Proximal Endovascular Occlusion for Carotid Artery Stenting
Results From a Prospective Registry of 1,300 Patients

Eugenio Stabile, MD, PhD, Luigi Salemme, MD, Giovanni Sorropago, MD, Tullio Tesorio, MD, Wail Nammas, MD, Marianna Miranda, MD, Grigore Popusoi, MD, Angelo Cioppa, MD, Vittorio Ambrosini, MD, Linda Cota, MD, Giampaolo Petroni, MD, Giovanni Della Pietra, MD, Angelo Ausania, MD, Arturo Fontanelli, MD, Giancarlo Biamino, MD, Paolo Rubino, MD
Mercogliano, Italy

Objectives
This single-center registry presents the results of proximal endovascular occlusion (PEO) use in an unselected patient population.

Background
In published multicenter registries, the use of PEO for carotid artery stenting (CAS) has been demonstrated to be safe and efficient in patient populations selected for anatomical and/or clinical conditions.

Methods
From July 2004 to May 2009, 1,300 patients underwent CAS using PEO. Patients received an independent neurological assessment before the procedure and 1 h, 24 h, and 30 days after the procedure.

Results
Procedural success was achieved in 99.7% of patients. In-hospital, major adverse cardiac or cerebrovascular events included 5 deaths (0.38%), 6 major strokes (0.46%), 5 minor strokes (0.38%), and no acute myocardial infarction. At 30 days of follow-up, 2 additional patients died (0.15%), and 1 patient had a minor stroke (0.07%). The 30-day stroke and death incidence was 1.38% (n = 11005119). Symptomatic patients presented a higher 30-day stroke and death incidence when compared with asymptomatic patients (3.04% vs. 0.82%; p < 0.05). No significant difference in 30-day stroke and death rate was observed between patients at high (1.88%; n = 112) and average surgical risk (1.07; n = 7) (p = NS). Operator experience, symptomatic status, and hypertension were found to be independent predictors of adverse events.

Conclusions
The use of PEO for CAS is safe and effective in an unselected patient population. Anatomical and/or clinical conditions of high surgical risk were not associated with an increased rate of adverse events. (J Am Coll Cardiol 2010;55:1661–7) © 2010 by the American College of Cardiology Foundation

Carotid artery stenting (CAS) is considered to be a reasonable alternative to carotid endarterectomy (CEA), particularly in patients at high risk for CEA (1). Although there are no randomized studies comparing CAS with and without embolic protection devices (EPDs), consensus among experts suggests their use in reducing the risk of stroke during CAS (2). Among the EPDs that are in clinical use, proximal EPDs have the theoretical advantage of providing embolic protection during all phases of the intervention (2).

It has been reported that the use of proximal endovascular occlusion (PEO) during CAS, as a proximal EPD, is associated with a reduced amount of Doppler-detected microembolic signals when compared with distal protection devices (3). In multicenter registries, the use of PEO for CAS has been demonstrated to be safe and efficient in selected patient populations (4–7). This registry presents the results of a single-center experience on the use of PEO for CAS in 1,300 patients.

Methods

Patient selection. From July 2004 to May 2009, 1,437 patients underwent CAS at our institution. Of these, 1,300 CAS procedures were performed using PEO for neuroprotection.

Among the PEO-protected CAS procedures, 89 were performed from July 2004 to December 2004 in selected patients (59.8% of a total of 149 CAS procedures performed in the same period of time), and 1,211 were performed from...
January 2005 to May 2009 in consecutive patients (93.9% of a total of 1,288 CAS procedures performed in the same period of time). From July 2004 to December 2004, patients were selected for PEO only if they did not present with contralateral internal carotid artery occlusion/stenosis or any stenosis of the ipsilateral external carotid artery. PEO was used from January 2005 to May 2009 in all consecutive patients undergoing CAS.

Inclusion criteria were the degree of internal carotid artery (ICA) stenosis, determined by angiography, according to North American Symptomatic Carotid Endarterectomy Trial Criteria (8,9): 1) asymptomatic stenosis ≥80%; and 2) symptomatic stenosis ≥50%. Symptomatic is defined as carotid stenosis associated, within 6 months before the procedure, with amaurosis fugax, ipsilateral hemispheric transient ischemic attack, or ipsilateral ischemic stroke not resulting in a major residual neurological deficit (stroke scales: Barthel ≤60, National Institutes of Health ≥15, or Rankin >3).

Patients with the following criteria were excluded: 1) presence of a critical stenosis of the ipsilateral common carotid artery (CCA); 2) occlusion of ipsilateral external carotid artery (ECA); 3) an international normalized ratio >1.3; 4) contraindication to aspirin and thienopyridines; and 5) refused informed consent before enrollment.

**Concomitant therapy.** All patients received aspirin (75 to 160 mg/day) and should have been on ticlopidine (250 mg twice daily) for at least 7 days. Alternatively, patients received clopidogrel preload (300 mg) 24 h before the procedure. After the procedure, thienopyridines were continued for 3 months, whereas aspirin was continued for life.

For anticoagulation, 70 to 100 IU/kg of heparin was administered before wiring the ECA, with intention to achieve activated clotting time (ACT) >250 s. Additional heparin was administered at operator discretion according to ACT values (2). Forty-five patients received 0.75 mg/kg of bivalirudin as intravenous bolus (Angiomax, The Medicines Company, New York, New York) followed by infusion at a rate of 2.5 mg/kg/h, discontinued at the end of the procedure (8).

**Technique of the CAS procedure.** All procedures were performed percutaneously, with the patient under local anesthesia. The physicians performing the procedures fulfilled reported qualification for training (10). Institutional experience was considered level 1 for the first 50 cases, level 2 for the following 250 cases, and level 3 for the remaining 1,000 cases.

At the start of the procedure, an 8 to 10 F, 25-cm long, introducer sheath (Terumo, Tokyo, Japan) was inserted in the infrarenal aorta via the common femoral artery. Coronary angiography was performed in all patients before CAS due to the high incidence of concomitant coronary artery disease (1). Presence of at least one >70% angiographic coronary artery stenosis was considered as significant coronary artery disease.

After aortic arch angiography, a selective bilateral carotid artery catheterization was performed using a 5-F JR4 diagnostic catheter advanced over a 0.035-inch soft hydrophilic wire (Standard Glidewire, Terumo).

Once diagnostic angiography was completed, the wire was advanced in one of the ECA distal branches, the diagnostic catheter was advanced in the distal ECA, and then the hydrophilic wire was exchanged for a 300-cm, 0.035-inch stiff wire (Hi-Torque Supracore, Abbott Vascular, Abbott Park, Illinois). The Mo.Ma system (Invatec, Roncadelle, Italy) was guided over the stiff wire until the radiopaque marker of the distal balloon was located in the ECA, at approximately 1 cm beyond bifurcation and in proximity to or at the superior thyroidal artery (3). Then the distal balloon was inflated in the ECA and the proximal balloon in the CCA, thus blocking the antegrade and the retrograde flow across the target vessel. A 0.014-inch wire was then navigated through the ICA stenosis. Lesion pre-dilation was left to the operator’s discretion, and self-expanding carotid stents were deployed. Stents were classified according to alloy: nitinol or stainless steel (Carotid Wallstent, Boston Scientific, Natick, Massachusetts). Nitinol stents were divided according to cell design: 1) closed (X-Act, Abbott Vascular); 2) open (Precise, Cordis, Miami, Florida; Acculink, Abbott Vascular); and 3) hybrid, with closed cells in the center and open at the sides (Cristallo Ideale, Invatec) (11).

After dilation, at least 60 ml of blood was aspirated and filtered through sieves, checking for visible plaque debris. Blood flow was restored only after 3 consecutive aspirations free of debris, deflating first the distal balloon and then the proximal balloon. The final angiography included ipsilateral biplane carotid and intracranial views (4–7).

**Post-procedural patient management.** Femoral sheaths were removed when ACT was <150 s in the heparin group and 2 h after the procedure, independently of ACT values, in the bivalirudin group (12).

Access site hemostasis was achieved by manual compression in all patients. If clinical signs of limb ischemia occurred on the side of femoral access, sheaths were removed independently of post-procedural time and ACT values. Femoral sheath-induced leg ischemia was classified as major if it required thromboembolectomy and minor if it resolved by sheath removal (13).

A complete blood count was obtained before the CAS procedure and before hospital discharge. An independent neurologist assessed all patients (13).
Definitions. Occlusion time (time of flow blockage) was defined as the time from the inflation to the deflation of the proximal balloon in the CCA. Occlusion intolerance was defined as any transient neurological deficit observed during occlusion time, but showing a complete recovery within 20 min after restoring antegrade flow. Procedural time was defined as the time from the completion of diagnostic angiography and final intracranial views (4–7). Device success was defined as the ability to position, deploy, and retrieve the intact Mo.Ma device during the index procedure (5). Protection success was defined as complete blockage of antegrade blood flow in the ICA throughout the entire procedure (5). Technical success was defined as device success and the ability to successfully implant a carotid stent with a residual ICA stenosis <30% (5). Procedural success was defined as technical success without the occurrence of any major adverse cardiac or cerebrovascular event or unresolved occlusion intolerance during the index procedure.

The primary end point of the study was the incidence of death and any stroke in-hospital and at 30 days.

Neurological complications were classified as one of the following: 1) minor stroke, defined as a new neurological deficit that either resolves completely within 30 days or increased National Institute of Health Stroke Scale by ≤3; and 2) major stroke, defined as a new neurological deficit that persists for >30 days and increased National Institute of Health Stroke Scale by ≥4 (1,14,15).

Patients were considered at high surgical risk if presenting with at least 1 or more high-risk criteria in either medical comorbidities (age >80 years; Canadian Cardiovascular Society angina class III or IV or unstable angina; congestive heart failure class III or IV; left ventricular ejection fraction <30%; left main and/or ≥2-vessel coronary; urgent [<30 days] heart surgery; recent myocardial infarction [<30 days]; severe chronic lung disease; severe renal disease) or anatomical criteria (high cervical lesion; lesion below clavicle, prior radical neck surgery or radiation; CEA restenosis; contralateral carotid occlusion; tracheotomy; contralateral laryngeal nerve palsy) (2).

Follow-up. All patients received a follow-up visit at 1 month. Clinical examination assessed overall general conditions, neurological signs and symptoms, medications, hospitalizations, or any type of complication that occurred after the procedure (13).

Statistics. Nominal and categorical variables were presented as contingency tables with frequencies and percentages. Continuous variables were reported as the mean with SD and compared by t test for normally distributed values (p < 0.05 was considered statistically significant). Proportions were compared by chi-square or Fisher exact tests. A 2-tailed p value <0.05 was considered statistically significant for superiority analysis, and a 1-tailed p value <0.05 was considered statistically significant for noninferiority analysis. Through the program SPSS version 16.1 (SPSS Inc., Chicago, Illinois), we built a general linear model using the 9 outcomes as logistic binary variables and all the following variables as the predictors: experience level, high surgical risk, sex, age >80 years, smoking history, hypertension, diabetes, low-density lipoprotein >100 mg, critical stenosis of the ipsilateral external carotid artery, symptomatic status, and type of stent design (16).

Results

Patient demographic characteristics are presented in Table 1. The vast majority of patients were male, with a robust incidence of atherosclerosis risk factors. Concomitant coronary artery disease was present in 66.0% of the patients. Age >80 years was relatively frequent (9.3%), and almost 50% of patients were at high surgical risk (2). Almost one-quarter of the patients were symptomatic.

![Table 1 Patient Demographic Characteristics](image)

| Male | 71.5 (930) |
| Age (yrs) | 69.9 ± 7.6 |
| High surgical risk | 49.8 (648) |
| Anatomical criteria | 7.9 (135) |
| Medical comorbidities | 79.2 (967) |
| Both anatomical and medical | 12.9 (84) |
| Age >80 yrs | 9.3 (121) |
| Smoking history | 69.6 (904) |
| Diabetes | 24.6 (319) |
| Hypertension | 88.9 (1,156) |
| LDL >100 mg/dl | 76.2 (995) |
| History of CAD | 35.4 (460) |
| Presence of significant CAD | 66.0 (857) |
| Symptomatic patients | 27.8 (361) |

Date presented as % (n) or mean ± SD.
CAD = coronary artery disease; LDL = low-density lipoprotein.

![Table 2 Procedural Characteristics](image)

| Contralateral ICA stenosis >70% | 8.2 (107) |
| Contralateral ICA occlusion | 4.3 (56) |
| Ipsilateral ICA stenosis >70% | 7.2 (93) |
| Type of stent | |
| Stainless steel | 1.85 (24) |
| Open cells, nitinol | 58.70 (763) |
| Closed cells, nitinol | 5.23 (68) |
| Hybrid cells, nitinol | 34.22 (445) |
| Pre-dilation | 68.9 (896) |
| Post-dilation | 100 (1,300) |
| Procedural time (min) | 17.4 ± 7.8 |
| Occlusion time (s) | 198 ± 59 |
| Occlusion intolerance | 19.9 (257) |
| Immediate intolerance | 0.3 (4) |
| Mo.Ma size (F) | |
| 10 | 42.5 (553) |
| 9 | 26.6 (346) |
| 8 | 30.8 (401) |

Date presented as % (n) or mean ± SD.
ECA = external carotid artery; ICA = internal carotid artery.
Procedure-related characteristics are presented in Table 2. Severe contralateral disease was present in 12.5% of patients, and in 56 patients (4.6% of total), the contralateral ICA was occluded.

Table 2 indicates the use of different types of stent. In particular, stainless steel stents were used in 24 patients (1.8%), stents with open-cell design were used in 763 patients (58.7%), and stents with hybrid-cell design were used in 445 patients (34.2%).

Protection success was achieved in 99.7% of patients (n = 1,296). There was no difficulty in removing the deflated distal balloon from underneath the stent struts.

In 4 cases (0.3%), patients showed immediate intolerance to balloon occlusion. In 2 cases (0.15%), we decided to deflate the proximal balloon and use the Mo.Ma system as a guiding catheter. Briefly, the Mo.Ma was left in place with only the distal balloon inflated, and a filter was advanced in the internal carotid artery through the Mo.Ma working channel. Once the filter device was correctly placed distally to the lesion, stenting was completed under distal protection. In the other 2 patients (0.15%), the procedure was completed using the intermittent occlusion technique; the occlusion balloons were deflated and blood aspirated after each procedural step (pre-dilation, stent release, and post-dilation). In all these patients, procedural success was achieved, and none experienced any adverse events.

In no cases were the ECA or CCA diameters large enough that blood flow could not be blocked completely. In 7.2% of the cases, a critical stenosis of the ipsilateral ECA was present, but this did not preclude procedural success. In the presence of significant stenosis of the ECA, the balloon was inflated distal to the lesion.

The average time of endovascular occlusion was 198 ± 59 s (range 94 to 845 s).

Occlusion intolerance was observed in 257 patients (19.9%); in all cases, CAS could be concluded under cerebral protection. Despite the fact that 12.5% of the patients had a total occlusion or a critical stenosis of the contralateral ICA, the presence of this anatomical condition did not preclude protection success.

In most of these cases, symptoms of occlusion intolerance started after stent post-dilation, during blood aspiration. The average back pressure of the group of patients presenting with occlusion intolerance was lower than the corresponding average back pressure of the group of patients presenting with started after stent post-dilation, during blood aspiration. The did not preclude protection success.

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In most of these cases, symptoms of occlusion intolerance started after stent post-dilation, during blood aspiration. The average back pressure of the group of patients presenting with occlusion intolerance was lower than the corresponding patients tolerant to occlusion (42.9 ± 13.2 mm Hg vs. 66.0 ± 14.0 mm Hg; p < 0.001). Despite this, neurological symptoms resolved in each case within 20 min after restoration of the antegrade blood flow.

Filtered blood showed the presence of visible atherosclerotic debris in 33.4% of patients.

Technical and procedural success was achieved in all patients (100%). The primary end point occurred in 18 patients (1.38%) (Table 3).

During in-hospital stay, 2 patients died due to cardiovascular reasons: 1 patient had a pulmonary edema 4 h after the procedure and died due to ventricular fibrillation, and the other patient experienced cardiac arrest the day after the procedure. This patient had an acute coronary syndrome 4 h after the procedure because of an acute occlusion of the left anterior descending artery that was treated with a percutaneous coronary intervention. Unfortunately, because of a concomitant severe aortic stenosis (for which he was scheduled for transcatheter aortic valve implantation) and compromised left ventricular function, the patient died the day after.

One patient died due to a hemorrhagic stroke that occurred 4 h after the procedure. Another 2 patients died due to noncardiovascular reasons: 1 because of multiorgan failure triggered by post-procedural acute renal failure and 1 because of subarachnoid bleeding probably caused by anti-coagulation. During in-hospital stay, 5 patients had a minor stroke and 6 patients had a major, nonfatal stroke. The cumulative in-hospital incidence of death and stroke was 1.15%.

During the 30-day follow-up period, 2 additional patients died. One patient died due to a drug-resistant pneumonia, and the other patient died due to a contrast-induced nephropathy that resulted in acute renal failure.

During the 30-day follow-up period, 1 additional patient had a minor stroke; the patient presented with transient monocular blindness, and a computed tomography scan showed the presence of a new ischemic lesion in the visual cortex. Symptoms disappeared by the 30-day follow-up visit.

The incidence of events was higher in symptomatic patients than in asymptomatic patients (3.0% vs. 0.8%; p < 0.01). No difference was observed between patients with (1.78%) and without (1.44%) contralateral carotid artery occlusion (p = NS). The incidence of events was higher in patients at high surgical risk than in those at average surgical risk (1.85% vs. 1.07%; p < 0.05). No significant differences were observed among patients having anatomical criteria or medical comorbidities, or both, of high surgical risk (Fig. 1).

The general linear model showed that independent predictors of events were level of institutional experience and symptomatic status, both in hospital and at 30 days of follow-up. From level 1 to 3 of institutional experience, the in-hospital death and stroke incidence progressively decreased (level 1: 4%; level 2: 1.5%; level 3: 1.0%); 30-day stroke and death incidence similarly decreased (level 1: 4%; level 2: 1.5%; level 3: 1.2%). The higher the experience level, the lower the risk of in-hospital stroke (odds ratio [OR]:
and death and stroke (OR: 0.28; 95% CI: 0.14 to 0.59; p < 0.01) (Fig. 2); the effect is similar of institutional experience on cumulative (in-hospital plus 0 to 30 days) incidence of stroke (OR: 0.30; 95% CI: 0.13 to 0.67; p < 0.01), death (OR: 0.30; 95% CI: 0.11 to 0.82; p < 0.05), and death and stroke (OR: 0.30; 95% CI: 0.16 to 0.67; p < 0.01) (Fig. 2).

Asymptomatic patients had a lower risk of in-hospital (OR: 4.25; 95% CI: 1.28 to 14.09; p < 0.05) and 30-day stroke (OR: 2.32; 95% CI: 1.73 to 17.97; p < 0.01). Similarly, asymptomatic patients had a lower risk of in-hospital cumulative events (OR: 3.56; 95% CI: 1.23 to 10.27; p < 0.05) and 30-day cumulative events (OR: 1.52; 95% CI: 0.33 to 6.28; p < 0.01) (Fig. 2).

Hypertensive condition (concomitant antihypertension medications or blood pressure >140/90 mm Hg at hospital admission) was also shown to be an independent predictor of 30-day stroke (OR: 0.27; 95% CI: 0.08 to 0.91; p < 0.05) and death and stroke (OR: 0.34; 95% CI: 0.12 to 0.94; p < 0.05) (Fig. 2).

In all patients, access site hemostasis was obtained by manual compression. Despite the use of 8- to 10-F introducer sheaths, only 1 patient required a femoral percutaneous transluminal angioplasty for an occlusive dissection. The overall incidence of femoral artery pseudoaneurysm was 1.3%; all were managed by manual compression. Surgical repair of a pseudoaneurysm or blood transfusions were never necessary.

Discussion

The results of this registry confirm that PEO is a safe and effective neuroprotection system during CAS. It allowed us to obtain an excellent rate of procedural success with a low incidence of death and stroke at 30 days, both in selective and consecutive patients. Even the presence of historical anatomical contraindications to PEO use (i.e., contralateral occlusions) did not increase incidence of post-procedural events. Patients at high surgical risk had a greater incidence of events, even though this condition was not an independent events predictor in the general linear model.

In this registry, the PEO, which combines the functions of a working sheath and cerebral protection in a single system, was successfully positioned in all patients. Only 19.1% of patients were intolerant to blood flow arrest. The incidence of occlusion intolerance in this registry is higher than the range described (5–7). A clinical explanation for this could not be
identified, but it could be related to the robust presence of patients with contralateral carotid artery occlusion, the elevated number of patients with age >80 years, and the significant presence of medical comorbidities. However, occlusion intolerance did not interfere with procedural success.

In 2 cases, because of the immediate onset of occlusion intolerance, we had to use a distal filter through the sheath of the Mo.Ma device, thus confirming that the PEO does not preclude the use of alternative or adjunctive methods of distal protection (7). In the other 2 cases of immediate intolerance, we adopted the intermittent occlusion technique. Adverse events were not observed in these 4 patients. Therefore, intolerance to proximal protection systems can easily be overcome in order to obtain a successful and "protected" stent procedure without complications.

There were no device-related complications at the balloon inflation sites or difficulties in retrieving the device with deflated balloons. Despite the use of 8- to 10-F sheaths, the incidence of access site complications in the present registry was low (2.3%); percutaneous transluminal angioplasty of the femoral artery was necessary only in 1 case (0.09 %), and in no case was surgical repair or blood transfusion required.

Described limitations of proximal protection devices are the presence of a critical stenosis or extremely large diameter of the ECA. In this study, the presence of a critical stenosis of the ECA did not preclude procedural success, and in no cases was the ECA diameter so large that blood flow could not be arrested after distal balloon inflation.

In contrast to reported multicenter studies, patients with contralateral ICA occlusion were not excluded from the study. This anatomical condition did not preclude procedural success and did not increase incidence of events both in-hospital and at 30 days of follow-up.

The observed in-hospital stroke/death rate (1.15%) (Table 3) confirmed the efficacy and safety of PEO. The origin of the embolic events leading to ischemic strokes, all documented by computed tomography, is uncertain. In all cases, the evaluation with color Doppler showed a patent stent.

Figure 2 General Linear Model of Predictors for In-Hospital and 30-Day Events After PEO-Protected CAS

(A) In-hospital stroke; (B) in-hospital death; (C) in-hospital death and stroke; (D) 30-day stroke; (E) 30-day death; (F) 30-day death and stroke (black circles, p = NS; red circles, p < 0.05). X-axis legend: 1 = stent design; 2 = experience level; 3 = octogenarians; 4 = high surgical risk; 5 = sex; 6 = smoking history; 7 = hypertension; 8 = diabetes; 9 = low-density lipoprotein >100 mg/dl; 10 = symptomatic; 11 = ipsilateral external carotid artery stenosis >70%. CAS = carotid artery stenting; PEO = proximal endovascular occlusion.
The 1.38% 30-day stroke/death rate in the present registry is comparable to that of other studies evaluating CAS with endovascular occlusion. The incidence of events according to symptomatic status is well below the limits set by guidelines (2).

Worthy of mention is the fact that the general linear model showed that level of institutional experience, symptomatic status, and hypertension are the only predictors for post-procedural events. Despite the fact that the operators fulfilled the appropriate criteria of training (10), the higher the institutional experience with PEO use, the lower is the risk of events. The hypertensive status showed to be a negative predictor of 30-day events. The clinical significance of this protective effect requires further investigation.

Finally, it must be noted that the present study included a first group of patients who are selected according to available inclusion and exclusion criteria and a second group of consecutively treated patients. The outcome of these 2 groups is similar, thus demonstrating that PEO can successfully be applied during CAS in the vast majority of patients.

Reprint requests and correspondence: Dr. Eugenio Stabile, Invasive Cardiology Laboratory, Cardiology Division, Clinica Montevergine, Via Mario Malzoni 1, Mercogliano 83013, Italy. E-mail: geko50@hotmail.com.

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