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TOPIC ARTICLE

Complication rate in unprotected carotid artery stenting with closed-cell stents

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Abstract
Introduction The discussion on the use of protection devices (PDs) in carotid artery stenting (CAS) is gaining an increasing role in lowering the periprocedural complication rates. While many reviews and reports with retrospective data analysis do promote the use of PDs the most recent multi-centre trials are showing advantages for unprotected CAS combined with closed-cell stent designs.
Methods We retrospectively analysed 358 unprotected CAS procedures performed from January 2003 to June 2009 in our clinic. Male/female ratio was 2.68/1. The average age was 69.3 years. Seventy-three percent (261/358) showed initial neurological symptoms. All patients were treated on a standardised interventional protocol. A closed and small-sized cell designed stent was implanted in most cases (85.2%). One hundred seventy-one (47.8%) were controlled by Doppler ultrasonography usually at first in a 3-month and later in 6-month intervals.
Results The peri-interventional and 30-day mortality/stroke rate was 4.19% (15/358). These events included three deaths, five hyperperfusion syndromes (comprising one death by a secondary fatal intracranial haemorrhage), one subarachnoid haemorrhage and seven ischaemic strokes. Only 20% (3/15) of all complications occurred directly peri-interventional. The overall peri-interventional complication rate was 0.8% (3/358). Most complications occurred in initial symptomatic patients (5.36%). The in-stent restenosis rate for more than 70% was 7% (12/171) detected at an average of 9.8 month.
Conclusion Our clinical outcome demonstrates that unprotected CAS with small cell designed stents results in a very low procedural complication rate, which makes the use of a protection device dispensable.

Keywords Carotid artery · Stent · CAS · Protection devices · CEA

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Introduction

The beginning of internal and common carotid artery (ICA/CCA) treatment due to atherosclerotic stenosis by percutaneous transluminal angioplasty dates back to the late 1970s and the early 1980s [1–5]. In 1995 Diethrich et al. [6] published a long-term follow-up of a patient treated with a balloon expandable Palmaz–Schatz stent for postsurgical recurrent stenosis. Since then the numbers of publications and CAS treatments are increasing annually. However, with the beginning of interventional treatment of carotid artery stenosis the discussion on indication and complication rates compared to the long established carotid endarterectomy (CEA) was led with impetus. This discussion led to several multi-centre studies comparing the procedures in a controlled randomised prospective way [7–11].

In the late 1990s the development of so-called protection devices was promoted to reduce the peri-interventional risks of embolism and to gain an additional advantage over CEA. In recent years several interventional groups attempted to show the benefit of protection devices to reduce the

most obvious major complication: the periprocedural embolism rate [12–15]. Many of these reports resulted in a strong recommendation to use PDs. The positive effect of these PDs seemed so evident that even the safety committee of the French multi-centre study EVA-3s made PDs mandatory during the study and changed the study protocol. For many interventionalists the discussion on benefit and risks of PDs was already concluded. But besides several uncontrolled single-centre publications, mostly comparing historical cohorts of unprotected CAS with their recently performed CAS using PDs, no hard evidence on the superiority of CAS using PDs existed.

Recently performed subanalyses of pooled data of the multi-centre trials (EVA-3S, SPACE I) could not show a benefit of so-called protected CAS compared to unprotected CAS referring to peri-interventional complications [16]. Preliminary results of the MR-substudy of the ICSS trial, recently presented at the ESC 2009 in Stockholm, showed again a benefit for unprotected stenting. Last-mentioned studies maintain our practical experience at the application of unprotected CAS.

Beyond this ongoing discussion we present the outcome-data of our patients treated with a standardised protocol in a neuro-interventional centre with unprotected CAS and stents with closed and small cell design.

Material and methods

We retrospectively analysed data from patients treated with carotid stenting in our institute. All patients were independently neurological controlled before and after the procedure. Patients were treated with a standardised stenting protocol over the years.

Data collection

The data were collected retrospectively from neuroradiological interventional reports, neurologist's letters, angiographic, CAT and MR images as well as scanned patient charts. All information was cross-checked.

Patient collective

From January 2003 to June 2009 data of 337 patients, treated with unprotected CAS, were evaluated retrospectively. Of these, 21 patients had two independent stent implantations, resulting in 358 interventions conducted in our clinic. As in some cases two stents were placed in one session, 371 stents were implanted in total. These were either the same stent model or two different models. Left carotids were treated more often than right carotids (191 left/167 right).

Males had a 2.68 higher proportion in the collective than females. The average age of the patient pool was 69.3 years, with the oldest patient being 99 years and the youngest 43 years old (Table 1).

Fifty-four percent of the patients had an initial stenosis degree of 70–90%, higher degrees (>90%) and lower degrees (50–70%) occurred in 24% and 22%, respectively. Of this collective 73% (261/358) showed neurological symptoms within the last 180 days, which could be related to the carotid stenosis and were defined therefore as symptomatic carotid stenosis.

Out of the 358 procedures 32 (8.9%) had a recurring stenosis after CEA. Thirteen of these 32 (40.6%) showed neurological symptoms for their restenoses. Furthermore, vascular examination of the contralateral ICA showed occlusion in 58 (16.2%) cases, 50–70% stenosis in 18 (5%), 70–90% stenosis in eight (2.2%) and >90% stenosis in three (0.8%) cases. The initial and residual stenoses were measured in the angiograms with NASCET criteria.

Medication

The pharmacological and interventional regime was similar for all patients. Two to 3 days prior to intervention all patients were treated with at least 100 mg aspirin (ASS) and 75 mg clopidogrel. Preceding to the intervention, 1 mg atropine s.c. was administered. During the intervention an i. v. heparin bolus (3,000–5,000 IU) was given to increase the ACT to 250–350 s. Before inflating the post-dilatation balloon 1 mg atropine i.v. was injected. Post-stenting oral administration of 75 mg clopidogrel per day was continued for at least 6 weeks, that of 100 mg ASS per day for at least 6 months. In case of further diseases necessitating other coagulation compromising medication, an individual concept for anti-platelet and anti-coagulation therapy was developed.

Intervention

Of the 358 interventions, the majority (341) were performed via the femoral access route. Seventeen were performed via a transbrachial approach. The puncture of the femoral or brachial artery was carried out under local anaesthesia (5 ml Scandicain 3%). After placement of a short 5F sheath a 0.035-in. guidewire (Terumo Radifocus guidewire M angled, Terumo Corp., Tokyo, Japan) was introduced and a 90-cm-long 6F sheath (Super Arrow Flex Sheath, Arrow International Inc., Bernville, PA, USA) was exchanged.

Via a 5F multi-purpose catheter (Supertorque MP A2, Cordis Europe, the Netherlands), or a 6F sidewinder catheter (Sidewinder II Supertorque special, Cordis Europe, the Netherlands) the CCA of the corresponding side was

Table 1 Classification of the patient collective into female and male, showing the clinical picture pre-stenting.

		Female (95)	Male (263)
Vessel	ICA	90	256
	CCA	5	7
Treated side	Left	49	142
	Right	46	121
Initial stenosis (NASCET)	>90%	23	64
	70–90%	52	140
	50–70%	20	59
	<50%	0	0
Initial clinical Symptoms	Asymptomatic	28	69
	Symptomatic	67	194
s/p CEA	Ipsi-lateral	6	13
	Bilateral	2	11
Contralateral stenosis (NASCET)	None	75	196
	Occlusion	12	46
	>50%	5	13
	>70%	2	6
	>90%	1	2

All stenoses measured in angiograms with NASCET criteria

reached and the sheath, under guidance of the catheter and additional stabilisation with the guidewire, advanced into the distal CCA.

After injection of an i.v. heparin bolus to increase the ACT, the stenosis was passed by a 0.014-in. Choice PT wire (Boston Scientific Corp., Natick, MA, USA). In more than 50% of the cases, an adequate lumen allowed a primary passage with the stent. In the case of subocclusive and very high-grade (>85% NASCET) stenosis, a pre-dilatation was made beforehand using a 2.5- to 3-mm balloon catheter (166 patients; 46.4%, Ryujin or Hayate, Terumo Corp., Shibuya-ku, Tokyo, Japan). The most frequently used stent was the Carotid Wallstent (Table 2) with the preferred size of 7/30 mm (206/316; 65.2%).

Twelve of the used stents were balloon-mounted stents and were either placed if the ICA stenosis could not be reached or passed by a self-expanding stent or, in case of the Omnilink stents, used for treatment of proximal CCA stenoses. In this collective 1 TIA occurred.

In almost every intervention (94.4%) a post-dilatation with usually a 5-mm balloon catheter was conducted. Mostly utilised was a Maverick 5/20 (189, 55.9%, Boston Scientific Corp., Natick, MA, USA) and a Submarine 5/20 (93, 27.5%; Krauth Cardio-Vascular GmbH, Hamburg, Germany). After neurological examination of the patient and angiographic control of the extra- and intracranial vessels the catheters were removed and a femoral arterial sealing device (Angioseal 54.8%, Vasoseal 32.6%) was used in most cases of femoral access. The transbrachial approach was handled with manual compression.

All patients were monitored and ordered bed rest for 24 h at the stroke unit and controlled neurologically. After 24 h the patient was again clinically examined by the interventionalist.

Table 2 Applied stent models in the study.

Model (Manufacturer)	*n* (%)
Carotid Wallstent Monorail (Boston Scientific Corp., Natick, MA, USA).	316 (85.2%)
Optimed (OptiMed, Ettlingen, Germany)	33 (8.9%)
Driver Medtronic, Santa Rosa, CA, USA)	7 (1.9%)
N/A	4 (1.1%)
Omnilink (Guidant, Indianapolis, IN, USA)	4 (1.1%)
Acculink (Guidant Corporation, Santa Clara, CA, USA)	2 (0.5%)
Precise (Cordis Endovascular, Warren, NJ, USA)	2 (0.5%)
AVE (Medtronic, Santa Rosa, CA, USA)	1 (0.3%)
BostonNexstent (Boston Scientific Corp., Natick, MA, USA)	1 (0.3%)
Cristallo ideale (Invatec, Roncadelle, Italy)	1 (0.3%)

Pre-existent risk factors

Only 15% of the patients showed none of the classical risk factors like high blood pressure, extensive use of nicotine, diabetes or impaired lipometabolism. In the remaining patients at least one risk factor was determined, and 48% of the cases had a combination of two to four of these factors. In only two patients no information on risk factors were stated in the patient records.

Preceding radiotherapy

Prior to stenting 6.9% of the patients had undergone a radiation therapy in the head and neck area, mostly due to a brain or head and neck cancer.

Timing of CAS

Initially symptomatic patients were usually treated within a time range of 10 days after occurrence of neurological symptoms. Initially asymptomatic patients were treated within 30 days after interdisciplinary decision for CAS treatment

Results

In none of the procedures angiographically visible emboli occurred. The peri-interventional and 30-day event rate was 4.19% (15/358). These events included

- three deaths
- five hyperperfusion syndromes (comprising one death by a secondary fatal intracranial haemorrhage (ICH) and is included in the three deaths)
- one subarachnoid haemorrhage (SAH)
- seven ischaemic strokes

Most of the complications occurred in primarily symptomatic patients (Table 3) resulting in a complication rate of 5.3% (14/261) vs. 1% (1/97) for the asymptomatic group.

All cases of death had a neurological symptomatic ICA stenosis of more than 90% (NASCET). Causes of death were: (1) a fatal subdural haematoma after clipping of an intracranial aneurysm 14 days before CAS and anti-platelet therapy for the stenting procedure, (2) a fatal intracranial haemorrhage caused by a hyperperfusion syndrome, and (3) pneumonia 25 days after intervention, presumably originating from cardiac insufficiency and pulmonary congestion.

Regarding the patients with hyperperfusion syndromes, all but one had an initial stenosis of >90%. This particular patient had an initial stenosis of 50–70% and was the only with hyperperfusion syndrome and no ICH. The patient with fatal ICH was the only female with hyperperfusion syndrome and had an additional occlusion of the contralateral ICA.

One minor SAH was diagnosed in a post-interventional CAT head scan in a patient under coumarin therapy.

Two ischaemic strokes occurred during the interventional procedures: One caused by repeated partial deployment and recapturing of the stent due to difficult anatomical conditions, and the second occurred without any obvious procedural related problems.

Out of the remaining five strokes, one was the consequence of an emergency aortocoronary bypass 21 days after CAS and a second resulted from a myocardial infarction 1 month after intervention. One occurred in a patient with known atrial arrhythmia under adjustment of the anti-platelet medication to heparin after neck of femur fracture. The two remaining incidents were related to the conducted stenting procedure. One of these was found in a patient with prior radiation of the head.

In conclusion, the overall peri-interventional complication rate was 0.8% (3/358), meaning 20% (3/15) of all complications.

Neurological symptoms were defined as TIA if they could be referred to the hemisphere of the treated side and showed complete remission within 24 h. In this respect eight additional TIAs occurred in these 358 procedures, seven during intervention and one post-interventional.

Omitting not procedure-related complications (e.g., cardiac and pulmonary events due to pre-existing diseases as well as the subdural haematoma due to a prior clipped aneurysm), a procedure-related complication rate of 3.35% (12/358) for unprotected CAS resulted.

Table 3 Cases with a peri-interventional or 30-day event after stent-implantation.

		Stroke	SAH	Hyperperfusion	Death
Total patients		7	1	5	3[a]
Gender (m/f)		5/2	0/1	4/1	2/1
Inital stenosis (NASCET)	1=90%	2	1	4	3
	2=70–90%	5	–	–	–
	3=50–70%	–	–	1	–
Symptomatic		6	1	5	3
Asymptomatic		1	–	–	–

[a] Comprising one hyperperfusion patient

In 15 cases (4.19%), residual stenoses of more than 30% were found after the post-dilatation procedure. None of the patients with prior radiation showed a residual stenosis over 30%. Dissection due to the post-dilatation of the stenting procedure demanding an additional stenting occurred in one case.

Twenty-four patients showed complications in the femoral access route. These can be subclassified into eight insufficient closures by a sealing device, ten haematomas, five aneurysms and one obstruction of the femoral artery. Three aneurysms as well as the obstruction of the femoral artery had to be revised surgically.

One hundred eighty-seven (52.2%) of the 358 patients were either lost (179) to long-term follow-up or were recently treated with CAS (eight). The remaining 171 (47.8%) were controlled by Doppler ultrasonography usually at first in a range from 3 to 60 months with an average of 12.8 months. An overall in-stent restenosis rate of more than 50% (ECST criteria) was measured in 12.3% (21 Patients). These can be differentiated into restenosis grades of 50–70% (9/171, 5.3%) and >70% (12/171, 7%). The average time of occurrence in the >70% group was 9.83 months (6–19 months); 91.6% (11/12) were retreated interventionally. Ten of these were treated with balloon angioplasty solely, in the remaining case an additional stenting was performed without any complication.

Three of the restenosis (2>70%, 1<70%) occurred in patients with a history of CEA prior to stenting. Only seven of the 32 patients with prior CEA treatment had a long-term follow-up. On this basis 28.5% (2/7) of this small collective showed a high-grade restenosis.

For the subgroup with radiation therapy no sufficient long-term follow-up information could be reconstructed.

Discussion

Our results clearly indicate that the so-called unprotected stenting, with a peri-interventional and 30-day complication rate of 4.19% (3.35% procedure-related complications, respectively), is more than comparable to the results of the so-called protected CAS as published in large-scale studies promoting PDs [13–15]. Cremonesi et al. showed a respectable 3.4% overall complication rate in 442 protected CAS. However, in their collective only 57% of the patients were clinically symptomatic for the treated stenoses. These results are therefore not directly comparable to our collective with 73% symptomatic stenoses. The results comparing the 30-day complication rates for asymptomatic patients showed 3.7% complications in Cremonesi's collective, while we could demonstrate a rate of 1% in our asymptomatic patients.

The results of Cremonesi et al. were undercut by a meta-analysis of Kaastrup et al. demonstrating in a literature review that the combined stroke and death rate within 30 days was 1.8% in 896 CAS performed with cerebral protection devices. In the patient group treated with PDs, 64% were symptomatic for the treated stenoses. This extreme low complication rate does not appear to be very reliable, especially when comparing these data with recently published data of the PRO-CAS registry. Theiss and co-workers presented in their analysis a combined in-hospital mortality and stroke rate of 3.6% in 5,341 interventions. However, even in their collective, only 54.8% of the patients had a symptomatic ICA stenosis [16]. In comparison to our data, no follow-ups by independent stroke neurologists were performed for any of the evaluated CAS procedures of the three previously mentioned studies.

Recently Garg and co-workers published a review of the literature from 1995 to 2007 with 134 articles meeting their inclusion criteria. Using pooled analysis of all 134 reports, the relative risk (RR) for stroke was 0.62 (95% CI 0.54 to 0.72) in favour of protected CAS. Subgroup analysis revealed a significant benefit for protected CAS in both symptomatic (RR 0.67; 95% CI 0.52 to 0.56) and asymptomatic (RR 0.61; 95% CI 0.41 to 0.90) patients ($p<0.05$). Meta-analysis of 24 studies reporting data on both protected and unprotected stenting demonstrated a relative risk of 0.59 (95% CI 0.47 to 0.73) for stroke, again favouring protected CAS ($p<0.001$) [17].

Just as the previous mentioned studies, further authors published their uncontrolled mono-centre results mainly by comparing older data from unprotected interventions with patient groups where protection devices were applied [14, 15, 18–21]. Almost all of these studies concluded that PDs appear to reduce the thromboembolic complication rate and are strongly recommended in CAS procedures [14, 15]. But the comparison of historic with concurrent cohorts implements the individual and cumulative learning curve of an institution, which affects the outcome of those studies.

The influence of the standardisation of the anti-thrombotic regiment on the periprocedural complication rate too has to be taken into account when comparing older unprotected cohorts with recently treated protected ones.

In conclusion the positive effects imputed to PDs might be caused by many other factors.

In this regard the subanalysis data of two most recent multi-national, multi-centre trials present results veering toward the same direction of our outcome: favouring unprotected CAS. The results in SPACE I specified a complication rate in the unprotected vs. the protected group of 6.5% vs. 8.3% [22]. A pooled data analysis of SPACE I and EVA-3S evaluated the complication rate between patients treated with or without a PD. The unprotected

group had a 30-day event rate of 7.3% (5.1–10.2%) vs. 8.1% (5.5–11.3%) in the protected group with a 95% CI.

All these data are demonstrating that (1) there is no convincing evidence revealing the explicit benefit of PDs and (2) most complications during unprotected CAS do occur post-stenting, and could have not been prevented by PDs. Our single-centre results of unprotected CAS as well indicate that most complications occurred after and not during the stenting procedure, concluding that direct intra-interventional complications are considerably low. The evaluation of the case record forms of the SPACE I, trial reveals that about half of the complications evolved during the actual stenting and angioplasty procedure. Forty-one percent of the primary outcome events (pOEs; including 10% hyperperfusion syndromes) occurred when the catheter devices were displaced from the treated carotid and 10% of the pOEs during the navigation procedure at the aortic arch. These complications cannot be avoided by the use of a PD. Even in patients treated with a PD, half of the complications occurred immediately peri-interventional, revealing that PDs do not eliminate periprocedural embolic events. Comparable to these results again only 20% of the complications in our collective occurred intra-interventional.

It would seem reasonable to use a PD to reduce thromboembolic events caused by debris in CAS while a carotid stenosis is being dilated [23, 24]. But in protected as well as in the so-called unprotected CAS, pre-dilatation is often necessary before the PD is placed. In addition, after stent placement and post-dilatation, removing the PD can cause microembolisation. Therefore, from a procedural point of view, PDs may reduce, but certainly do not eliminate plaque embolisation, as demonstrated by periprocedural monitoring with transcranial Doppler [18, 25]. On the other hand, PDs do have the potential to produce complications such as vasospasms or dissections associated with temporary or permanent carotid occlusion [26]. All in all, the advantages and disadvantages of PDs seem to compensate one another, which may result in the equivalence as shown in the secondary analysis of the SPACE I data.

The timing of CAS might also have an impact on the complication rate. Most of the symptomatic patients were treated within 10 days after first occurrence of neurological symptoms. To cause neurological symptoms it can be assumed that the plaque surface is active and leads to embolisation, or the stenosis is pre-occlusive with insufficient collateralisation of the depending brain parenchyma. Treating these stenoses within 10 days might increase the risk for intra-interventional embolisations. Secondary analysis of the data from SPACE I, EVA-3S and the ICSS trial, focussing on this issue, could give more information for the best timing to treat symptomatic carotid artery stenoses with CAS.

Furthermore, as already discussed in studies comparing CAS vs. CEA, the procedural experience of a centre with particular instruments and materials seems to have a more dominating influence on the outcome than the method itself. A conclusion of these issues is that there is no reason to stipulate PDs for CAS procedures [27].

The ‘Multi-Centre Carotid World Registry’ reports of a combined 30 days stroke and mortality rate of 5.8% in 6,734 CAS. In this registry data no differentiation was made between symptomatic and asymptomatic or protected vs. unprotected CAS [28, 29]. This complication rate is well undercut by the results of unprotected CAS in our patient collective. In our opinion the most important issue to reduce the complication rate in carotid artery stenting is a standardised protocol, and the long-term experience of the interventionalist, while the use of a protection device seems to have no or even a negative impact.

But the discussion focussed on protected vs. unprotected CAS still does not describe the entire problem in endovascular therapy of atherosclerotic stenosis in carotid arteries. In addition the stent design seems to be an important factor in influencing the complications. Regrettably only little substantial information is provided in the current literature. Our results as well do not provide enough information on this issue as the majority of stenoses were treated with one stent model, the Carotid Wallstent Monorail. Especially high-risk lesions were treated with this kind of stent because of its small cell size and therefore its expected positive effect on plaque stabilisation. Not unexpected, most strokes occurred in this high-risk patient collective. However, we are convinced that our low overall complication rate is an effect of the small cell design resulting in a high level of protection.

In this context a further pitfall is the insufficient vocabulary describing stent designs mostly under the criteria of open or closed-cell design, cell size and wall thickness. Still there is no adequate classification system to describe the potential risk of embolisation connected with a certain stent design. Though it is possible to produce a smaller cell size in an open cell design than in a closed one, the risk of strut free areas is immanent in open cell designs. Especially when the vasculature shows a high tortuosity, small-closed cell designs seem favourable.

Recently, Schillinger and co-workers published a consecutive patient series treated at ten European centres to analyse the impact of different stent designs on neurological adverse events and mortality [30]. In contrast to the SPACE I data analysis they found no superiority of a specific stent design. However, in their population up to 90% of all patients were treated with a protection system, which may blot out the influence of a different stent design.

A very important, but rarely discussed problem is the occurrence of hyperperfusion syndromes. Brantley and co-

workers [31] recently presented a publication of seven patients with hyperperfusion syndromes (HPS) in 482 patients (1.45%) which is close to our rate of HPS (1.39%). But unlike their results all but one of our patients had an initial stenosis of >90% (NASCET). In Brantley's cohort none of the patients had an ICH and all patients recovered within 6 to 24 h while ours showed ICH in four cases one of them fatal. We do see a correlation between filiform stenoses and post-procedural high blood pressure as risk factors for HPS while Brantley and co-workers concluded that the clinical predictors of HPS and the optimum management remain to be determined.

The restenosis rates requiring interventional therapy show a broad variance in the literature. The articles are seldom comparable because of different cutoff values for the Doppler ultrasonographic (DUS) estimation of in-stent restenosis, the different threshold values for restenoses between 50% and 80% and the broad range of post-interventional follow-up intervals.

The cutoff values for the DUS derived parameters to estimate the degree of restenosis in stented arteries are not yet standardised. An unstented carotid artery has a more elastic vessel wall than a stented one, even if stenosis is present. That means that the cutoff criteria for the degrees of an in-stent stenosis, based on blood velocity parameters, are different from the established cutoffs used for unstented arteries. This leads to an over-estimation of the degree of in-stent restenosis, if common DUS criteria are applied [32, 33]. Spies and co-workers showed different cutoff values for several stent models concluding that the stent type might have a significant impact on DUS-derived velocity signals [34].

Under these limitations of the values in the literature, as well as our own values, the in-stent restenosis of 7% at an estimated degree of 70% (ECST) and more is comparable with the average percentage of restenoses presented in the literature which is between 5% and 8% for stenoses over 80% [35–38].

Regrettably the limited size of the group with long-term follow-up (171/358) restricts the information on the rate of restenoses in smaller subgroups such as patients with CEA or radiation in their history.

We hope that in the near future probably the development of new stent designs and materials will provide interventionalists with lower periprocedural complication rates and lower long-term restenosis rates.

A limitation of our study is certainly the retrospective aspect and accompanied problems. Our data are therefore (as the data of other retrospective analysis) not entirely comparable to the SPACE I or EVA-3S data. Additionally, some complications might not be adequately documented in the files or might have been missed as some patients were quickly referred to rehabilitation centres or other hospitals. However, all of our patients were independently controlled by a neurologist and not by the interventionalist. We therefore believe that the data in this report are credible and provide a precious contribution to the recent discussion about carotid stenting.

Conclusion

Our data support the assumption that carotid artery stenting without the use of a protection device is safe and an acceptable alternative to carotid endarterectomy. The use of stents with a small cell design in the hands of well-experienced interventionalists is requested to offer CAS therapy to patients with high-grade carotid stenoses.

Conflict of interest statement We declare that we have no conflict of interest.

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