

The Fate of the External Carotid Artery after Carotid Artery Stenting. A Follow-up Study with Duplex Ultrasonography[☆]

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Objective. To evaluate the long-term effect of carotid angioplasty and stenting (CAS) of the internal carotid artery (ICA) on the ipsilateral external carotid artery (ECA).

Subjects and Methods. We prospectively registered the pre- and post-interventional duplex scans obtained from 312 patients (mean age 70 years) who underwent CAS. Duplex scans were scheduled the day before CAS, 3 and 12 months post-procedurally and yearly thereafter, to study progression of obstructive disease in the ipsilateral ECA compared to the contralateral ECA. The duplex ultrasound criteria used to identify ECA stenosis $\geq 50\%$ were Peak Systolic Velocities of ≥ 125 cm/s.

Results. Preprocedural evaluation of the ipsilateral ECA demonstrated $\geq 50\%$ stenosis in 32.7% of cases vs 30% contralateral. Both ipsilateral and contralateral 3 (1%) ECA occlusions were noted. After stenting 5 (1.8%) occlusions were seen vs 1% contralateral. No additional ipsilateral occlusions and 2 additional contralateral occlusions were noted at extended follow-up. The prevalence of $\geq 50\%$ stenosis of the ipsilateral ECA (Kaplan-Meier estimates) progressed from 49.1% at 3, to 56.4%, 64.7%, 78.2%, 72.3%, and 74% at 12, 24, 36, 48, and 60 months respectively. Contralateral prevalences were 31.3%, 37.7%, 41.7%, 43.1%, 46.0%, and 47.2% respectively ($p < 0.001$). Progression of stenosis was more pronounced in 234 patients (75%) with overstenting of the carotid bifurcation ($p = 0.004$).

Conclusion. Our results show that significant progression of $\geq 50\%$ stenosis in the ipsilateral ECA occurs after CAS. There was greater progression of disease in the ipsilateral compared with the contralateral ECA. Progression of disease in the ECA did not lead to the occurrence of occlusion during follow up.

Keywords: CAS; External carotid artery; Duplex US; Follow-up stenosis.

Introduction

Carotid Angioplasty and Stenting (CAS) has emerged as an alternative to carotid endarterectomy (CEA) in treatment of carotid artery occlusive disease.¹ Despite promising early results, recurrent stenosis and its management are reported disadvantages of the method. Another possible disadvantage of CAS might be the covering of the external carotid artery (ECA)

orifice. This might be a further argument against carotid stenting.

Most high-grade arteriosclerotic lesions are located at the carotid bifurcation, usually at the distal common carotid artery (CCA) and the proximal internal carotid artery (ICA), and frequently the ECA is also involved. In many cases stents are placed from the ICA, extending into the CCA thereby covering the ECA origin.

The ipsilateral ECA can potentially provide an important collateral pathway for retinal and cerebral blood flow in the presence of occlusion or severe stenosis of the ICA, especially in patients with an incomplete circle of Willis. In contrast to the ICA, evaluations of development of ECA stenosis have been rarely described.^{2–4} The fate of the ipsilateral ECA has been investigated with² and without³

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additional external endarterectomy. A comparison of the development of obstructive disease between the ipsilateral ECA and the contralateral ECA after CEA has, to our knowledge, never been performed. In most studies on ECA patency, duplex-scan-based flow criteria were used to grade ECA stenosis.^{2–5}

As far as we know, only one study has been published so far with data concerning the effect of carotid stent placement on the ipsilateral ECA immediately after the procedure and during a limited 2 years of follow-up.⁵ Furthermore, this study did not differentiate between overstented and non-overstented bifurcations.

Therefore, in the present study the following four questions were addressed: 1) What is the prevalence of primary stenosis and occlusion of the ipsilateral and contralateral ECA before carotid stent placement? 2) Is there further development of obstructive disease in the ipsilateral ECA immediately after stenting and during follow-up compared with the contralateral side? 3) Is there a difference in the development of ECA stenosis between overstented and non-overstented bifurcations? 4) Is there a relationship between development of ECA stenosis and development of in-stent restenosis?

To answer these questions, we performed a follow up study with annual duplex US of both the ipsi- and contralateral carotid arteries, in patients treated with CAS.

Methods

Patients

Between December 1998 and 2002 all patients scheduled for CAS in our institution were prospectively entered in a computerized database. Patients had their CAS performed for either primary carotid bifurcation stenosis or restenosis after previous CEA. Patients with preceding contralateral CAS were excluded from this study, as were patients in which no stent was placed during the procedure. A total of 312 patients were included in this study. Median age was 70 years (range 47–89), 221 (70.6%) were male. In 173 patients (55.4%) the left carotid artery was treated. The study was approved by the local ethics committee, and written informed consent was obtained from all patients in accordance with institutional guidelines.

Seventy (22.4%) had been symptomatic of the ipsilateral carotid artery stenosis (Transient Ischemic Attack, Transient Monocular Blindness or minor stroke) in the 4 months preceding CAS. In 242 patients (77.6%) CAS was performed in the work-up before

coronary artery bypass grafting (CABG), or other cardiothoracic reconstructive surgery. These patients were treated to prevent perioperative complications and most had not been symptomatic of the ipsilateral carotid bifurcation stenosis.

The degree of stenosis was assessed by duplex ultrasound scanning and intra-arterial digital subtraction angiography prior to endovascular treatment. Symptomatic patients were treated if the degree of stenosis at the carotid bifurcation exceeded 70%, according to the North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria.⁶ For asymptomatic patients the cut-off point for treatment was a diameter reduction of 80%. Preoperative and postoperative carotid artery duplex examination that specifically evaluated the degree of ECA stenosis were available for review on all 312 CAS procedures performed during this period. Patients were monitored at the recovery room and, barring any complication, discharged the following day.

Carotid angioplasty and stenting procedure

In all patients CAS was performed in accordance with our previously described CAS protocol.^{7,8} All procedures were performed under local anesthesia, from a groin approach. All procedures were performed by either an experienced interventional cardiologist or an experienced interventional radiologist. The choice of stent type, and the decision whether or not to use a cerebral protection device (CPD) were at the discretion of the treating interventionalist. As most procedures were performed before CPD's had become available, no protection device was used in 267 cases (85.3%). Several different types of appropriately sized self expandable stents were used (Table 1). Overstenting of the carotid bifurcation was defined as covering of the ECA origin by stent placement from the ICA extending into the CCA. Aspirin (80–100 mg/day) was given prior to CAS and continued indefinitely. Clopidogrel (75 mg/day) was started 72 h before the procedure and continued for at least 4 weeks. Patients re-entered the carotid surveillance

Table 1. Types and numbers of stents used

Stent type	Manufacturer	N (%)
Carotid Wallstent	Boston Scientific, Natick, MA	219 (70)
Easy Wallstent	Boston Scientific, Natick, MA	82 (26)
Peripheral Wallstent	Boston Scientific, Natick, MA	1 (0.3)
Acculink	Guidant, Indianapolis, IN	2 (0.6)
Carotid SE	Medtronic, Minneapolis, MN	7 (2.2)
Precise	Cordis J&J, Miami Lakes, FL	1 (0.3)
Total		312

programme, with duplex US at 3 and 12 months and yearly thereafter.

Duplex ultrasound scanning

All patients were evaluated initially preprocedurally and during follow-up with duplex ultrasonography of the ipsi- and contralateral CCA, ICA, and ECA. The duplex criteria used in our vascular laboratory (HP/Agilent, Sonos 2500 or 4500, Andover, USA) are based on the Strandness criteria (20–49%/50–70%/70–90%/90–99%/occlusion). In terms of classification of the degree of ICA and ECA stenosis with duplex ultrasound, we used the same velocity criteria in the post-stenting as for the pre-stenting situation.

Endpoints

Endpoints in the analyses were development of ECA occlusion or >50% ECA stenosis during follow-up assessed by Duplex US scanning.

Statistics

Statistical analysis was performed using the statistical software package SPSS (SPSS, Inc., Chicago, IL). Actuarial survival analysis was performed by using Kaplan-Meier life tables. A p -value of <0.05 (log-rank) was considered statistically significant for all analyses.

Results

Preprocedural evaluation of the ipsilateral ECA demonstrated $\geq 50\%$ stenosis in 32.7% of cases vs 30% contralateral. Three ipsilateral and 3 contralateral ECA occlusions (1%) were noted ($p = \text{NS}$). After stenting 2 new ECA occlusions (0.8%) were seen vs 0 contralateral. These 2 additional ipsilateral and asymptomatic occlusions occurred immediately after the procedure, both in patients in which the carotid bifurcation was overstented. Contralaterally, no new occlusion was noted immediately after the procedure. No additional ipsilateral ECA occlusions and two additional contralateral ECA occlusions (at 24 and 36 months respectively) were noted at extended follow-up.

A comparison of progression of disease of the ipsilateral ECA ($n = 312$) and contralateral ECA, as demonstrated by duplex US, is shown in Fig. 1. On the day preceding stenting the ipsilateral and contralateral ECA did not differ significantly. The prevalence of $\geq 50\%$ stenosis of the ipsilateral ECA progressed from 49.1% at 3 months, to 56.4%, 64.7%, 68.2%,

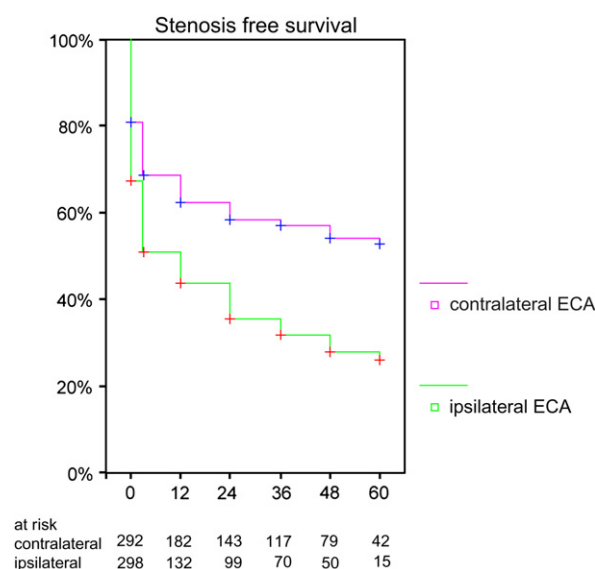


Fig. 1. Kaplan Meier estimates of ECA stenosis free survival: ipsilateral ECA vs contralateral ECA ($N = 312$) ($p < 0.001$). Time schedule: BASELINE – 3 m–12 m–24 m–36 m–48 m–60 m. *Ipsilateral*: 67.3% (baseline) – 50.9% – 43.6% – 35.3% – 31.8% – 27.7% – 26.0%. The Standard Error (SE) was 0.0296, 0.0297, 0.0296, 0.0294, 0.0301 and 0.0329 at 3 to 60 months respectively. Number of events (stenosis $\geq 50\%$) was 194 with a mean stenosis free follow-up of 23.3 months 95% CI (20.2–26.3) SE 1.56. *Contralateral*: 70% (baseline) – 68.8% – 62.4% – 58.5% – 57% – 54.2% – 52.9%. The SE was 0.0274, 0.0289, 0.0299, 0.0303, 0.0320, and 0.0336 at 3 to 60 months respectively. Number of events (stenosis $\geq 50\%$) was 124 with a mean stenosis free follow-up of 36.5 months 95% CI (33.3–39.7) SE 1.64.

72.3%, and 74% at 12, 24, 36, 48, and 60 months respectively. The contralateral prevalences were 31.2%, 37.6%, 41.5%, 43.1%, 45.8%, and 47.1% respectively. Compared with preprocedural data, the pronounced increase in stenosis rate of the ipsilateral ECA and the moderate increase in the contralateral ECA was statistically significant ($p < 0.001$).

In 234 patients (75%) the carotid bifurcation was overstented. Prevalence of $\geq 50\%$ ECA stenosis in non-overstented cases was 20.4% pre-CAS and 25%, 29.9%, 37.1%, 42.9%, 53.6%, and 53.6% at 3, 12, 24, 36, 48 and 60 months follow-up respectively. In patients with overstented bifurcations the prevalence was 35.4% pre-CAS, and 53.4%, 61.4%, 70.2%, 73.2%, 75.9%, and 77.9% at 3, 12, 24, 36, 48 and 60 months follow-up respectively (Fig. 2). This difference, with the overstented bifurcations showing more disease progression of the ipsilateral ECA was statistically significant ($p = 0.004$).

So far we looked at the complete patient group including those with a more than 50% ECA stenosis at baseline. In fact it would be more fair to look at the development of truly new stenoses. If patients with

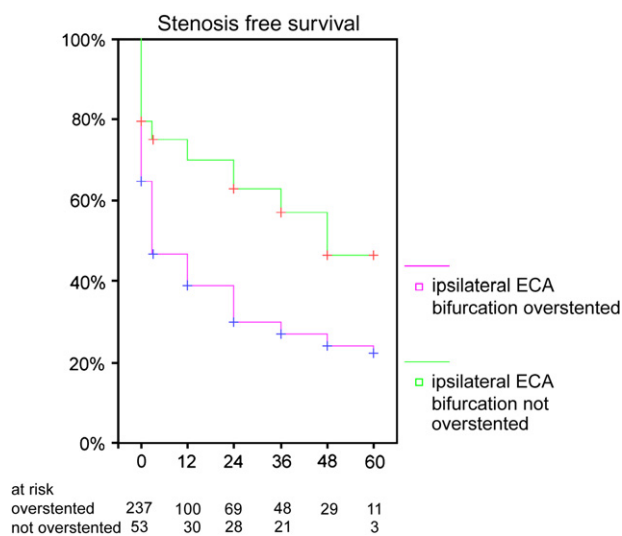


Fig. 2. Kaplan Meier estimates of ECA stenosis free survival: ipsilateral non-overstented ECA ($N = 54$) vs ipsilateral overstented ECA ($N = 238$) ($p = 0.0004$). Time schedule: BASELINE – 3 m–12 m–24 m–36 m–48 m–60 m.

Ipsilateral (All): 67.3% (baseline) – 50.9% – 43.6% – 35.3% – 31.8% – 27.7% – 26.0%. The Standard Error (SE) was 0.0296, 0.0297, 0.0296, 0.0294, 0.0301 and 0.0329 at 3 to 60 months respectively. Number of events (stenosis $\geq 50\%$) was 194 with a mean stenosis free follow-up of 23.3 months 95% CI (20.2–26.3) SE 1.56.

Non-overstented: 79.6% (baseline) – 75.0% – 70.1% – 62.9% – 57.1% – 46.4% – 46.4%. The SE was 0.0608, 0.0658, 0.0711, 0.0752, 0.0827 at 3 to 48 months respectively. Number of events (stenosis $\geq 50\%$) was 23 with a mean stenosis free follow-up of 37.5 months 95% CI (30.3–44.7) SE 3.67.

Overstented: 64.6% (baseline) – 46.6% – 38.6% – 29.8% – 26.8% – 24.1% – 22.1%. The SE was 0.0327, 0.0323, 0.0316, 0.0312, 0.0318, 0.0350 at 3 to 60 months respectively. Number of events (stenosis $\geq 50\%$) was 167 with a mean stenosis free follow-up of 20.6 months 95% CI (17.3–23.9) SE 1.68.

a preprocedural $\geq 50\%$ ECA stenosis were excluded from analysis, 176 patients remained (Fig. 3). In these 176 patients with no $\geq 50\%$ stenosis at baseline, the prevalence of $\geq 50\%$ stenosis of the ipsilateral ECA progressed from 0% pre-CAS to 24.6% at 3 months, 35.3%, 47.3%, 52.8%, 58.9%, and 61.5% at 12, 24, 36, 48, and 60 months respectively.

In 37 of 176 patients with no overstenting of the bifurcation prevalence of $\geq 50\%$ stenosis of the ipsilateral ECA progressed from 0% at baseline to 5.9%, 11.9%, 21.1%, 28.2%, 41.7%, 41.7% at 3, 12, 24, 36, 48, and 60 months respectively (Fig. 4). In patients with overstented bifurcations prevalence of $\geq 50\%$ stenosis of the ipsilateral ECA progressed from 0% at baseline to 29.9%, 41.1%, 53.8%, 58.5%, 62.7%, 65.8% at 3, 12, 24, 36, 48, and 60 months respectively ($N = 139$).

During follow-up in 48 patients (15.5%) an in-stent stenosis $\geq 50\%$ of the ipsilateral ICA occurred (Fig. 5). Comparison of ECA and ICA stenosis progression demonstrated a correlation between the two. There

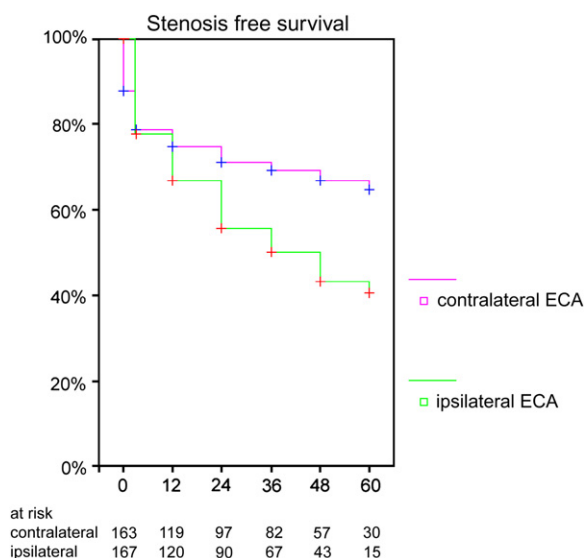


Fig. 3. Kaplan Meier estimates of ECA stenosis free survival in selected patients with no stenosis at baseline: ipsilateral ECA vs contralateral ECA ($N = 167$) ($p = 0.0043$). Time schedule: BASELINE – 3 m–12 m–24 m–36 m–48 m–60 m.

Ipsilateral: 0% (baseline) – 75.4% – 64.7% – 52.4% – 47.2% – 41.1% – 38.5%. The Standard Error (SE) was 0.0327, 0.0373, 0.0406, 0.0418, 0.0444 and 0.0492 at 3 to 60 months respectively. Number of events (stenosis $\geq 50\%$) was 82 with a mean stenosis free follow-up of 36 months 95% CI (32.1–39.8) SE 1.97.

Contralateral: 0% (baseline) – 90.1% – 82.6% – 76.8% – 74.0% – 69.6% – 67.3%. The SE was 0.0323, 0.0345, 0.0367, 0.0377, 0.0401 and 0.0442 at 3 to 60 months respectively. Number of events (stenosis $\geq 50\%$) was 50 with a mean stenosis free follow-up of 43.6 months 95% CI (39.7–47.5) SE 2.0.

were more ECA stenoses in patients who developed an in-stent ICA stenosis (38/48; 79%), compared to those who did not (94/264; 36%) ($p = 0.026$).

In separate analyses no statistically significant correlation was found between ECA stenosis development in symptomatic versus asymptomatic patients, or in primary versus post-CEA restenosis, nor was there a statistically significant correlation between ECA stenosis development and CPD use, or the type of stent used.

Discussion

This study reports the long term fate of the ECA after carotid stenting. Our results show that significant progression of disease in the ipsilateral ECA occurs after overstenting. Furthermore, there was greater progression of disease in the ipsilateral stented ECA compared with the untreated contralateral ECA. However, this did not have an adverse impact on either the patency of the ECA or the clinical outcome of the patient.

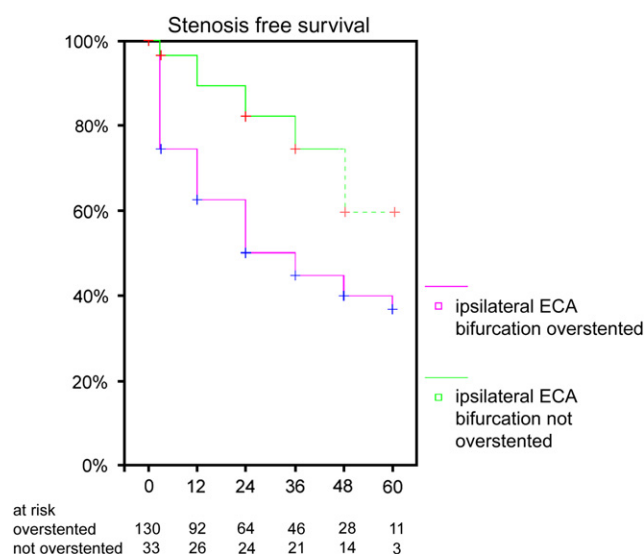


Fig. 4. Kaplan Meier estimates of ECA stenosis free survival in patients with no ECA stenosis at baseline: ipsilateral non-overstented ECA ($N=37$) vs ipsilateral overstented ECA ($N=139$) ($p=0.004$). Time schedule: BASELINE – 3 m-12 m-24 m-36 m-48 m-60 m.

Ipsilateral (All $N=167$): % (baseline) – 75.4% – 64.7% – 52.4% – 47.2% – 41.1% – 38.5%. The Standard Error (SE) was 0.0327, 0.0373, 0.0406, 0.0418, 0.0444 and 0.0492 at 3 to 60 months respectively. Number of events (stenosis $\geq 50\%$) was 82 with a mean stenosis free follow-up of 36 months 95% CI (32.1–39.8) SE 1.97.

Non-overstented: 0% (baseline) – 94.1% – 88.1% – 78.9% – 71.8% – 58.3% – 58.3%. The SE was 0.0339, 0.0579, 0.0720, 0.0838 and 0.102 at 3 to 48 months respectively. Number of events (stenosis $\geq 50\%$) was 10 with a mean stenosis free follow-up of 48 months 95% CI (41.8–54.8) SE 3.32.

Overstented: 0% (baseline) – 71.1% – 58.9% – 46.2% – 41.5% – 37.3% – 34.2%. The SE was 0.0382, 0.0428, 0.0462, 0.0471, 0.0492, and 0.0553 at 3 to 60 months respectively. Number of events (stenosis $\geq 50\%$) was 70 with a mean stenosis free follow-up of 33.4 months 95% CI (29.0–37.8) SE 2.24.

The role of the ECA as a collateral to the brain is worthy of discussion. The ipsilateral ECA is thought by many to be an important source of cerebral blood flow in the presence of occlusion or severe stenosis of the ICA, that might also serve as significant conduit for vascular reconstruction.⁹ As the severity of ICA disease increases, the contribution from the extracranial collateral circulation is expected to become greater up to 10 to 15% of middle cerebral artery blood flow.¹⁰ Others doubt if the contribution of the ECA collaterals to cerebral perfusion is substantial.^{11,12} Still, many surgeons routinely perform some kind of ECA endarterectomy during standard CEA,² to preserve ECA patency and hereby collateral supply in cerebral perfusion in the event of recurrent ICA stenosis. Management of ECA stenosis during routine CEA is however controversial, in part because of high residual stenosis rate as well as early and late recurrent stenosis rate.² Thus some surgeons have

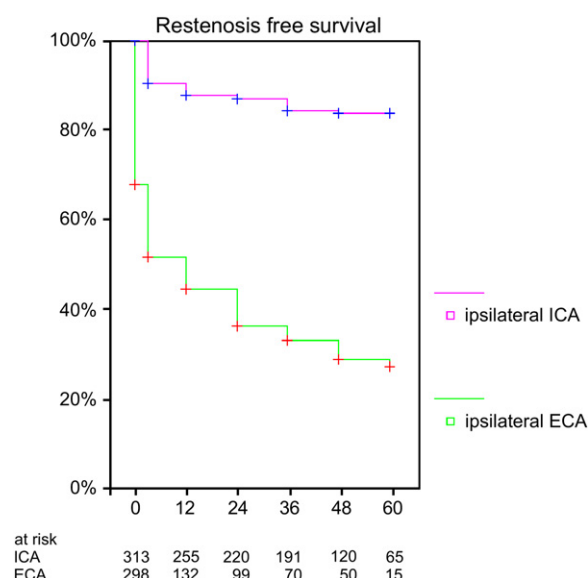


Fig. 5. During follow-up in 48 patients stenosis $\geq 50\%$ of the ipsilateral Internal Carotid Artery occurred. The incidence of in-stent recurrent stenosis ($\geq 50\%$) therefore was 15.5% in the present study after a mean follow-up of 44 months. The correlation between ICA and ECA reached significance when patients with ECA stenosis at baseline were included (Chi-Square test; $p=0.026$). If patients with a preprocedural $\geq 50\%$ ACE stenosis were abandoned from analysis the correlation was non significant ($p=0.09$).

recommended leaving the diseased ECA intact during CEA.³ According to the guidelines of an international consensus meeting¹³ CAS is also recommended without intervention at the ECA.

The prevalence of ECA stenosis depends on definition and measurement tool. ECA stenosis ($>50\%$) was found in 22% of patients indicated for CEA.³ Willfort found 17.5% of patients with $>70\%$ ECA stenosis in patients preceding CAS.⁵ The preprocedural ipsi- and contralateral prevalence of ECA stenosis in our study group was 32.7% vs 30% respectively using duplex with a cut-off point of $\geq 50\%$ stenosis (PSV > 125 cm/s).

Ascer *et al.*,³ being the first to compare pre- and post-operative duplex evaluation of the ECA, found no significant early or late influence of CEA on disease progression in the ipsilateral ECA. Postoperative occlusion of the ECA following CEA showed to be rare, and even in the presence of significant preoperative ECA stenosis, postoperative occlusion did not occur despite intentionally leaving plaque within the ECA. ECA stenoses showed relatively stable and only a minority progressed to severe stenosis. More importantly, those that did progress to severe stenosis did not appear to confer additional risk of neurologic complication in their series. In Willforts study, the clinical significance of disease progression in the ipsilateral ECA during the first year after CAS was

limited.⁵ Only one patient with presumed embolic ECA occlusion immediately after stent placement had transient jaw claudication. Similarly we found 2 patients with occlusion postprocedurally also without symptoms. No other ipsilateral occlusions occurred during follow-up.

Both in Willfort's and in our study a significantly higher progression of disease in the ipsilateral versus the contralateral ECA after CAS was found.⁵ We also showed that progression was more significant with overstenting of the carotid bifurcation. During stent placement atheromatous material might be pushed from the CCA/ICA into the origin of the ECA. Furthermore, it is assumed that flow turbulence caused by passage through the meshes of the stent wall to the ECA might be a plausible explanation for the increased narrowing of the ECA. Although prospective with a follow-up of 121 carotid arteries, the duration of Willfort's study was limited to 24 months. After 1 year, based on Wallstents only, some kind of steady state seemed to be achieved. Our results however, with longer follow-up, clearly show that development of ECA stenosis is an ongoing process and therefore probably not only caused by early flow turbulence, but by true disease progression.

Comparison of ECA and ICA stenosis progression post-endarterectomy demonstrated no correlation between the two.³ In Ascers study only 8% of cases showed $\geq 70\%$ stenosis of both the ICA and ECA. Moreover, progression of disease within the ECA after CEA did not lead to restenosis of the ICA, suggesting the independence of disease within these two vessels. Interestingly, for the post-stenting situation we found a correlation between ECA and ICA stenosis progression [Fig. 5]. In-stent restenosis, reported 3.5% using only Wallstents with overstenting of the bifurcation¹⁴ reduces the impact of the ECA as a source of collateral supply to the brain. In case of higher incidence of carotid stent recurrent stenosis, as published in CAVATAS or our own experience^{1,15,16} the dynamics of ECA disease and the importance of the ECA as a collateral seem even more limited. On the other hand, in-stent restenosis of nitinol stents that are being used increasingly and can be placed selectively in the ICA, does probably not affect the origin of the ECA, and will subsequently lead to increased flow through the ECA which emphasizes the importance of the ECA as a collateral.

Two characteristics that make the carotid bifurcation somewhat unique are the different blood flow requirements and waveforms of the ICA and the ECA.^{17,18} Probably both phenomena are induced by the different resistances found in the runoff beds for each artery, high in the case of the ECA and low in

the case of the ICA. In case of significant ICA stenosis and thus high resistance in the ICA, an increasing percentage of ECA flow is speculated to be diverted through collateral paths into the bed normally supplied by the ICA. When endarterectomy relieves bifurcation stenosis, CCA blood flow is redistributed preferentially to the ICA.¹⁹ The proportionate change in total flow has a positive increase in the ICA, whereas flow in the ECA is likely to decrease. Duplex ultrasonography is the primary non-invasive screening procedure for evaluation of ICA stenosis to select patients for angiography.²⁰ In contrast to the ICA, evaluations of degree of ECA stenosis have been rarely described.²⁻⁴ The duplex US findings concerning ECA stenosis have been handled in the same manner as ICA stenoses and the same Doppler criteria have been used to evaluate them.⁴ Ascer and Archie used PSV of the ECA to grade ECA stenosis. Paivansalo found the peak systolic flow ratio ECA/CCA to be superior for grading ECA stenosis, which was also used by Willfort. In terms of classification of the degree of ICA and ECA stenosis with ultrasound, we used the same velocity criteria in the post-stenting as for the pre- and post-endarterectomy situation. However, measurement of external carotid stenosis is more complicated and less accurate as a result of its smaller transverse diameter, as compared with ICA lesions.³ Furthermore, ipsilateral ICA stenosis affects the flow parameters of the ECA. Thus, ECA flow values must be considered carefully.²¹

Our study has several limitations. It was a non-randomized study using different types of stents. Our analysis did not show a relation between stenosis development and used type of stent. However, 96% of the stents used in this cohort were Wallstents, and analysis of a more balanced mix of stent types might discover such a relationship. Furthermore, in our analysis we used the PSV as the only measurement tool as discussed above. However, we believe that the clear trend shown by our results is independent of the measurement technique used.

Conclusion

Our results show that significant progression of stenosis in the ipsilateral ECA occurs after CAS. Progression is more pronounced with bifurcation overstenting. In the opposite ECA non or mild progression was found. In other words, there was greater progression of disease in the ipsilateral ECA compared with the contralateral ECA. Finally, progression of disease in the ECA did not have an adverse impact on the

patency of the ECA. Even in the presence of preprocedural ECA stenosis, post-CAS occlusion did not occur.

Appendix.

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References

- 1 CAVATAS Investigators. Endovascular versus surgical treatment in patients with carotid stenosis in the Carotid and Vertebral Artery Transluminal Angioplasty Study (CAVATAS): a randomised trial. *Lancet* 2001;**357**:1729–1737.
- 2 ARCHIE Jr JP. The outcome of external carotid endarterectomy during routine carotid endarterectomy. *J Vasc Surg* 1998;**28**:585–590.
- 3 ASCER E, GENNARO M, POLLINA RM, SALLES CUNHA S, LORENSON E, YORKOVICH WR *et al.* The natural history of the external carotid artery after carotid endarterectomy: implications for management. *J Vasc Surg* 1996;**23**:582–586.
- 4 PÄIVÄNSALO MJ, SINILUOTO TMJ, TIKKAKOSKI TA, MYLLYLÄ V, SURAMO IJ. Duplex US of the external carotid artery. *Acta Radiol* 1996;**37**:41–45.
- 5 WILLFORT-EHRINGER A, AHMADI R, GRUBER D, GSCHWANDTNER ME, HAUMER A, HEINZ G *et al.* Effect of carotid artery stenting on the external carotid artery. *J Vasc Surg* 2003;**38**:1039–1044.
- 6 BARNETT HJM, TAYLOR DW, ELIASZIW M, FOX AJ, FERGUSON GG, HAYNES RB *et al.* Benefit of carotid endarterectomy in patients with symptomatic moderate or severe stenosis. *N Engl J Med* 1998;**339**:1415–1425.
- 7 Antonius Carotid Endarterectomy AaSSG. Transcranial Doppler monitoring in angioplasty and stenting of the carotid bifurcation. *J Endovasc Ther* 2003;**10**:702–710.
- 8 ACKERSTAFF RGA, SUTTROP M-J, VAN DEN BERG JC, OVERTOOM TTHC, VOS JA, BAL ET *et al.* Prediction of early cerebral outcome by TCD monitoring in carotid bifurcation angioplasty and stenting. *J Vasc Surg* 2005;**41**:618–624.
- 9 DIETRICH EB, LIDDICOAT JE, MCCUTCHEN JJ, DE BAKEY ME. Surgical significance of the external carotid artery in the treatment of cerebrovascular insufficiency. *J Cardiovasc Surg* 1968;**15**:213–223.
- 10 FEARN SJ, PICTON AJ, MORTIMER AJ, PARRY AD, MCCOLLUM CN. The contribution of the external carotid artery to cerebral perfusion in carotid disease. *J Vasc Surg* 2000;**31**:989–993.
- 11 SILLESEN H. The haemodynamic value of external carotid artery collateral blood supply in carotid artery disease. *Eur J Vasc Surg* 1988;**2**:309–313.
- 12 MCINTYRE KE, ELY III RL, MALONE JM, BERNHARD VM, GOLDSTONE J. External carotid artery reconstruction: its role in the treatment of cerebral ischaemia. *Am J Surg* 1985;**150**:58–64.
- 13 VEITH FJ, AMOR M, OHKI T, BEEBE HG, BELL PRF, BOLIA A *et al.* Current status of carotid bifurcation angioplasty and stenting based on a consensus of opinion leaders. *J Vasc Surg* 2001;**33**(Suppl): S111–S116.
- 14 WILLFORT-EHRINGER A, AHMADI R, GSCHWANDTNER ME, HAUMER M, LANG W, MINAR E. Single-center experience with carotid stent restenosis. *J Endovasc Ther* 2002;**9**:299–307.
- 15 CHRISTIAANS MH, ERNST JMPG, SUTTROP MJ, VAN DEN BERG JC, OVERTOOM TTHC, KELDER JC *et al.* Restenosis after carotid angioplasty and stenting: a follow-up study with duplex ultrasonography. *Eur J Vasc Endovasc Surg* 2003;**26**:141–144.
- 16 CHAKHTOURA EY, HOBSON RW, GOLDSTEIN J, SIMONIAN GT, LAL BK, HASER PB *et al.* In-stent restenosis after carotid angioplasty-stenting: incidence and management. *J Vasc Surg* 2001;**33**:220–225.
- 17 HYUN S, KLEINSTREUER C, ARCHIE JP. Computational analysis of effects of external carotid artery flow and occlusion on adverse carotid bifurcation hemodynamics. *J Vasc Surg* 2003;**37**:1248–1254.
- 18 PHILLIPS DJ, GREENE FM, LANGLOIS Y *et al.* Flow velocity patterns in the carotid bifurcations of young, presumed normal subjects. *Ultrasound Med Biol* 1983;**9**:39–49.
- 19 GORDON IL, STEMMER EA, WILSON SE. Redistribution of blood flow after carotid endarterectomy. *J Vasc Surg* 1995;**22**:349–360.
- 20 JAHROMI AS, CINA CS, LIU Y, CLASE CM. Sensitivity and specificity of color duplex ultrasound measurement in the estimation of internal carotid artery stenosis: a systematic review and meta-analysis. *J Vasc Surg* 2005;**41**:962–972.
- 21 ZWIEBEL WJ, AUSTIN CW, SACKETT JF, STROTHER CM. Correlation of high-resolution, B-mode and continuous wave Doppler sonography with arteriography in the diagnosis of carotid stenosis. *Radiology* 1983;**149**:523–532.

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