Carotid Artery Stenting Protected With an Emboli Containment System

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Background and Purpose—Fear of distal embolization and stroke has aroused concern regarding carotid stenting. Devices to protect the cerebral circulation may make carotid stenting safer.

Methods—A multidisciplinary study group tested a balloon occlusion-aspiration emboli entrapment device in conjunction with carotid stenting. The device consists of an elastomeric balloon on a steerable wire with a detachable adapter that inflates and deflates the distal temporary occlusion balloon. An aspiration catheter is used to remove trapped emboli after stenting and before occlusion balloon deflation.

Results—Seventy-five patients with severe internal carotid artery stenosis were treated with stents deployed with this cerebrovasculature protection system. All 75 patients (100%) had grossly visible particulate material aspirated, and all were treated successfully without major or minor stroke or death at 30 days. Preintervention stenosis was 81±10%, and residual stenosis was 5±7%. Nine patients (12%) had angiographic evidence of thrombus before intervention, but no patient had thrombus or vessel cutoff after the procedure. Four patients (5%) developed transient neurological symptoms during protection balloon occlusion, but symptoms resolved with balloon deflation. The 22 to 667 particles aspirated per patient ranged from 3.6 to 5262 μm in maximum diameter (mean, 203±256 μm). These particles included fibrous plaque debris, lipid or cholesterol vacuoles, and calcific plaque fragments.

Conclusions—Protected carotid stenting was performed successfully and safely in this study early in the experience with cerebrovascular protection devices. Particulate emboli are frequent with stenting, and cerebral protection will likely be necessary to minimize stroke. Randomized trials comparing protected carotid stenting with endarterectomy are warranted. **(*Stroke*. 2002;33:1308-1314.)**

Key Words: carotid artery diseases ■ carotid endarterectomy ■ stents ■ stroke

Carotid artery atherosclerosis is a major cause of disabling stroke and death. Compared with medical therapy, surgical endarterectomy has been proven to decrease stroke in both symptomatic[1–3] and asymptomatic[4] patients with severe stenosis. However, carotid plaque is friable, and stroke can occur despite meticulous technique and placement of intraoperative shunts.[5,6] In addition, patients with carotid disease are frequently elderly with significant comorbid disease. Even in experienced centers, carotid endarterectomy has an acceptable but significant risk of perioperative arrhythmia, congestive failure, myocardial infarction, and death.

Because of the success of percutaneous stenting of atherosclerotic lesions in other arterial systems,[7–10] several centers have investigated the less invasive alternative of stenting severe carotid stenoses.[11–16] However, fear of distal embolization of plaque fragments to the brain has generated concern regarding the safety and wisdom of this approach, especially considering the established low risk and durability of endarterectomy.[17,18] The incidence of stroke generally reported with carotid stenting is 5% to 10%,[11–16] although 1 center reported stroke in 71% of an early series.[19] Transcranial Doppler studies suggest the universal occurrence of emboli associated with this procedure.[20] Therefore, most clinicians have avoided carotid stenting except in patients with high surgical risk or surgically inaccessible lesions.

Systems to guard against distal embolization during percutaneous intervention have recently been developed. The GuardWire (Percusurge Inc) emboli entrapment and aspiration system was tested in vitro, used in animals, and subsequently used in human saphenous vein graft interventions.[21–23] A randomized trial recently confirmed a 40% to 50% reduction in major complications during protected versus unprotected saphenous

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vein graft stenting.[24] The first protected carotid artery intervention cases with this system began in 1998.[25] A multidisciplinary group of physicians (a surgeon, an interventional neuroradiologist, 3 interventional radiologists, and 3 interventional cardiologists) treating patients with carotid disease agreed to evaluate this emboli containment and aspiration system in patients undergoing carotid artery stenting. These are the results of this investigation.

Methods

Study Population

Patients with symptomatic carotid artery lesions ≥60% by angiography or asymptomatic patients with lesions ≥70% were considered for this study. Patients with a stroke within 1 week and patients with 100% occlusion of the ipsilateral carotid artery were excluded. The study protocol was approved by the institutional review board at each institution, and the patient signed informed consent to undergo this investigational procedure.

Carotid Stenting Procedure

Patients were treated with aspirin 81 to 325 mg/d for at least 24 hours and underwent an independent neurological examination at baseline. The patient was taken to the angiographic laboratory with an intravenous line infusing. A 7F to 9F sheath or guiding catheter was placed in the common carotid artery according to standard techniques[11–16] after local anesthesia. The patient was anticoagulated with 50 to 100 U/kg heparin until the activated clotting time was >250 seconds. Angiograms were recorded in at least 2 projections. Intracranial views were also documented.

The cerebral protection system used consists of 3 components: an exchange length guidewire, a MicroSeal adapter, and a monorail aspiration catheter. The wire is a 0.014- or 0.018-in angioplasty-style wire with a segment of hollow nitinol hypotube. The distal wire segment is shapeable, radiopaque, and steerable. Just proximal to this platinum distal segment is a compliant latex balloon capable of occluding blood flow when inflated. The proximal end of the hypotube wire incorporates a moveable seal allowing inflation and deflation of the balloon via the detachable adapter.

After the distal tip is shaped, the guidewire is advanced through the guide catheter under fluoroscopic guidance. The wire is steered through the carotid stenosis and advanced at least 3 cm beyond the target lesion (Figure 1). The intended predilatation balloon, stent, and postdilatation balloon are all prepped and available. The first device to be used in the lesion (predilatation balloon or stent, at operator discretion) is passed over the wire into the distal guiding catheter. The MicroSeal adapter is then attached, and the latex balloon is inflated to 5.5-mm diameter. Contrast is injected to confirm complete occlusion of distal flow. If flow is still present, then an additional 0.5 cm^3 diluted contrast is added to enlarge the balloon until complete occlusion is achieved. The MicroSeal is then closed, and the adapter is removed. The predilatation balloon, if used, is passed across the lesion and inflated until any indentation of the balloon is resolved. The dilatation balloon is then deflated and removed. The stent is then placed over the guidewire and deployed in the lesion. The stent delivery system is removed, and the postdilatation balloon is then passed over the guidewire into the lesion. A single balloon inflation of 8 to 15 atm for 15 to 20 seconds is performed. The balloon is deflated and then removed. The aspiration catheter is placed up to the level of the distal occlusion balloon under fluoroscopy. Fifteen to 45 mL of blood is then aspirated from the area between the carotid artery bifurcation and the occlusion balloon. The aspiration catheter is removed. The occlusion balloon is then deflated, restoring antegrade flow.

Most procedures were completed with only 1 occlusion balloon inflation, aspiration, and deflation. In the first 3 patients and in 4 additional patients in the series, aspiration was performed and the occlusion balloon was deflated after 1 to 2 steps of a sequential procedure, ie, after predilatation followed by aspiration and occlusion balloon deflation, after stent placement followed by aspiration and occlusion balloon deflation, and after stent postdilatation followed by aspiration and occlusion balloon deflation. With the sequential approach, the occlusion time in each step could be minimized at operator discretion.

After final occlusion balloon deflation and removal, angiography was repeated, confirming adequate lumen diameter in at least 2 orthogonal views. Intracranial views were repeated to check for vessel cutoff or flow abnormality. The patient underwent frequent brief neurological assessments during the procedure (question and answers, hand and foot movement) and a detailed neurological evaluation at the end of the procedure.

The patient was taken to a recovery area where the sheath was removed when the activated clotting time was <170 seconds. The patient remained on bed rest for 6 to 9 hours. The following morning, a detailed neurological examination by an independent observer was repeated. Discharge medications included aspirin 325 mg/d indefinitely and ticlopidine 250 mg BID or clopidogrel 75 mg/d for at least 2 weeks. The patient returned at 30 days for a repeated independent neurological examination.

Particulate Analysis

Aspirated blood was injected from a syringe into a container through a 40-μm filter. The filter was rinsed by injection of 20 to 60 cm^3 saline and then immersed in either 10% buffered formaldehyde or 2.5% glutaraldehyde in cacodylate buffer. Particulate debris from 31 consecutive patients was sent to a core pathology laboratory for analysis (J.M.).

After 24-hour fixation, the mesh of the filters was carefully cut from its frame and mounted on glass slides, stained with toluidine blue 1%, and covered with a coverslip. Digitized black-and-white images were used for measurement. The Optimas Analysis Program, version 5.0, was used. Each of 4215 particles was manually selected, and the maximum diameter of the particles was measured. In 2000 particles, area was also measured. In 7 patients, particles floating in the fixative were centrifuged, and a smear was stained with hematoxylin and eosin.

Data Analysis

The primary end point of the study was the composite of death or nonfatal stroke at 30 days. Secondary end points were balloon occlusion intolerance, transient ischemic attack, arrhythmia, congestive heart failure, myocardial infarction, pulmonary complications extending hospitalization, and access site complications. The study was designed as a prospective registry to determine the feasibility and complications of protected carotid stenting. Data are reported as mean±SD. Investigators agreed to compile the data and analyze the results after the first 50 to 75 cases had been completed. An independent group of monitors audited and source verified the data at each institution before analysis.

Results

The first 75 consecutive eligible patients were enrolled at the participating centers. Baseline patient characteristics are included in Table 1. Of the 75 patients, 56% had previous transient hemispheric or ophthalmic symptoms. Twenty-three patients (31%) had significant contralateral or intracranial carotid artery disease. Lesion characteristics are listed in Table 2. Nine lesions had angiographic evidence of thrombus, and lesion length was >20 mm in 27 patients (36%).

Procedural details are compiled in Table 3. Four patients (5%) had balloon occlusion intolerance with transient neurological symptoms that resolved after balloon deflation. One of these intolerant patients had significant contralateral carotid stenosis. One patient had adjunctive thrombus aspiration with the AngioJet (Possis Inc). No angiographic evidence of vessel cutoff or flow abnormality was seen.

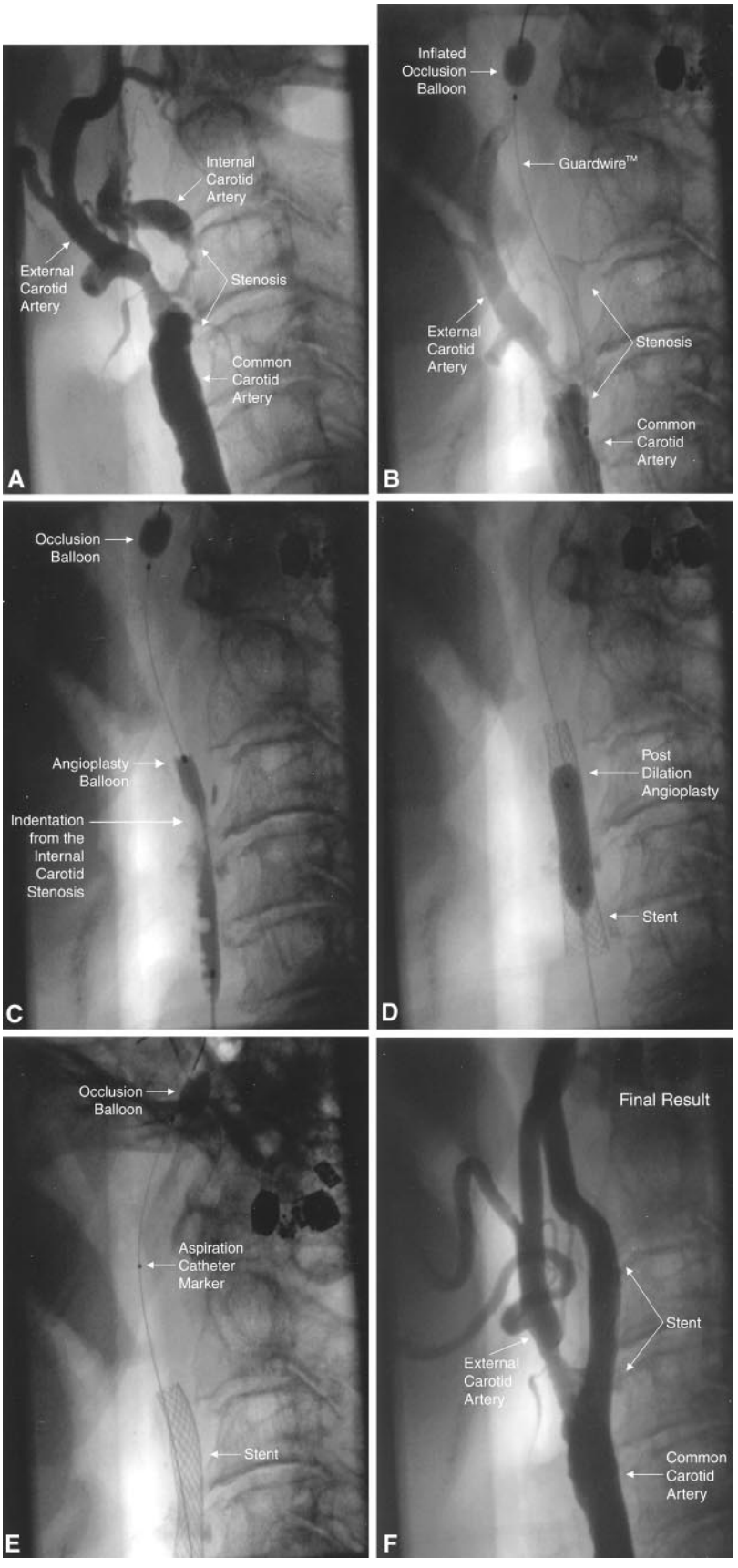

Figure 1. A, This preintervention angiogram shows a complex ulcerated plaque at the carotid bifurcation. B, Occlusion balloon is inflated in the internal carotid artery, blocking antegrade flow. C, Angioplasty is performed. D, Stent is deployed and dilated. E, Emboli trapped in the internal carotid artery are aspirated. F, Completion angiography after occlusion balloon deflation and removal.

TABLE 1. Baseline Patient Characteristics

	n	%
Age, y	67±10	
Men/women	54/21	72/28
Contralateral CVA	11	15
Ipsilateral CVA	10	13
Ipsilateral TIA	26	35
Ipsilateral retinal embolus	6	8
Symptomatic (within 4 mos)	42	56
Contralateral carotid stenosis 70% to 99%	19	25
Contralateral carotid 100% occlusion	4	5
Vertebrobasilar stenosis ≥70%	14	19
Angina	27	36
Congestive heart failure	12	16
Hypertension	52	69
Diabetes	25	33
Renal insufficiency (Cr >2.0 mg%)	2	3
Previous neck radiation	2	3
Previous ipsilateral carotid endarterectomy	5	7

CVA indicates cerebrovascular accident; TIA, transient ischemic attack; and Cr, creatinine.

Neurological examinations (immediately after the procedure and the next day) confirmed no evidence of stroke after the procedure. One patient with sustained hypotension (systolic blood pressure ≤70 mm Hg for 4 hours) and intracranial ipsilateral and contralateral carotid stenoses >70% had a transient ischemic attack resolved with restoration of systolic blood pressure >100 mm Hg. No unstable angina, ventricular arrhythmia, or myocardial infarction occurred. One patient developed pulmonary edema after being given a rapid infusion of saline for relative hypotension (systolic blood pressure, 90 to 100 mm Hg) immediately after stenting. This patient responded to diuresis and medical management. Three patients required transfusion for bleeding. Two of these patients required surgical femoral artery repair.

At 30 days, repeated neurological examination revealed no incidence of cerebrovascular accident, and no patient reported symptoms consistent with transient ischemic attack after hospital discharge. No patients died or were rehospitalized at 30 days.

Particulate debris was visible in all aspiration samples (Figure 2). The number of particles analyzed per patient ranged from 22 to 667. The mean maximum diameter was 203±256 μm. Particles ranged from 3.6- to 5262-μm maximum diameter. Forty-one percent of particles were <100 μm in maximum dimension, 64% were <200 μm, and 35% were 200 to 1000 μm. Particles ranged from 41.2 to 5157 μm^2 in area. The mean area of the particles was 279.4 μm^2.

On microscopic examination, these particles consisted of lipid vacuoles with cholesterol clefts surrounded by fibrin and platelets, fibrous eosinophilic staining material consistent

TABLE 2. Carotid Artery Lesion Characteristics

Right/left, n (%)	34/41 (45/55)
Lesion length, mm	18.4±10.9
Diameter stenosis, %	80.8±11.1
Ulceration >5 mm, n (%)	40 (53)
Thrombus, n (%)	9 (12)
Calcification, n (%)	46 (64)

TABLE 3. Procedural Data and 30-Day Complications

Procedural data	
Multiple balloon occlusions/aspiration sequences, n (%)	7 (9)
Single balloon occlusion/aspiration sequence, n (%)	68 (91)
Continuous occlusion time,*, min	15.3±5.5 (3.1–25.2)
Segmented occlusion times, min	
First occlusion	7.4±3.0
Second occlusion	7.8±5.1
Predilation, n (%)	60 (80)
Maximum predilation balloon size, mm	4.1±0.5
Maximum predilation pressure, atm	8.9±2.0
Stent(s) used, n (%)	
Wallstent	60 (80)
Palmaz	2 (3)
SMART	2 (3)
Other	11 (15)
Postdilation balloon size, mm	5.8±0.5
Postdilation maximum balloon pressure, atm	10.5±2.1
Residual stenosis, %	5.4±6.9
Dissection, n (%)	4 (5)
Dissection requiring second stent, lesion site	3 (4)
Guardwire dissection requiring additional stent	1 (1)
Intracranial vessel cutoff, n	0
Procedural and 30-d complications	
Occlusion balloon intolerance, n (%)	4 (5)
Symptoms (resolved after balloon deflation)	Transient dysarthria
	Loss of consciousness, focal seizure
	Blurred vision
	Confusion
Bradycardia requiring medication, n (%)	8 (11)
Bradycardia requiring temporary pacing, n (%)	5 (7)
Hypotension requiring pressor, n (%)	4 (5)
Transient ischemic attack, n (%)	1 (1)
Minor CVA (NIHSS <4), n (%)	0 (0)
Major CVA, n (%)	0 (0)
Death, n (%)	0 (0)
Myocardial infarction, n (%)	0 (0)
Congestive heart failure, n (%)	1 (1)
Bleeding requiring transfusion, n (%)	3 (4)
Surgical wound closure, n (%)	2 (3)
Hospitalization after procedure (mean), d	2.7±1.5

CVA indicates cerebrovascular accident; NIHSS, National Institute of Health Stroke Scale score.

*Minimum and maximum values are given in parentheses.

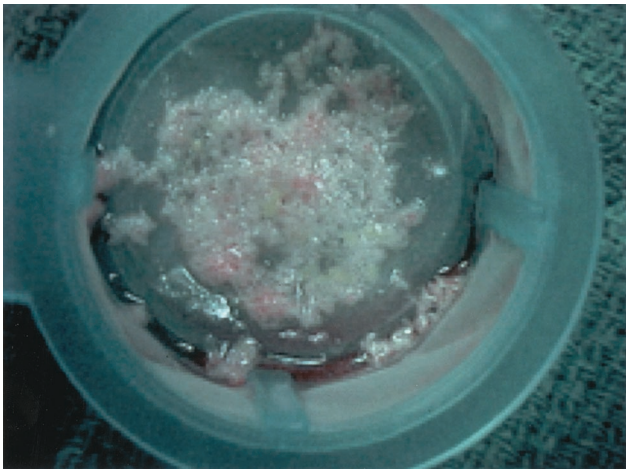

Figure 2. Aspirated particles from a patient with restenosis after a prior endarterectomy that were collected and rinsed with saline on a 40-μm filter.

with connective tissue from the cap of a plaque, calcific plaque fragments, lymphocyte clusters, and platelet clumps (Figure 3).

Discussion

Fear of embolizing plaque fragments distally into the brain during carotid stenting has dampened enthusiasm for this technique, especially considering the established safety and efficacy of carotid endarterectomy. One small randomized study of carotid stenting compared with surgery[19] was prematurely terminated because of an unacceptable incidence of stroke with stenting (71%), although most clinical reports are more encouraging.[11–16] In a nonmonitored, voluntary registry of 2048 carotid stenting cases, the incidence of death and stroke was reported to be 5.8%.[15] Despite the absence of neurological deficits in most patients, transcranial Doppler monitoring suggests that emboli are a frequent sequelae of wire manipulation, balloon dilatation, and stenting. Plaque material embolized to the cerebrovasculature is likely the predominant cause of stroke with carotid stenting. The debris aspirated in every patient in this study supports this contention. The large maximum diameter of many particles (200 to 1000 μm in 35%) and the number of retrieved emboli are a cause for concern.

Several devices to trap emboli have been conceived and developed. Theron and colleagues reported their experience with the first cerebral protection device, but that device has not been universally adopted for carotid stenting because of its limited steerability and relatively large profile. This first-generation device also did not eliminate emboli or clinical complications.[26,27]

Since those pioneering efforts, emboli containment devices have evolved.[25] We examined and scrutinized the system tested in this study and found it encouraging enough to warrant a clinical trial with carotid stenting. The data collected are strikingly positive. We judged the 100% success rate and the elimination of stroke in 75 consecutive cases of protected carotid stenting to be a major clinical advance. The presence of visible particles aspirated from all patients and

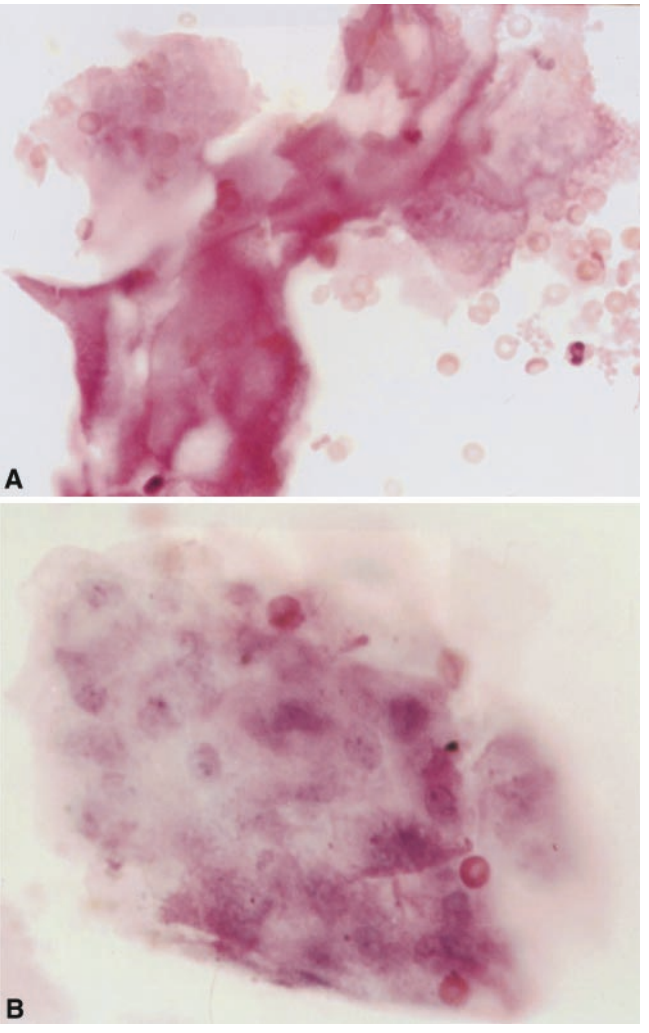

Figure 3. Microscopic view of a calcified particle (A) and a cluster of endothelial cells (B).

the absence of stroke as a complication support the use of an emboli containment system in cases of percutaneous carotid intervention. The ease of use of the balloon on a wire and its adaptability into the clinical practices of interventionists in vascular surgery, radiology, neuroradiology, and cardiology were established in this study.

The emboli containment device tested does have the limitation of not stopping distal embolization until the occlusion balloon is inflated. Crossing the lesion with this balloon on a wire does create transcranial Doppler signals suggesting microemboli, but the number of emboli counts is only a fraction of those found with unprotected stenting.[28] In addition, this device does transiently interrupt antegrade flow in the carotid artery. However, most patients (95% in this series) tolerate temporary balloon occlusion without symptoms, and all patients could be treated successfully with the serial inflation-deflation cycles used in those patients manifesting transient symptoms.

Diffusion-weighted MRI of the brain was not done in this study. However, sensitive imaging techniques are needed in future studies to determine whether subclinical emboli are prevented by this and other cerebral protection devices.

Likewise, diffusion-weighted MRI may be useful in determining whether prolonged occlusion times may cause subclinical defects. However, the long occlusion times used in this study did not cause clinical stroke.

Interestingly, significant contralateral carotid stenosis was not associated with balloon occlusion intolerance in any patient in this series. Online transcranial Doppler monitoring of middle cerebral artery flow velocity, used in only a small subset of patients in this series, might prove useful in predicting patients at high risk of balloon occlusion intolerance, but clinical parameters did not allow the operators to predict which patients might benefit from brief, sequential occlusion balloon cycles rather than 1 prolonged inflation.

Every new device used in percutaneous interventional procedures adds to the complexity and cost of the procedure. The cerebral protection system used in this study was added to the carotid stenting procedure without causing major clinical complications. However, the case involving a Guardwire balloon-induced dissection requiring an additional stent ≈4 to 5 cm above the carotid bifurcation illustrated that this device does have the potential for causing vascular complications even while limiting emboli to the brain. This complication, plus the price of the device ($1575 for the saphenous vein graft GuardWire System; Medtronic, Inc), underscores the importance of larger trials and cost-effectiveness analysis to determine the ultimate role of this new tool for the treatment of patients with carotid disease. The carotid protection device used in this study is not approved or available for routine clinical use in North America, but a similar device is approved and marketed for use in saphenous vein graft intervention.

Pathological analysis of the aspirated material suggests that emboli are an expected consequence of carotid stenting. The data imply that an effective, atraumatic protection device will be necessary to limit carotid stenting complications, possibly to the level expected with the established gold standard of carotid endarterectomy. Clearly, there will be further improvements in carotid stenting equipment and cerebrovascular protection devices over the next few years. However, the device studied in this trial has evolved sufficiently to warrant routine use as an adjunct to carotid stenting.

The emboli entrapment-aspiration system tested appears to provide adequate protection to the cerebral circulation to make carotid stenting a viable alternative to endarterectomy in symptomatic patients with high surgical risk, and perhaps even in asymptomatic patients who are routine surgical candidates. Randomized trials of protected carotid stenting versus carotid surgery should be expedited.

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