remaining six hospitals all had O/E ratios of <1, indicating lower 30-day stroke/death rates than expected; in two of these hospitals, these differences were statistically significant.

Differences across hospitals were not attributable to differences in hospital or surgeon volume (Fig 3). In fact, the hospital with the highest volume in our analysis (975 CEAs performed by 10 surgeons) had the highest 30-day stroke/death rate, and a hospital with relatively small vol-

Table V. Comparison of observed and expected 30-day stroke/death rates by hospital[a]

Variable	*Overall*	*Hospital*							
		1	*2*	*3*	*4*	*5*	*6*	*7*	*8*
CEAs, No.	3092	162	975	244	385	329	90	285	561
Risk of stroke/death									
Expected	1.8%	2.0%	2.0%	1.9%	1.8%	1.5%	1.6%	1.5%	1.6%
Observed	1.8%	4.3%	3.0%	1.2%	1.3%	0.9%	0.0%	1.1%	0.5%
O/E ratio[b]		2.1	1.5	0.63	0.73	0.60	0.0[c]	0.67	0.32[c]
P (observed vs expected)		.132	.06	.316	.412	.252	.001[c]	.415	.002[c]

CEA, Carotid endarterectomy.

[a]Only hospitals performing at least 50 CEAs during the study period were included in this analysis; therefore, 3 centers representing a total of 61 CEAs were eliminated from this portion of the analysis.

[b]Ratio of observed-to-expected 30-day stroke/death rates. A number >1 indicates a higher stroke/death rate than expected, and a number <1 indicates a lower stroke/death rate than expected.

[c]Indicate observed stroke/death rates significantly higher or lower than expected.

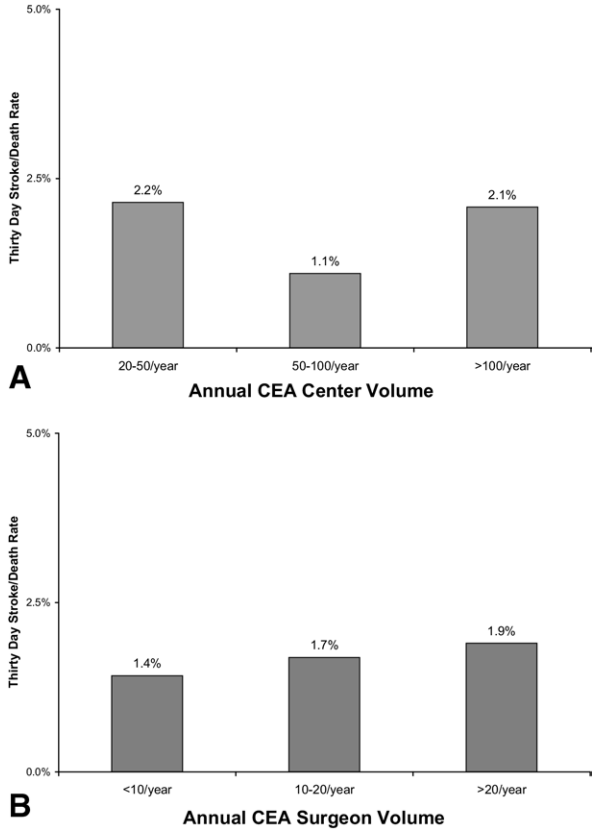

Fig 3. Thirty-day stroke/death rate by **(A)** annual hospital volume of carotid endarterectomies *(CEA; P* = .143 across volume categories) and **(B)** annual surgeon volume (*P* = .642 across volume categories).

ume (90 CEAs performed by 5 surgeons) had no strokes or deaths during the study period.

DISCUSSION

In contrast with previous models for predicting stroke or death after CEA, our model was derived from a prospective collection of contemporary data derived from 50 surgeons in 11 hospitals that varied in size from 25 to 615 beds and comprised both academic and community centers. We identified emergency procedure, contralateral ICA occlusion, preoperative cortical stroke, congestive heart failure, increased age, and lack of antiplatelet agent use as predictive of postoperative 30-day stroke or death after CEA.

Our model describes several risk factors reported in previous studies (Table I), with similar effect sizes. Two prior models reported an OR of 1.8 for 30-day stroke or death for contralateral ICA occlusion, which is similar in magnitude to our OR of 2.8.[2,3] These same studies[2,3] also reported that ipsilateral cerebral symptoms increased the risk of stroke and death nearly twofold, similar to our finding of an OR of 2.4 for preoperative ipsilateral stroke. We did not find that preoperative cortical TIA or ocular symptoms were significantly predictive of stroke or death; only preoperative ipsilateral stroke predicted 30-day postoperative stroke or death. Although we suspect that this represents a type II error, this finding may also reflect the increased impact of previous stroke, compared with TIAs. Patients with preoperative stroke before CEA have been shown to be at higher risk for postoperative stroke, independent of whether or not they have TIAs at the time of CEA.[27] Lastly, emergency CEA, usually indicated for free-floating thrombus or crescendo symptoms, has been well described as having worse surgical outcomes compared with routine or urgent CEA,[28] in keeping with our findings. Common risk factors with similar effect sizes to previous studies lend credence to the generalizability of our model.

Our finding of a protective effect of preoperative ASA or clopidogrel is not surprising given previous research. The benefit of ASA to reduce postoperative stroke or death was first shown in a secondary analysis from the North American Symptomatic Carotid Endarterectomy Trial[29] in 1991. Subsequently, a small, randomized trial showed that stroke rates were reduced with lower-dose perioperative ASA (81 mg/d) vs placebo.[9] The ASA and Carotid Endarterectomy (ACE) trial[12] further emphasized the benefit of low-dose ASA by randomizing 2849 patients to escalating

aspirin doses, noting that the combined end point of stroke, death, or myocardial infarction was lowest in the lower-dose (81 and 325 mg) groups than with higher doses. Finally, Kresowik et al[6] noted a 30% reduction in stroke or death with the use of preoperative antiplatelet therapy in review of >10,000 Medicare beneficiaries undergoing CEA between 1995 and 1996. Other antiplatelet agents have not been as extensively studied in terms of their benefit for stroke reduction after CEA, but recent studies of clopidogrel provide evidence that perioperative embolization is reduced.[10,11] Given this body of evidence, it is not surprising that our risk prediction model, based on clinic-level detail, noted a protective effect from antiplatelet therapy.

Clearly, perioperative antiplatelet medication use is simple method to substantially reduce stroke and death after CEA, and many consider its administration "routine." However, a recent study from the Netherlands indicated that only 66% of patients undergoing CEA were receiving preoperative antiplatelet agents, showing the opportunity for improvement using this simple process.[30] Even within our region, before starting our quality improvement efforts, only 73% of patients undergoing CEA were taking antiplatelet agents. However, this percentage has increased to >93% after dedicated efforts to improve this process measure across our participating centers.[31]

After risk adjustment for underlying patient characteristics, we found residual differences in outcome across hospitals, which are likely due to differences in processes of care. Our analysis did not disclose any significant influence of operative variables such as shunt use, arteriotomy closure method, dextran infusion, or use of intravenous antihypertensives after surgery. Our plan is to use benchmarking site visits to identify processes of care that differ across surgeons and hospitals, with the hope of identifying "best practices" in CEA. After identifying the processes of care that produce the best results, we hope to implement these processes to improve care across all centers, a technique that has been successfully used by cardiac surgeons in our region.[32]

Although our study found that outcome was not influenced by surgeon or hospital volume, this is likely a type II error given prior publications on this topic.[21-23] Most studies that found a surgeon volume effect for CEA outcome required hundreds of surgeons and tens of thousands of patients to find a relatively small effect,[21,22] whereas we studied only 50 surgeons performing 3000 CEAs. Nonetheless, our analysis found no dramatic influence on outcome differences between centers in our region according to volume.

Our subgroup analysis of asymptomatic patients found two important predictors of stroke or death after CEA: contralateral ICA occlusion and age >70. Stroke or death rates were acceptable in patients with only one (or none) of these risk factors, but patients with both contralateral ICA occlusion and age >70 had a predicted stroke risk of >4%. Although conclusions are limited by the small number of patients meeting these criteria in our population, these data warrant further investigation, because a stroke or death rate of 4% in asymptomatic patients is outside the clinical guidelines for performing CEA.[33] If confirmed in larger analyses, these data would indicate the need for better patient selection in the treatment of asymptomatic carotid stenosis.

Our study has several limitations. First, our relatively low event rate may have resulted in a type II error, limiting our ability to identify other predictors of outcome after CEA, such as a benefit for statins, as has been shown in other series.[34-36] However, the significant risk factors that we identified agree with previous reports and are likely valid.

Second, 30-day follow-up was missing in our database in 11% of patients. We used the Social Security Death Index to ensure that there were no unreported deaths in this group. We estimate that we may have missed one to two strokes in this period, based on our analysis of patients with complete follow-up, in which only 10% of postoperative strokes occurred after discharge but <30 days. This could have increased our postoperative stroke/death rate from 1.8% to 2.0 to 2.2%, but should not have affected our risk adjustment calculations.

CONCLUSIONS

We identified factors associated with 30-day stroke or death after CEA by using our regional prospective database. Surgeons can easily preoperatively "risk-stratify" patients by considering these easily available variables—emergency nature of the procedure, contralateral ICA occlusion, preoperative ipsilateral cortical stroke, congestive heart failure, and age—which may aid decision making for patients who are at the borderline for benefit from CEA. In addition, by using perioperative antiplatelet agents, surgeons can impart measurable risk reduction. Finally, risk adjustment models like this allow valuable benchmarking among different centers.

AUTHOR CONTRIBUTIONS

Conception and design: PG, JC
Analysis and interpretation: PG, DL, JC
Data collection: DL, JC
Writing the article: PG, JC
Critical revision of the article: PG, DL, JC
Final approval of the article: PG, DL, JC
Statistical analysis: PG, DL
Obtained funding: JC
Overall responsibility: PG

REFERENCES

1. Musser DJ, Nicholas GG, Reed JF 3rd. Death and adverse cardiac events after carotid endarterectomy. J Vasc Surg 1994;19:615-22.
2. Rothwell PM, Warlow CP. Prediction of benefit from carotid endarterectomy in individual patients: a risk-modelling study. European Carotid Surgery Trialists' Collaborative Group. [see comment]. Lancet 1999; 353:2105-10.
3. Tu JV, Wang H, Bowyer B, Green L, Fang J, Kucey D, et al. Risk factors for death or stroke after carotid endarterectomy: observations from the Ontario Carotid Endarterectomy Registry. [see comment]. Stroke 2003;34: 2568-73.
4. Sacco RL, Benjamin EJ, Broderick JP, Dyken M, Easton JD, Feinberg WM, et al. American Heart Association Prevention Conference. IV.

Prevention and Rehabilitation of Stroke. Risk factors. Stroke 1999;28:1507-17.
5. Kakkos SK, Nicolaides A, Griffin M, Sabetai M, Dhanjil S, Thomas DJ, et al. Factors associated with mortality in patients with asymptomatic carotid stenosis: results from the ACSRS Study. Int Angiol 2005;24: 221-30.
6. Kresowik TF, Bratzler D, Karp HR, Hemann RA, Hendel ME, Grund SL, et al. Multistate utilization, processes, and outcomes of carotid endarterectomy. J Vasc Surg 2001;33:227-34; discussion 234-5.
7. Goldstein LB. Extracranial carotid artery stenosis. Stroke 2003;34: 2767-73.
8. Frawley JE, Hicks RG, Woodforth IJ. Risk factors for peri-operative stroke complicating carotid endarterectomy: selective analysis of a prospective audit of 1000 consecutive operations. Aust N Z J Surg 2000; 70:52-6.
9. Lindblad B, Persson NH, Takolander R, Bergqvist D. Does low-dose acetylsalicylic acid prevent stroke after carotid surgery? A double-blind, placebo-controlled randomized trial. Stroke 1993;24:1125-8.
10. Payne DA, Jones CI, Hayes PD, Naylor AR, Goodall AH. Therapeutic benefit of low-dose clopidogrel in patients undergoing carotid surgery is linked to variability in the platelet adenosine diphosphate response and patients' weight. Stroke 2007;38:2464-9.
11. Payne DA, Jones CI, Hayes PD, Thompson MM, London NJ, Bell PR, et al. Beneficial effects of clopidogrel combined with aspirin in reducing cerebral emboli in patients undergoing carotid endarterectomy. [see comment]. Circulation 2004;109:1476-81.
12. Taylor DW, Barnett HJ, Haynes RB, Ferguson GG, Sackett DL, Thorpe KE, et al. Low-dose and high-dose acetylsalicylic acid for patients undergoing carotid endarterectomy: a randomised controlled trial. ASA and Carotid Endarterectomy (ACE) Trial Collaborators. [see comment]. Lancet 1999;353:2179-84.
13. Halm EA, Lee C, Chassin MR. Is volume related to outcome in health care? A systematic review and methodologic critique of the literature. [summary for patients in Ann Intern Med. 2002;137:I52; PMID: 12230383]. Ann Intern Med 2002;137:511-50.
14. Pine M, Jordan HS, Elixhauser A, Fry DE, Hoaglin DC, Jones B, et al. Enhancement of claims data to improve risk adjustment of hospital mortality. JAMA 2007;297:71-6.
15. Teres D, Higgins TL. What if your hospital informatics department could provide a severity adjuster? [comment]. Crit Care Med 2000;28: 3570-1.
16. El-Tamer MB, Ward BM, Schifftner T, Neumayer L, Khuri S, Henderson W. Morbidity and mortality following breast cancer surgery in women: national benchmarks for standards of care. [see comment]. Ann Surg 2007;245:665-71.
17. Brass EP, Hiatt WR. Review of mortality and cardiovascular event rates in patients enrolled in clinical trials for claudication therapies. Vasc Med 2006;11:141-5.
18. Wyse RKH, Taylor KM. Using the STS and multinational cardiac surgical databases to establish risk-adjusted benchmarks for clinical outcomes. Heart Surg Forum 2002;5:258-64.
19. Peterson ED, DeLong ER, Muhlbaier LH, Rosen AB, Buell HE, Kiefe CI, et al. Challenges in comparing risk-adjusted bypass surgery mortality results: results from the Cooperative Cardiovascular Project. J Am Coll Cardiol 2000;36:2174-84.
20. Cronenwett JL, Russell MT, Likosky DS, Eldrup-Jorgensen J, Stanley AC, Nolan BW, et al. A regional registry for quality assurance and improvement: the Vascular Study Group of Northern New England (VSG-NNE). J Vasc Surg 2007;46:1093-101.
21. Birkmeyer JD, Stukel TA, Siewers AE, Goodney PP, Wennberg DE, Lucas FL. Surgeon volume and operative mortality in the United States. [see comment]. N Engl J Med 2003;349:2117-27.
22. Cowan JA Jr, Dimick JB, Thompson BG, Stanley JC, Upchurch GR Jr. Surgeon volume as an indicator of outcomes after carotid endarterectomy: an effect independent of specialty practice and hospital volume. [see comment]. J Am Coll Surg 2002;195:814-21.
23. Holt PJ, Poloniecki JD, Loftus IM, Thompson MM. Meta-analysis and systematic review of the relationship between hospital volume and outcome following carotid endarterectomy. Eur J Vasc Endovasc Surg 2007;33:645-51.
24. Anonymous. Endarterectomy for asymptomatic carotid artery stenosis. Executive Committee for the Asymptomatic Carotid Atherosclerosis Study. [see comment]. JAMA 1421;273:1421-8.
25. Ballotta E, Renon L, Da Giau G, Barbon B, Terranova O, Baracchini C. Octogenarians with contralateral carotid artery occlusion: a cohort at higher risk for carotid endarterectomy? J Vasc Surg 2003;39:1003-8.
26. Tu JV, Hannan EL, Anderson GM, Wu K, Vranizan K, Popp AJ, et al. The fall and rise of carotid endarterectomy in the United States and Canada. N Engl J Med 1998;339:1441-47.
27. Eliasziw M, Streifler JY, Spence JD, Fox AJ, Hachinski VC, Barnett HJ. Prognosis for patients following a transient ischemic attack with and without a cerebral infarction on brain CT. North American Symptomatic Carotid Endarterectomy Trial (NASCET) Group. Neurology 1995;45:428-31.
28. Bhatti AF, Leon LR Jr, Labropoulos N, Rubinas TL, Rodriguez H, Kalman PG, et al. Free-floating thrombus of the carotid artery: literature review and case reports. J Vasc Surg 2007;45:199-205.
29. Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. North American Symptomatic Carotid Endarterectomy Trial Collaborators. [see comment]. N Engl J Med 1991;325:445-53.
30. Assadian A, Eidher U, Senekowitsch C, Rotter R, Löchli S, Hagmüller GW, et al. Antiplatelet therapy prior to carotid endarterectomy–still room for improvement. Vasa 2006;35:96-100.
31. Cronenwett JL, Likosky DS, Russell MT, Eldrup-Jorgensen J, Stanley AC, Nolan BW, et al. A regional registry for quality assurance and improvement: the Vascular Study Group of Northern New England (VSGNNE). J Vasc Surg 2007;46:1093-1101; discussion 1101-2.
32. O'Connor GT, Plume SK, Olmstead EM, Morton JR, Maloney CT, Nugent WC, et al. A regional prospective study of in-hospital mortality associated with coronary artery bypass grafting. The Northern New England Cardiovascular Disease Study Group. [see comment]. JAMA 1991;266:803-9.
33. Endarterectomy for asymptomatic carotid artery stenosis. Executive Committee for the Asymptomatic Carotid Atherosclerosis Study. JAMA 1991;273:1421-8.
34. Perler BA. The effect of statin medications on perioperative and long-term outcomes following carotid endarterectomy or stenting. Semin Vasc Surg 2007;20:252-8.
35. McGirt MJ, Perler BA, Brooke BS, Woodworth GF, Coon A, Jain S, et al. 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors reduce the risk of perioperative stroke and mortality after carotid endarterectomy. [see comment]. J Vasc Surg Nov 2005;42:829-836; discussion 836-827.
36. Kennedy J, Quan H, Buchan AM, Ghali WA, Feasby TE. Statins are associated with better outcomes after carotid endarterectomy in symptomatic patients. [see comment]. Stroke 2005;36:2072-6.

Submitted Feb 17, 2008; accepted May 4, 2008.