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Carotid artery stenting has increased risk of external carotid artery occlusion compared with carotid endarterectomy

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Abstract

Objective—The external carotid artery (ECA) can be an important source of cerebral blood flow in cases of high-grade internal carotid artery stenosis or occlusion. However, the treatment of the ECA is fundamentally different between carotid endarterectomy (CEA) and carotid artery stenting (CAS). CEA is routinely associated with endarterectomy of the ECA, whereas CAS excludes the ECA from direct flow. We hypothesize that these differences make ECA occlusion more common after CAS. Further, the impact of CAS on blood flow into the ECA is interesting because the flow from the stent into the ECA is altered in a way that may promote local inflammation and may influence instant restenosis (ISR). Thus, our objective was to use our institutional database to identify whether CAS increased the rate of ECA occlusion and, if it did, whether ECA occlusion was associated with ISR.

Methods—Patients undergoing CAS or CEA from February 2007 to February 2012 were identified from our institutional carotid therapy database. Preoperative and postoperative images of patients who followed up in our institution were included in the analysis of ECA occlusion and rates of ISR.

Results—There were 210 (67%) CAS patients and 207 (60%) CEA patients included in this analysis. Despite CAS patients being younger (68 vs 70 years), having shorter follow-up (12.5 vs 56.2 months), and being more likely to take clopidogrel (97% vs 35%), they had an increased rate
of ECA occlusion (3.8%) compared with CEA patients (0.4%). CAS patients who went on to ECA occlusion had an increased incidence of prior neck irradiation (50% vs 15%; \( P = .03 \)), but we did not identify an association of ECA occlusion with ISR >50%.

**Conclusions**—Whereas prior publications have identified increased rates of external carotid stenosis, this is the first demonstration of increased ECA occlusion after CAS. However, ECA occlusion is uncommon (~4%) and did not have an association with ISR >50%. Future work modeling ECA flow patterns before and after CAS will be used to further test this interaction.

Stroke is a major cause of morbidity and mortality and is the fourth leading cause of death in the United States.\(^1\) Trials such as the North American Symptomatic Carotid Endarterectomy Trial and the European Carotid Surgery Trial have definitively established carotid endarterectomy (CEA) to be the “gold standard” therapy for high-grade symptomatic carotid artery stenosis.\(^2,3\) Carotid artery stenting (CAS) is an alternative method of treating carotid artery stenosis that has a role in certain clinical scenarios.\(^4,5\) CEA and CAS use very different methods to relieve carotid stenosis. CEA enables complete vascular control before manipulation of the plaque, whereas CAS routinely uses a distal protection device that must cross the lesion before placement. This may in part explain the increased stroke rate with CAS.\(^6\) Still, CAS is relatively favored in certain situations, such as restenosis after prior CEA, after neck irradiation, or with anatomically prohibitive lesions.\(^7–9\)

In addition to the different risks and benefits associated with CAS and CEA, CAS and CEA have fundamentally different approaches to the external carotid artery (ECA). CEA routinely treats stenotic ECAs as part of the endarterectomy, whereas CAS intentionally covers the ECA. The ECA primarily functions as a muscular artery to the face and scalp, but it can be a source of collateral blood flow in the presence of high-grade internal carotid artery stenosis or occlusion.\(^10–12\) This exclusion of the ECA after CAS leads to aberrant flow patterns out of the stent struts into the ECA. Thus, we hypothesize that these differences would increase the risk of ECA occlusion after CAS compared with CEA. Because altered flow patterns can increase the inflammatory profile of endothelial cells,\(^13\) we further hypothesized that ECA occlusion may coincide with an increased rate of instent restenosis (ISR).

**METHODS**

Medical records from patients undergoing CAS and CEA from February 2007 to March 2012 were used to identify patients who had their follow-up carotid duplex examinations in our division’s vascular laboratory. After identification of patients, their records were retrospectively evaluated from a single institution’s database under an approved Institutional Review Board protocol, which waived the need for patient consent.

Patient demographics, comorbidities, antiplatelet regimen, imaging results, and clinical outcomes were collected and analyzed. The degree of preoperative ECA stenosis was assessed by duplex velocities, angiography, or axial imaging before the procedure, and both symptomatic and asymptomatic patients were included in the study. Indications to undergo stenting included medical comorbidities, contralateral occlusion, prior history of ipsilateral neck surgery including CEA, prior irradiation of the neck, and anatomically challenging lesions. “Surgical high risk” for CEA included congestive heart failure (class III/IV), severe
chronic obstructive pulmonary disease, and severe coronary artery disease that could not be revascularized. The decision to undergo CAS vs CEA was made by the treating physicians and the patients.

Patients in both the CEA and CAS groups underwent duplex imaging of their carotid arteries at 1 month, 6 months, and 1 year after their index operation and yearly thereafter. Patients without follow-up imaging in our division’s vascular laboratory were excluded from the analysis. The most recent duplex imaging on follow-up was used to determine patency of the ECA, and our vascular laboratory’s carotid duplex ultrasound criteria or axial imaging (magnetic resonance angiography or computed tomography angiography) was used to determine degree of ISR in patients undergoing CAS. ISR >50% was defined as peak systolic velocity >175 cm/s, and ISR >70% was defined as peak systolic velocity >300 cm/s and end-diastolic velocity >140 cm/s. For patients undergoing reoperation on the same vessel, the last documented ultrasound study before reoperation was used. For patients undergoing CAS, intraoperative angiography was used to determine prestent ECA patency, and preoperative axial imaging or duplex ultrasound was used to analyze the degree of preoperative ECA stenosis. For those undergoing CEA, operative reports and preoperative duplex ultrasound were used to determine preoperative ECA patency, and axial imaging or duplex ultrasound was used to analyze the degree of preoperative ECA stenosis. Seven patients in the CAS cohort and three patients in the CEA cohort with ECA occlusion before the procedure were excluded from the analysis.

The objective of this study was to compare the incidence of ECA occlusion after CEA and CAS. Secondary objectives included analyzing the impact of ECA occlusion on ISR. ECA patency in patients undergoing CAS was compared with that in patients undergoing CEA. The CAS group was then subdivided into patients with ECA occlusion and those without ECA occlusion to compare rates of ISR between the two groups.

Categorical variables were compared by $\chi^2$ or Fisher exact test (dependent on sample size) to determine statistical significance, and continuous variables were compared by Student $t$-test. $P < .05$ was used as the threshold for statistical significance.

RESULTS

Between February 2007 and March 2012, 312 CAS (299 patients) and 344 CEA (335 patients) procedures were performed at our institution. From this group of patients, 210 (67%) CAS patients had follow-up duplex imaging available at the time of the analysis, and 207 (60%) CEA patients had follow-up carotid duplex imaging in our vascular laboratory (60%) (Fig 1). Patients were similarly matched with the exception of variables known to be associated with CAS (younger, symptomatic, cervical irradiation, prior CEA). In addition, the CAS cohort had a shorter follow-up and increased use of clopidogrel (Table I).

There was a significantly increased rate of ECA occlusion in the CAS group compared with the CEA group (CAS, eight arteries [3.8%]; CEA, one artery [0.4%]; $P = .04$). All other postoperative outcomes were similar between groups (Table II). Interestingly, the preoperative velocities in the ECA were higher in the CAS-treated patients compared with
the CEA patients. However, within the CAS group, the preoperative ECA peak systolic velocities were similar between the patients who developed ECA occlusion and those who did not, but the end-diastolic velocity was higher in the ECA occlusion group (44 cm/s compared with 31 cm/s; \( P < .001 \)) (Table III). There was also an increased frequency of neck irradiation within the CAS patients who developed ECA occlusion but not with other indications for CAS (Table IV).

The increased rate of ECA occlusion was not associated with an increased rate of ISR >50% (Table V), but patients undergoing CAS with subsequent ECA occlusions were more likely to have had cervical irradiation compared with those undergoing CAS without ECA occlusions (four [50%] vs 30 [15%]; \( P = .03 \)). No ISR in patients with ECA occlusions required repeated intervention, and no ECA occlusions resulted in symptoms. Five patients underwent reoperation on the same artery in the CAS group and five underwent reoperation in the CEA group in this time period.

**DISCUSSION**

The ECA is an interesting artery in that it is part of the robust collateral system in the neck, but it rarely causes symptoms until it is occluded. Even with ECA ligation, symptoms are rare. However, in the setting of high-grade internal carotid artery stenosis or occlusion, the ECA can contribute meaningfully to the cerebral blood flow in up to 80% of these patients.\(^{14}\) Also, isolated ECA endarterectomy in patients with internal carotid artery occlusion is well established in the literature.\(^{15,16}\) Thus, the ECA can be an important part of cerebral perfusion in these patients. Because CAS is commonly performed in patients with higher anatomic risk, including contralateral occlusion and prior CEA or neck irradiation, the ECA may be particularly important in this cohort of patients. Thus, our objective in this study was to analyze our institution’s high-volume carotid therapy results to determine if CAS was associated with increased rates of ECA occlusion.

Whereas previous studies have shown an increased rate of ECA stenosis with CAS,\(^{17–19}\) this is the first study to find an increased rate of ECA occlusion. ECA exclusion as a result of CAS has recently been demonstrated to alter the arterial flow patterns into the ECA, in part owing to poor apposition of the stent to the ECA orifice. These altered flow patterns alter wall shear stress, contributing to proinflammatory and proatherosclerotic arterial wall remodeling.\(^{13}\) We speculated that these effects not only may influence ECA occlusion rates but also may play a role in ISR, which is known to be a persistent and variable problem with CAS [ISR rates of 3%–17%].\(^{20,21}\) but we did not identify an association of ECA occlusion with ISR >50%. This may be due in part to the low rate of ECA occlusion (~4%) in the CAS group. Further, there are a number of other factors that contribute to ISR in addition to changes in wall shear stress.\(^{22}\)

Reichmann et al recently demonstrated, as others have before, that CAS is associated with increased ECA duplex velocities, but they did not identify increased rates of ECA occlusion.\(^{17}\) Whereas their numbers were substantially lower than ours, the lack of even a trend (one post-CAS occlusion; two post-CEA occlusions) suggests differences between our respective patient populations; but somewhat surprisingly, this increased rate of irradiation...
did not correlate with increased rate of ISR. Because neck irradiation was significantly
different for CAS patients who went on to ECA occlusion compared with those who did not,
this is one important difference between our data and the data of Reichmann and de
Borst,\textsuperscript{17, 18} in which the incidence of irradiation was not included. The report of Woo et al
had 10 patients with cervical irradiation of 101 CAS patients (and 165 CEAs). They reported
two occluded ECAs after CAS and none in the CEA group. Unfortunately, this report did not
address whether the patients with occluded ECAs had irradiation.\textsuperscript{19} Whereas the
preoperative ECA peak systolic velocities were similar between CAS patients regardless of
whether ECA occlusion occurred, the end-diastolic velocity increased in those who went on
to occlude. Here the absolute difference was small (44 cm/s vs 31 cm/s), and we are not sure
what role a small increase in end-diastolic velocity in the ECA had on our findings. In
addition, the patients seen in the northeastern part of the United States and the Netherlands
are likely to be very different from the patients in the southeastern part of the United States.
In fact, our institution is in the very middle of the stroke heat map for the United States.\textsuperscript{23}
Although prospective data collections from patients/practitioners of different regions will
likely bring much information to the local delivery of quality health care, the Society for
Vascular Surgery’s Vascular Quality Initiative data are most mature in the Northeast. Thus, it
is of limited value to our population currently. However, we are members of the Georgia-
Florida Vascular Quality Initiative group, and future analyses of these data, along with
comparisons in other regions, will be helpful in clarifying the differences that exist and
create the discrepancies in the literature.

Finally, this study does have significant limitations that need to be addressed. The data
collected were analyzed in a retrospective fashion. However, the data collected were from a
“real-world” experience and included all patients seen in our institution, thus not biasing
results toward one specialty or another. In fact, the CAS group were younger and had shorter
length of follow-up but an increased rate of clopidogrel use. Logically, these differences
should prejudice the results in favor of CAS and the potential of a type II statistical error.
Given our finding of increased ECA occlusion rates after CAS, we believe longer and more
complete follow-up would only bias the results in support of this finding. In contrast, we did
not identify an association of ECA occlusion with an increased rate of ISR. This leaves open
to debate whether altered arterial flow patterns and shear stress in the ECA contribute to
ISR. Ongoing work in our division is currently developing a modeling program and ex vivo
culture model to better characterize the impact of altered flow patterns on the ECA through
the stent struts. Second, the percentage of follow-up patients in this data set was 67% (CAS)
and 60% (CEA). Some of this is explained by including only duplex results from our
division’s Intersocietal Accreditation Commission vascular laboratory and that ECA
velocities were not routinely documented before our transition to an electronic storage
system in 2010. Our institution is a quaternary referral center with many of our patients
being referred for therapy by vascular surgeons and interventionalists from the surrounding
area. This leads to follow-up regimens that include outside physicians and various vascular
laboratories. To secure the most reliable information, we chose to restrict the duplex follow-
up studies to our laboratory. Further, the demographics of the patients in the CAS and CEA
groups were separately reported between those with and without follow-up. Here we found
small differences between those who followed up and those who did not. In the CAS group,
there were fewer male patients in the lost to follow-up group. For CEA, the lost to follow-up patients were younger and more likely to be symptomatic. There were no other significant differences between those patients who followed up and those who did not (Table I). Further, our referral pattern has also led our institution to have a disproportionate number of CAS procedures, but the number of CEA and CAS procedures was stable during the years interrogated in this manuscript (Fig 2).

CONCLUSIONS

CAS is associated with increased rates of ECA occlusion over CEA, and patients with ECA occlusion after CAS had an increased incidence of neck irradiation compared with CAS patients who had patent ECAs. Ongoing work in our laboratory modeling altered flow through the stent into the ECA by use of clinical imaging and ex vivo arterial culture will help better understand the biology of this phenomenon. Finally, the impact of arterial flow through stent struts on the arterial wall biology is important to all vascular trees and not limited only to ECA occlusion. We hope this work will help spur further investigation of this finding.

Acknowledgments

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References


Fig 1.
Overview of data analysis. Flow diagram showing cohort selection algorithm and exclusion criteria. CAS, Carotid artery stenting; CEA, carotid endarterectomy; ECA, external carotid artery; N, number of procedures. *P < .05.
Fig 2.
Annual carotid therapy case volume. Procedure numbers segregated as carotid artery stenting (CAS) and carotid endarterectomy (CEA) per year during the course of the study. The year 2012 was not included as only January and February were included in the study.
Table I
Baseline demographics of carotid artery stenting (CAS) and carotid endarterectomy (CEA) treatment groups

<table>
<thead>
<tr>
<th>Variable</th>
<th>CAS (n = 312)</th>
<th>CEA (n = 344)</th>
<th>P value(^c), CAS vs CEA</th>
<th>P value(^a), CAS no f/u, No. (%)</th>
<th>P value(^b), CEA no f/u, No. (%)</th>
<th>P value(^e), CAS vs CEA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Demographics</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Patients</td>
<td>210 (67)</td>
<td>102 (33)</td>
<td>1</td>
<td>207 (60)</td>
<td>137 (40)</td>
<td>0.001(^e)</td>
</tr>
<tr>
<td>Age, years</td>
<td>68</td>
<td>68</td>
<td>1</td>
<td>70</td>
<td>67</td>
<td>0.03(^e)</td>
</tr>
<tr>
<td>Follow-up, months</td>
<td>12.5</td>
<td>NA</td>
<td>NA</td>
<td>56.2</td>
<td>NA</td>
<td>NA</td>
</tr>
<tr>
<td>Gender, male</td>
<td>134 (64)</td>
<td>51 (53)</td>
<td>0.03(^e)</td>
<td>127 (61)</td>
<td>81 (60)</td>
<td>7</td>
</tr>
<tr>
<td>Symptomatic</td>
<td>91 (43)</td>
<td>46 (52)</td>
<td>0.8</td>
<td>77 (37)</td>
<td>66 (48)</td>
<td>0.05(^e)</td>
</tr>
<tr>
<td>Comorbidities</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smoking</td>
<td>67 (32)</td>
<td>38 (37)</td>
<td>0.4</td>
<td>67 (32)</td>
<td>36 (26)</td>
<td>2</td>
</tr>
<tr>
<td>Diabetes</td>
<td>74 (36)</td>
<td>35 (34)</td>
<td>0.9</td>
<td>69 (33)</td>
<td>48 (35)</td>
<td>8</td>
</tr>
<tr>
<td>HTN</td>
<td>188 (89)</td>
<td>93 (91)</td>
<td>0.7</td>
<td>192 (93)</td>
<td>122 (89)</td>
<td>2</td>
</tr>
<tr>
<td>HLD</td>
<td>153 (72)</td>
<td>78 (77)</td>
<td>0.6</td>
<td>160 (77)</td>
<td>106 (77)</td>
<td>1</td>
</tr>
<tr>
<td>CKD</td>
<td>20 (10)</td>
<td>9 (9)</td>
<td>1</td>
<td>25 (12)</td>
<td>14 (10)</td>
<td>9</td>
</tr>
<tr>
<td>Cervical irradiation</td>
<td>31 (15)</td>
<td>8 (8)</td>
<td>0.1</td>
<td>4 (2)</td>
<td>1 (1)</td>
<td>0.6</td>
</tr>
<tr>
<td>Prior CEA</td>
<td>55 (26)</td>
<td>25 (25)</td>
<td>0.8</td>
<td>16 (8)</td>
<td>12 (9)</td>
<td>0.8</td>
</tr>
<tr>
<td>Antiplatelet regimen</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASA</td>
<td>175 (84)</td>
<td>83 (85)(^d)</td>
<td>0.7</td>
<td>184 (89)</td>
<td>116 (87)(^d)</td>
<td>0.6</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>197 (94)</td>
<td>89 (92)(^d)</td>
<td>0.6</td>
<td>73 (35)</td>
<td>41 (31)(^d)</td>
<td>0.4</td>
</tr>
</tbody>
</table>

ASA, Aspirin; CKD, chronic kidney disease; f/u, follow-up; HLD, hyperlipidemia; HTN, hypertension; NA, not applicable.

Baseline demographics of patients who underwent CAS and CEA (comparison in far right column). CEA patients were older, had longer follow-up, were less likely to have had irradiation or prior CEA, and were less likely to be taking clopidogrel. CAS and CEA patients were then segregated by those who followed up and those who did not. For CAS, there were more male patients in the follow-up group. For CEA, the follow-up patients were older and less likely to be symptomatic than those who did not follow up.

\(^a\)P value of CAS compared with CAS without follow-up.

\(^b\)P value of CEA compared with CEA without follow-up.

\(^c\)P value of CAS compared with CEA.
Discharge medications percentage and $P$ value calculated excluding in-hospital deaths (CAS, $n = 5$; CEA, $n = 3$).

Statistically significant.
Table II

Clinical outcomes of patients undergoing carotid artery stenting (CAS) compared with carotid endarterectomy (CEA)

<table>
<thead>
<tr>
<th>Variable</th>
<th>CAS, No. (%)</th>
<th>CAS no f/u, No. (%)</th>
<th>P value&lt;sup&gt;a&lt;/sup&gt;</th>
<th>CEA, No. (%)</th>
<th>CEA no f/u, No. (%)</th>
<th>P value&lt;sup&gt;b&lt;/sup&gt;</th>
<th>P value&lt;sup&gt;c&lt;/sup&gt;, CAS vs CEA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients</td>
<td>210</td>
<td>102</td>
<td></td>
<td>207</td>
<td>137</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ECA occlusion</td>
<td>8 (4)</td>
<td>NA</td>
<td>NA</td>
<td>1 (0.4)</td>
<td>NA</td>
<td>NA</td>
<td>.04&lt;sup&gt;d&lt;/sup&gt;</td>
</tr>
<tr>
<td>30-day stroke/death/MI</td>
<td>8 (4)</td>
<td>NA</td>
<td>NA</td>
<td>4 (2)</td>
<td>NA</td>
<td>NA</td>
<td>.4</td>
</tr>
<tr>
<td>CVA</td>
<td>5 (2)</td>
<td>4 (4)</td>
<td>.5</td>
<td>3 (1.4)</td>
<td>5 (4)</td>
<td>.3</td>
<td>1</td>
</tr>
<tr>
<td>TIA</td>
<td>2 (0.9)</td>
<td>0 (0)</td>
<td>.6</td>
<td>2 (0.9)</td>
<td>1 (1)</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Death</td>
<td>5 (2)</td>
<td>0 (0)</td>
<td>.2</td>
<td>3 (1)</td>
<td>0 (0)</td>
<td>.3</td>
<td>.7</td>
</tr>
<tr>
<td>MI</td>
<td>0 (0)</td>
<td>1 (1)</td>
<td>.3</td>
<td>0 (0)</td>
<td>2 (1)</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Bradycardia/hypotension</td>
<td>10 (4.7)</td>
<td>10 (10)</td>
<td>.1</td>
<td>5 (2.4)</td>
<td>2 (2)</td>
<td>1</td>
<td>.3</td>
</tr>
<tr>
<td>Hyperperfusion syndrome</td>
<td>2 (0.9)</td>
<td>0 (0)</td>
<td>.6</td>
<td>2 (0.9)</td>
<td>1 (1)</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Wound site hematoma</td>
<td>2 (0.9)</td>
<td>2 (2)</td>
<td>.6</td>
<td>1 (0.4)</td>
<td>3 (2)</td>
<td>.3</td>
<td>1</td>
</tr>
</tbody>
</table>

CVA, Cerebrovascular accident; ECA, external carotid artery; f/u, follow-up; MI, myocardial infarction; NA, not applicable; TIA, transient ischemic attack.

Clinical outcomes comparing CAS with CEA and differences between those who followed up in our vascular laboratory and those who did not among CAS and CEA patients. There were no differences within the groups, but ECA occlusion was significantly more common in the CAS group.

<sup>a</sup>P value for CAS compared with CAS without follow-up.

<sup>b</sup>P value for CEA compared with CEA without follow-up.

<sup>c</sup>P value for CAS compared with CEA.

<sup>d</sup>Indicates clinical significance.
Table III

Average preoperative external carotid artery (ECA) velocities

<table>
<thead>
<tr>
<th>Variable</th>
<th>ECA occlusion, average velocity</th>
<th>No ECA occlusion, average velocity</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>CAS group</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PSV</td>
<td>201 cm/s</td>
<td>203 cm/s</td>
<td>1</td>
</tr>
<tr>
<td>EDV</td>
<td>44 cm/s</td>
<td>31 cm/s</td>
<td>&lt;.001&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>CEA group</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PSV</td>
<td>246 cm/s</td>
<td>150 cm/s</td>
<td>NA&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>EDV</td>
<td>48 cm/s</td>
<td>23 cm/s</td>
<td>NA&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

CAS, Carotid artery stenting; CEA, carotid endarterectomy; EDV, end-diastolic velocity; PSV, peak systolic velocity.

Preoperative PSV among those in the CAS group was not statistically different among those with and without ECA occlusion, but preoperative EDV was significantly higher among CAS patients who went on to ECA occlusion. The statistical difference could not be calculated among the CEA group because there was only one occluded ECA in the occlusion group.

<sup>a</sup> Indicates clinical significance.

<sup>b</sup> Only one value for velocity in CEA group with ECA occlusion; therefore, Student t-test and P value could not be calculated.
### Table IV

Indications for stenting in carotid artery stenting (CAS) group

<table>
<thead>
<tr>
<th>Variable</th>
<th>ECA occlusion, No. (%)</th>
<th>No ECA occlusion, No. (%)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients</td>
<td>8</td>
<td>196</td>
<td></td>
</tr>
<tr>
<td>Cervical irradiation</td>
<td>4 (50)</td>
<td>30 (15)</td>
<td>.03a</td>
</tr>
<tr>
<td>Prior CEA</td>
<td>1 (13)</td>
<td>56 (27)</td>
<td>.44</td>
</tr>
<tr>
<td>Prohibitive anatomy</td>
<td>2 (25)</td>
<td>34 (17)</td>
<td>.63</td>
</tr>
<tr>
<td>Medical comorbidities</td>
<td>1 (13)</td>
<td>3 (0.2)</td>
<td>.15</td>
</tr>
</tbody>
</table>

*CEA, Carotid endarterectomy; ECA, external carotid artery.

Prior cervical irradiation was more common in the CAS patients who went on to ECA occlusion than in those who did not. There were no other interactions between indications for CAS and ECA occlusion.

*a* Indicates clinical significance.
Table V

In-stent restenosis (ISR) among carotid artery stenting (CAS) patients with and without external carotid artery (ECA) occlusion

<table>
<thead>
<tr>
<th>Variable</th>
<th>ECA occlusion, No. (%)</th>
<th>No ECA occlusion, No. (%)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients</td>
<td>8 (3.8)</td>
<td>193 (96.2)</td>
<td></td>
</tr>
<tr>
<td>ISR &gt;50%</td>
<td>2 (25)</td>
<td>27 (14)</td>
<td>.6</td>
</tr>
<tr>
<td>ISR &gt;50–69%</td>
<td>2 (25)</td>
<td>18 (9)</td>
<td>.2</td>
</tr>
<tr>
<td>ISR &gt;70%</td>
<td>0 (0)</td>
<td>8 (4)</td>
<td>1</td>
</tr>
<tr>
<td>Stent occlusion</td>
<td>0 (0)</td>
<td>1 (0.5)</td>
<td>1</td>
</tr>
</tbody>
</table>

ISR >70%: axial imaging or peak systolic velocity >175 cm/s and end-diastolic velocity >140 cm/s. ISR 60–69%: peak systolic velocity >175 cm/s.

There was no interaction identified between ECA occlusion and ISR or CAS occlusion.