Outcomes After Carotid Artery Stenting and Endarterectomy in the Medicare Population

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Background and Purpose—Carotid artery stenting (CAS) is an alternative to carotid endarterectomy (CEA) for stroke prevention. The value of this therapy relative to CEA remains uncertain.

Methods—In 10 958 Medicare patients aged 66 years or older between 2004 and 2006, we analyzed in-hospital, 1-year stroke, myocardial infarction, and death outcomes and the effects of potential confounding variables.

Results—CAS patients (87% were asymptomatic) had a higher baseline risk profile, including having a higher percentage of coronary and peripheral arterial disease, heart failure, and renal failure. In-hospital stroke rate (1.9% CAS versus 1.4% CEA; P=0.14) and mortality (CAS 0.9% versus 0.6% CEA; P=0.20) were similar. By 1 year, CAS patients had similar stroke rates (5.3% CAS versus 4.1% CEA; P=0.12) but higher all-cause mortality rates (9.9% CAS versus 6.1% CEA; P<0.001). Using Cox multivariable models, there was a similar stroke risk (hazard ratio, 1.28; 95% CI, 0.90–1.79) but CAS patients had a significantly higher mortality (HR, 1.32; 95% CI, 1.02–1.71). Sensitivity analyses suggested that unmeasured confounders could be responsible for the mortality difference. In multivariable analysis, stroke risk was highest in the patients symptomatic at the time of revascularization.

Conclusions—CAS patients had a similar stroke risk but an increased mortality rate at 1 year compared with CEA patients, possibly related to the higher baseline risk profile in the CAS patient group. (Stroke. 2011;42:2019-2025.)

Key Words: carotid artery stenting ■ carotid endarterectomy ■ stroke

Carotid artery stenosis is a major risk factor for ischemic stroke.1 Approximately half of patients with ischemic stroke have carotid stenosis and approximately one-third have no warning symptoms.1 Previous studies have determined that carotid endarterectomy (CEA) is superior to medical therapy alone in patients with symptomatic and asymptomatic high-grade carotid stenosis.2–6 Carotid artery stenting (CAS) is increasingly performed as a CEA alternative. Results from randomized clinical trials comparing CAS and CEA are limited; in particular, it is not clear if CAS has a clear advantage over CEA in asymptomatic patients with average surgical risk.7–13 Studies on CAS outcomes in elderly (80 years or older) patients have shown increased perioperative stroke or death risk compared with younger patients.14–17 “Real world” data on long-term outcomes and predictors of adverse outcomes after CAS in elderly patients are limited.

In October 2004, the Centers for Medicare and Medicaid Services announced the reimbursement for CAS in Food and Drug Administration postapproval investigational stent studies18 and in March 2005 broadened coverage by reimbursing CAS procedures in selected patients at high surgical risk.19 This decision reimbursed for asymptomatic carotid artery stenosis ≥80% or symptomatic carotid artery stenosis ≥50%. The minimally invasive nature of CAS makes it an appealing treatment option. This fact could lead to its increased and possibly inappropriate use. The purpose of this national population-based study was to determine the relative effectiveness of CAS compared with CEA in the management of patients with carotid artery stenosis using the population-based Medicare database.

Patients and Methods

Data sources were the Centers for Medicare and Medicaid Services 5% Medicare Provider Analysis Review and Denominator files from 2003 to 2006, which contain a 5% nationwide random sample of Medicare claims. The study population consisted of all patients aged 66 years or older (to ensure available Medicare claims data for a full year before the procedure) who underwent CAS or CEA between January 1, 2004 and December 31, 2006 and were continuously enrolled in the Medicare Part A (inpatient) services program without health maintenance organization (HMO) for 1 year before the procedure. For in-hospital end points, all patients were included and for 1-year outcomes all patients from years 2004 and 2005 were included. Because all patients were hospitalized for CAS or CEA, study cohorts were identified by discharge diagnosis and procedural codes based on the presence of International Classification of...
Figure 1. Flow diagram for identification of study cohort. A, Carotid artery stenting (CAS); B, Carotid endarterectomy (CEA). ICD-9 indicates International Classification of Diseases, 9th revision; HMO, health maintenance organization.

Diseases, 9th revision, clinical modification (ICD-9CM) codes.20 Procedure codes for CEA (38.12), CAS (00.63, 00.64, 00.61), and insertion of noncoronary stents (39.50, 39.90) were used in conjunction with the diagnostic codes for carotid artery stenosis (433.11, 433.10, 433.1, 435.9). If patients had multiple procedures during the study period, then only the first carotid artery revascularization procedure was included. We also excluded patients with both CAS and CEA in the 12-month window (Figure 1). Patients were classified as asymptomatic or symptomatic. If the patient’s principal discharge diagnosis was “carotid artery stenosis with stroke” or a secondary diagnosis code included a history of previous stroke (ICD-9 codes 342:34200 to 34202, 3421, 34210, 34211, 34212, 34280 to 34282, 34290 to 34292, or 438: 4380, 43810 to 43812, 43819 to 43822, 43830 to 43832, 43840 to 43842, 43850 to 43853, 4386, 4387 43881 to 43885, 43889, 4389), TIA (435 or 781.4), or amaurosis fugax (362.34 or 368.12), then patients were classified as asymptomatic. If the principal discharge diagnosis was “carotid artery stenosis” without mention of stroke and with no accompanying secondary diagnoses for TIA or history of stroke or amaurosis fugax, then the patient was classified as asymptomatic.21

Primary outcome measures were the occurrence of stroke, myocardial infarction (MI), and all-cause death rates after procedure by 1 year. Stroke was defined by discharge ICD-9 codes of in-hospital “postoperative stroke” (997.00 to 997.02, 997.09, 997.0).21,22 Post-procedure death was defined as any death during the same hospital stay regardless of postprocedure interval. Teaching hospital status was determined based on linked Centers for Medicare and Medicaid Services teaching hospital identification number.

Discrete data are reported as frequencies and compared by $\chi^2$ and Fisher exact tests as appropriate. Continuous data are reported as mean±SD and compared by Student t test. Time to first occurrence of study outcomes (death, stroke, MI) were estimated according to the Kaplan-Meier method. In these analyses, for stroke and MI analyses, patients were censored at death, at loss of Medicare Part A coverage, at switching to a health maintenance organization, or at the end of 1-year follow-up. Multivariable Cox proportional hazards model was used to identify independent predictors of 1-year stroke, death, MI, and combined end point. Adjustment was made for age, sex, medical comorbidities, admission diagnosis, presentation type (symptomatic versus asymptomatic), treatment year, and hospital type (teaching versus nonteaching) for determining outcome predictors. We tested the assumption of proportionality in the Cox model by determining that the logarithm of the baseline cumulative hazard rates and the Schoenfeld residuals were proportional with follow-up time. Additionally, propensity analyses were conducted to balance independent risk factors for outcomes between CAS and CEA.23 The propensity that a patient would receive CAS was generated from a logistic regression model that incorporated the potential confounders included in the Cox model. Patients were grouped into 5 strata representing quintiles of the propensity score. The Cochran-Mantel-Haenszel $\chi^2$ test was used to determine whether the covariance was balanced after adjusting for propensity quintiles. Covariates that retained a significant difference between patients receiving CAS and CEA were adjusted together with the propensity score in the Cox models. Sensitivity analyses were also performed to assess the potential effects of unmeasured confounders on the associations observed between outcomes and different treatments.24 The unmeasured confounders in our study could be, for example, the degree of carotid stenosis, the use of protection devices during stenting procedure, and operator’s experience. Prevalence of the unmeasured confounder was set to different levels for patients receiving CAS and CEA. For each prevalence level, effect size of the unmeasured confounder on outcomes ranged from hazard ratio of 1.1 to 1.5. The estimated hazard ratio for CAS versus CEA from Cox models was further adjusted for the unknown confounders. All analyses were performed using SAS version 9.1 (Cary, NC). For all comparisons, a 2-sided value of $P<0.05$ was considered statistically significant.

Results
During 2004 to 2006, 10,958 patients underwent either CEA or CAS identified from the 5% nationwide random sample of Medicare claims (MedPAR file). The majority were treated for asymptomatic disease (87.5%). Overall, a greater percentage underwent CEA (n=9635; 87.9%) than CAS (n=1323; 12.1%). CAS performance as a percentage of CAS and CEA treatments increased from 8.2% in year 2004 to 16.8% in year 2006 (Table 1). Patients (n=7461) who underwent either CEA (n=737) or CEA (n=6724) in year 2004 to 2005 with completed 1-year data were included for analysis of 1-year outcomes.

Patient Characteristics by Procedure
Table 1 shows the baseline characteristics that, in general, were similar in the 2 groups. However, the CAS group had a higher percentage of patients with age 80 years or older (35% CAS versus 31.1% CEA; $P=0.0045$) and a higher risk profile including coronary artery disease or MI history (65.6% CAS versus 50.9% CEA; $P<0.0001$), heart failure (18.8% CAS versus 12.7% CEA; $P<0.0001$), renal failure (10.6% CAS...
versus 5.9% CEA; \( P < 0.0001 \), and peripheral vascular disease (49.5% CAS versus 23.5% CEA; \( P < 0.0001 \)).

**In-Hospital Outcomes**

In-hospital outcomes were assessed in 10 958 patients who underwent either CAS (n = 1323) or CEA (n = 9635; Table 2). There were similar in-hospital stroke (1.9% CAS versus 1.4% CEA; \( P = 0.14 \)) and all-cause death (0.9% CAS versus 0.6% CEA; \( P = 0.20 \)) rates. In-hospital all-cause mortality (3.0% CAS versus 1.2% CEA; \( P = 0.07 \)) and stroke risk (6.6% CAS versus 3.5% CEA; \( P = 0.053 \)) trended higher in symptomatic CAS patients (CAS, 168; CEA, 1207), but not in the asymptomatic group (CAS, 1155; CEA, 8428; mortality: 0.6% CAS versus 0.5% CEA, \( P = 0.71 \); stroke: 1.2% CAS versus 1.1% CEA, \( P = 0.65 \)).

**1-Year Outcomes and Their Predictors in the Entire Cohort**

At 1 year, the 2 groups had a similar stroke rate (5.3% CAS versus 4.1% CEA; \( P = 0.12 \)), but CAS had an increased all-cause death rate (9.9% CAS versus 6.1% CEA; \( P < 0.001 \); Figure 2). A significant higher stroke rate in the CAS group was observed in the symptomatic (CAS, 93; CEA, 644; 18.9% CAS versus 10.3% CEA; \( P = 0.016 \)) but not in the asymptomatic (CAS, 848; CEA, 5876; 3.4% CAS versus 3.2% CEA; \( P = 0.79 \)) patients. There was also an increased MI rate in the CAS group (4.8% CAS versus 2.5% CEA; \( P < 0.001 \)). The combined end point of death, stroke, or MI was higher in the CAS group (16.7% CAS versus 11.0% CEA; \( P < 0.001 \); Table 2).

By multivariate analysis of the entire cohort (n = 7461), after adjustment for other predictors (all the variables listed in the Table 3 under the adjusted model), CAS was not an

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**Table 1. Baseline Patients Characteristics**

<table>
<thead>
<tr>
<th>Factor</th>
<th>CAS (%)</th>
<th>CEA (%) (n=9635)</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (mean±SD)</td>
<td>77±6</td>
<td>76±6</td>
<td>0.19</td>
</tr>
<tr>
<td>Age groups</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;80 y</td>
<td>860 (65.0)</td>
<td>6636 (68.9)</td>
<td>0.01</td>
</tr>
<tr>
<td>≥80 y</td>
<td>463 (35.0)</td>
<td>2999 (31.1)</td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>761 (57.5)</td>
<td>5519 (57.3)</td>
<td>0.89</td>
</tr>
<tr>
<td>Female</td>
<td>562 (42.5)</td>
<td>4116 (42.7)</td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>1222 (92.4)</td>
<td>9077 (94.2)</td>
<td>0.01</td>
</tr>
<tr>
<td>Not white</td>
<td>101 (7.6)</td>
<td>558 (5.8)</td>
<td></td>
</tr>
<tr>
<td>Year</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2004</td>
<td>314 (23.7)</td>
<td>3526 (36.6)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>2005</td>
<td>423 (32.0)</td>
<td>3215 (33.4)</td>
<td></td>
</tr>
<tr>
<td>2006</td>
<td>586 (44.3)</td>
<td>2894 (30.0)</td>
<td></td>
</tr>
<tr>
<td>Presentation type</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Asymptomatic</td>
<td>1155 (87.3)</td>
<td>8428 (87.5)</td>
<td>0.86</td>
</tr>
<tr>
<td>Symptomatic</td>
<td>168 (12.7)</td>
<td>1207 (12.5)</td>
<td></td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>409 (30.9)</td>
<td>2878 (29.9)</td>
<td>0.44</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1026 (77.6)</td>
<td>7589 (78.8)</td>
<td>0.31</td>
</tr>
<tr>
<td>COPD</td>
<td>291 (22.0)</td>
<td>2003 (20.8)</td>
<td>0.31</td>
</tr>
<tr>
<td>CAD/MI</td>
<td>866 (65.6)</td>
<td>4899 (50.9)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Heart failure</td>
<td>248 (18.8)</td>
<td>1219 (12.7)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Renal failure</td>
<td>140 (10.6)</td>
<td>564 (5.9)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Obesity</td>
<td>57 (4.3)</td>
<td>438 (4.6)</td>
<td>0.70</td>
</tr>
<tr>
<td>PVD</td>
<td>655 (49.5)</td>
<td>2260 (23.5)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Teaching hospital</td>
<td>859 (64.9)</td>
<td>5163 (53.6)</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

CAD indicates coronary artery disease; CAS, carotid artery stenting; CEA, carotid endarterectomy; COPD, chronic obstructive pulmonary disease; MI, myocardial infarction; PVD, peripheral vascular disease.

*Data are n (%) unless otherwise indicated.

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**Table 2. Outcomes in Patients by Treatments**

<table>
<thead>
<tr>
<th>In-Hospital Outcomes</th>
<th>CAS (n=1323)</th>
<th>CEA (n=9635)</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death</td>
<td>12 (0.9; 0.4–1.4)</td>
<td>58 (0.8; 0.5–0.8)</td>
<td>0.20</td>
</tr>
<tr>
<td>Stroke</td>
<td>25 (1.9; 1.2–2.6)</td>
<td>132 (1.4; 1.1–1.6)</td>
<td>0.14</td>
</tr>
<tr>
<td>Outcomes at 1 y</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Death</td>
<td>73 (9.9; 8.0–12.3)</td>
<td>412 (6.1; 5.6–6.7)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Stroke</td>
<td>39 (5.3; 3.9–7.2)</td>
<td>277 (4.1; 3.7–4.6)</td>
<td>0.12</td>
</tr>
<tr>
<td>MI</td>
<td>35 (4.8; 3.5–6.7)</td>
<td>165 (2.5; 2.1–2.9)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Combined (stroke/MI/death)</td>
<td>123 (16.7; 14.2–19.6)</td>
<td>739 (11.0; 10.3–11.8)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Data are n (%; 95% confidence interval).

CAS indicates carotid artery stenting; CEA, carotid endarterectomy; MI, myocardial infarction.

**Figure 2.** The Kaplan-Meier curves for development of stroke (A) and all-cause death (B) by year in carotid artery stenting (CAS) and carotid endarterectomy (CEA) patients.
independent predictor of stroke (hazard ratio, 1.28; 95% CI, 0.9–1.79). However, CAS was associated with significantly increased risk of death, MI, and combined events compared to CEA (Table 3).

### Propensity Score Analysis

To further adjust for potential confounders, propensity analyses were performed. Those covariates that were statistically unbalanced at baseline between the CAS and CEA groups as shown in Table 1 were adjusted with propensity scoring. Propensity score adjustments yielded no substantive differences relative to traditional multivariate Cox analyses (Table 3).

### Sensitivity Analysis

Sensitivity analyses to estimate the potential effect of unmeasured confounders on the study outcomes of mortality and acute MI were also performed. Results controlling the unmeasured confounder based on different prevalence and hazard ratio of an unknown factor are shown in Table 4. In Table 4, the first row is the result from our current statistical model after adjusting known risk factors but without adjusting for any unmeasured confounding factor (assumption is that there is no unmeasured confounder, 0% prevalence of unmeasured confounder for both treatments and effect size hazard ratio=1.0). The rest of the rows are based on the following assumptions: if there is an unmeasured confounder, if the prevalence of the confounder was as shown in the right 2 columns for each treatment, and if the effect size (hazard ratio) was as shown in the third column, and we included this unmeasured confounder (with our assumed prevalence and effect size on hazard ratio) in the multivariate models that adjusted other known risk factors to calculate. Unmeasured confounders with either a very small effect (hazard ratio, 1.10) on mortality and somewhat large difference (30%) in prevalence, or with a relative large effect (hazard ratio, 1.30) on mortality and small difference (10%) in prevalence could alter the significance of the difference on mortality between CAS and CEA. For acute MI, it would require both somewhat larger effect on mortality and large difference to change the study conclusion.

### Examination for Interaction

Interactions were tested on the basis of the Cox model presented in Table 3. There were no statistically significant interactions between age, gender, presentation type (symptomatic disease versus asymptomatic disease), and treatment choices (CAS versus CEA) on any studied 1-year outcomes.

### Discussion

The present analysis of carotid revascularization in a random sample of Medicare claims is the largest population-based series with 1-year outcome comparing CAS and CEA. There are several important observations.

The CAS group had a significantly worse baseline risk profile, ie, a higher percentage of patients with comorbidities, including...
coronary and peripheral arterial disease, heart failure, and renal failure (Table 1). However, there was no significant difference in in-hospital stroke and death rates. By 1 year, the CAS stroke rate was not significantly different from CEA. However, all-cause mortality was significantly higher with CAS. After adjustment of those covariates that were statistically unbalanced at baseline between the CAS and CEA groups, a similar stroke risk and higher mortality at 1 year with CAS were maintained. Although it is difficult to compare in-hospital and 1-year results from the present study to other investigations because of differences in methodology and study population, the rates of in-hospital stroke and mortality in the present study are similar to those reported in CAS registries and randomized, controlled, clinical trials. It may be that mortality is truly increased with CAS relative to CEA. There are reasons, however, to believe that there are more cogent explanations for the mortality difference. If CAS truly caused higher long-term mortality, then it might be expected that postprocedure adverse outcomes would be higher with CAS than CEA, which would then have predisposed to worsened long-term outcomes from these complications. In fact, stroke rate was similar. If stent implantation per se were associated with increased mortality, then the expectation would be that late stent thrombosis and fatal stroke would be the putative mortality mechanism. This possibility is unlikely insofar as nonfatal stroke was similar in the 2 groups. Further, the incidence of CAS late stent thrombosis reported in the literature is quite low. More likely is the possibility that CAS patients were at higher baseline risk for mortality. Propensity analysis attempts to correct for baseline inequality, but this type of analysis can only account for a proportion of baseline differences. Further, the Medicare database does not allow for elucidation of the cause of death. In view of the higher MI rate in all CAS subgroups and higher baseline coronary artery disease prevalence risk, it would not be surprising if the excess in death rate were attributable, at least in part, to an increase in cardiac-related deaths. Sensitivity analyses suggest that unmeasured confounders could account for the measured mortality difference.

Despite the broad technical feasibility of CAS, controversy surrounds patient selection, particularly in relation to stroke risk. Several studies have demonstrated that increasing age is an important predictor of complications with CAS. The randomized multicenter Carotid Revascularization Endarterectomy versus Stent Trial (CREST) lead-in phase showed that elderly (80 years or older) patients had a markedly elevated 30-day combined periprocedural stroke or death rate of 12.1% versus 3.2% in younger patients. A large observational study showed that in octogenarians, CAS had a higher rate of in-hospital postoperative stroke (2.1% versus 0.9%; \( P < 0.001 \)) and mortality (1.3% versus 0.4%; \( P < 0.001 \)) compared with CEA. Most recently, an interim analysis of International Carotid Stenting Study (ICSS) showed that the incidence of stroke, death, or procedural MI was higher with CAS at 120 days (8.5% CAS versus 5.2% CEA; \( P = 0.006 \)). In contrast, our study showed a similar stroke rate. By 1 year, the similar stroke risk in both older (66–79 years) and very old (80 years or older) patients was maintained with CAS versus CEA, providing increased assurance for CAS use in elderly patients (Table 3).

The presence of symptoms related to carotid stenosis was the strongest risk factor for stroke in our study. In this subgroup, CEA appeared to be significantly better than CAS,

### Table 4. Sensitivity Analyses of the Hazard Ratio of Mortality and Acute Myocardial Infarction for Patients Receiving Carotid Artery Stenting Compared to Those Receiving Carotid Endarterectomy Controlling for an Unmeasured Binary Confounder

<table>
<thead>
<tr>
<th>Prevalence of Unmeasured Confounder</th>
<th>Unmeasured Confounder HR</th>
<th>HR (95% CI) of Outcomes, Adjusted for Unmeasured Confounder</th>
</tr>
</thead>
<tbody>
<tr>
<td>CAS (%) CEA (%)</td>
<td></td>
<td>Mortality</td>
</tr>
<tr>
<td>0 0</td>
<td>1.00</td>
<td>1.32 (1.02)</td>
</tr>
<tr>
<td>60 70</td>
<td>1.10</td>
<td>1.31 (1.01)</td>
</tr>
<tr>
<td>50 70</td>
<td>1.10</td>
<td>1.30 (1.00)</td>
</tr>
<tr>
<td>40 70</td>
<td>1.10</td>
<td>1.28 (0.99)</td>
</tr>
<tr>
<td>60 70</td>
<td>1.20</td>
<td>1.30 (1.00)</td>
</tr>
<tr>
<td>50 70</td>
<td>1.20</td>
<td>1.27 (0.98)</td>
</tr>
<tr>
<td>40 70</td>
<td>1.20</td>
<td>1.25 (0.97)</td>
</tr>
<tr>
<td>60 70</td>
<td>1.30</td>
<td>1.29 (0.99)</td>
</tr>
<tr>
<td>50 70</td>
<td>1.30</td>
<td>1.29 (0.99)</td>
</tr>
<tr>
<td>40 70</td>
<td>1.30</td>
<td>1.24 (0.96)</td>
</tr>
<tr>
<td>60 70</td>
<td>1.50</td>
<td>1.32 (1.02)</td>
</tr>
<tr>
<td>50 70</td>
<td>1.50</td>
<td>1.29 (0.99)</td>
</tr>
<tr>
<td>40 70</td>
<td>1.50</td>
<td>1.24 (0.96)</td>
</tr>
</tbody>
</table>

AMI indicates acute myocardial infarction; CAS, carotid artery stenting; CEA, carotid endarterectomy; CI, confidence interval; HR, hazard ratio.
when stroke risk with CAS was almost 3-times as high as CEA, although the sample size of this subgroup was small. Conversely, there was no significant difference in stroke rate in the asymptomatic group who represent the majority of patients with carotid stenosis in the real world. The expected decrease in annual stroke rate with CEA in the asymptomatic group was in the 1% range. Our study showed that the greatest risk factors for mortality and stroke were the presence of comorbid conditions (Table 3). In view of the relatively small decrease in stroke risk with revascularization and the influence of multiple comorbidities on stroke risk in asymptomatic patients, it may be reasonable to consider the possibility that state-of-the-art intensive medical treatment without revascularization may be an appropriate therapeutic option in some patients rather than revascularization, but this hypothesis requires formal testing.

Comparison With the CREST Study
Most recently, the primary outcome results from the randomized CREST trial have been published. It should be pointed out that, in addition to the study design, there are several important differences of CREST compared with our study, particularly that CREST had >50% symptomatic patients, which may explain in part the higher incidence of stroke in the CREST trial compared to our study. Stroke rate periprocedurally was significantly higher in the CAS cohort, whereas it was not in the current study. However, <15% of our patients were symptomatic. In our symptomatic subgroup, there was a significant increase (P = 0.016) with CAS. Thus, the results of the 2 studies in this regard may be similar, ie, the risk of periprocedural stroke in symptomatic patients may be higher with CAS than CEA. The relatively lower incidence of stroke in both groups in our study compared with CREST (4.1% CAS versus 2.3% CEA) may also be partly attributable to the lack of mandatory postprocedure neurological evaluation as was mandated in CREST.

Long-term stroke rate (median follow-up, 2.5 years) was similar in the CREST study, which is compatible with our data. However, the MI rate, particularly the in-hospital rate, was higher in the CEA group in CREST, which may be explained by the excellent baseline matching in CREST and the expected higher stress from surgery with CEA. In the present study, CAS patients had higher coronary artery disease prevalence, so the increased baseline risk likely cancelled out any beneficial decrease in MI development in the CAS group. Mortality was similar in the 2.5-year follow-up in the CREST trial. The differences in mortality outcome between CREST and the present study may be attributable to hidden biases, encouraging higher-risk patients to undergo less invasive procedure. A fuller description of the CREST mortality results may help to explain mortality differences observed.

Study Limitations
This study has limitations related to the use of an administrative database and baseline inequalities. Many anatomic, procedural, and clinical factors are not available, and neither is the ability to review individual patient-specific characteristics. As an example, the use and duration of antiplatelet therapy in the 2 groups, particularly thienopyridine in addition to aspirin, cannot be ascertained. To minimize overlooking cases of symptomatic carotid stenosis, all secondary diagnoses of TIA, previous stroke, or amaurosis fugax were included as a means to identify the symptomatic patients. Because coding for carotid stenosis is performed at discharge, it is possible that patient admission presentation data may have been misclassified. Coding errors related to patient factors are also possible; to minimize coding inaccuracies, we used established comorbidity ICD9-CM codes. Likewise, to identify stroke and MI, we used established ICD9-CM codes. In this study, we used multivariable Cox proportional hazards model and propensity analyses to adjust unbalanced risk factors between treatment choices. However, as in all registries, the selection of a particular therapy may be related to hidden biases, which may explain the procedural mortality differences rather than the procedure itself. Although this study was a look at the real world of treating carotid artery stenosis, the Centers for Medicare and Medicaid Services policy of limiting payment to “high-risk” patients only suggests the possibility that many of the CAS patients were enrolled in clinical registries, whereas the CEA patients were treated outside of an investigation, and that CAS patients treated as part of a clinical investigation had a higher baseline risk compared with the CEA patients. This difference, in turn, may explain the mortality difference. The sensitivity analyses results also supported the hypothesis that the mortality difference may be related to residual hidden biases. Finally, although this study represents the largest registry analysis on carotid artery stenosis treatment, some of the subgroup analyses are limited by small sample size.

Conclusions
We found that Medicare CAS patients (87% of whom were asymptomatic) had a higher risk profile than CEA patients. CAS patients had similar in-hospital postoperative stroke and in-hospital mortality rates. By 1 year, CAS patients had a similar stroke risk but an increased mortality rate compared with CEA, likely because of increased baseline mortality risk.

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Disclosures
None.

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