**Data supplement**

**Title: Influence of stent design and use of protection devices on outcome of carotid artery stenting – a pooled analysis**

Content:

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**Statistical analysis plan**

**Supplementary table 1: Patient baseline characteristics for implanted stent type.**

|  |  |  |  |
| --- | --- | --- | --- |
|  | Total  (n=1557) | Open Cell Stent  (n=595) | Closed Cell Stent  (n=962) |
| Age (years; mean, SD) | 69.1 (±8.9) | 69.5 (±9.1) | 68.9 (±8.8) |
| Male sex (n, %) | 1104 (71) | 409 (69) | 695 (73) |
| History of hypertension (n, %) | 1106 (72) | 421 (72) | 685 (72) |
| Systolic blood pressure at randomisation  (mm Hg; mean, SD) | 145 (±21.1) | 146.9 (±23) | 143.9 (±19.8) |
| History of diabetes (n, %) | 370 (24) | 134 (23) | 236 (25) |
| History of hypercholesterolaemia (n, %)\* | 592 (61) | 275 (60) | 317 (63) |
| History of smoking (n, %) | 988 (64) | 372 (63) | 616 (65) |
| History or coronary heart disease (n, %) | 358 (23) | 146 (25) | 212 (22) |
| History of peripheral artery disease (n, %)\* | 158 (16) | 74 (16) | 84 (17) |
| Stenosis on the left side (n, %) | 815 (53) | 325 (55) | 490 (51) |
| Ipsilateral degree of stenosis |  |  |  |
| Moderate (50-69%) (n, %) | 305 (20) | 96 (16) | 209 (22) |
| Severe (70-99%) (n, %) | 1243 (80) | 497 (84) | 746 (78) |
| Contralateral severe carotid stenosis or occlusion (n, %) | 213 (15) | 94 (17) | 119 (14) |
| Qualifying event |  |  |  |
| Amaurosis fugax or retinal stroke (n, %) | 280 (18) | 120 (20) | 160 (17) |
| Transient ischemic attack (n, %) | 535 (35) | 203 (34) | 332 (35) |
| Hemispheric stroke (n, %) | 725 (47) | 266 (45) | 459 (48) |
| History of stroke prior to qualifying event (n, %)\* | 162 (17) | 92 (20) | 70 (14) |

Legend: Patients with deployed stents and available data on stent type are included. Data are n/N (%), unless otherwise indicated. \*Data were not gathered in the Stent-Protected Angioplasty versus Carotid Endarterectomy (SPACE) trial.

**Supplementary table 2: Procedure-related characteristics in contributing trials.**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
|  | Total  (n=1557) | EVA-3S  (n=246) | SPACE  (n=573) | ICSS  (n=738) |
| Stent design (n, %) |  |  |  |  |
| Open-cell | 595 (38.2) | 101 (41.1) | 127 (22.2) | 367 (49.7) |
| Precise RX (Cordis) | 263 (16.9) | 26 (10.6) | 35 (6.1) | 202 (27.4) |
| Acculink (Guidant/Abbott) | 232 (14.9) | 70 (28.5) | 92 (16.1) | 70 (9.5) |
| Protégé (EV3) | 82 (5.3) | 0 (0.0) | 0 (0.0) | 82 (11.1) |
| Next Stent (Boston Scientific) | 3 (0.2) | 0 (0.0) | 0 (0.0) | 3 (0.4) |
| Exponent (Medtronic) | 3 (0.2) | 0 (0.0) | 0 (0.0) | 3 (0.4) |
| Zilver (Cook Medical) | 5 (0.3) | 5 (2.0) | 0 (0.0) | 0 (0.0) |
| S.M.A.R.T. (Cordis) | 7 (0.4) | 0 (0.0) | 0 (0.0) | 7 (0.9) |
| Closed-cell | 962 (61.8) | 145 (58.9) | 446 (77.8) | 371 (50.3) |
| Carotid Wallstent (Boston Scientific) | 899 (57.7) | 145 (58.9) | 436 (76.1) | 318 (43.1) |
| Xact (Abbott) | 58 (3.7) | 0 (0.0) | 10 (1.7) | 48 (6.5) |
| Cristallo ideale (Invatec/Medtronic) | 5 (0.3) | 0 (0.0) | 0 (0.0) | 5 (0.7) |
| Protection device used (n, %) | 950 (61.0) | 227 (92.3) | 153 (26.7) | 570 (77.2) |
| Distal filters\* | 827 (87.1) | 160 (70.5) | 133 (86.9) | 534 (93.7) |
| Accunet (Guidant) | 15 (1.6) | 5 (2.2) | 4 (2.6) | 6 (1.1) |
| Angioguard (Cordis) | 124 (13.1) | 21 (9.3) | 16 (10.5) | 87 (15.3) |
| Emboshield (Abbott) | 131 (13.8) | 24 (10.6) | 18 (11.8) | 89 (15.6) |
| Filterwire (Boston Scientific) | 374 (39.4) | 61 (26.9) | 66 (43.1) | 247 (43.3) |
| Interceptor (Medtronic) | 1 (0.1) | 0 (0.0) | 0 (0.0) | 1 (0.2) |
| Mednova Neuroshield (Abbott) | 29 (3.1) | 0 (0.0) | 5 (3.3) | 24 (4.2) |
| Spider (EV3) | 153 (16.1) | 49 (21.6) | 24 (15.7) | 80 (14.0) |
| Distal occlusion\* | 86 (9.1) | 67 (29.5) | 16 (10.5) | 3 (0.5) |
| Percusurge (Medtronic) | 86 (9.1) | 67 (29.5) | 16 (10.5) | 3 (0.5) |
| Flow reversal\* | 34 (3.6) | 0 (0.0) | 4 (2.6) | 30 (5.3) |
| Neuroprotection System (GORE) | 22 (2.3) | 0 (0.0) | 0 (0.0) | 22 (3.9) |
| Mo.Ma (Invatec) | 5 (0.5) | 0 (0.0) | 4 (2.6) | 1 (0.2) |
| Parodi AES (Arteria) | 7 (0.7) | 0 (0.0) | 0 (0.0) | 7 (1.2) |
| Type of device unknown\* | 0 | 0 | 0 | 3 (0.5) |
| Pre-dilatation performed (n, %)† | 760 (48.8) | 41 (16.7) | 194 (33.9) | 525 (71.1) |
| Post-dilatation performed (n, %)† | 1177 (89.8) | 0 (0.0) | 552 (96.3) | 625 (84.7) |
| Double APT used (n, %)† | 1357 (88.8) | 191 (77.6) | 546 (95.3) | 620 (87.0) |

Legend: Patients with deployed stents and available data on stent type and protection device use are included. Data are n/N (%), unless otherwise indicated.\*Percentages of all protection devices. †Percentages exclude missing data; missing data were: pre-dilatation (n=1 patient), post-dilatation (n=246), antiplatelet therapy (n=29).

**Supplementary table 3: Risk of procedural stroke or death in the stenting versus endarterectomy groups at study centers according to the frequency of closed-cell stent use**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Frequency of closed-cell stent use at center | CAS | CEA | RR for CAS versus CEA (95% CI) | Interaction p-value for trend |
|  | Events / patients | Events / patients |  |  |
| >80% closed-cell stents | 42/677 (6.2%) | 33/700 (4.7%) | 1.31 (0.84, 2.03) |  |
| 20-80% closed-cell stents | 54/615 (8.8%) | 29/640 (4.5%) | 1.93 (1.25, 3.00) | P=0.062 |
| <20% closed-cell stents | 19/231 (8.2%) | 6/238 (2.5%) | 3.24 (1.32, 7.96) |  |

Legend: Crude risks (number of events divided by number of patients) and binomial regression estimates of risk ratios (RR) and 95% confidence intervals (CI) of any stroke or death within 30 days of treatment are provided for patients treated with stenting compared with patients treated with endarterectomy. Centers were excluded if they had <4 CAS procedures with known stent design. CAS, carotid artery stenting; CEA, carotid endarterectomy.

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**Supplementary figure 1:** Effect of stent treatment with versus without use of protection devices in contributing trials.

Percentages are number of events divided by number of patients. Squares and horizontal bars represent within-trial treatment risk ratios and 95% CIs, respectively, with unprotected stenting as the reference group, on a log scale. The size of squares represents study weight. The diamond represents the pooled risk ratio and 95% CI, adjusted for source trial. In the investigation of heterogeneity, the interaction p value represents the significance of the interaction between source trial and treatment effect in the regression model (likelihood ratio test); a significant p value suggests heterogeneity. EVA-3S=Endarterectomy versus Angioplasty in Patients with Symptomatic Severe Carotid Stenosis. SPACE=Stent-Protected Angioplasty versus Carotid Endarterectomy. ICSS=International Carotid Stenting Study.

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**Supplementary figure 2:** Effect of stent treatment with versus without use of protection devices on risk of procedural stroke or death according to stent design and patient age.

Percentages are number of events divided by number of patients. Squares and horizontal bars represent treatment risk ratios and 95% CIs within subgroups, respectively, with unprotected stenting as the reference group, on a log scale, adjusted for source trial. The diamond represents the overall risk ratio and 95% CI for protected versus unprotected stenting, adjusted for source trial.

**Statistical analysis plan**

Background

Rationale

Since NASCET1 had proven carotid revascularisation by means of endarterectomy (CEA) to significantly reduce the risk of stroke in symptomatic carotid disease, CEA has become the gold standard of treatment. Besides a risk of peri-procedural stroke however, CEA carries some risk of general complications related to surgery and anaesthesia, as well as morbidity related to incision in the neck such as cranial nerve palsy and haematoma. In recent years, endovascular treatment with placement of a stent has been advocated as a less invasive alternative to endarterectomy. Carotid artery stenting (CAS) has potential advantages over endarterectomy: general and local surgical complications could be avoided and hospital stay may be shortened. However, stenting may also cause stroke during the intervention, either due to thrombo-embolism or release of atheromatous emboli. The development of thrombotic material on the disrupted vessel wall or at the foreign body, the stent, had drastically been reduced by combined platelet aggregation inhibition (aspirin and clopidogrel).2,3In contrast, there is ongoing uncertainty whether the use of cerebral protection devices reduces the risk of embolism of atheromatous or thrombotic material during CAS. The advocates of protection declare, that filters like bow nets in the distal ICA would be necessary, in order to protect the brain by means of removing emboli from the circulation. The advocates of unprotected stenting plead against the additional manipulation of endovascular material as it might increase the risk of dislodging emboli or injuring the vessel wall with subsequent thrombus formation or dissection. Furthermore the observation was made, that seemingly different stents - possibly depending on the stent design - are associated with a different frequency of complications. This supported the hypothesis that the tight, “closed” stent design caused less often adverse outcome events than wide mash “open” stent design.4

In three large randomised clinical trials of symptomatic carotid disease which contribute to the Carotid Stenting Trialists Collaboration (CSTC), stenting has been performed with and without protection. In the French Endarterectomy versus Angioplasty in Patients with Symptomatic Severe Carotid Stenosis trial (EVA-3S)5, the use of protection devices became mandatory after unfavourable initial results in 33 patients treated without protection; 227 patients were treated with protection devices. In the German, Swiss, and Austrian Stent-Protected Angioplasty versus Carotid Endarterectomy in symptomatic patients trial (SPACE)6, 154 patients were treated with, and 424 were treated without protection devices, among those receiving stenting as randomly allocated (per-protocol). The International Carotid Stenting Study (ICSS)7, reported on 593 protected and 208 unprotected per-protocol CAS procedures. The unprotected cohort in EVA 3S5 was too small for a comparative analysis of the data. However the analysis for ICSS data demonstrated a short-term rate for ipsilateral stroke and death with protection versus non-protection of 6.1% versus 4.6%, and for SPACE the results were 8.3% versus 6.2%.

In the North American Carotid Revascularization Endarterectomy versus Stenting Trial (CREST)8, which recruited both patients with symptomatic and patients with asymptomatic carotid stenosis, use of protection devices was mandatory from the beginning. The rate any stroke or death up to 30 days after treatment in the stenting arm was 4.4%. Among patients with symptomatic carotid disease however the peri-procedural stroke-or-death rate in CREST was 6.0%, which was similar to the combined results of the European trials. The currently available evidence therefore does not warrant the necessity of protection devices for CAS procedures.

The observation that the design of the stent might influence the OE rate has been observed by Bosiers and Cremonesi in 2006 already, who found in symptomatic carotid disease a rate of 2.2% for closed and 7.0% for open cell design.4 Jansen et al. analysed the data for SPACE patients and found an OE rate for closed cell stent: (5.6%, 95% CI: 3.7 to 8.2%]) and open cell stent (11.0%, 95% CI: 6.2 to 17.8%).9 The hypothesis generated on the basis of these findings was that the tight close cell design might cover the plaque better than the wide mesh of open cell stents and hence restrain more debris from escaping into the blood stream.

It may not only be the actual placement of the stent onto the stenosis, which causes mechanical impact on intact intima and the atheromatous plaque. Single or multiple balloon dilatations of the stenosis before or after stent insertion (so-called pre- and postdilatation) have been suspected to be a potential cause of embolic events, which might be even more important in combination with open or closed cell design.10

The influence of clinical and demographic patient characteristics on the risk of peri-procedural stroke in stenting has been an important focus of research: most consistently, increasing age has been associated with increasing stroke risk.11 The initial pooled analysis of the European trials in the CSTC showed that the risks of stroke or death in the short-term were similar between CAS and CEA in patients younger than 70 years, whereas the comparison strongly favoured CEA in the older patient group. As this was a completely unexpected finding at the time these trials were designed, age-related anatomical and physiological data have not been prospectively defined and systematically collected in the trials. Therefore, the exact mechanism mediating the interaction between age and the relative risk of complications with stenting versus endarterectomy remains to be determined; it is conceivable that an increase in the general burden of atherosclerosis (including the aortic arch) or changes in the vascular anatomy of supra-aortic arteries or in the composition of atherosclerotic plaques render elderly patients more susceptible to thromboembolic complications during CAS. Other clinical variables, such as type of the most recent ischaemic event before randomisation and delay between the event and treatment might be associated with plaque instability. Any of these factors might therefore influence the impact of stent cell design, use or protection devices or pre- or postdilatation on procedural risks, and should therefore be analysed in conjunction with the technical variables.

Due to limitations in sample size, such analyses could not be performed with sufficient statistical power at the level of individual trials.

Objective

To overcome the limitations of single trials, the investigators of the European-led stenting trials (EVA-3S, SPACE, and ICSS)5–7have set up the Carotid Stenting Trialists’ Collaboration (CSTC) with the purpose of conducting a combined analysis of outcome data from individual patients randomised for CAS in these trials. The objectives of the present analysis are to investigate the effect of the use of protection devices and the cell design of stents, as well as other procedure-related, clinical or demographic variables on the peri-procedural risk of stroke or death associated with stent treatment for symptomatic carotid stenosis. The following protocol for this analysis has been agreed by the CSTC Steering Committee before the data from the contributing trials were pooled and analysed.

Methods

Contributing trials

The individual patient data meta-analysis of EVA-3S (NCT 00190398), SPACE (ISRCTN 57874028), and ICSS (ISRCTN 25337470) was prospectively agreed at the design stage of the trials. Patient eligibility criteria, interventions, collected baseline characteristics, and definitions of outcome events were therefore broadly similar between the contributing trials. All three were open clinical trials with blinded outcome adjudication, randomising patients with moderate or severe carotid stenosis (≥50% reduction of the lumen diameter, measured according to the method used in NASCET) and associated recent, non-disabling ocular or cerebral ischaemic events, who were equally suited for either procedure, to undergo treatment by stenting or endarterectomy. The use of approved cerebral protection devices was optional in SPACE and ICSS. In contrast protection devices were made mandatory in EVA-3S after an interim analysis revealed a higher risk of procedural stroke with unprotected stenting compared with protected stenting (see above).

Definition of outcome events

The primary outcome event of the present analysis is any stroke or death occurring between the day of procedure and 30 days thereafter. The exact time of the onset of outcome events in relation to the procedure was not systematically collected in the contributing trials; for the present analysis, we therefore assumed that outcome events occurring on the day the procedure took place occurred as an immediate complication thereof. In order to investigate whether associations between technical variables and the 30-day risk of stroke or death were driven by immediate complications of the procedure, we performed a sensitivity analysis including only stroke and death outcomes occurring on the day of procedure. Outcome events are defined in the protocol for the CSTC pooled analysis of short-term outcome events, which was published in the supplementary web-appendix to the original CSTC paper.12

Per protocol definition

The analysis will be done per-protocol, including only patients who were randomised to stenting and in whom a stent was deployed across the stenosis. Patients who did not receive stent treatment as randomly allocated will be excluded (i.e., those randomised to stenting in whom the procedure was abandoned before a stent was inserted; those randomised to stenting and crossing over to surgery, those randomised to surgery and crossing over to stenting; those remaining on medical treatment without any attempt at revascularisation; and those who died before treatment).

Selection of variables

The association between peri-procedural outcome events and the following primary procedure-related variables of interest will be assessed: use of protection device and type of stent cell design (open-cell versus closed-cell). We defined closed-cell designs for stents that show a small open area (< 5•0 mm2) between the cell struts and therefore a higher coverage rate and where all stent-struts are interconnected; open cell designs for stents showing a greater open area (> 5•0 mm2) and without interconnection between all stent struts (Table II).4 We included all types of protection devices and defined five different categories (distal filter, distal balloon, proximal balloon, flow reversal, others) (Table III). Other procedure-related variables include predilatation, postdilatation and type of peri-procedural antithrombotic treatment (Table I). In addition, the association between peri-procedural outcome events and the following clinical and demographic variables will be assessed: exact age at the time of treatment (calculated as the difference between date of birth and date of treatment), sex, history of diabetes, hypertension, hypercholesterolaemia, smoking (current or past), coronary heart disease, and peripheral artery disease; type of the most recent ischaemic event (retinal ischaemia including transient monocular blindness or retinal infarct, hemispheric transient ischaemic attack, or hemispheric ischaemic stroke) in the region supplied by the ipsilateral carotid artery before randomisation; history of stroke before the most recent ipsilateral ischaemic event; systolic blood pressure at randomisation; level of functional disability at randomisation measured by the modified Rankin Score; degree of ipsilateral carotid stenosis determined at randomisation according to NASCET1 criteria or non-invasive equivalent (moderate [50-69% luminal diameter reduction], or severe [70-99%]) and side of the stenosis (left/right); and presence of contralateral severe carotid stenosis or occlusion. All collected patient and procedure related characteristics will serve as variables in analysis. Table I lists all pre-defined variables

Statistical analysis

As the protocols of the three contributing trials are broadly similar, statistical heterogeneity between the treatment effects in individual trials is expected to be small. Individual patient data will therefore be pooled and primarily analysed with fixed-effect binomial regression models including source trial terms as covariables, in order to obtain an overall unadjusted estimate of risk ratios (RR) and 95% confidence intervals of major outcome events as primary aggregate measures of treatment effect. Nevertheless, potential heterogeneity of the individual trial treatment effects will be examined by testing for interactions between source trial and treatment effect in the binomial regression model. If substantial heterogeneity is found, secondary random effects analyses will also be performed.

Associations between the pre-defined variables and peri-procedural outcome events will first be assessed on a univariable level (unadjusted risk ratios). Secondly, RR will be adjusted for (A) only those factors that change the crude RR by more than 5% on a relative scale; (B) simultaneously for the three factors that have the most influence on the RR, as well as (C) for all factors that change the RR. Differences between the effects of protection devices on the primary outcome measure in (1) older versus younger patients and (2) in open-cell versus closed-cell stenting will be investigated by formal testing of statistical interaction. The former interaction will be adjusted for stent design; the latter will be adjusted for age.

Discussion

The prospective meta-analysis of individual patient data from three large randomised trials in the Carotid Stenting Trialists’ Collaboration represents a powerful tool to analyse the impact of procedural variables on the safety of stent treatment for symptomatic carotid stenosis. The pooled analysis has the potential to identify particular patient groups in whom stenting in general or specific techniques of stenting are associated with increased procedural risks. Furthermore, hypotheses for future technical approaches of carotid endovascular interventions may be generated.

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**Table I: Variables**

|  |  |
| --- | --- |
| Technical variables | Predilatation |
| Postdilatation |
| Stent design (open cell vs. closed cell design) |
| Stent model |
| Single versus double peri-procedural antiplatelet therapy |
| Use of protection device |
| Duration of CAS |
| Clinical and demographic variables | Age at treatment |
| Coronary heart disease |
| Degree of treated stenosis |
| History of diabetes |
| Hypercholesterolaemia |
| Hypertension |
| Presence of contralateral stenosis or occlusion |
| Sex |
| Side of stenosis (left/right) |
| Smoking (current or past) |
| Type of the most recent ipsilateral ischaemic event before randomisation |

**Table II:** Types and categorisation of stents used in EVA-3S, SPACE and ICSS

|  |  |
| --- | --- |
| Closed-cell design | Carotid Wallstent (Boston Scientific) |
| Xact (Abbott) |
| Cristallo ideale (Invatec) |
| Open-cell design | Precise RX (Cordis) |
| Acculink (Guidant/Abbott) |
| Protégé (EV3) |
| Next Stent (Boston Scientific) |
| Exponent (Medtronic) |

**Table III:** Types of Protection-Devices used in EVA-3s, SPACE and ICSS

|  |  |
| --- | --- |
| Distal Filter | Spider (EV3) |
| FilterWire EZ (Boston Scientific) |
| Accunet (Abbott/Guidant) |
| EmboShield (Abbott) |
| Angioguard (Cordis) |
| Neuroshield (MedNova) |
| Distal balloon | GuardWire Plus (Medtronic) |
| Proximal balloon | MO.MA (Invatec) |
| Flow reversal | PercuSurge (Medtronic) |
| Gore Neuroprotection System (Gore) |
| Other | Parodi anti embolism system (Arteria Medical Science) |