Severe contralateral carotid stenosis or occlusion does not have an impact on risk of ipsilateral stroke after carotid endarterectomy

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ABSTRACT

Objective: This study evaluates the impact of severe (>70%) contralateral carotid stenosis or occlusion (SCSO) on outcomes after carotid endarterectomy (CEA).

Methods: Clinical data for all patients undergoing CEA at a single center were prospectively gathered and retrospectively reviewed, with the sample population stratified according to the presence of SCSO. Perioperative outcomes of CEA in the presence of SCSO were analyzed using univariate and multivariate methods.

Results: During a 17-year study period, 2945 CEAs were performed on 1843 patients, including 736 (25%) patients with SCSO. Patients identified with SCSO had a higher rate of positive intraoperative electroencephalographic changes (30% vs 16%; $P < .0001$) and use of an intraoperative shunt (40% vs 28%; $P < .0001$). Univariate analysis identified SCSO as a risk factor for any stroke (2.8% vs 1.5%; $P = .02$), death (2.2% vs 1.1%; $P = .02$), and any stroke/death (4.3% vs 2.4%; $P < .0079$) but not ipsilateral stroke (1.5% vs 1.2%; $P = .38$). Multivariable regression demonstrated SCSO as an independent predictor of any stroke (odds ratio [OR], 1.8; 95% confidence interval [CI], 1.0-3.3; $P = .05$) and any stroke/death (OR, 1.7; 95% CI, 1.1-2.7; $P = .02$), without increasing risk of ipsilateral stroke (OR, 1.3; 95% CI, 0.6-2.7; $P = .54$). The presence of SCSO was also associated with a higher risk of late mortality (hazard ratio, 1.3; 95% CI, 1.1-1.4; $P < .01$).

Conclusions: Although the presence of SCSO is a risk factor for any stroke/death with CEA, it does not increase the risk of ipsilateral stroke. These data suggest that increased attention to perioperative medical and hemodynamic management should be especially considered in this cohort of patients as the observed strokes do not occur in the territory at risk from the surgical procedure. (J Vasc Surg 2018;67:1744-51.)

Carotid endarterectomy (CEA) is the "gold standard" surgical treatment for prevention of cerebrovascular events in symptomatic and asymptomatic patients with severe stenosis of the internal carotid artery[1-4]; however, the reported effect of severe (>70%) contralateral carotid stenosis or occlusion (SCSO) on perioperative and long-term outcome of CEA is variable.

The North American Symptomatic Carotid Endarterectomy Trial (NASCET)[5,6] reported an increased risk of perioperative stroke after CEA in patients with contralateral carotid artery occlusion. Despite increased perioperative risk, surgical intervention in the NASCET population of patients provided greater benefit than medical management alone. In post hoc analysis of the Asymptomatic Carotid Atherosclerosis Study (ACAS), CEA in asymptomatic patients with contralateral carotid occlusion increased the risk of perioperative stroke compared with those with patent contralateral vessels.[7]

Several multicenter contemporary studies have demonstrated that pre-existing SCSO is a risk factor for inferior outcomes after CEA.[8-14] Others have reported marginally higher stroke risk in patients with contralateral carotid disease,[15,16] whereas many single-center studies found comparable perioperative results for both groups.[17-22]

The goal of this study was to determine the early and late outcomes of CEA in patients with SCSO compared with those with a normal artery and those with moderate contralateral carotid artery stenosis.

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METHODS

Study design. The Institutional Review Board at the Massachusetts General Hospital reviewed and approved this clinical protocol. Given the retrospective nature of the study and use of deidentified data, informed consent was waived by the Institutional Review Board.

All consecutive CEAs performed from January 1989 to December 2005 by the Massachusetts General Hospital Division of Vascular Surgery were prospectively collected and retrospectively analyzed. We identified 2945 CEAs performed on 1843 patients for whom contralateral carotid artery disease and perioperative outcomes data were available. Patients who underwent concurrent procedures, such as coronary artery bypass graft or proximal common carotid artery stenting (CAS), at the time of CEA were excluded from this study. Decisions about technical management of the operation were left to the individual surgeon. For patients undergoing selective shunting, a 10% change in electroencephalographic (EEG) monitoring was used as an indication for shunt placement.

Patients were retrospectively stratified into two groups. The nonsevere group (n = 2209) included those patients with a patent contralateral internal carotid artery and those with mild (<50%) or moderate (50%-70%) contralateral carotid artery disease. The severe group (n = 736) included those with severe stenosis (70%-89%), very severe stenosis (90%-99%), and an occluded contralateral internal carotid artery. Ultrasound imaging was performed to measure the degree of carotid artery stenosis; our institution used measuring guidelines set forth by the Intersocietal Accreditation Commission of Vascular Testing.

Patient demographics, clinical data, and perioperative and intraoperative factors were obtained through retrospective review of hospital medical records. Long-term stroke, restenosis, re-exploration, and readmission data were gathered from all outpatient clinic visits, hospital admissions, carotid noninvasive studies, and relevant radiologic studies.

Four to 6 weeks after the index procedure, patients underwent carotid noninvasive studies. Neurologic examination was performed by the treating surgeon at the 1-month, 6-month, and 1-year period to evaluate for signs of adverse neurologic event. Patients were observed annually thereafter at the discretion of the surgeon. Hospital medical records and the Social Security Death Index, when necessary, were used to determine long-term survival.

Definitions and end points. Symptomatic disease was defined as ipsilateral stroke, transient ischemic attack, or amaurosis fugax occurring within the 6 months preceding CEA. Antilipid treatment included statin and nonstatin therapy. Primary study end points to identify major determinants of morbidity and mortality included 30-day and long-term ipsilateral stroke as well as death and ipsilateral stroke/death; the secondary end point was any stroke/death.

ARTICLE HIGHLIGHTS

- **Type of Research:** Single-center retrospective analysis of prospectively collected data
- **Take Home Message:** After 2945 carotid endarterectomies in 1843 patients, the odds of any stroke (2.8%; odds ratio, 1.8), any stroke/death (4.3%; odds ratio, 1.7), and late mortality (hazard ratio, 1.3) were increased in patients with contralateral severe carotid artery stenosis or occlusion, whereas the incidence of ipsilateral stroke was no different.
- **Recommendation:** This study suggests that the presence of contralateral severe carotid stenosis or occlusion in patients undergoing carotid endarterectomy increases the risk of stroke and death but not the risk of ipsilateral stroke.

Statistical analysis. All statistical calculations were performed using SAS 9.2 software (SAS Institute, Cary, NC). Univariate analysis was performed to compare baseline clinical and demographic features, operative details, and perioperative outcomes. Continuous variables are presented as mean value and 95% confidence intervals (CIs). Discrete variables are presented as number of events and population percentages. Logistic regression models were used to determine factors associated with 30-day any stroke, ipsilateral stroke, death, and any stroke/death. A Kaplan-Meier product limit estimator curve was plotted to compare survival functions between patients with SCSO and those without severe contralateral disease. A multivariable risk-adjusted Cox proportional hazards model was used to obtain hazard ratios (HRs), 95% CIs, and *P* values for independent predictors of late mortality. Statistical significance was defined as a two-tailed *P* value of $<.05$.

RESULTS

During the 17-year study period beginning in 1989 and ending in 2005, 2945 CEA procedures were performed on 1843 patients with contralateral carotid stenosis and available perioperative outcomes data. The mean age of patients included in this study was 71 years (standard deviation, ±8 years); 60% were male. After stratification based on the presence or absence of contralateral carotid artery disease, 736 procedures were performed on patients with SCSO (11.1% severe stenosis, 5.3% very severe stenosis, 8.6% occluded). The remaining 2209 procedures were classified as having nonsevere contralateral internal carotid artery disease (25.4% patent, 25.4% mild stenosis, 24.1% moderate stenosis).

Demographic and clinical features are reported in Table I. The two groups were comparable in age, gender,

Table I. Clinical features of patients undergoing carotid endarterectomy (*CEA*) comparing those with vs those without severe contralateral carotid stenosis or occlusion (SCSO)

Variable	All procedures (N = 2945)	Nonsevere, <70% (n = 2209)	Severe, >70% (n = 736)	*P* value
Age, years	71.6 ± 8.7	71.2 ± 8.7	71.3 ± 8.6	.86
Male gender	60.8	59.6	64.5	.06
History of CAD	55.7	45.3	41.2	.12
History of HTN	82.4	82.0	83.4	.51
History of COPD	14.7	15.0	13.6	.45
History of diabetes	23.6	23.9	22.5	.55
History of XRT	2.5	2.6	2.4	.85
Current smoker	21.6	20.7	24.5	.099
Any lipid treatment	70.3	72.2	64.5	.0017
Contralateral CEA	27.3	21.6	44.4	<.0001
Redo (of past CEA)	3.6	3.8	3.1	.42
Symptomatic presentation	28.8	28.0	31.0	.12

CAD, Coronary artery disease; *COPD*, chronic obstructive pulmonary disease; *HTN*, hypertension; *XRT*, neck irradiation.
Categorical variables are presented as percentages. Continuous variables are presented as mean ± standard deviation.

and rate of symptomatic presentation. Patients without severe disease had higher rate of any lipid treatment (72.2% vs 64.5%; $P = .0017$). No significant univariate differences were found between both groups with regard to common cardiovascular risk factors.

Procedural details are presented in Table II. Patients with SCSO had a higher rate of primary closure (38.4% vs 26.9%; $P < .0001$), positive intraoperative EEG changes (30.5% vs 16.7%; $P < .0001$), and intraoperative shunt placement (40.3% vs 28.1%; $P < .0001$).

Perioperative outcomes. Perioperative outcomes are presented in Table III. For all patients, the 30-day any stroke rate was 1.8% and ipsilateral stroke rate was 1.3%. The 30-day mortality rate for all patients was 1.4%, with a combined any stroke/death rate of 2.9%. Compared with nonsevere disease, SCSO had significantly higher rates of any stroke (2.8% vs 1.5%; $P = .024$), death (2.2% vs 1.1%; $P = .027$), and any stroke/death (4.3% vs 2.4%; $P = .0079$). There was no significant difference for ipsilateral stroke in both groups (1.5% vs 1.2%; $P = .38$). Perioperative rates of myocardial infarction (MI; 3.5% vs 1.7%; $P = .05$), surgical re-exploration (4.9% vs 1.0%; $P < .001$), and hospital readmission (7.3% vs 3.4%; $P < .002$) were significantly higher in the SCSO group.

Multivariable logistic regression modeling found that SCSO was an independent predictor of 30-day any stroke (odds ratio [OR], 1.8; 95% CI, 1.0-3.2; $P = .05$) and 30-day any stroke/death (OR, 1.7; 95% CI, 1.1-2.7; $P = .02$). SCSO was not an independent predictor of 30-day ipsilateral stroke (OR, 1.3; 95% CI, 0.6-2.7; $P = .54$) or 30-day death (OR, 0.9; 95% CI, 0.4-2.1; $P = .86$).

Table IV reports the multivariate predictors of 30-day outcomes after CEA for our primary end points. Symptomatic presentation and male gender were independent predictors of any stroke, ipsilateral stroke, and any stroke/death at 30 days after CEA. In addition, redo CEA and history of diabetes were predictors of 30-day any stroke and ipsilateral stroke. Current smoking status was the only predictor of 30-day death after CEA.

Late outcomes. Survival after CEA in patients without severe contralateral disease was 95.2%, 85.6%, 71.5%, and 43.5% ± 1% at 1 year, 3 years, 5 years, and 10 years. Long-term survival after CEA in patients with SCSO was 91.5%, 78.1%, 63.9%, and 34.1% ± 2% at 1 year, 3 years, 5 years, and 10 years. SCSO was associated with a statistically lower ($P < .001$) late survival rate (Fig).

Multivariable Cox proportional hazards regression modeling was performed to evaluate predictors of late mortality (Table V). SCSO significantly increased the risk of late mortality (HR, 1.3; 95% CI, 1.1-1.4; $P < .0001$). Additional predictors of late mortality included symptomatic presentation before CEA, age, and smoking status and history of coronary artery disease, chronic obstructive pulmonary disease, diabetes mellitus, and prior neck irradiation. Any lipid therapy and male gender were protective for late mortality after CEA.

DISCUSSION

Our data demonstrate that SCSO is a predictor of early and late adverse neurologic and cardiovascular outcomes after CEA. SCSO is an independent predictor of 30-day any stroke and 30-day any stroke/death. These findings are consistent with previously reported results.[23] Using the Vascular Study Group of Northern New England regional quality data set, Goodney et al[11] analyzed 30-day stroke/death rates in 3092 CEAs performed across 11 hospitals from 2003 to 2007. In multivariate analysis, they found that contralateral carotid occlusion was an independent predictor of any perioperative stroke/death (OR, 2.8; 95% CI, 1.3-6.2; $P = .009$).

Table II. Procedural details of patients undergoing carotid endarterectomy (CEA) comparing those with vs those without severe contralateral carotid stenosis or occlusion (SCSO)

Variable	All patients (N = 2945), %	Nonsevere (n = 2209), %	Severe (n = 736), %	*P* value
Neck dissection	2.9	3.2	2.0	.1
Local anesthesia	3.4	3.5	2.8	.37
Primary closure	29.8	26.9	38.4	<.0001
EEG changes	19.3	16.7	30.5	<.0001
Shunt placement	31.2	28.1	40.3	<.0001

EEG, Electroencephalographic.

Table III. Perioperative outcomes of all patients undergoing carotid endarterectomy (CEA) comparing those with vs those without severe contralateral carotid stenosis or occlusion (SCSO)

Variable	Nonsevere (n = 2209), %	Severe (n = 736), %	*P* value
Any stroke	1.5	2.8	.024
Ipsilateral stroke	1.2	1.5	.38
Death	1.1	2.2	.027
Any stroke/death	2.4	4.3	.0079
MI	1.7	3.5	.05
Re-exploration	1	4.9	<.001
Readmission	3.4	7.3	.002

MI, Myocardial infarction.

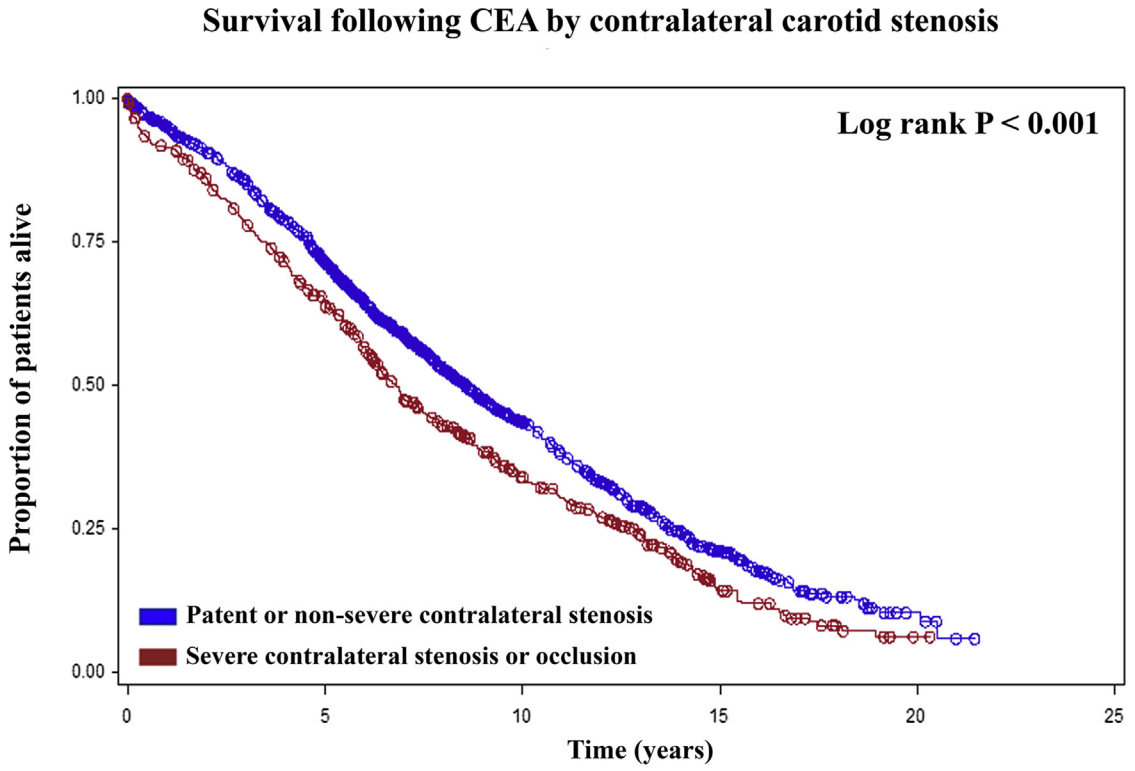
Table IV. Independent predictors of 30-day outcomes after carotid endarterectomy (*CEA*)

Variable	OR (95% CI)	*P* value
Any stroke		
Symptomatic presentation[a]	2.8 (1.5-4.9)	<.001
Male gender	2.2 (1.2-4.0)	.007
Redo CEA	4.9 (1.9-12.1)	<.001
Diabetes	2.6 (1.4-4.7)	.001
SCSO	1.8 (1.0-3.3)	.05
Ipsilateral stroke		
Symptomatic presentation	2.5 (1.2-4.9)	.008
Male gender	3.6 (1.7-7.4)	<.001
Redo CEA	5.9 (2.1-16.3)	<.001
Diabetes	2.3 (1.1-4.6)	.02
Death		
Current smoker	2.4 (1.1-4.9)	.02
Any stroke/death		
Symptomatic presentation	1.9 (1.2-3.0)	.003
Male gender	1.7 (1.1-2.6)	.02
SCSO	1.7 (1.1-2.7)	.02

CI, Confidence interval; *OR*, odds ratio; *SCSO*, severe contralateral stenosis or occlusion.
[a]Symptomatic presentation is defined as ipsilateral stroke, transient ischemic attack, or amaurosis fugax within 6 months preceding CEA.

Other large-scale population-based studies using the Ontario CEA Registry,[10] Vascunet[8] (a collaboration of national and regional registries of 10 European countries), and Swedvasc[12] (the Swedish Vascular Registry) have similarly identified contralateral carotid occlusion as an independent risk factor for perioperative stroke and stroke/death (Table VI). Similarly, a meta-analysis of 30 published articles found that contralateral carotid artery occlusion is an independent predictor of 30-day stroke (OR, 1.6; 95% CI, 1.3-2.1; $P < .001$) and 30-day death (OR, 1.7; 95% CI, 1.2-2.6; $P = .004$).[13] However, several single-center studies have reported no significant difference in perioperative outcomes after CEA in patients with contralateral carotid disease.[17-22] Although these studies showed no significant difference in perioperative outcomes, the study cohorts were small, with contralateral carotid artery occlusion groups composing only 6% to 14% of the entire study cohort, potentially resulting in a type I error.[24]

Whereas SCSO was a predictor of 30-day any stroke, it was not an independent predictor of 30-day ipsilateral stroke or 30-day mortality. Perioperative stroke in our population of patients occurred in regions outside of the surgically treated area. Similarly, AbuRahma et al[25] reported 0% stroke ipsilateral to the operative side in patients with contralateral disease, whereas stroke on the contralateral side occurred in 3.2% of their cohort. This is likely due to decreased cerebral perfusion, particularly after carotid cross-clamping in patients with compromised collateral cerebral circulation. As another measure of decreased cerebral perfusion, it has been reported that patients with SCSO had significantly lower intraoperative cerebral stump pressures compared with those with a patent artery or mild to moderate contralateral artery disease ($P = .02$).[26] Similarly, previous studies monitoring cerebral perfusion during CEA have reported an inverse correlation between cerebral collateral perfusion pressure and degree of contralateral stenosis.[27] Our study showed significantly higher rates of intraoperative EEG changes ($P < .0001$) and shunt placement ($P < .0001$) in patients with SCSO compared with those with nonsevere contralateral carotid disease. This is consistent with a majority of our reviewed literature, which showed significantly higher rates of intraoperative shunt placement in patients with contralateral carotid



	1 Year	5 years	10 years	15 years
NO CONTRALATERAL STENOSIS OR OCCLUSION (N=2209)				
At risk (N)	2001	1340	333	92
Survival (%)	95	72	44	21
Error (%)	0.5	1	1.4	1.5
SEVERE CONTRALATERAL STENOSIS OR OCCLUSION (N=736)				
At risk (N)	661	422	158	6
Survival (%)	91	64	34	6
Error (%)	1	2	2	2

Fig. Survival. Kaplan-Meier curves plotting the fraction of the study population alive over time after carotid endarterectomy (*CEA*) comparing those with vs those without severe contralateral carotid stenosis or occlusion (SCSO).

occlusion.[15-22,26-33] These studies in combination with our findings demonstrate decreased collateral cerebral circulation as an important distinction in SCSO pathophysiology.[34] Selective shunting with routine EEG or cerebral stump pressure monitoring has been shown to reduce the need for shunting in patients compared with routine shunting for all patients. Continuous neurologic monitoring in the awake patient undergoing CEA with regional anesthesia has had even lower shunt use.[35]

In our study, SCSO was found to be an independent predictor of late mortality (HR, 1.27; 95% CI, 1.1-1.4; $P < .0001$), with long-term survival rates significantly worse for patients with SCSO ($P < .001$). The predictors of late mortality identified in our study highlight the adverse effects of cardiovascular burden in patients with SCSO. As an additional marker of increased cardiovascular burden, our study found that SCSO is associated with significantly higher rates of perioperative MI,

Table V. Independent predictors of long-term survival after carotid endarterectomy (CEA)

Variable	HR (95% CI)	*P* value
Symptomatic presentation	1.2 (1.03-1.28)	.01
Age (per year)	1.1 (1.06-1.08)	<.0001
Male gender	0.9 (0.78-0.96)	.0098
CAD	1.5 (1.3-1.7)	<.0001
COPD	1.4 (1.2-1.6)	<.0001
Diabetes	1.8 (1.5-1.9)	<.0001
XRT	1.7 (1.2-2.2)	.0004
Current smoker	1.2 (1.08-1.4)	.0012
Lipid therapy[a]	0.7 (0.60-0.76)	<.0001
SCSO	1.3 (1.1-1.4)	<.0001

CAD, Coronary artery disease; *CI,* confidence interval; *COPD,* chronic obstructive pulmonary disease; *HR,* hazard ratio; *SCSO,* severe contralateral stenosis or occlusion; *XRT,* neck irradiation.
[a]Lipid treatment includes statin and nonstatin drugs.

Table VI. Independent effect of severe contralateral stenosis or occlusion (SCSO) on 30-day any stroke/death rate

Study	OR (95% CI)	*P* value
Present	1.7 (1.1-2.9)	.02
Goodney et al[11]	2.8 (1.3-6.2)	.009
Menyhei et al[8]	1.89 (1.3-2.6)	.002
Ricotta et al[9]	1.31 (1.25-2.38)	.04
Tu et al[10]	1.72 (1.25-2.38)	.001
Kragsterman et al[12]	5.27 (2.02-13.07)	.0005

CI, Confidence interval; *OR,* odds ratio.

surgical re-exploration, and hospital readmission. In analyzing CEA outcomes in Stenting and Angioplasty with Protection in Patients at High Risk for Endarterectomy (SAPPHIRE)-eligible high-risk patients, postoperative MI (3.1% vs 0.9%; $P < .05$) as well as a composite cluster of adverse events of death/stroke/MI (9.3% vs 1.6%; $P < .005$) occurred more frequently in the high-risk group.[16] Interestingly, our study found male gender to be a predictive factor for 30-day any stroke, 30-day ipsilateral stroke, and 30-day any stroke/death, despite most studies reporting a higher stroke rate in the female population. Our study did, however, find that male gender was protective for late mortality in patients with SCSO.

This study shows that SCSO is an independent risk factor for adverse early and late outcomes after CEA. However, SCSO should not be used as an indication for CAS over traditional surgery.[36] Paraskevas and Veith[37] have challenged the notion that contralateral occlusion should be used as an indication for CAS over CEA. These findings are supported by our literature review, which found that patients with SCSO actually had similar or better outcomes after CEA compared with CAS. Yang et al[34] found that patients with contralateral carotid artery occlusion and vertebral artery occlusion experienced more postoperative neurologic complications including transient ischemic attacks and stroke after CAS compared with CEA. Another study evaluated the perioperative outcomes in 147 patients with contralateral carotid artery occlusion; its findings "refuted the assumption" that contralateral carotid occlusion should be used as a criterion for treating with CAS over CEA.[38]

Whereas CEA performed on the carotid artery contralateral to the major occlusion (ie, the side deemed to bear lower burden of disease) did not affect the natural history of carotid disease, it did reduce stroke rate in the surgical territory.[24,34,35] However, in comparing 5-year stroke rate in the population of asymptomatic patients with contralateral carotid artery disease, surgical treatment increased the absolute risk of stroke by 2%.[7] This is a key distinction that should be considered in this subpopulation of individuals with contralateral carotid artery disease.

This study is limited by its retrospective design and potential for referral bias, given the catchment of a single institution. Postoperative events were largely documented from hospital record reviews and therefore lack independent adjudication. Although other studies have performed routine preoperative cerebral angiography and magnetic resonance imaging to identify the level of collateral perfusion, routine imaging was not performed for our study cohort. In addition, routine postoperative cerebral angiography and magnetic resonance imaging were not performed, possibly allowing missed diagnosis of silent infarcts. In addition, the data from our study may not reflect contemporary outcomes as the study period was concluded in December 2005. This was necessary because of the discontinuation of our prospectively maintained database at that time as well as to evaluate long-term survival in our population of patients. Since the conclusion of our study period, medical management of atherosclerotic disease has changed, potentially leading to a difference in current perioperative stroke rates and long-term survival. On the contrary, surgical management and execution of CEA have remained largely unchanged. As a result, analysis with contemporary data may show lower stroke rates but is more likely to show improvement in long-term survival related to better medical management. Despite these limitations, our study remains one of the largest single-center cohorts evaluating the impact of SCSO after CEA, with 25% of our cohort making up the SCSO group.

CONCLUSIONS

Our data add to the discussion of SCSO effects on early and late outcomes after CEA in contemporary literature. We have identified SCSO as an independent risk factor for early MI, perioperative stroke, and stroke/death without increasing the risk of ipsilateral stroke. Our

findings highlight the fragility of cerebral perfusion in patients with SCSO and the increased risk of perioperative stroke without simultaneous increase in ipsilateral stroke rate after CEA. Furthermore, SCSO is associated with late mortality after CEA. These patients have a higher systemic cardiovascular disease burden and therefore should have aggressive risk factor control to maximize their long-term survival. Patients with SCSO undergoing CEA may benefit from aggressive perioperative hemodynamic management and consideration for selective intraoperative shunt placement.

AUTHOR CONTRIBUTIONS

Conception and design: VP
Analysis and interpretation: PP, VP
Data collection: GL, RL, WC, CK, MC, RP, VP
Writing the article: PP
Critical revision of the article: PP, GL, RL, WC, CK, MC, RP, VP
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