

David Fiorella, M.D., Ph.D.

Departments of Neuroradiology
and Neurosurgery,
Cleveland Clinic,
Cleveland, Ohio

Henry H. Woo, M.D.

Departments of Neuroradiology
and Neurosurgery,
Cleveland Clinic,
Cleveland, Ohio

Felipe C. Albuquerque, M.D.

Department of Neurosurgery,
Barrow Neurological Institute,
Phoenix, Arizona

Peter K. Nelson, M.D.

Departments of Neuroradiology
and Neurosurgery,
New York University
Medical Center,
New York, New York

Reprint requests:

David Fiorella, M.D., Ph.D.
Departments of Neuroradiology
and Neurosurgery,
Cleveland Clinic,
9500 Euclid Avenue, L10,
Cleveland, OH 44195.
Email: david.fiorella@Adelphia.net

Received, April 19, 2007.

Accepted, September 11, 2007.



ONLINE
DIGITAL
VIDEO

The Pipeline embolization device (PED; Chestnut Medical, Menlo Park, CA) is a flexible, microcatheter-delivered, self-expanding, endovascular “stent-like” construct engineered specifically for the treatment of cerebral aneurysms (Fig. 1). The device consists of a braided mesh cylinder composed of individual platinum and cobalt chromium microfilaments. When fully deployed, the implants are designed to provide approximately 30 to 35% metal surface coverage at nominal expansion, which is a much higher percentage of coverage than that provided by conventional (noncovered) intravascular stents. When delivered across the neck of an aneurysm, the device expands to oppose the walls of the parent vessel. Whereas conventional endovascular strategies for the treatment of cerebral aneurysms aim solely

DEFINITIVE RECONSTRUCTION OF CIRCUMFERENTIAL, FUSIFORM INTRACRANIAL ANEURYSMS WITH THE PIPELINE EMBOLIZATION DEVICE

OBJECTIVE: The Pipeline embolization device (PED; Chestnut Medical, Menlo Park, CA) is a new endovascular construct designed to exclude aneurysms from the parent cerebrovasculature. We report the results of the first two human implantations of this device in North America.

CLINICAL PRESENTATION: Two patients presenting with large, symptomatic, circumferential, fusiform intracranial vertebral artery aneurysms were treated with the PED. In both cases, more traditional open microneurosurgical and neuroendovascular treatment strategies had either failed or were associated with unacceptably high risk.

INTERVENTION: Three PEDs were placed across the aneurysms in each of the patients to achieve reconstruction of a new parent vessel through the center of a circumferential aneurysm. In the first patient, who had previously been treated with stent-supported coil embolization, the PED construct alone was sufficient to achieve parent vessel reconstruction and exclusion of the recurrent aneurysm. In the second patient, a microcatheter was jailed within the saccular portion of the aneurysm and the parent vessel was reconstructed with three telescoped PEDs. Although the PED construct dramatically reduced flow into the aneurysm, the lesion remained patent. Coiling of the saccular portion of the aneurysm was subsequently performed via the jailed microcatheter. Follow-up angiography performed 72 hours after the procedure demonstrated occlusion of the aneurysm with cylindrical reconstruction of the affected vascular segment. Neither patient has experienced any complication in the periprocedural period (30 d) or during subsequent long-term (>1 year) follow-up.

CONCLUSION: The PED represents an important advance in the endovascular therapy of cerebral aneurysms, targeting primary parent vessel reconstruction rather than endosaccular occlusion as a means by which to achieve exclusion of the aneurysm and definitive anatomic reconstruction of the parent artery.

KEY WORDS: Cerebral aneurysm, Fusiform, Reconstruction, Stent

Neurosurgery 62:1115–1121, 2008

DOI: 10.1227/01.NEU.0000313128.12325.14

www.neurosurgery-online.com

to obliterate the saccular portion of the aneurysms by filling them with an embolic material (e.g., coils, Onyx; Micro Therapeutics, Inc., Irvine, CA), the PED is designed to provide sufficient metal coverage across the neck of the aneurysm to physiologically exclude the lesion from the circulation. At the same time, the device is intended to be porous enough to preserve the patency of any branch vessels covered by the construct.

We report the results of the first human implantations of the PED for the treatment of complex, circumferential, fusiform aneurysms. Both cases were performed under a United States Food and Drug Administration exemption to allow the treatment of aneurysms that were without other acceptable treatment options.

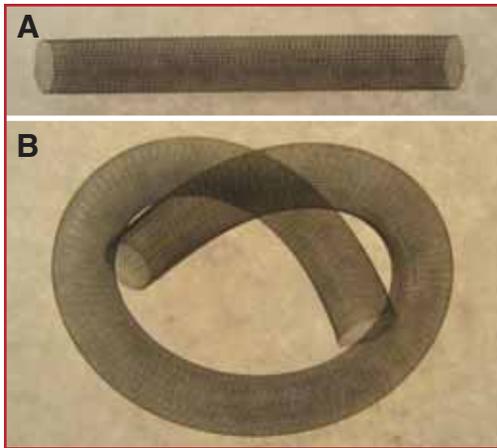


FIGURE 1. A, *ex vivo* image of the Pipeline embolization device (PED; Chestnut Medical, Menlo Park, CA), a flexible, microcatheter-delivered, self-expanding, endovascular stent-like construct engineered specifically for the treatment of cerebral aneurysms. The device is a braided, mesh cylinder composed of individual multialloy microfilaments. When fully deployed, the implants are designed to provide approximately 30 to 35% metal surface coverage at nominal expansion, a much higher percentage of coverage than that provided by conventional (noncovered) intravascular stents. When delivered across the neck of an aneurysm, the device expands to oppose the walls of the parent vessel. B, the PED is flexible enough to be manipulated into a “knot” configuration in its fully expanded state.

Case Reports

(see video at web site)

Patient 1

History and Previous Treatment. A 47-year-old man presented with severe headaches and neck pain. Computed tomography (CT) and CT angiography (CTA) revealed an 18-mm long fusiform aneurysm of the dominant left vertebral artery. The saccular component of the aneurysm measured 14 mm in width at its greatest diameter. The aneurysmal segment was positioned between the origins of the posterior inferior cerebellar artery proximally and the anterior spinal artery distally. The contralateral right vertebral artery was patent, but it was also small and nondominant. These CTA findings were verified by conventional angiography (Fig. 2A). Consideration was given to surgical and endovascular deconstruction of the aneurysmal segment as well as reconstructive endovascular treatment with stent-supported coil embolization.

For several reasons, including the diminutive size of the contralateral vertebral artery, the absence of significant anterior to posterior circulation collaterals, and the possibility that the aneurysm was the result of a spontaneous dissection and indicative of a diffuse underlying vasculopathy that could predispose the patient to dissection of the contralateral vertebral artery at some point in the future, a reconstructive treatment plan was pursued.

After an SL-10 microcatheter (Boston Scientific, Fremont, CA) was positioned within the saccular component of the aneurysm, a 4 × 28 mm Multilink Vision stent (Abbott Vascular, Abbott Park, IL) was deployed across the aneurysmal segment of the vertebral artery, trapping the

microcatheter within the aneurysm. A 4 × 20 mm Hyperglide balloon (EV3, Minneapolis, MN) was then manipulated into the lumen of the stent. Using a balloon-in-stent technique, coil embolization of the aneurysm was performed with the introduction of 188 cm of 0.010-inch Cerecyte embolization coils (Micrus Corp., Sunnyvale, CA). After coil embolization, partial occlusion of the aneurysm was achieved (Fig. 2B).

Angiographic follow-up performed 2 months later demonstrated partial remodeling of the aneurysmal segment with progressive thrombosis around the coil mass. For this reason, two additional Neuroform stents (4.5 × 30 mm and 4.5 × 15 mm, Boston Scientific) were placed within the coronary stent in an attempt to augment the degree of flow redirection and endovascular remodeling provided by the original coronary stent construct.

Angiographic follow-up performed 16 months after the initial treatment demonstrated that the previously visualized patent portions of the aneurysm were essentially unchanged. However, a new segment along the anterior wall of the aneurysmal segment demonstrated progressive aneurysmal dilation (Fig. 2C). The three-stent construct now precluded catheterization of this aneurysmal segment. Endovascular deconstruction of the aneurysm may have been difficult given the relatively short length of the segment between the posterior inferior cerebellar artery and the anterior spinal artery, which could be safely occluded by coils. Surgical deconstruction would have also been difficult due to the stent construct, which spanned the involved segment. For these reasons, it was decided to pursue treatment with the PED.

Pipeline Treatment. Twenty-four months after the original treatment was completed, PED reconstruction of the left vertebral artery was performed (Video 1). The patient was pretreated with aspirin and clopidogrel. Responsivity to both agents was confirmed with platelet aggregometry. After a Mass Transit microcatheter (Cordis Neurovascular, Warren, NJ) was manipulated across the indwelling stent construct over a 0.014-foot Transcend soft-tip microwire (Boston Scientific) (Fig. 2D), three telescoped PEDs were strategically overlapped across the aneurysmal segment to provide maximal metallic coverage of the aneurysm while limiting stent coverage of the anterior spinal artery near the vertebrobasilar junction. Serial angiography performed after placement of each of the three devices demonstrated progressively diminished flow into the aneurysm, leading to its abrupt occlusion after deployment of the third device (Video 1). Follow-up angiography performed 72 hours and 3 weeks after treatment confirmed stable exclusion of the aneurysm. The patient experienced no periprocedural (<30d) neurological complications.

Follow-up angiography performed at 4.5 months demonstrated stable exclusion of the aneurysm with only trace filling along the distal aspect of the aneurysm neck, which persisted late into the venous phase of angiography (Fig. 2G). All branch vessels covered by the PED remained patent (Fig. 2F). More than 12 months after treatment, the patient remained neurologically normal and without any neurological complications.

Patient 2

A 57-year-old man initially presented with acute onset of gait instability and left sided neck pain. At that time, magnetic resonance imaging, magnetic resonance angiography, and CTA revealed an acute left posterior inferior cerebellar artery territory infarction and mild focal irregularity of the left vertebral artery (Fig. 3A). The patient was treated with Coumadin (DuPont Pharmaceuticals, Wilmington, DE) for 3 months and was then converted to aspirin. After approximately 3 months, the patient returned to his neurological baseline. Eleven months after the initial episode, the patient experienced the acute onset of left hemibody numbness and paresthesias. Repeat magnetic resonance imaging revealed regions of chronic infarction but no new regions of

restricted diffusion. Magnetic resonance angiography and, later, CTA were performed, which both demonstrated that the right vertebral artery had completely occluded and that the focus of irregularity involving the V4 segment of the left vertebral artery had expanded into a 14×9 mm circumferential aneurysm. The length of the aneurysmal segment was 23 mm (Fig. 3B). Posterior communicating arteries were present bilaterally, but they were very small. These findings were confirmed by conventional angiography (Fig. 3C; Video 2).

Given the lack of adequate collateral circulation, deconstructive treatment options were not considered. Constructive treatment with conventional stent-supported coiling was determined unlikely to provide durable and complete occlusion of the aneurysm. For these reasons, parent vessel reconstruction with PED was pursued.

The patient was pretreated with aspirin and clopidogrel before the procedure. Responsivity to both agents was confirmed with platelet aggregometry. A high-flow Renegade catheter (Boston Scientific) was manipulated across the aneurysmal segment over a 0.014-inch Synchro microwire (Boston Scientific). An SL-10 microcatheter was then manipulated over the 0.014-inch Synchro microwire into the saccular component

of the aneurysm. Three telescoping PEDs were then strategically overlapped through the Renegade catheter, including one measuring 3.5×20 mm (distal), another measuring 4.25×20 mm (proximal), and finally one measuring 4×20 mm (central), to create a construct which spanned the aneurysmal V4 segment, creating a new prosthetic parent vessel through the central portion of the aneurysm and jailing the SL-10 microcatheter within the aneurysm. The saccular portion of the aneurysm was subsequently coiled with 101.5 cm of 0.010-inch Cerecyte embolization coils (Micrus Corporation, Sunnyvale, CA). Post-embolization angiography of the left vertebral artery demonstrated sluggish persistent filling through the construct and into the interstices between the embolization coils (Fig. 3, E and F; Video 2). However, a delayed angiogram performed 72 hours later, before discharge, demonstrated anatomic reconstruction of the parent artery with complete occlusion of the aneurysm (Fig. 3G; Video 2).

Follow-up angiography performed at 6 months (Fig. 3H) revealed stable anatomic reconstruction of the parent vessel, complete occlusion of the aneurysm, and no evidence of in-stent stenosis. The patient has experienced no periprocedural or delayed complications and remains neurologically normal more than 12 months after treatment.

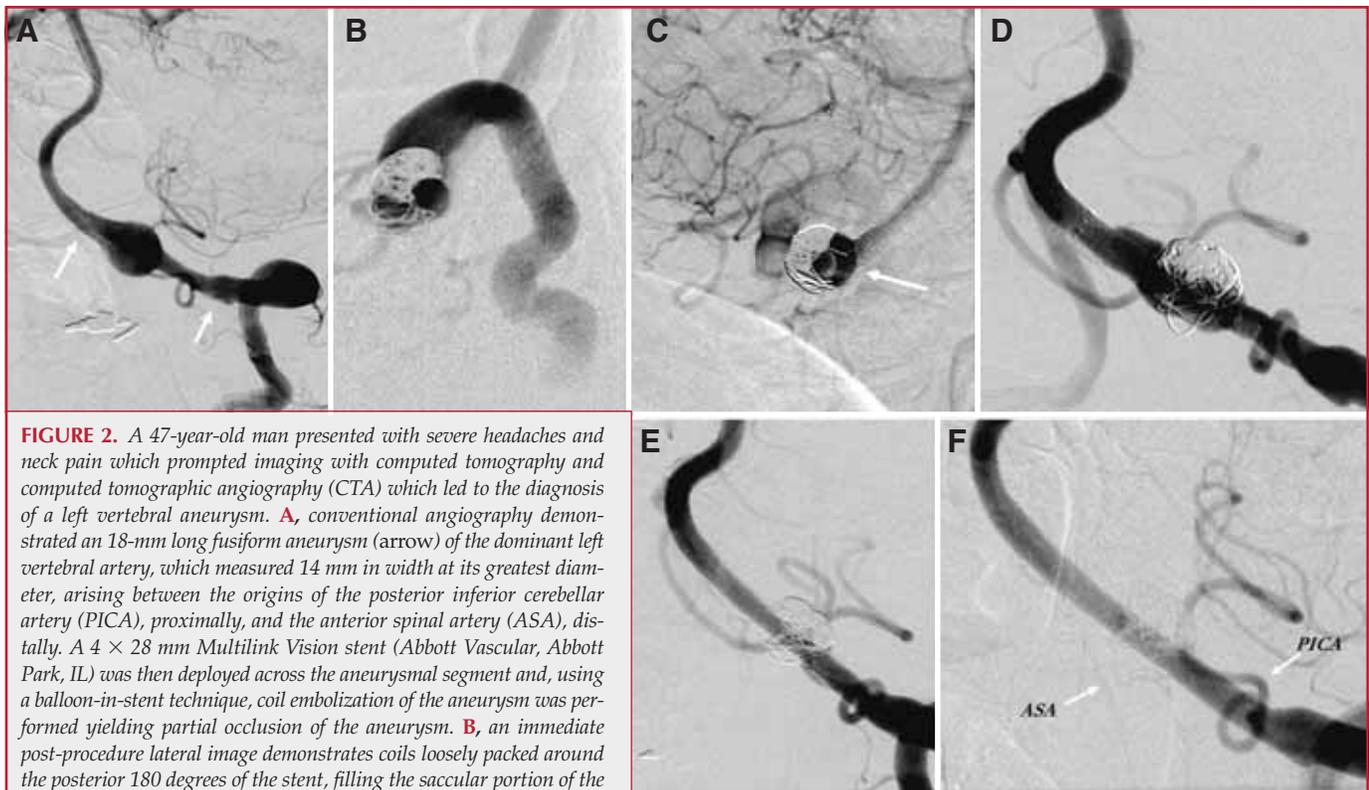


FIGURE 2. A 47-year-old man presented with severe headaches and neck pain which prompted imaging with computed tomography and computed tomographic angiography (CTA) which led to the diagnosis of a left vertebral aneurysm. **A**, conventional angiography demonstrated an 18-mm long fusiform aneurysm (arrow) of the dominant left vertebral artery, which measured 14 mm in width at its greatest diameter, arising between the origins of the posterior inferior cerebellar artery (PICA), proximally, and the anterior spinal artery (ASA), distally. A 4×28 mm Multilink Vision stent (Abbott Vascular, Abbott Park, IL) was then deployed across the aneurysmal segment and, using a balloon-in-stent technique, coil embolization of the aneurysm was performed yielding partial occlusion of the aneurysm. **B**, an immediate post-procedure lateral image demonstrates coils loosely packed around the posterior 180 degrees of the stent, filling the saccular portion of the fusiform aneurysm. Initial follow-up angiography at 2 months demonstrated progressive thrombosis with decreased filling within the coil mass; for this reason, two Neuroform stents (Boston Scientific, Fremont, CA) were placed across the aneurysm in an attempt to encourage further thrombosis. Follow-up angiography in the lateral (**C**) and posteroanterior (**D**) projections performed at 16 months demonstrated interval new expansion of a saccular component along the anterior wall of the aneurysm (**C**, arrow) despite the continued progressive occlusion along the posterior and inferior aspect of the aneurysm (**D**). Since the growing aneurysm along the anterior wall could not be accessed through the indwelling stents, reconstruction with the PED was performed. A Hi-Flow Renegade catheter (Boston Scientific) was positioned within the parent vessel across the stent-coil construct. Three PEDs were delivered through this catheter: one distally, one proximally, and one overlapping both constructs within the mid-distal portion of the diseased segment. **E**, following the deployment of three PEDs across the aneurysmal segment, the aneurysm was nearly completely occluded. On delayed imaging, trace filling was noted just proximal to the coil mass. **F**, angiographic follow-up at one year shows anatomical reconstruction of the previously aneurysmal segment. The PICA and ASA, both jailed by the PED construct remain patent. The patient remains without neurological symptoms now more than one year after treatment.

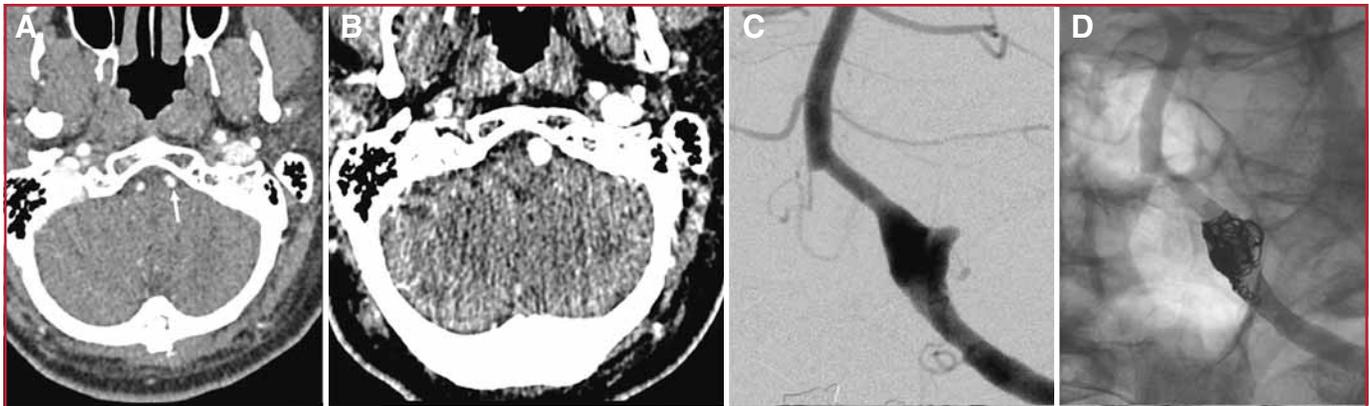


FIGURE 3. A 57-year-old male initially presented with acute onset of gait instability and left-sided neck pain. **A**, CTA evaluation at that time revealed an acute left PICA territory infarction and mild focal irregularity of the left vertebral artery (arrow). The patient was treated with Coumadin (DuPont Pharmaceuticals, Wilmington, DE) for 3 months and was then converted to aspirin. **B**, CTA source image from an exam performed 11 months later for recurrent symptoms demonstrated that the right vertebral artery had completely occluded and that the focus of irregularity involving the V4 segment of the left vertebral artery had expanded into a 14 × 9 mm circumferential aneurysm. **C**, conventional left vertebral angiography confirmed the occlusion of the right vertebral artery and interval growth of a circumferential, fusiform, large, left vertebral artery aneurysm, which involved a 23-mm long segment of the parent artery. Following parent vessel reconstruction with three PEDs, coil embolization of the saccular component of the aneurysm was performed through a microcatheter which had been jailed within the saccular component of the aneurysm (outside of the PED construct). **D**, native images demonstrate a coil mass distributed circumferentially about the PED construct. **E**, angiography immediately after coil embolization demonstrated markedly delayed flow-through into the aneurysm sac with stasis of contrast around the indwelling coil mass, which persisted on delayed imaging. **F**, follow-up angiography performed at 72 hours, demonstrated anatomical reconstruction of the involved segment with no residual filling of aneurysm. **G**, follow-up angiography at one year demonstrated a durable cure of the aneurysm. The patient remains neurologically normal more than one year after treatment.

DISCUSSION

Modern endovascular strategies for the treatment of cerebral aneurysms have rapidly evolved since the introduction of the Guglielmi detachable coil system (Boston Scientific/Target, Fremont, CA) in 1992 and the subsequent United States Food and Drug Administration approval of the device in 1995. Technological advances in coil design (e.g., three-dimensional coils, coils of varying stiffness), and methodological innovations (e.g., balloon remodeling technique) have facilitated the treatment of more complex aneurysms and simplified the treatment of all cerebral aneurysms.

In 1997, Higashida et al. (5) reported the application of an intravascular stent as an adjunctive device to support the endosaccular occlusion of a cerebral aneurysm. Five years later, the introduction of the Neuroform stent, the first stent specifically designed for use as an adjunct to support the coil embolization of wide-necked intracranial aneurysms, led to a marked increase in the number of aneurysms treated with a

stent-supported technique. Concurrently, other investigators began studying the impact of stents on flow dynamics in silicone aneurysm models and experimental animal sidewall aneurysms (1). These sentinel events stimulated a shift in focus from endosaccular aneurysm occlusion alone to strategies that incorporated intravascular stents, not only to support endosaccular occlusion, but also to achieve endoluminal remodeling of the diseased vascular segment (2–4).

The presence of an intravascular stent construct provides several theoretical advantages in addition to physically supporting the introduction of coils into the aneurysm. First, the stent construct across the aneurysm neck redirects flow along the normal course of the parent vessel, disrupting flow into the aneurysm (1–5). Second, the stent implantation may change the configuration of the parent vessel, changing the anatomy of the parent vessel-aneurysm complex and the aneurysm inflow zone. Third, implantation of the stent within the affected vessel ideally acts as a stimulus and provides a scaffolding to support neointimal overgrowth across the aneurysm neck defect,

thereby facilitating the biological remodeling of the deficient segment of the parent artery (4, 7).

The magnitude of the biological and, particularly, hemodynamic effects of the available intracranial stents on the aneurysm-parent vessel complex are somewhat mitigated by the limited metal surface area coverage provided by the available devices. For example, the Neuroform stent provides only 6.5 to 9.5% metal surface area coverage when deployed within an appropriately size matched vessel. Although this amount of coverage may be sufficient to improve the durability of aneurysm occlusion after coil embolization (2, 3) and may, in selected cases, induce the remodeling of very shallow blood blister-like and dissecting pseudoaneurysms (4), these stents do not represent a reliable stand-alone therapy for the majority of intracranial aneurysms which require dense coil packing of the aneurysm neck in order to provide a sufficient substrate for neointimal overgrowth of the aneurysm neck defect (8).

The PED represents an important advance of the endovascular remodeling strategy for aneurysm treatment. Whereas the predicate devices were designed primarily to achieve or support endosaccular aneurysm occlusion, the PED represents the first device designed primarily to hemodynamically exclude the aneurysm from the parent artery and to provide sufficient scaffolding to support neointimal repaving of the neck defect. The PED provides approximately 30% percent metal surface area coverage. The amount of coverage can be manipulated during deployment by maintaining forward pressure on the delivery wire and “packing” the device in the region of the aneurysm neck. In addition, multiple devices can be strategically overlapped or telescoped to further augment the coverage provided by a single device. Unlike a covered stent, the interstices between the strands that compose the PED are designed to allow enough flow through the construct to maintain the patency of both large branch vessels as well as small perforators arising along the course of the parent vessel bearing the aneurysm.

Kallmes et al. (6) recently studied the efficacy of the PED as a stand-alone device to induce the occlusion of experimental elastase-induced sidewall aneurysms in rabbits. Complete or near complete occlusion was achieved in 15 out of 17 experimental aneurysms after the placement of a single PED. Aneurysms harvested at 6 months demonstrated “thick neointima” growing over the PED struts and across the aneurysm neck. The PEDs were placed across the vertebral artery origin as well as within the rabbit aorta across the origin of the lumbar arteries. In all cases, these small branch vessels that had been covered by the PEDs remained patent at the time of long-term follow-up.

The PED strategy offers several significant advantages over existing technologies. First, as demonstrated by the current cases, the device provides a straightforward means by which to achieve definitive endovascular exclusion of aneurysms that would not otherwise be amenable to constructive treatment with other available technologies. Second, the deployment of the PED represents a technically easier strategy for the treatment of selected aneurysms than endosaccular approaches. The aneurysm itself, in some cases, may not have to be catheterized, essentially eliminating the risk of procedural rupture. In addition,

other complications related to the introduction and manipulation of multiple aneurysm coils (coil stretching or breaking, coil prolapse into the parent vessel, etc.) may be avoided. Third, complete aneurysm occlusion achieved with the PED should be more durable than endosaccular occlusion with coils. If the hemodynamic alteration induced by the placement of the construct results in the exclusion of the aneurysm during the initial treatment, the treatment should only become more durable with time as the construct undergoes endothelialization. In addition, the primary factors thought to stimulate aneurysm growth and recurrence (e.g., the “water-hammer” effect, wall shear stress, etc.) are likely disrupted, if not completely eliminated, when an aneurysm is angiographically excluded with the PED.

At the same time, use of the PED imposes several important limitations compared with conventional stent-supported coil embolization. First, due to its small pore size, placement of the PED precludes future coil embolization of the aneurysm sac. For this reason, and given the limited information regarding the efficacy of the PED as stand-alone therapy for various classes of aneurysms, if the aneurysm to be treated has a large saccular component, we recommend jailing a microcatheter during placement of the PED construct to facilitate coiling after the construct is in place, should this be necessary. Second, while effective in achieving the exclusion of side wall and circumferential aneurysms, it is not clear how effective the device would be to achieve embolization of bifurcation aneurysms. Third, the application of the device requires dual antiplatelet therapy, complicating its use in the setting of acute subarachnoid hemorrhage.

CONCLUSION

The PED represents an important advance in the evolution of devices available for the endovascular treatment of cerebral aneurysms. The PED treatment strategy signals a fundamental paradigm shift from predicate approaches designed to achieve endosaccular occlusion alone to one that elicits aneurysm exclusion as the end product of definitive parent vessel reconstruction. The PED provides a means by which to achieve safe, technically straightforward, and definitive treatment of aneurysms that have either failed or are not amenable to conventional treatment strategies.

Disclosure

Peter K. Nelson, M.D., is a stockholder and consultant for Chestnut Medical.

REFERENCES

1. Cantón G, Levy DI, Lasheras JC, Nelson PK: Flow changes caused by the sequential deployment of stents across the neck of sidewall cerebral aneurysms. *J Neurosurg* 103:891–902, 2005.
2. Fiorella D, Albuquerque FC, Han P, McDougall CG: Preliminary experience using the Neuroform stent for the treatment of cerebral aneurysms. *Neurosurgery* 54:6–17, 2004.
3. Fiorella D, Albuquerque FC, Deshmukh VR, McDougall CG: Usefulness of the Neuroform stent for the treatment of cerebral aneurysms: Results at initial (3–6-mo) follow-up. *Neurosurgery* 56:1191–1202, 2005.
4. Fiorella D, Albuquerque FC, Deshmukh VR, Woo HH, Rasmussen PA, Masaryk TJ, McDougall CG: Endovascular reconstruction with the

Neuroform stent as monotherapy for the treatment of uncoilable intradural pseudoaneurysms. *Neurosurgery* 59:291–300, 2006.

5. Higashida RT, Smith W, Gress D, Urwin R, Dowd CF, Balousek PA, Halbach VV: Intravascular stent and endovascular coil placement for a ruptured fusiform aneurysm of the basilar artery. Case report and review of the literature. *J Neurosurg* 87:944–949, 1997.
6. Kallmes DF, Ding YH, Dai D, Kadirvel R, Lewis DA, Cloft HJ: A new endoluminal, flow-disrupting device for treatment of saccular aneurysms. *Stroke* 38:2346–2352, 2007.
7. Lopes D, Sani S: Histological postmortem study of and internal carotid artery aneurysm treated with the Neuroform stent. *Neurosurgery* 56:E416, 2005.
8. Nelson PK, Sahlein D, Shapiro M, Becske T, Fitzsimmons BF, Huang P, Jafar JJ, Levy DI: Recent steps toward a reconstructive endovascular solution for the orphaned, complex-neck aneurysm. *Neurosurgery* 59 [Suppl 3]:S77–S92, 2006.

COMMENTS

A group of authors with extensive experience using intracranial stents for the treatment of intracranial aneurysms has reported the use of the Pipeline embolization device (PED) (Chestnut Medical, Menlo Park, CA) in two patients with fusiform aneurysms of the dominant vertebral artery. The PED, in conjunction with bioactive coils placed circumferentially around the PED, was effective in achieving endoluminal parent vessel reconstruction while excluding the aneurysm from the circulation. The high metal surface area coverage provided by the PED, augmented by the technique of overlapping three devices, probably provides a scaffold for endothelial repaving and flow redirection across the neck of the aneurysm.

The enthusiasm for this “quantum advance” in endovascular therapy must be tempered by the extremely short clinical (30 days) and angiographic (3 weeks) follow-up in these two patients, the paucity of human subjects in which it has been placed, the limited (potentially nonexistent) data from animal models, and other obvious considerations. The long-term patency and thromboembolic risk of the PED, especially when more than one is used in an overlapping and telescoping fashion as in these two patients, has yet to be determined. The optimal regimen and duration of antiplatelet therapy remain to be clarified. The other drawbacks, including limitations on the use of the PED in ruptured aneurysms because of the need for dual antiplatelet medication, is well discussed in the article.

Despite these potential concerns, I concur with the authors that the PED represents an extremely exciting and promising technology. The drift away from mere endosaccular occlusion of the aneurysm lumen (e.g., with deconstructive techniques) toward endoluminal parent vessel reconstruction is intuitive and encouraging.

Arun P. Amar
Stanford, California

The authors describe a new endovascular construct, the PED, to aid in the treatment of complex aneurysms. The authors used this stent in two patients with very complex fusiform aneurysms with impressive technical, angiographic, and clinical success. The follow-up is limited, and only two patients have been treated, but the 6-month results are encouraging. One factor confounding the results is the previous endovascular treatment in each patient. The concept of the PED with its higher metal-to-vessel ratio is attractive, but more evidence of aneurysm remodeling is needed. The authors’ resounding enthusiasm for this device as a way to avoid direct manipulation of the aneurysm sac is dampened with the disclaimer of microcatheterization of the saccular components of these aneurysms. This is important because the immediate technical disadvantage of this device is losing access to the

aneurysm once the PED is deployed. The PED has been designed with the intention that coiling is no longer necessary. There may be some reluctance to deploy the PED as a stand-alone construct until we see much more experience and the results of long-term follow-up. We will be eager to see the benefits and limitations of this device as this experience is gained.

Ramachandra P. Tummala
Minneapolis, Minnesota

L. Nelson Hopkins
Buffalo, New York

Since the original application of a Palmaz-Schatz balloon-expandable stent to treat a ruptured fusiform basilar artery aneurysm in 1997 (3), metallic stents have become an integral tool in the endovascular treatment of cerebral aneurysms. Boston Scientific was the first manufacturer to bring a dedicated cerebral stent, the Neuroform, to the commercial market in 2002. Over the past 5 years, more than 16,000 Neuroform stents have been applied under Food and Drug Administration guidelines. In May 2007, Johnson & Johnson Cordis brought its own version of a cerebral reconstruction device, Enterprise, to the commercial market with Food and Drug Administration humanitarian device exception approval. In fact, there are currently a number of different proprietary designs at various stages of development. These devices primarily provide structural support so that adjunctive devices such as coils or polymers can be used to fill the adjacent aneurysm. Now, the “Holy Grail” becomes vascular flow-remodeling. On the basis of experimental data, many investigators believe that cerebral aneurysms, in part, represent a response to deleterious shear stress, pathological blood flow patterns, in the vessel wall. Remodeling of cerebral blood flow may treat the underlying cause of some aneurysms. Experimental data also show that the Neuroform stent causes modest changes in blood flow within the stented artery and adjacent aneurysm. Moreover, layering additional stents adds a marginal reduction in intra-aneurysmal flow vortex and aneurysm wall shear stress (1, 2). Chestnut Medical’s PED represents an iterative step in the direction of greater stent surface area to augment vascular flow remodeling. Fiorella et al. suggest that the PED increases stent surface area to 30% (a 3- to 5-fold increase over the Neuroform) without causing the occlusion of adjacent parent artery perforator branch vessels. The authors have boldly applied the PED in two human cases. Their technical and treatment success with the absence of complications at 30 days bodes well for the next step in cerebral stent design and vascular flow remodeling.

Sean D. Lavine
Michael Chen
E. Sander Connolly, Jr.
Robert A. Solomon
Philip M. Meyers
New York, New York

1. Canton G, Levy DI, Lasheras JC: Hemodynamic changes due to stent placement in bifurcating intracranial aneurysms. *J Neurosurg* 103:146–155, 2005.
2. Canton G, Levy DI, Lasheras JC, Nelson PK: Flow changes caused by the sequential placement of stents across the neck of sidewall cerebral aneurysms. *J Neurosurg* 103:891–902, 2005.
3. Higashida RT, Smith W, Gress D, Urwin R, Dowd CF, Balousek PA, Halbach VV: Intravascular stent and endovascular coil placement for a ruptured fusiform aneurysm of the basilar artery. Case report and review of the literature. *J Neurosurg* 87:944–949, 1997.

The authors introduce a novel intravascular device (PED) for the treatment of intracranial aneurysms and present the results of the first two human implantations in the treatment of two fusiform aneurysms. This device does not function as previous devices in that it is not designed to provide support to keep material within an aneurysm dome; instead it appears to function by excluding the aneurysm from the circulation. This is a significant departure from the way we have viewed the intravascular treatment of aneurysms. If the PED is as effective with more traditional type lesions such as sidewall aneurysms, we may see a dramatic decrease in the complications associated with coiling aneurysms such as aneurysm rupture and coil

migration. The device should eliminate aneurysm remnants and aneurysm recanalization which we see with the current endovascular aneurysm strategies. Certainly, a trial will demonstrate the advantages of the PED as well as identify any of its limitations. The PED may represent an important step in how we treat intracranial aneurysms endovascularly, but additional studies will be needed to evaluate both its long-term safety and effectiveness, particularly for its use in other types of aneurysms.

Howard A. Riina
New York, New York

INFORMATION FOR SUBSCRIBERS

NEUROSURGERY (ISSN 0148-396x), a peer-reviewed journal, is a program of the Congress of Neurological Surgeons and is published monthly by Lippincott Williams & Wilkins, 16522 Hunters Green Parkway, Hagerstown, MD 21740-2116. Business offices are located at 530 Walnut Street, Philadelphia, PA 19106-3621. Periodical postage paid at Hagerstown, MD, and at additional mailing offices. Copyright © 2007 by the Congress of Neurological Surgeons.

POSTMASTER: Send address changes to **NEUROSURGERY**, P.O. Box 1550, Hagerstown, MD 21740.

CNS MEMBERSHIP SERVICES

Congress of Neurological Surgeons
10 North Martingale Road, Suite 190
Schaumburg, IL 60173

TEL: 847/240-2500; **FAX:** 847/240-0804

TOLL-FREE: 877/517-1CNS

EMAIL: info@1cns.org

EDITORIAL CORRESPONDENCE

Managing Editor, Rodrick A. Faccio

NEUROSURGERY

1420 San Pablo Street, PMB A-106

Los Angeles, CA 90033

TEL: 323/442-3001; **FAX:** 323/442-3002

EMAIL: neurosurgery-journal@hsc.usc.edu

ANNUAL SUBSCRIPTION RATES

UNITED STATES—\$496.00 Individual, \$715.00 Institution, \$247.00 In-training; **REST OF WORLD**—\$728.00 Individual, \$969.00 Institution, \$430.00 In-training; Single copy rate \$71.00. (All prices include a handling charge.)

Subscriptions outside of North America must add \$23.00 for airfreight delivery. United States residents of AL, CO, DC, FL, GA, HI, IA, ID, IN, KS, KY, LA, MD, MO, ND, NM, NV, PR, RI, SC, SD, UT, VT, WA, WV add state sales tax. The GST tax of 7% must be added to all orders shipped to Canada (Lippincott Williams & Wilkins' GST Identification #895524239, Publications Mail Agreement #617687). Subscription prices outside the United States must be prepaid. Prices subject to change without notice. (Visit us online at: www.lww.com)

Members of the Congress of Neurological Surgeons receive the journal as a benefit of membership.

Please indicate in-training status and name of institution. Institutional rates apply to libraries, hospitals, corporations, and partnerships of three or more individuals. Subscriptions will begin with currently available issue unless otherwise requested. Copies will be replaced without charge if the publisher receives a request within 90 days of the mailing date.

Individual and in-training subscription rates include print and access to the online version. Institutional rates are for print only; online subscriptions are available via Ovid. Institutions can choose to purchase a print and online subscription together for a discounted rate. Institutions that wish to purchase a print subscription, please contact Lippincott Williams & Wilkins, 16522 Hunters Green Parkway, Hagerstown, MD 21740-2116; **TEL:** 800/638-3030 (outside the United States 301/223-2300); **FAX:** 301/223-2400. Institutions that wish to purchase an online subscription or online with print, please contact the Ovid Regional Sales Office near you or visit: www.ovid.com/site/index.jsp and select Contact and Locations.

ADDRESS FOR NON-MEMBER SUBSCRIPTION INFORMATION, ORDERS, OR CHANGE OF ADDRESS: Lippincott Williams & Wilkins, P.O. Box 1580, Hagerstown, MD 21741-1580; **TEL:** 800/638-3030 (outside the United States 301/223-2300); **FAX:** 301/223-2400.

IN JAPAN, CONTACT: LWW Igaku-Shoin Ltd., 3-23-14 Hongo, Bunkyo-ku, Tokyo 113-0033; **TEL:** 81/3-5689-5400; **FAX:** 81/3-5689-5402.

IN BANGLADESH, INDIA, NEPAL, SRI LANKA, AND PAKISTAN, CONTACT: Globe Publications Pvt. B-13, 3rd Floor, A Block, Shopping Complex, Naraina Vihar, Ring Road, New Delhi 110028, India; **TEL:** 91/11-579-3211; **FAX:** 91/11-579-8876.

REPRINTS: Individual articles are available only from the authors. Reprints in large quantities, for commercial or academic use, may be purchased from the Publisher. For information and prices, call 800/341-2258.

BOUND VOLUMES FOR VOLUMES 58 & 59 will be available in early 2007 to subscribers only (U.S.—\$253.00; **FOREIGN**—\$271.00). Orders must be received by the Publisher before December 1, 2006.

DISCLAIMER

The statements and opinions contained in the articles of **NEUROSURGERY** are solely those of the individual authors and contributors and not of the CNS or LWW. The appearance of advertisements in the journal is not a warranty, endorsement, or approval of the products or their safety. The CNS and LWW disclaim responsibility for an injury or property resulting from any ideas or products referred to in the articles or advertisements.

INDEXING ABSTRACT SERVICES: The journal is currently included by the following services in print and/or electronic format: Index Medicus, Current Contents/Life Sciences, Clinical Medicine, and Science Citation Index.

COPYRIGHT & PERMISSIONS

NEUROSURGERY is copyrighted by the CNS. No portion(s) of the work(s) may be reproduced without written consent from LWW. Permission to reproduce copies of articles for noncommercial use may be obtained from the Copyright Clearance Center, 222 Rosewood Drive, Danvers, MA 01923; **TEL:** 978/750-8400. Copyright © 2007 by the Congress of Neurological Surgeons.