Protected Carotid Stenting
Clinical Advantages and Complications of Embolic Protection Devices in 442 Consecutive Patients

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Background and Purpose—Periprocedural embolization of debris during carotid stenting interventions may result in neurological deficit. This study was designed to evaluate in-hospital and 30-day adverse events in patients percutaneously treated for carotid artery disease with embolic protection devices.

Methods—From 1999 to June 2002, a total of 442 consecutive patients underwent percutaneous angioplasty and/or stenting of the extracranial carotid artery. The endovascular procedure was conducted under embolic protection devices.

Results—The percutaneous procedure was successful in 440 of 442 patients (99.5%). No periprocedural death occurred with any embolic protection device. All in-hospital stroke/death and 30-day ipsilateral stroke/death rate was 1.1%. The overall complication rate was 3.4%. Major adverse events included 1 major stroke (0.2%), 4 intracranial hemorrhages (0.9%), 1 carotid artery wall fissuration (0.2%), and 1 diffuse cardioembolism (0.2%). Minor adverse events included 4 minor strokes (0.9%) and 4 transient ischemic attacks (0.9%). The cerebral protection device–related complications were 4 (0.9%): 1 case of abrupt closure of the internal carotid artery because of spiral dissection (0.2%), 1 case of trapped guide wire (0.2%), and 2 cases of intimal dissection (0.5%). Transient loss of consciousness, tremors, and fasciculations were present in 6 of 40 patients (15%) in whom occlusive protection devices were used.

Conclusions—Our data suggest that percutaneous stenting of the carotid artery when a cerebral protection device is used is feasible and effective but not without potential complications. However, a long learning curve may exist for the proper use of some embolic protection devices. (Stroke. 2003;34:1936-1943.)

Key Words: carotid arteries ■ protective devices ■ stents

Carotid angioplasty and stenting is being used widely to treat severe carotid obstructive disease, and it is now accepted as a less invasive technique that provides an alternative for some patients, particularly those with significant comorbidities. However, the most important acute complication of carotid angioplasty and stenting is related to the distal embolization of particles generated during the endovascular procedure, with the risk of definitive stroke.1–6

At present, distal cerebral microembolization is demonstrated by transcranial Doppler monitoring and diffusion-weighted MRI; both techniques have revealed significant and increased dislocation of microemboli during carotid angioplasty and stenting. The microemboli are greater in percutaneous treatment compared with a surgical approach.7–9

In the last 3 years, numerous cerebral protection systems have been proposed to limit the acute neurological complications related to distal embolization. The aim of this study was to investigate the feasibility, safety, and efficacy of different endovascular protection systems during elective stenting of the extracranial carotid arteries.

Subjects and Methods
From December 1999 to June 2002, a total of 442 consecutive patients underwent percutaneous angioplasty and stenting of the extracranial carotid artery protected by embolic protection devices (EPDs). Written, informed consent for intervention was obtained from all patients. The demographic and clinical data, angiographic evaluation, and neurological history characteristics of the study group are summarized in Tables 1 and 2.

Patient Inclusion Criteria
Inclusion criteria included the following: age >50 years; monolateral or bilateral carotid critical stenosis (>75% carotid lesion); symptomatic for the culprit carotid lesion; asymptomatic for the culprit carotid lesion but with a positive cerebral CT scan for silent cerebral focal ischemia ipsilateral to the culprit carotid lesion; and refusal by surgeons as high-risk surgical subsets.
Carotid de novo lesion 383 86.7
Carotid postendarterectomy restenosis 58 13.1

Angiographic evaluation

Male 350 79.2
Population study 442 100.0
Age (mean ± SD), y 73 ± 7.6
Carotid post-endarterectomy restenosis 58 13.1
Carotid post-PTA/stenting restenosis 1 0.2

Angiographic evaluation

Right carotid artery 252 57.0
Left carotid artery 190 43.0
Diameter stenosis, (mean ± SD), % 83 ± 6
Lesion length (mean ± SD), mm 16 ± 9
Bilateral carotid disease >70% 110 24.9
Contralateral carotid occlusion 48 10.9

PTA indicates percutaneous transluminal angioplasty.

Patient Exclusion Criteria

Exclusion criteria included thrombocytopenia, leucopenia, neutropenia, or gastrointestinal bleeding in the previous 3 months; allergy to aspirin, clopidogrel, ticlopidine; angiographic appearance of fresh thrombus at the carotid lesion site; and angiographic appearance of carotid chronic total occlusion or long preocclusive lesion (‘string sign’ lesion).

Patient Assessment

Before treatment, all patients underwent careful neurological examination performed by an independent board-certified neurologist (including National Institutes of Health Stroke Scale [NIHSS]), echo/color-flow Doppler (lesion site and intracranial cerebral blood flow assessment), cerebral CT scan, and angiographic evaluation.

Within 24 hours after the procedure and at the 30-day follow-up, all patients underwent another neurological examination performed by the same independent board-certified neurologist and a complete echo/color-flow Doppler evaluation. A procedure cerebral CT scan was performed only in patients with documented neurological complications.

Preprocedure Echo Analysis of Carotid Plaques

Plaque echogenicity as assessed by B-mode ultrasound has been found to reliably predict the content of soft tissue and the amount of calcification in carotid plaques. Plaque morphology in terms of echogenicity, defined as reflectance of the emitted ultrasound signal, was assessed in a modified version of the classification proposed by Gray-Weale et al and graded from 1 to 4 as echolucent, predominantly echolucent, predominantly echogenic, or echogenic. 

Echo plaque characteristics and complexity were checked in all patients. Additionally, echo/color-flow Doppler showing the absence of significant residual stenosis and pathological acceleration in blood flow (<1.5 m/s). Transient ischemic attack (TIA) was defined as a focal retinal or hemispheric event from which the patient made a complete recovery within 24 hours. Minor stroke was defined as a new neurological deficit that either resolved completely within 30 days or increased the NIHSS by ≥3. Major stroke was defined as a new neurological deficit that persist for >30 days and increased the NIHSS by ≥4. Finally, fatal stroke was defined as death attributed to an ischemic stroke or intracerebral hemorrhagic stroke.

Medical Treatment

Before the Procedure

All patients were treated with acetysalicylic acid at a mean dosage of 125 mg/d and ticlopidine at a mean dosage of 500 mg/d at least 4 to 5 days before admission.

During the Procedure

The mean dosage of sodium heparin used during the procedure was 100 U/kg. A mean of 1 mg atropine was given to patients before balloon inflation.

Postprocedure

Ticlopidine (500 mg/d) was continued for at least 30 days after the interventional procedure (hemochrome and white blood count were checked 7 to 10 days after the percutaneous intervention). Antiplatelet therapy with aspirin is being continued indefinitely.

Description of the Procedure

All procedures were carried out via puncture of the right and/or left femoral artery. The vascular approach did not change, depending on the use of different types of cerebral protection devices. The common carotid artery was selectively engaged directly by use of a primary guiding catheter. When use of a primary guiding catheter was not possible because of the particular anatomy of supra-aortic vessels, we placed a stiff wire into the external carotid artery for positioning of a long sheath or a guiding catheter into the common carotid artery.

Once the common carotid artery was engaged, all patients underwent an angiographic examination of the culprit carotid lesion in 2 different projections and an angiographic examination of the intracranial circulation in the anteroposterior and/or lateral projection. The same angiographic checkup was performed at the end of the
Procedural success was achieved in 440 of 442 subjects (99.5%). In 1 failure, the endovascular treatment was ineffective because of spiral dissection of the left internal carotid artery (major asymptomatic complication) distal to the stenosis site as a result of Medtronic PercuSurge occlusive balloon vessel injury. In the other failure, despite a good angiographic final result, the endovascular procedure was ineffective because the 0.014 wire of the Johnson & Johnson-Cordis AngioGuard system remained trapped in the proximal edge of a Johnson & Johnson-Cordis Palmaz-Schatz P205 stent. The system was easily retrieved via surgical cutdown of the carotid artery.

### In-Hospital and 30-Day Adverse Events in the Study Group

All in-hospital stroke/death and 30-day ipsilateral stroke/death rate was 1.1%. The overall complication rate was 3.4% (Tables 4 and 5). In summary, there were 7 major adverse events: 1 major stroke (0.2%), 4 intracranial hemorrhages (0.9%), 1 carotid artery wall fissuration (0.2%), and 1 diffuse cardioembolism (0.2%). Minor adverse events included 4 minor strokes (0.9%) and 4 TIs (0.9%). No periprocedural death occurred in the study group.

The cases complicated by intracranial hemorrhage achieved complete resolution of symptoms (hemisensory loss, hemiplegia, and epileptic seizures) and radiological findings (CT and MR) within 30 days. One case was complicated by carotid artery fissuration (very small and limited carotid artery wall rupture) with a periarterial extravasation immediately after stent postdilatation. This acute complication was successfully treated by sealing the wall rupture with long and repeated low-pressure balloon inflations inside the carotid stent. The patient left the laboratory asymptomatic, without angiographic patterns of residual significant periarterial extravasation.

One case was complicated by an unusual peripheral embolic event temporarily involving 3 different arterial sites (left common carotid artery, left brachial artery, right common femoral artery) occurring 12 hours after carotid stenting. This event, reported as diffuse cardioembolism, was related to the detachment of emboli from left atrium in an elderly patients with chronic atrial fibrillation determined by the full anticoagulation needed for the endovascular procedure. The patient was successfully treated via surgical multivascular approach.

### In-Hospital and 30-Day Adverse Events in Symptomatic Patient Subset

All in-hospital stroke/death and 30-day ipsilateral stroke/death rate was 1.2%. The overall complication rate was 3.2% (Table 5). The major adverse events were as follows: 1 major stroke.
stroke (0.4%), 3 intracranial hemorrhages (1.2%), and 1 diffuse cardioembolism (0.4%). There were 3 minor adverse events: 2 minor strokes (0.8%) and 1 TIA (0.4%).

In-Hospital and 30-Day Adverse Events in Asymptomatic Patient Subset
All in-hospital stroke/death and 30-day ipsilateral stroke/death rate was 1.1%. The overall complication rate was 3.7% (Table 5). The 2 major adverse events included 1 intracranial hemorrhage (0.5%) and 1 carotid wall fissuration (0.5%). Minor adverse events included 2 minor strokes (1.1%) and 3 TIAs (1.6%).

Temporal Distribution of Embolic Complications
Timing of any clinical appearance of neurological symptoms (major stroke, minor stroke, TIA) related to cerebral embolization was measured in all 442 study patients. Acute embolic complications were defined as neurological events occurring during the endovascular procedure or a few minutes after retrieval of the protection device. Late embolic complications were defined as neurological events occurring after the endovascular procedure completion, during the patient’s hospital stay and in the 30 following days. Temporal distribution analysis showed the following data.

Complications Related to the Use of EPDs
There were 4 (0.9%) EPD-related complications: dissection treated with an additional stent (2, 0.5%), vessel occlusion by spiral dissection (1, 0.2%), and “trapped” guide wire (1, 0.2%).

EPD Major Complications
Two major adverse events were reported, both asymptomatic. The first was a case of spiral dissection of the left internal carotid artery distal to the stenosis site as a result of Medtronic PercuSurge occlusive balloon vessel injury. This injury was attributed to diffuse artery disease also involving the distal portion of the internal carotid artery. Although we were unable to cross the spiral dissection and the intracranial left internal carotid artery was completely occluded, the patient did not complain of any symptoms. Neurological examination did not show any neurological deficit in either the Catheterization Laboratory or the ward. CT and MRI evaluation excluded the presence of any lesion. This behavior resulted because the hemisphere supplied by the occluded carotid artery received sufficient blood flow from the contralateral carotid circulation and intracranial posterior circulation.

The second event occurred during the endovascular treatment of a subocclusive ostial stenosis of the left common carotid artery. This procedure, because of the complete occlusion of the contralateral internal carotid artery, was protected by the use of a 7-mm Johnson & Johnson-Cordis AngioGuard system. At the end of stent delivery (Johnson & Johnson-Cordis Palmaz-Schatz P205 manually crimped on 7/20-mm balloon), the 0.014 wire of the AngioGuard system remained trapped in the proximal edge of the stent. This probably occurred because of the significant acute angle between the aortic arch and the takeoff of the internal carotid artery or the high-pressure stent dilation carried out with a balloon on a second stiff wire placed parallel to the Angio-Guard wire. Because we were unable to recapture the nitinol basket and its potential embolic content, we referred the patient to the vascular surgical team to remove the system. The protection device was easily retrieved via surgical cutdown of the distal portion the common carotid artery. The patient was asymptomatic, and neurological examination did not show any neurological deficit.

EPD Minor Complications
Two cases of tear dissection of the right internal carotid artery distal to the stenosis site as a result of the Medtronic PercuSurge occlusive balloon vessel injury occurred. The localized wall dissection did not cause flow impairment and was easily treated by implanting an adjunctive self-expandable Boston Scientific Easy Wallstent above the first one to cover the intimal flap.

EPD Side Effects
Transient spasm of the intracranial segment of the carotid artery, presumably related to the protection device (distal occlusive balloon, filter), was observed in 35 cases (7.9%). In all cases, spasm was resolved after intracarotid nitroglycerin administration.

Carotid flow impairment resistant to nitroglycerin administration occurred in 58 cases (13.1%) protected by filter devices. All patients remained asymptomatic during the episode. Flow was completely restored after filter removal. A large amount of macroscopically visible debris was noticed inside the filters and considered to be responsible for the significant flow impairment.

Transient loss of consciousness, tremors, and fasciculations were present during 6 of 40 carotid procedures protected by either distal or proximal occlusive systems (PercuSurge, Parodi, MO.MA). In all cases, the neurological symptoms appeared after a mean cerebral occlusion time of 6±2.5 minutes. Immediately after deflation of the occlusive system, all patients returned to baseline neurological conditions.

Distal Protection Debris
In 304 patients (69.0%), macroscopically visible plaque debris was captured and retrieved. Plaque debris ranged from 75 to 6000 μm in diameter. In 17.7% of patients with visible particles (54 cases), the embolic debris was ≥2 mm in diameter.

Histopathological analysis was performed on all filters with macroscopically visible plaque debris with light microscopy after staining with hematoxylin-eosin and Azan-Mallory. The debris consisted of cholesterol crystals, fibrin material, atheromatous plaque, and macrophage foam cells.

Discussion
Some recent studies have demonstrated that endovascular treatment of carotid pathologies is correlated with a risk of cerebral ischemic events and, more generally, with a rate of complication that are no higher than those observed with traditional surgery. As demonstrated in published studies, EPDs have the potential to reduce the incidence of intracranial debris embolization, therefore reducing major...
adverse neurological events and providing results comparable to those for carotid endarterectomy.14,18,19

Comparing our reported data with recently published literature6,13,15–17 shows that the use of cerebral protection systems can be considered positive in terms of high procedural success, low device-related complications, and low in-hospital/30-day complications.

No peri-procedural death occurred in this neuroprotected group, with a composite all stroke/death rate of 1.1% confirmed at 30-day follow-up. The overall complication rate did not exceed 3.4%.

When we compare the overall complication rate in symptomatic and asymptomatic patients, no significant difference was demonstrated (symptomatic, 3.2%; asymptomatic, 3.7%). The overall stroke/death analysis gave the same result (symptomatic, 1.1%; asymptomatic, 1.2%).

When comparing all major and minor complication analysis in symptomatic and asymptomatic patients, we found more frequent major events (major stroke and intracranial hemorrhage) in the symptomatic subgroup than in asymptomatic group (2.0% versus 1.1%); conversely, minor events (minor stroke, TIA) were more frequent in the asymptomatic subgroup than in the symptomatic subgroup (2.6% versus 1.2%).

Analysis of temporal distribution of embolic complications (timing of any clinical appearance of neurological symptoms: major stroke, minor stroke, TIA related to cerebral embolization) demonstrated that neuroprotection devices allowed us to protect the procedure but did not affect late embolic events. We reported in the present series 5 acute embolic events (1.2%) and 4 late ones (0.9%).

Intracranial hemorrhage was encountered in 4 patients (0.9%) and was the most dangerous nonembolic complication reported in this series. In 3 cases, the event happened in very symptomatic and hypertensive patients with subocclusive carotid lesions and was attributed to cerebral reperfusion syndrome. In 1 case, the intracranial hemorrhage complicated a quite simple procedure in an asymptomatic patient and was not attributed to any specific clinical variable but to the full heparinization during the procedure and the prior acetylsalicylic acid and ticlopidine regimen.

The use of protection devices was not free of complications; at least 2 potentially dangerous situations (0.5%) related to the use of 2 very different protection devices occurred. Although both complications were managed without negative results to the patients—they did not complain of any symptoms and did not show any neurological deficit—we are aware of the objective danger these situations might involve.

The present study was not designed either to compare various protection systems or to detect differences between balloon occlusive devices, filter devices, and reversal flow devices. The primary end point was to evaluate the clinical impact of protection devices during carotid endovascular interventions. From our experience, we can state that occlusive balloon devices and reversal flow devices have a favorable crossing profile and do not require lesion crossing. However, balloon inflation may cause transient cerebral ischemia, which is not always predictable or well tolerated by all patients.

Transient loss of consciousness, tremors, and fasciculations were present in 6 cases (1.3% of the entire protected group, 15% of the occlusive system subset) of carotid procedures protected by either distal or proximal occlusive systems. In contrast, filter devices, although they have higher crossing profiles and more stiffness, maintain antegrade cerebral flow and allow visualization, which may result in more precise stent placement. In the present series, filter devices were well tolerated by all patients.

Study Limitations

The patients did not undergo transcranial Doppler monitoring during the endovascular carotid procedure and did not receive a postprocedure CT scan or MRI examination. For this reason, we are not able to provide any objective information about the degree of embolization occurring while crossing the lesion with wires, balloons, stents, and protection devices; the comparison between the 2 groups in terms of degree of embolization; and the efficiency of the protection systems in capturing all the particles produced during carotid angioplasty and stenting.

Conclusions

Our experience demonstrates that the extensive use of cerebral protection devices is feasible and effective in preventing distal embolization during carotid endovascular procedures. Despite the fact that the immediate peri-procedural and in-hospital results are encouraging, we are aware that EPDs allowed operators to protect the procedure but unfortunately did not affect late embolic events. Moreover, our experience suggests that the correct use of all protection systems requires a proper learning curve and that the use of protection devices is not currently complication free. Cerebral protection will most likely decrease the complication rate of carotid stent procedures further, possibly turning this intervention into the therapy of choice for patients in whom carotid artery angioplasty and stenting is indicated.

References

Carotid Artery Stenting With or Without Protection Devices? Strong Opinions, Poor Evidence!

In recent years, endovascular treatment of carotid artery stenosis has profited from substantial technical improvements, but the dominant point of discussion circles around the so-called cerebral protection devices. In this issue of *Stroke*, Cremonesi et al.\(^7\) report their impressive single-center experience with protected carotid artery stenting (CAS) in 442 patients. The overall complication rate was 3.4%, and the 30-day ipsilateral stroke/death rate was 1.1%. The authors conclude that protection devices are feasible and effective in preventing distal embolization. In a recent *Stroke* issue, Kastrup et al.,\(^2\) who systematically reviewed single-center CAS studies, concluded that protection devices appear to reduce thromboembolic complications during CAS.

The appearance of debris during CAS and carotid endarterectomy (CEA) is a common event.\(^1\) At first glance, it seems reasonable to apply protection systems to catch particles by means of occlusive balloon systems or filtration baskets in the internal carotid artery. The beneficial use of such devices seems to be supported by a growing number of publications, mostly from the field of cardiology, reporting declining neurological complication rates. Despite the lack of further controlled studies, the use of protection devices has even become obligatory in centers with high-grade carotid stenosis.\(^1\)

In unprotected CAS, procedural steps include guide wire passage (0.36 mm), stent placement (1.67 mm), and postdilatation. Predilatation of the stenosis has been deemed necessary in only \(\approx 2\%\) of the cases.\(^7\) In transcranial Doppler (TCD) monitoring, our own experience (unpublished data) and data from the literature\(^6\) indicate that 60% of microembolic signals appear during the procedural steps before postdilatation. In another TCD study during unprotected CAS, only the detection of embolic signals in the step of primary guide wire passage was related to neurological symptoms. A primary passage must always be an unprotected maneuver.

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In unprotected CAS, predilatation is often necessary (37% of patients in the present study) before the protection device (0.9 to 1.67 mm) is placed. After stent placement and postdilatation, removal of protection devices causes additional microembolization.\(^8\) From a procedural point of view, protection devices may reduce but certainly do not eliminate plaque embolization, as demonstrated by TCD\(^9\) and MRI studies.\(^10\)

Supplementing the recent dramatic technical progress (eg, less traumatic, self-expandable stent devices, more friction-resistant introducer catheters, and better guide
wire systems leading to marked improvement of CAS), 2 other factors quietly evolved: the associated learning curve of active interventionalists and the improved periprocedural anticoagulation regimens using combined platelet inhibitors (acetylsalicylic acid, clopidogrel), low-molecular-heparin at least 3 days before the procedure, and full heparinization (activated clotting time >250 seconds) during CAS. These improvements may have been important cofactors in preventing cerebral embolism during the recent era of protected CAS studies.

The work of Cremonesi et al specifically addresses the risk of protection devices. A remarkably low number of technical complications such as dissection of the internal carotid artery (0.7%) or trapped guide wire needing surgical approach (0.2%) were observed, and all these events were clinically well tolerated. Even the authors probably would not expect such a favorable outcome in these events. Hemodynamic intolerance in occlusive balloon systems (5% to 15% of patients) and congested nets are other typical problems. Neither Cremonesi et al nor Kastrup et al reported on the failure rate on application of protection systems, alluding that the success rate was 100%. This uncommonly high success rate, together with the small number of observed technical complications, indicates extraordinary skill in this group, which is especially spectacular compared with study groups reputed for their skills and long experience, in which a failure rate of up to 20% was reported.

In a former publication by Cremonesi et al on a CAS cohort of 119 patients treated without protection devices, the ischemic neurological event rate was 2.5% (2 minor strokes, 1 transient ischemic attack). In the present study, 6 patients suffered transient neurological symptoms resulting from intolerance of occlusive protection devices. Adding these events (n=6), transient ischemic attacks (n=4), minor strokes (n=4), and the 1 major stroke, the ischemic neurological event rate in the present report would also come to 2.5%. The 0.9% asymptomatic technical complication rate should simply be kept in mind.

The review by Kastrup et al from the neurological perspective included a variety of single-center studies from 1996 to 2003, with patients being treated under very heterogeneous conditions. The combined stroke and death rate within 30 days was 1.8% in CAS with protection and 5.5% in CAS without protection. From a neuroradiological perspective, concentrating our attention on the 10 studies appearing since 2002,13–21 when most of 966 treated vessels in 923 patients were probably treated under more comparable conditions, including technical and anticoagulation progress, the combined stroke and death rate within 30 days in protected CAS4,19–21 was 2.0% and in unprotected CAS was 3.2%.13–18 In our view, this does not justify the strong recommendation of the use of protection devices.

The NASCET trial eliminated the prejudice that the complication rate in CEA was only ~1% as reported in the vast majority of uncontrolled single-center studies. Apparently, the scientific debate on CAS in general and protection devices in particular is still in a “pre- NASCET stage.” Future attempts should concentrate all efforts on finishing the pending trials comparing CEA and CAS (CREST in the United States, EVA3S in France, ICESS or CAVATAS2 in the United Kingdom, and SPACE in Germany). All European studies have the same primary endpoint and are designed to promote secondary analysis. A later subgroup analysis of protection device use could be carried out because in SPACE and CAVATAS2 the use of protection devices is only optional.

We must all confess that our decisions to date are simply not evidence based. It is too early to draw binding conclusions with all clinical and economic consequences. We feel that the ongoing trials with a NASCET analog design should be supported, the results awaited, and then the conclusions implemented.

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Editorial Comment—Carotid Artery Stenting With or Without Protection Devices?
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