Restenosis Is More Frequent After Carotid Stenting Than After Endarterectomy
The EVA-3S Study

Caroline Arquizan, MD; Ludovic Trinquart, MSc; Pierre-Jean Touboul, MD; Anne Long, MD; Séverine Feasson, MD; Béatrice Terriat, MD; Marie-Pierre Gobin-Metteil, MD; Brigitte Guidolin, MD; Serge Cohen, MD; Jean-Louis Mas, MD; for the EVA-3S Investigators

Background and Purpose—Carotid angioplasty and stenting (CAS) may be more often associated with residual or recurrent stenosis than carotid endarterectomy (CEA). We compared the rates of restenosis in patients treated with CAS or CEA in the EVA-3S trial.

Methods—Five hundred seven patients (242 treated by CAS and 265 by CEA) had carotid ultrasound follow-up (mean carotid ultrasound follow-up, 2.1 years) according to a predefined protocol. Carotid restenosis of 50% to 69% was diagnosed on planimetry, whereas carotid restenosis of ≥70% or occlusion was diagnosed using either planimetry or velocity criteria.

Results—The rate of carotid restenosis of ≥50% or occlusion was significantly higher after CAS (12.5%) than after CEA (5.0%; time ratio, 0.16; 95% CI, 0.03–0.76; \( P = 0.02 \)). The rates of severe restenosis of ≥70% or occlusion were low and did not differ significantly between the 2 groups (3-year rates are 3.3% in the CAS group and 2.8% in the CEA group). Age at baseline was the only vascular risk factor significantly associated with carotid restenosis. Our study could not detect any effect of carotid restenosis on ipsilateral stroke.

Conclusions—The short-term rate of carotid restenosis of ≥50% or occlusion is 2.5-times more common after CAS than after CEA, a difference accounted for by an excess risk in moderate restenosis. More data with longer follow-up are needed to assess the rates of late severe restenosis and to determine the relation between restenosis and recurrent stroke over time. (Stroke. 2011;42:1015-1020.)

Key Words: angioplasty ■ carotid endarterectomy ■ carotid stenosis ■ Doppler ultrasound ■ stenting

Carotid angioplasty and stenting (CAS) is being evaluated as a potential alternative to carotid endarterectomy (CEA) in patients with severe carotid artery stenosis. CAS appears to be more often associated with residual or recurrent stenosis than endarterectomy, which might restrict its long-term efficacy for stroke prevention. However, more data from randomized clinical trials are needed to better evaluate the relative risk of carotid restenosis in patients treated with CAS as compared with those treated with CEA, to identify risk factors for restenosis and to assess the impact of restenosis on the rate of recurrent ipsilateral strokes. We report on a predefined carotid ultrasound follow-up study of patients enrolled in the Endarterectomy vs Angioplasty in Patients with Symptomatic Severe Carotid Stenosis (EVA-3S) trial.

Materials and Methods
The methods and results of the EVA-3S study have been reported previously. This multicenter, randomized, open, assessor-blinded, noninferiority trial was approved by the ethics committee of Cochin Hospital (Paris, France). All patients gave written informed consent. Patients were eligible if they were aged 18 years or older, had experienced a hemispheric or retinal TIA or a nondisabling stroke (or retinal infarct) within 120 days before enrollment, and had an atherosclerotic stenosis of 60% to 99% of the symptomatic carotid artery, as determined by the North American Symptomatic Carotid Endarterectomy Trial (NASCET) method by means of catheter angiography or both duplex scanning and MRA.

Carotid Ultrasound
The study protocol recommended that carotid ultrasound should be performed at 1, 6, 12, 18, 24, and 36 months after treatment and results should be recorded on a standardized study report form. The
carotid ultrasound protocol included Doppler velocity measurements and B-mode imaging assisted by color-coded Duplex. The following parameters had to be systematically recorded: lumen diameters of the operated carotid artery at the point of maximum stenosis (if any) and in the normal artery distal to the stenosis; lumen diameters of the stented carotid artery measured at the site of maximum in-stent stenosis (if any), in the distal portion of the stent, and in the normal artery distal to the stent; peak systolic velocities of the internal carotid artery (ICA); and presence of contralateral carotid stenosis of ≥70% or occlusion. Patients who could not attend all carotid ultrasound visits at their participating center were asked to have carotid ultrasound performed outside the center (786 of 2273 carotid examinations). All study report forms and reports of examinations performed outside participating centers were collected by the coordinating center. One investigator (C.A.) ascertained the degree of stenosis on the basis of predefined criteria and made the final classification of stenosis. No other arterial investigation was required by the protocol to confirm the degree of restenosis.

Carotid restenosis of 50% to 69% was diagnosed on planimetry, whereas carotid restenosis of ≥70% or occlusion was diagnosed when it was present on either planimetry or velocity parameters. The degree of carotid stenosis on planimetry was measured according to the NASCET method, ie, by comparing the lumen diameter of the carotid artery (at the narrowest point of stenosis) to the lumen diameter of the normal ICA distal to the stenosis. When the distal carotid artery could not be examined on planimetry in patients treated with CAS, we measured the degree of in-stent stenosis by comparing the in-stent lumen diameter of the carotid artery (at the narrowest point of stenosis) to the lumen diameter of the distal portion of the stent. ICA peak systolic velocities of >210 cm/s in patients treated with CEA7 and of >300 cm/s in those treated with CAS8–10 were used to define carotid restenosis of ≥70%. Restenosis cannot result from poor initial result of revascularization (residual stenosis) or recurrent stenosis after successful revascularization. Recurrent stenosis was defined as the absence of residual stenosis at 1 month, with subsequent development of ≥50% stenosis or occlusion.

Statistical Analysis
We performed a per protocol analysis of patients who completed CAS or CEA and had follow-up data with carotid ultrasound. We assessed agreement between NASCET and in-stent methods, and between the NASCET method and velocity criteria, for the degree of carotid restenosis. We computed kappa coefficients and indices of average positive agreement and average negative agreement.

Because the development of restenosis was assessed at periodic follow-up visits, the time from revascularization to restenosis was interval-censored. Restenosis occurred between the follow-up visit when restenosis was first noted and the previous visit. We used an extension of the Kaplan-Meier estimation to interval-censored times to estimate the cumulative probability of restenosis.11 Score tests were performed to test for any difference between the 2 groups. We used these methods to assess the rates of ipsilateral restenosis of ≥50% and ≥70%, as well as the rates of contralateral stenosis of ≥70%.

We assessed the effect of treatments and baseline characteristics on the time from revascularization to restenosis using an accelerated failure–time model, which handles interval-censored data. Results from this analysis are expressed using time ratios. They indicate the degree to which a covariate accelerates (or decelerates) the time to restenosis. For instance, an estimated time ratio of 0.20 for CAS vs CEA means that the time to restenosis after CEA is estimated to be 20% of that after CEA, or that CAS is estimated to shorten time to restenosis by 80%. Each model included the treatment group and 1 of the following variables: age, sex, hypertension, diabetes (recorded at randomization); smoking, antihypertensive, antiplatelet, and lipid-lowering therapy (recorded at the 1-month follow-up visit); and type of stent (closed-cell vs open-cell). A 2-sided P≤0.05 was considered statistically significant. All analyses were performed using SAS and R software.

### Results
Among 527 patients enrolled in EVA-3S, 265 were randomly assigned to CAS and 262 were assigned to CEA. Of the 265 patients assigned to CAS, 4 did not have carotid revascularization, 1 crossed over to CEA, and in 13 stenting was converted intraoperatively to CEA. Of the 262 patients randomly assigned to CEA, 3 did not have carotid revascularization and 2 crossed over to CAS. Thirteen patients did not have carotid ultrasound follow-up, leaving 507 patients for the present study, of whom 242 were treated by CAS and 265 were treated by CEA.

Baseline clinical characteristics of patients included in the present study were similar to those of the whole population of EVA-3S, and there were no significant differences between those assigned to CAS and those assigned to CEA (data not shown). There was no difference between the 2 groups with regard to the numbers of carotid ultrasound examinations and duration of carotid ultrasound follow-up (Table 1).

The agreement between NASCET and in-stent measurements of the degree of stenosis in patients treated with CAS was good (kappa coefficient, 0.74; 95% CI, 0.51–0.88; Table 2). There was a nonsignificant tendency for the NASCET method to overestimate the degree of stenosis compared to

### Table 1. Carotid Duplex Ultrasound During Follow-Up

<table>
<thead>
<tr>
<th>Time from treatment</th>
<th>Carotid Angioplasty and Stenting (n=242)</th>
<th>Carotid Endarterectomy (n=265)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1–14 days</td>
<td>185</td>
<td>190</td>
</tr>
<tr>
<td>14 days–3 mo</td>
<td>63</td>
<td>76</td>
</tr>
<tr>
<td>3–9 mo</td>
<td>198</td>
<td>219</td>
</tr>
<tr>
<td>9–15 mo</td>
<td>179</td>
<td>183</td>
</tr>
<tr>
<td>15–21 mo</td>
<td>172</td>
<td>175</td>
</tr>
<tr>
<td>21–27 mo</td>
<td>167</td>
<td>168</td>
</tr>
<tr>
<td>27–33 mo</td>
<td>55</td>
<td>46</td>
</tr>
<tr>
<td>33–39 mo</td>
<td>99</td>
<td>98</td>
</tr>
<tr>
<td>Total of duplex examinations</td>
<td>1118</td>
<td>1155</td>
</tr>
<tr>
<td>N of carotid duplex ultrasound examinations during follow-up per patient, median (25%, 75% percentiles)</td>
<td>5 (4, 6)</td>
<td>5 (3, 6)</td>
</tr>
<tr>
<td>Mean duration of carotid duplex ultrasound follow-up, mo</td>
<td>26</td>
<td>25</td>
</tr>
</tbody>
</table>

### Table 2. Agreement Between the NASCET and the In-Stent Methods in Patients Treated With Carotid Angioplasty and Stenting

<table>
<thead>
<tr>
<th>Stenosis</th>
<th>&lt;50%</th>
<th>50%–69%</th>
<th>70%–100%</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>NASCET Method</td>
<td>403</td>
<td>3</td>
<td>0</td>
<td>406</td>
</tr>
<tr>
<td>50%–69%</td>
<td>6</td>
<td>2</td>
<td>1</td>
<td>9</td>
</tr>
<tr>
<td>70%–100%</td>
<td>0</td>
<td>0</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>Total</td>
<td>409</td>
<td>5</td>
<td>7</td>
<td>421</td>
</tr>
</tbody>
</table>

Weighted kappa coefficient, 0.74; 95% CI, 0.51–0.88.
the in-stent method. Figure 1 shows that the diameter of the stent was on average larger than the diameter of the ICA distal to the stent. Among 421 patients who had both measurements, the median difference was 0.2 mm (25% and 75% percentiles, −3.7 and 4.0; P < 0.0001). Although the overall agreement between the NASCET method and velocity criteria for the degree of stenosis was good, the observed proportion of positive agreement was poor for the detection of moderate stenosis (27.7%), in both the CEA and the CAS groups. By contrast, the agreement was excellent for the diagnosis of severe stenosis of ≥70% or occlusion (Table 3).

At 3 years, 27 patients with CAS had restenosis of ≥50% or occlusion compared to 12 patients with CEA, giving cumulative rates of 12.5% and 5.0%, respectively, with a time ratio of 0.16 (95% CI, 0.03–0.76; P = 0.02; Figure 2). Restenosis of ≥70% or occlusion was diagnosed in 5 patients treated with CAS (1 occlusion) and 7 patients treated with CEA (2 occlusions), giving 3-year cumulative rates of 3.3% and 2.8%, respectively. The time ratio was 3.30 (95% CI, 0.16–67.69; P = 0.44). Of the 12 patients who had severe restenosis or occlusion, 5 patients were treated by CAS during follow-up.

Among patients with restenosis, the majority had recurrent restenosis. Only 1 patient treated with CAS and 2 treated with CEA had residual stenoses of ≥50% at 1 month after the procedure. The rates of contralateral stenosis of ≥70% or occlusion were 19.8% in the CAS group and 18.5% in the CEA group. The time ratio was 0.98 (95% CI, 0.44–2.19; P = 0.96). Table 4 shows that age was the only independent predictor of carotid restenosis.

Among the 39 patients with carotid restenosis, 31 (79%) were receiving antihypertensive therapy, 30 (77%) were receiving lipid-lowering therapy, and 37 (95%) were receiving antiplatelet therapy, and 7 (18%) were smokers at the clinical follow-up visit closest to the carotid ultrasound examination showing carotid restenosis. These proportions were similar in the CAS and the CEA groups. These proportions were also similar to those observed in the whole study population, except for a lower proportion of smokers in the whole population (85%, 77%, 97%, and 10%, respectively).

Drug treatment and smoking status recorded at the time of restenosis detection and at the preceding carotid ultrasound examination were identical, except for 1 patient who started smoking before restenosis.

Table 3. Agreement Between the NASCET Method and Velocity Criteria

<table>
<thead>
<tr>
<th>NASCET Method</th>
<th>&lt;50%</th>
<th>50%–69%</th>
<th>70%–100%</th>
<th>Not Interpretable</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stenosis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;50%</td>
<td>1778</td>
<td>29</td>
<td>2</td>
<td>5</td>
<td>1814</td>
</tr>
<tr>
<td>50%–69%</td>
<td>26</td>
<td>10</td>
<td>0</td>
<td>19</td>
<td>55</td>
</tr>
<tr>
<td>70%–100%</td>
<td>0</td>
<td>0</td>
<td>29</td>
<td>0</td>
<td>29</td>
</tr>
<tr>
<td>Total</td>
<td>1804</td>
<td>39</td>
<td>31</td>
<td>24</td>
<td>1898</td>
</tr>
<tr>
<td>Carotid endarterectomy</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;50%</td>
<td>908</td>
<td>24</td>
<td>2</td>
<td>2</td>
<td>936</td>
</tr>
<tr>
<td>50%–69%</td>
<td>1</td>
<td>4</td>
<td>0</td>
<td>5</td>
<td>10</td>
</tr>
<tr>
<td>70%–100%</td>
<td>0</td>
<td>0</td>
<td>19</td>
<td>0</td>
<td>19</td>
</tr>
<tr>
<td>Total</td>
<td>909</td>
<td>28</td>
<td>21</td>
<td>7</td>
<td>965</td>
</tr>
<tr>
<td>Carotid angioplasty and stenting</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;50%</td>
<td>870</td>
<td>5</td>
<td>0</td>
<td>3</td>
<td>878</td>
</tr>
<tr>
<td>50%–69%</td>
<td>25</td>
<td>6</td>
<td>0</td>
<td>14</td>
<td>45</td>
</tr>
<tr>
<td>70%–100%</td>
<td>0</td>
<td>0</td>
<td>10</td>
<td>0</td>
<td>10</td>
</tr>
<tr>
<td>Total</td>
<td>895</td>
<td>11</td>
<td>10</td>
<td>17</td>
<td>933</td>
</tr>
</tbody>
</table>

*In patients with stenting, we used the in-stent method when distal carotid artery was not seen.

Weighted kappa coefficient (excluding noninterpretable results), 0.69; 95% CI, 0.65–0.73.

Weighted kappa coefficient (excluding noninterpretable results) for stenosis >70%, 0.97; 95% CI, 0.92–1.00.
From day 31 after the revascularization to 36 months, 17 patients had a recurrent stroke (10 in the CAS group and 7 in the CEA group), among which 6 had an ipsilateral stroke (3 in each group). Recurrent stroke occurred (after restenosis) in 1 (2.6%) of the 39 patients with restenosis compared to 16 (3.4%) of the 468 patients without restenosis ($P=0.99$). Four (10.3%) of the patients with restenosis and 25 (5.3%) of those without restenosis had a stroke or TIA during follow-up ($P=0.27$; Table 5).

### Discussion

In this predefined carotid ultrasound follow-up study of patients enrolled in EVA-3S, the 3-year rate of carotid restenosis of $\geq 50\%$ or occlusion was higher after CAS than after CEA, with 3-year cumulative risks of 12.5% and 5%, respectively. The excess rate of restenosis was driven by an excess rate of moderate restenosis. The rates of severe restenosis or occlusion were low and did not differ significantly between groups.

Although carotid ultrasound is considered the standard technique to detect restenosis in patients treated with CEA, there are no broadly accepted criteria for ultrasound of stented carotid arteries. The placement of a stent creates a...
“new” artery, with potential consequences on the planimetric and hemodynamic criteria used to estimate the degree of stenosis. Our study shows a good agreement between the degree of restenosis measured by the NASCET method and that measured by the in-stent method. The in-stent method could be an interesting alternative, particularly in patients in whom the normal ICA distal to the stent cannot be examined by ultrasound. There was a nonsignificant tendency for the NASCET method to overestimate the degree of stenosis compared to the in-stent method, which can be explained by our finding that the diameter of the ICA distal to the stent is on average smaller than the diameter of the stent (Figure 1). Whereas flow velocity criteria equate well with the presence of ≥70% carotid stenosis measured on catheter angiography with the NASCET method in patients with native carotid artery,7 velocity criteria corresponding to severe carotid stenosis of ≥70% have not been well-established in patients with stented carotid arteries. Reduction of compliance of stented carotid arteries could increase peak systolic velocities and lead to an overestimation of the degree of stenosis. Consequently, we used a higher threshold of peak systolic velocities (>300 cm/s)8–10 to identify severe (>70%) carotid restenosis in patients treated with CAS. However, we found no major differences in the rates of severe restenosis when analyses were performed with the same peak systolic velocities threshold in patients with CAS or CEA, as compared with analyses with different thresholds (data not shown). We used planimetry to diagnose carotid restenosis of 50% to 69% because it provides an angiographic-like carotid stenosis measurement, with a good sonographic–angiographic agreement.12–18 In addition, although there is a good agreement for severe stenosis between velocity parameters and planimetric data obtained by angiography or carotid ultrasound,9,19,20 moderate stenoses are quantified less accurately by velocity parameters,21 particularly in the setting of a multicenter study, which potentially increases variability of velocity measurements between centers.9 In our study, the observed proportion of positive agreement between velocity and planimetric data were poor for the category of 50% to 69% restenosis (Table 3).

Two previous randomized clinical trials have reported higher rates of restenosis in patients treated with CAS than in those treated with CEA. In the SPACE trial,1 the 2-year cumulative risk of severe restenosis or occlusion was 11% after stenting and 4.6% after endarterectomy. Approximately half of restenoses occurred in the first 6 months after treatment in both groups. The SPACE study protocol did not stipulate specific cut-off values. Stenoses were graded by way of hemodynamic parameters in the local ultrasound laboratory. In the CAVATAS study,2 the 5-year cumulative incidence of ≥70% restenosis was 30.7% in patients who had endovascular treatment and 16.5% in those (25.6%) who received a stent compared with 11% in the CEA group. For stenoses ≥50% or occlusion, the rates were 58.6% after endovascular treatment (36.6% in patients who received a stent) compared with 32% in patients treated with surgery. The lower rates of restenosis in our study compared with CAVATAS might be explained by differences in stenting techniques, medical treatment, ultrasound criteria for grading restenosis, and length of follow-up. Primary stenting is now the endovascular treatment of choice for severe carotid stenosis, whereas in CAVATAS most stenting procedures were performed after balloon dilatation was attempted. Improvement of medical treatment (in particular the use of statins) since CAVATAS was designed might have decreased the risk of restenosis after stenting. Finally, the use of identical flow velocity criteria to define stenoses after endovascular treatment and CEA may have led to overestimation of restenosis in patients who received stents.

In our study, the majority of restenoses after stenting as well as after CEA were recurrent stenoses, which contrasts with CAVATAS,22 in which residual stenosis accounted for more than one-third of severe restenoses, probably because most patients were treated with angioplasty alone. As in previous studies, most restenoses occurred in the first year after both CAS and CEA. This suggests that similar mechanisms are responsible for early restenosis after CAS and CEA. Age at baseline was the only factor associated with carotid restenosis. Unlike in CAVATAS,2 smoking recorded at the 1-month follow-up visit was not a significant predictor of restenosis. The type of stent did not seem to influence the rate of restenosis. The proportions of patients receiving antihypertensive, lipid-lowering, or antiplatelet therapy at the time of restenosis were similar to those in the whole study population. Smoking tended to be more frequent in patients with carotid restenosis than in the whole study population.

The rates of recurrent cerebral ischemic events did not differ significantly between patients with or without restenosis (Table 5), but the number of recurrent events was low, precluding any meaningful analysis. In SPACE,1 only 2 patients with recurrent stenosis after CAS had neurological symptoms during 2 years of follow-up. In CAVATAS,2 severe carotid restenosis or occlusion diagnosed within the first year after treatment was associated with an increased risk of subsequent ipsilateral stroke or TIA (5-year incidence of 23% vs 11%; P=0.04), but the increase in ipsilateral stroke alone was not significant (10% vs 6%).

Conclusions

In conclusion, the 3-year rate of restenosis of ≥50% or occlusion in EVA-3S was more than twice as high after CAS as after CEA. The excess rate of restenosis after CAS was attributable to an excess in moderate restenosis, whereas the rates of severe restenosis (≥70%) or occlusion was low and similar in both treatment groups. However, the proportion of patients with severe restenosis after CAS may increase over time. More data are needed to assess the rates of moderate and severe restenosis over time after CAS by means of identical ultrasound criteria to determine the relation between restenosis and recurrent stroke over time and to assess whether patients treated with endarterectomy or stenting require long-term follow-up with carotid ultrasound to detect restenosis.

Sources of Funding

Funding received from the French Ministry of Health (Programme Hospitalier de Recherche Clinique, Assistance Publique des Hôpitaux de Paris [AOM 97066]).

Disclosure

None.
References


Restenosis Is More Frequent After Carotid Stenting Than After Endarterectomy: The EVA-3S Study
Caroline Arquizan, Ludovic Trinquart, Pierre-Jean Touboul, Anne Long, Séverine Feasson, Béatrice Terriot, Marie-Pierre Gobin-Metteil, Brigitte Guidolin, Serge Cohen and Jean-Louis Mas

Stroke. 2011;42:1015-1020; originally published online February 10, 2011;
doi: 10.1161/STROKEAHA.110.589309
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2011 American Heart Association, Inc. All rights reserved.
Print ISSN: 0039-2499. Online ISSN: 1524-4628

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/42/4/1015

Data Supplement (unedited) at:
http://stroke.ahajournals.org/content/suppl/2012/03/12/STROKEAHA.110.589309.DC1

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org//subscriptions/
재협착은 내막절제술보다 경동맥 스텀트 이후 더 훈하다
EVA-3S 연구

Restenosis Is More Frequent After Carotid Stenting Than After Endarterectomy
The EVA-3S Study

Caroline Arquizan, MD; Ludovic Trinquart, MSc; Pierre-Jean Touboul, MD; Anne Long, MD; Séverine Feasson, MD; Béatrice Terriat, MD; Marie-Pierre Gobin-Metteil, MD; Brigitte Guidolin, MD; Serge Cohen, MD; Jean-Louis Mas, MD; for the EVA-3S Investigators

(Stroke. 2011;42:1015-1020.)

Key Words: angioplasty ■ carotid endarterectomy ■ carotid stenosis ■ Doppler ultrasound ■ stenting

배경과 목적
경동맥 혈관성형술과 스텀트 시술(carotid angioplasty and stenting, CAS)은 경동맥내막절제술(carotid endarterectomy, CEA)에 비하여 혈착이 남아 있거나 다시 생길 가능성이 더 높은 것이다. 저작들은 EVA-3S 시험에서 CAS나 CEA로 치료를 받은 환자들에서 재협착률을 비교하였다.

방법
507명의 환자(242명은 CAS, 265명은 CEA)가 미리 정한 프로토콜에 따라 경동맥 초음파 추적 검사(평균 2.1년의 추적 검사를 받았다. 50~69%의 경동맥 재협착은 planimetry로 진단하였고, 70% 이상의 경동맥 재협착이나 패색은 planimetry 또는 혈류 속도 기준을 이용하여 진단하였다.

결과
50% 이상의 경동맥 재협착이나 패색의 비율은 CEA (5.0%; time ratio, 0.16; 95% CI, 0.03~0.76; P=0.02)보다는 CAS (12.5%)가 더 높았다. 70% 이상의 심각한 재협착이나 패색의 비율은 낮았고, 두 군에서 의미 있는 차이는 없었다.3년 발생률은 CAS군에서 3.3%, CEA군에서는 2.8%). 시작 시점의 연령은 경동맥 재협착과 관련이 있는 유일한 혈관 위험인자였다. 본 연구에서 경동맥 재협착은 동측의 뇌졸중 빈도와 연관이 없었다.

결론
50% 이상의 경동맥 재협착이나 패색의 단기간 발생률도 CEA보다는 CAS가 약 2.5배 정도 더 높았고, 이는 중등도의 재협착의 위험이 증가하기 때문이다. 후기의 심한 재협착 발생률 및 재협착과 뇌졸중 재발의 관계에 대하여 조사하려면 더 긴 기간의 추적 조사 결과가 필요할 것이다.