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# First Clinical Experiences with an Endovascular Clamping System for Neuroprotection During Carotid Stenting

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***Objectives***. *This report evaluates the feasibility and efficacy of an endovascular blood flow blockage technique to prevent intracerebral embolization of plaque debris during carotid artery stenting.*
***Methods***. *Forty-two patients were enrolled in five clinical sites in Germany and Italy with either an asymptomatic internal carotid artery stenosis* $\geq$*75% (mean 87%) or a symptomatic stenosis* $\geq$*60% (mean 85%). Cerebral protection during the stenting procedure was achieved using an endovascular clamping technique, obtained by occlusion of the external and common carotid artery via two independently inflatable balloons integrated in the Mo.Ma® system. Blood with particulate plaque debris was aspirated before flow was restored. The patient's clinical and the neurological status were assessed during intervention, at discharge, and at 3 months follow-up.*
***Results***. *Stenting was performed in all but one patient. The mean flow occlusion time was* $10.6 \pm 6.5$ *min. Transient clamping intolerance was observed in five patients (12%). In two patients, neurological deficits persisted for 2 and 12 h, respectively. Two minor strokes (4,7%) occurred at 5 and 72 h after the procedure. No major strokes or deaths were observed at 3 months follow-up.*
***Conclusions***. *This first clinical experience with the Mo.Ma® device substantiates the feasibility of endovascular clamping in preventing cerebral embolization during carotid artery stenting.*

*Keywords: Carotid arteries; Carotid artery stenting; Cerebral protection device; Endovascular clamping.*

## Introduction

Cerebral protection devices currently used for carotid artery stenting are based on either balloon induced distal blockage of the ICA blood flow, filters deployed distally to the stenosis or proximal balloon occlusion of both external (ECA) and common carotid artery (CCA). The novel Mo.Ma® device emulates a surgical clamping technique by stagnating blood flow during the procedure and aspirating particulate debris before the restoration of blood flow during and/or at the end of the procedure. Here, we report the first clinical experience with this embolic protection system investigating its feasibility and safety.

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## Materials and Methods

### *Device*

The Mo.Ma® device (Invatec s.r.l., Roncadelle (BS), Italy), now CE marked, integrates the functional aspects of cerebral protection and guiding catheters in a single system. The basic component of the system is a 100 cm long catheter with a 2.1 mm (0.081 in.) central lumen that is wide enough for the insertion of interventional devices as well as the effective aspiration of particles. The exit port of the working channel is located between two independently inflatable low pressure compliant balloons (Fig. 1) that permit endovascular clamping of both the CCA ($\leq$13 mm) and the ECA ($\leq$6 mm). Antegrade blood flow is blocked before a guide wire is advanced across the lesion. Any particulate debris generated during wire advancement, stent advancement, pre-dilatation, stent deployment, or post-dilatation remains stagnate in the

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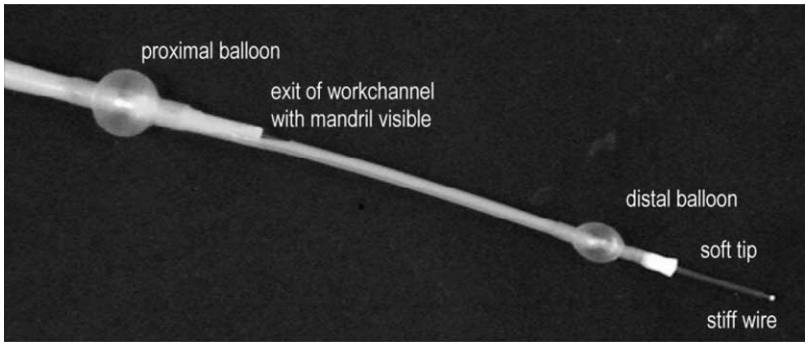


**Fig. 1.** The endovascular clamping device (Mo.Ma®, Invatec s.r.l., Roncadelle (BS), Italy).

vessel until it is aspirated prior to the restoration of blood flow.

*Patients*

Forty-two patients (30 males, 12 females) were enrolled in five clinical sites in Germany and Italy (Leipzig 20, Frankfurt 10, Neunkirchen 6, Mirano 4, Modena 2). The inclusion criteria were the presence of an asymptomatic ICA stenosis $\geq$75% or a symptomatic stenosis $\geq$60%. Patients were excluded from the study if they presented with a total occlusion of the contralateral ICA, had a stroke or acute myocardial infarction within 2 weeks prior to the intervention, had additional relevant lesions of the ECA and proximal CCA, presented with ICA stenoses after radiation or induced by an external tumor, had a known allergy to contrast media, renal insufficiency (Creatine $>$ 2.5 mg/dl), documented coagulopathy, or had vessel anatomy that precluded an endoluminal approach. The local ethics committee at each participating site approved the study and all patients gave informed consent.

*Intervention*

Patients received clopidogrel (300 mg) the day before the procedure and aspirin (500 mg IV) immediately before intervention if they were not on clopidogrel (75 mg/d) and aspirin (100 mg/d) already for at least 10 days. Diagnostic angiography with a 5 F catheter was performed to validate the ICA stenosis. A soft angled, coated guidewire and the diagnostic catheter were then gently navigated into the ECA. The soft guidewire was exchanged for a stiff 0.035 in. wire and the 5 F sheath was replaced by an 11 F, 25 cm long sheath. 10,000 IU heparin were administered. The Mo.Ma® system was advanced over the stiff wire until the radiopaque marker for the distal balloon was located in the ECA, around 0.5–1 cm beyond the bifurcation and proximal to or at the thyroidal branch (Fig. 2(A)). The distal ECA balloon was inflated to

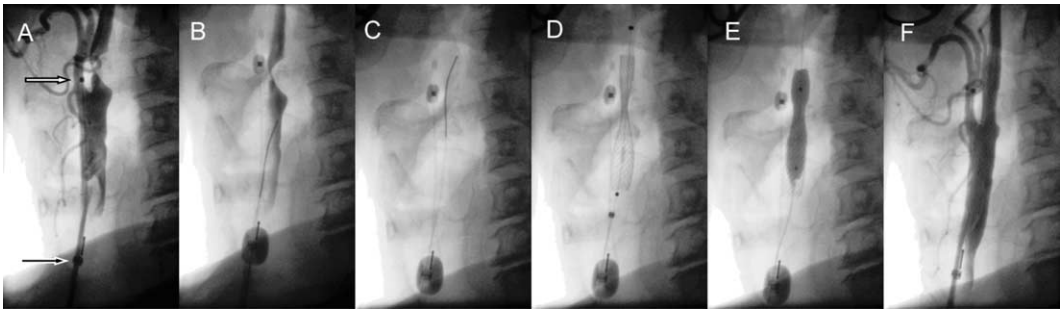


**Fig. 2.** Angiogram frames during carotid stenting using the Mo.Ma® system. (A) device *in situ*. Markers of proximal balloon (black arrow) visible in the CCA and the distal balloon (white arrow) visible in the ECA at the level of the ICA stenosis. (B) Dye stagnation after balloon inflation. (C) advancement of the 0.014 in. guide wire under protection. (D) Primary stent deployment (Carotid Wallstent 9$\times$30 mm). (E) Post-dilatation with a 6 mm balloon. (F) Final angiogram with balloons deflated after aspiration of blood.

anchor the system thereby avoiding accidental movement. A small valve at the tip of the system prevented reverse flow from the ECA as the stiff guide wire was removed. After the proximal balloon was inflated, a few millilitre contrast media was injected to verify the effective blockage of the antegrade flow (Fig. 2(B)). Immediately after documentation of flow blockage, the blood-dye mixture was aspirated through the working channel. Blood pressure in the CCA proximal to the lesion ('stump pressure'), maintained predominantly by the contralateral perfusion, was carefully monitored. A 0.014 in. guidewire was navigated through the lesion (Fig. 2(C)). Atropine was administered before a balloon or stent entered the ICA. Following pre-dilatation with a 4 mm balloon, an appropriately sized, self expanding stent (Carotid Wallstent®, Boston Scientific Inc. or Acculink®, Guidant Inc.) was deployed (Fig. 2(D)). Post-dilatation was performed with a 4–7 mm×20 mm balloon (Submarine Rapido®, Invatec s.r.l. or Viva®, Cordis) depending on vessel size (Fig. 2(E)). Intermittent aspiration after pre-dilatation and stent deployment was performed at the discretion of the operator. In cases where the patient developed neurological signs of intolerance, the procedure was performed in a step by step manner. After pre-dilatation or stent deployment, followed by blood aspiration to remove particulate, blood flow was restored until symptoms normalized. Clamping was then re-established for the remaining steps. In all cases, at least 40 ml blood was aspirated with a standard syringe before restoration of flow. The aspirates were filtered through a sieve to collect the plaque debris. After deflation of the ECA balloon, followed by the CCA balloon, the final result was documented (Fig. 2(F)). An intracranial angiogram in two perpendicular planes concluded the procedure. The Mo.Ma. system was then removed under fluoroscopy. The patient's neurological status was monitored throughout the procedure by continuous communication and testing motor function. The groin was closed by compression over 4 h. In cases of neurological deficits during or after procedure the patients underwent serial CT or MRA scans and neurological examinations.

### *Follow up*

The post-procedural hemodynamic and neurological status was carefully controlled. Repeat examinations according to the NIH Stroke Scale and Duplex ultrasound of the treated vessel were performed before discharge, 30 days after the procedure and at 3 months follow-up. The patients were discharged on clopidogrel (75 mg/d for 1 month) and continuous aspirin (100 mg/d).

## Results

### *Lesion characteristics*

The ratio of left to right sided lesions was 22/20. Forty-one (97.6%) lesions were *de novo* and one patient presented restenosis after endarterectomy. The mean lesion length was $14.3 \pm 6.0$ mm. The mean stenosis diameter was $85.1 \pm 8.0\%$ by visual estimate of angiogram according to ACCAS criteria (ratio between diameter of distal ACI and diseased area in the worst projection). In 11 patients (26.2%), a history of neurological symptoms was correlated to the target lesion. 13 patients (30.9%) had a significant stenosis (> 50%) of the contralateral ICA.

### *Procedure outcome*

The Mo.Ma® device was successfully used as intended and stenting of the stenosed ICA was performed in all but one patient. In this case, the complex anatomy of the arch prevented the stiff wire from advancing into the right ECA, a precondition to navigate the Mo.Ma® system into position. A Carotid Wallstent® was used in 38 cases (90.5%) and an Acculink® stent was used in three cases (7.1%). The mean time of flow blockage (clamping time) was $10.6 \pm 6.5$ min. Particulate debris was grossly visible in the filtered aspirate in 32 of the 41 completed procedures (76.1%). The mean duration of the total procedure was $51.0 \pm 28.3$ min. No minor or major strokes were observed during the procedure or as long the patients were in the vascular suite. Transient neurological deficits ('clamping intolerance') were observed in three patients (7.2%) who presented with symptoms ranging from limb weakness to total loss of consciousness in one patient during aspiration after post-dilatation. In two (4.8%) patients neurological deficits occurred during intervention not reversible within 20 min. These two events, patients suffered from facial paresis that lasted for 2 h and brachiofacial paresis that resolved after 12 h, respectively, appear to be of multifactorial causes and may be classified as TIA's. The serial CT-scan in these patients displayed no new lesions.

In one case, a minor stroke, likely embolic, occurred 5 h after stenting of an asymptomatic left ICA. The patient developed right-sided hemiparesis. The patient was discharged after 14 days with minor impairments persisting at 3 months follow-up (NIHSSS = 2). Another patient manifested distal ischemic symptoms on the punctured right leg 1 day after carotid stenting. Transbrachial angiography confirmed the suspected occlusion of the distal SFA

in the area of a previously implanted stent. The second day after local thrombolysis with rtPA, the patient developed frontolobal intracranial bleeding as demonstrated by CT scan. The hemorrhage was evacuated surgically and the patient recovered completely (NIHSSS = 0!) with only distinct changes in a sensitive personality test at 3 months follow-up. Patients were discharged after median of 2 days (range 2–14 days).

*Follow up*

All patients completed the 30 days as well as the 3 months follow-up without any further event.

## Discussion

The concept of endovascular clamping has unique technical advantages over distal balloon occlusion or filter protection devices used for cerebral protection during carotid artery stenting. First, endovascular clamping provides neuroprotection before the stenosis is 'touched' by any device, including the wire. Second, after deploying the stent, using the Mo.Ma® system, as much blood can be aspirated as is necessary to remove all particulate debris. A third advantage of endovascular clamping may be that full stagnation of blood flow is more easily confirmed and more stable than the precise, coaxial alignment of filters to the vessel wall. In our experience, the Mo.Ma® system was effective even in cases where the superior thyroid artery was not excluded by the distal balloon. In these cases, the back-flow from the superior thyroid artery did not cause washout of contrast dye. In sporadic procedures controlled by transcranial Doppler no high intensity transitory signals (HITS) were registered during flow blockage, even in cases where the superior thyroid artery was not covered. In addition, proximal occlusion with the Mo.Ma® system can be used in cases where an ICA with severe distal tortuosity precludes the safe insertion of distal protection devices.

The 'Parodi anti-embolism system',[9] shares the principle of occluding the ECA and CCA by balloons. However, there are distinct differences between the Mo.Ma® system and the 'Parodi anti-embolism system.' First, the Mo.Ma® device is a single sheath placed in a single advance over the stiff wire. Second, the Mo.Ma® system does not rely on the establishment of flow reversal, which is not possible to sustain throughout the entire procedure, as during stent advancement, placement, delivery, and post-dilatation. Further, flow blockage is easily confirmed by dye injection and complete blood aspiration is clearly visible.

Although this study was not powered to show equivalence to any other protection device, its complication rate is congruent with other studies investigating the impact of embolic protection devices on the adverse neurological event rate (Table 1).

One major concern with endovascular clamping, and shared with all occlusive devices, is the potential occurrence of 'clamping intolerance', or transient ischemic deficits, during flow blockage. In this study 'clamping intolerance' appeared in five patients (12%). In all of these cases the measured stump pressure, maintained by the collateral circulation, was below 35 mmHg. In two patients, which have to be classified as TIA's, with persisting neurological deficits (2 and 12 h), the systolic blood pressure had dropped below 60 mmHg after stent deployment and post-dilatation. This indicated that the most probable reason for the persisting deficits was the stimulation of the carotid

**Table 1. Impact of embolic protection devices on the adverse event rate**

| First author | Ref no. | Proc [*n*] | Protection devices used | Procedural stroke/death rate (%) | 30 Days major stroke/death rate (%) | 30 Days all stroke/death rate (%) |
|---|---|---|---|---|---|---|
| Henry | 1 | 53 | Guardwire® | 1.8 (retinal) | | |
| Parodi | 2 | 46 | Three different | 2.2 | | |
| Reimers | 3 | 88 | Three different filters | 1.2 | 0 | 1.2 |
| Al-Mubarak | 4 | 164 | Neuroshield®, Mednova® | | | 2.0 |
| Schlueter | 5 | 102 | Guardwire® | 3.1 | | |
| Castriota | 6 | 275 | Different filters | 1.3 | | |
| Macdonald | 7 | 150 | Neuroshield® | 2.7 | 2.7 | 4.0 |
| Adami | 8 | 30 | PAES | 0 | 0 | 0 |
| Cremonesi | 9 | 442 | Different | 1.1 | | 3.4 |
| Reimers | 10 | 753 | Different filters and occlusive devices | 2.2 | 1.2 | 3.3 |
| This study | | 42 | Mo.Ma® | 0 | 0 | 4.7 |

sinus followed by cardiovascular depression and not embolization of plaque debris.

The occurrence of 'clamping intolerance' may be minimized by administration of dopamine, if necessary, and by staging of the procedure where flow is restored after each step followed by aspiration.

This first clinical experience demonstrates that the Mo.Ma system is feasible, can be placed and retrieved safely, followed by successful stenting and the effective aspiration of particulate debris. Furthermore, the promising concept of endovascular clamping may be effective in the prevention of cerebral embolization during carotid artery stenting. Large controlled trials are required to confirm this preliminary result.

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