Stent-Assisted Reconstructive Endovascular Repair of Cranial Fusiform Atherosclerotic and Dissecting Aneurysms
Long-Term Clinical and Angiographic Follow-Up

Ajay K. Wakhloo, MD, PhD; Jake Mandell, MD; Matthew J. Gounis, PhD; Christopher Brooks, PAC; Italo Linfante, MD; Jesse Winer, MD; John P. Weaver, MD

Background and Purpose—The purpose of this study was to investigate the periprocedural morbidity, mortality, and long-term clinical and angiographic follow-up using stent-assisted coiling and stenting alone for treatment of cranial fusiform dissecting and atherosclerotic aneurysms.

Methods—The Institutional Review Board approved the study. A retrospective analysis was performed of 30 fusiform dissecting and atherosclerotic aneurysms treated in 28 patients (20 females; mean age, 52.6 years). Eleven aneurysms (37%) were located in the posterior circulation. Twenty-one (70%) originated from arterial dissection and 4 aneurysms (13%) presented with subarachnoid bleeding. Twenty-four (80%) aneurysms were treated with stents and coils, whereas 6 (20%) were treated with stents alone.

Results—Immediate postprocedural angiograms in 24 aneurysms treated with stent-assisted coiling showed complete occlusion in 12 and subtotal occlusion in 11 aneurysms, whereas no occlusion was seen in one aneurysm and in all 6 aneurysms treated with stents alone. A clinical improvement or stable outcome was achieved in 25 patients (89%). The 2 cases of permanent morbidity included a patient with a finger dysesthesia associated with a perforator stroke and another patient with hemiparesis and aphasia due to a delayed in-stent thrombosis. One patient died after treatment of a giant vertebrobasilar junction aneurysm. Angiographic follow-up was available in 23 of the 27 surviving patients (85%) at a mean of 16.2 months (range, 1 to 108 months). Recanalization in 4 patients (17%) at 3, 5, 24, and 36 months required retreatment in 3. In-stent stenosis of ≤50% was found in 3 patients.

Conclusion—Stent-assisted coil embolization is an attractive option for ruptured and nonruptured fusiform aneurysms with stable long-term outcome. However, recanalization observed up to 3 years after the initial obliteration emphasizes the need for long-term follow-up angiography. (Stroke. 2008;39:3288-3296.)

Key Words: dissecting aneurysm ■ fusiform aneurysms ■ intracranial aneurysms ■ new endovascular techniques ■ pseudoaneurysms ■ stent-assisted coiling ■ stents

Fusiform aneurysms of the intracranial circulation are rare, having an incidence less than 0.1%. They are significantly less common than saccular aneurysms but are more difficult to treat and have a devastating natural history with up to 80% mortality in 5 years if untreated. Fusiform aneurysms pose a challenge to surgical and endovascular treatment because (1) there is no defined neck to exclude from the parent artery; (2) critical perforating vessels frequently originate directly from the aneurysm; and (3) the location of the aneurysm is often nonamenable to surgical approach.

Patients with fusiform aneurysms commonly present with symptoms related to mass effect, eg, headaches, cranial nerve compression, paresis, or ataxia, ischemia secondary to dissection involving perforating branches or thromboembolic events, and less commonly, subarachnoid hemorrhage (SAH). The etiology of these aneurysms remains unclear. Both underlying atherosclerotic disease and recurrent arterial dissections secondary to hypertension have been implicated, causing pseudoaneurysms of a fusiform architecture.

Treatment of fusiform aneurysms by endovascular means is complex. Initially, endovascular approaches to fusiform aneurysms were limited to the “deconstructive” techniques of partial coil embolization, trapping, n-butylcyanoacrylate glue embolization, or parent artery occlusion after patient passing a balloon test occlusion. Clinical outcome has been dependent on characteristics and location of the aneurysm as well as presenting symptoms. Suitable patients who have adequate collateral circulation and tolerate balloon test occlusion with hypotensive challenge have reportedly a mortality rate as low
as 7%. However, mortality of the more treacherous vertebralbasilar dissecting aneurysms treated using these endovascular techniques has been reported at up to 20%.6

More recently, introduction of stents into the neurovascular system has enabled the preservation of the affected parent artery. This “reconstructive” technique is suggested to alter the intra-aneurysmal flow dynamics and lead to thrombus formation within the aneurysm.7–9 Stenting alone is best applied to smaller aneurysms.10 Within the past several years, however, stent-assisted coil embolization of complex and larger aneurysms is favored over endovascular parent artery occlusion, especially when balloon test occlusion fails and bypass surgery combined with vessel occlusion remains the only alternative.11–13 Using this technique, a single stent is first positioned inside the parent artery bridging the aneurysm. Multiple stents can also be overlapped in a telescopic fashion to form a larger structure. Detachable coils are then delivered into the aneurysm through the stent cells. The stent serves as a scaffold and prevents coil herniation into the parent vessel. Another commonly used method involves the initial placement of the microcatheter for coil delivery into the aneurysm and jailing of the catheter against the vessel wall by stenting (Figure 1). Despite early promise with these devices,14,15 no long-term angiographic and clinical follow-up are available.

In this study, multisite clinical experience and long-term angiographic and clinical follow-up using stenting only and stent-assisted coiling of fusiform-dissecting and atherosclerotic aneurysms are reported. Albeit of different etiology, both atherosclerotic and dissecting fusiform aneurysms were included because the parent artery reconstructive techniques of stenting or stent-assisted coiling are well suited to treat fusiform aneurysms of either origin.

**Materials and Methods**

**Patient Selection**

Between June 1996 and June 2007, stenting alone or stent-assisted coiling was used in the treatment of 30 aneurysms in 28 patients with atherosclerotic or dissecting aneurysms of fusiform architecture. An Institutional Review Board approval was obtained for the study from each university hospital where the endovascular procedures were conducted. Endovascular treatment was performed when it was considered the most appropriate treatment option after individualized evaluation by the vascular neurosurgeon and interventional neuroradiologist, taking into account collateral blood supply as determined by balloon test occlusion, aneurysm location and surgical accessibility, and patient preferences. Several patients were referred to us for endovascular treatment by experienced cerebrovascular neurosurgeons both within and outside the medical centers where the procedures were performed. Eight patients tolerated balloon test occlusion with the hypotensive challenge without any change in neurological status but were treated with stent-assisted coiling and preservation of the parent artery after careful evaluation by the treating team in consideration of each patient’s age and the potential of developing disease of the contralateral internal carotid artery. Stenting only was performed with dissecting sidewall aneurysms in suitable locations as proved effective in previous in vitro studies.16 Stent-assisted coiling was performed for all other fusiform aneurysms. A retrospective review was conducted of presenting clinical symptoms, angiographic features, MR studies if available, procedural studies, and clinical course as well as long-term angiographic and clinical follow-up.

There were 8 males and 20 females with an average age of 52.6 years (range, 29 to 75 years), the males being slightly older than the females (57.4 years versus 50.7 years; Table). Two patients had 2 separate fusiform aneurysms, which were treated in both cases. The most common clinical presentation was headaches (9 of 28 [32%]) and 4 aneurysms (13%) presented with SAH. Other symptoms included hemiparesis, hemiplegia, stroke, seizures, syncope, and

Figure 1. Case 17: A 59-year-old woman presented with severe headaches. A, Lateral angiogram of the left internal carotid artery (ICA) shows a giant fusiform aneurysm of the cavernous segment and a fetal origin of the posterior cerebral artery (double arrow). The patient failed a balloon test occlusion (not shown). Note previously coiled right ICA terminus aneurysm (arrowhead). B, Using the “jailing technique,” a micocatheter is placed into the aneurysm (arrow) and a stent (4 × 18-mm GFX coronary stent) is being deployed across the aneurysm (open arrow). C–D, Placement of coils around the stent; the cross-sectional view shows the reconstructed ICA and its involvement in the aneurysm (arrow). E–F, Posttreatment angiograms in oblique views demonstrate aneurysm filling, which progresses to obliteration at 60-month follow-up (G–H).
<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age/Sex</th>
<th>Presenting Symptoms</th>
<th>Aneurysm Location/Dissecting</th>
<th>Immediate Angiography</th>
<th>Long-Term Angiographic Follow-Up</th>
<th>Clinical Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Occlusion/Remnant</td>
<td>Months/Finding/Treatment</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>49/F</td>
<td>Growth of pseudoaneurysm</td>
<td>Petrous ICA/yes</td>
<td>&gt;90% Base</td>
<td>14/Mild coil compaction</td>
<td>14 months asymptomatic</td>
</tr>
<tr>
<td>2</td>
<td>43/F</td>
<td>Headaches, diplopia</td>
<td>Cavernous ICA/yes</td>
<td>&gt;90% Base</td>
<td>10/Stable, persistent base filling</td>
<td>10 months improving</td>
</tr>
<tr>
<td>3</td>
<td>48/F</td>
<td>Headaches</td>
<td>Paraclinoid ICA/no</td>
<td>100% None</td>
<td>3/No recanalization</td>
<td>9 months improving</td>
</tr>
<tr>
<td>4</td>
<td>55/M</td>
<td>Coil compaction of previous aneurysm</td>
<td>Intradural VA/yes</td>
<td>&gt;90% Base</td>
<td>5/No recanalization ≤50% ISS</td>
<td>9 months asymptomatic</td>
</tr>
<tr>
<td>5</td>
<td>36/F</td>
<td>Hemiplegia, hemisensory loss</td>
<td>Basilar trunk/yes</td>
<td>&gt;90% Dome</td>
<td>5/Small regrowth, healed dissection</td>
<td>156 months asymptomatic</td>
</tr>
<tr>
<td>6</td>
<td>57/F</td>
<td>Headaches</td>
<td>Paraceloid ICA/no</td>
<td>100% None</td>
<td>5/No recanalization</td>
<td>5 months asymptomatic</td>
</tr>
<tr>
<td>7</td>
<td>51/F</td>
<td>Headaches, diplopia</td>
<td>Cavernous ICA/yes</td>
<td>100% None</td>
<td>5/No recanalization</td>
<td>6 months asymptomatic</td>
</tr>
<tr>
<td>8</td>
<td>47/F</td>
<td>Transient quadriaparesis</td>
<td>Basilar trunk/yes</td>
<td>0% Dome</td>
<td>2/No recanalization ≤50% ISS</td>
<td>2 months asymptomatic</td>
</tr>
<tr>
<td>9</td>
<td>45/F</td>
<td>SAH</td>
<td>Paraceloid ICA/no</td>
<td>0% Dome</td>
<td>2/No recanalization</td>
<td>2 months asymptomatic</td>
</tr>
<tr>
<td>10</td>
<td>49/F</td>
<td>Headaches, neck pain</td>
<td>Basilar trunk/yes</td>
<td>&gt;90% Base</td>
<td>108/Small regrowth at 24 months</td>
<td>60 months finger hypesthesia</td>
</tr>
<tr>
<td>11</td>
<td>58/F</td>
<td>Neck pain</td>
<td>Petrous ICA/yes</td>
<td>0% Dome</td>
<td>6/No recanalization</td>
<td>6 months asymptomatic</td>
</tr>
<tr>
<td>12</td>
<td>29/M</td>
<td>SAH, headaches</td>
<td>Intradural VA/yes</td>
<td>0% Dome</td>
<td>14/No recanalization ≤50% ISS</td>
<td>14 months mild headache</td>
</tr>
<tr>
<td>13</td>
<td>75/M</td>
<td>Dizziness, gait disturbance</td>
<td>VB junction/no</td>
<td>&gt;90% Dome</td>
<td>60/Recanalization at 36 months; retreated at 40 months</td>
<td>66 months asymptomatic</td>
</tr>
<tr>
<td>14</td>
<td>39/F</td>
<td>SAH</td>
<td>Intradural VA/yes</td>
<td>100% None</td>
<td>36/No recanalization</td>
<td>36 months asymptomatic</td>
</tr>
<tr>
<td>15</td>
<td>58/F</td>
<td>Multiple ipsilateral strokes</td>
<td>Petrous ICA/yes</td>
<td>Express &gt;2/no</td>
<td>0/No recanalization</td>
<td>6 months asymptomatic</td>
</tr>
<tr>
<td>16</td>
<td>51/F</td>
<td>Headaches</td>
<td>Parapaliod ICA/none</td>
<td>100% None</td>
<td>6/No recanalization</td>
<td>18 months asymptomatic</td>
</tr>
<tr>
<td>17</td>
<td>59/F</td>
<td>Headaches</td>
<td>Cavernous ICA/yes</td>
<td>&gt;90% Dome</td>
<td>60/No recanalization</td>
<td>60 months asymptomatic</td>
</tr>
<tr>
<td>18</td>
<td>69/M</td>
<td>Ataxia</td>
<td>VB junction/no</td>
<td>30% Dome</td>
<td>6/No recanalization ≤50% ISS</td>
<td>60 months asymptomatic death 2 weeks postprocedure</td>
</tr>
<tr>
<td>19</td>
<td>66/M</td>
<td>Left hemispheric stroke from carotid bulb atherosclerotic occlusive disease</td>
<td>Basilar trunk/yes</td>
<td>100% None</td>
<td>8/No recanalization</td>
<td>13 months improving</td>
</tr>
<tr>
<td>20</td>
<td>72/M</td>
<td>Dizziness, gait disturbance</td>
<td>PICA/no/MCA bifurcation/yes</td>
<td>100% None</td>
<td>1/No recanalization</td>
<td>7 months mild headache</td>
</tr>
<tr>
<td>21</td>
<td>62/F</td>
<td>Syncopal episodes</td>
<td>M2–M3/yes</td>
<td>&gt;90% Dome</td>
<td>6.5/Mild coil movement</td>
<td>6.5 months asymptomatic</td>
</tr>
<tr>
<td>22</td>
<td>41/F</td>
<td>Seizures</td>
<td>MCA bifurcation/yes</td>
<td>&gt;90% Base</td>
<td>12/No recanalization</td>
<td>12 months asymptomatic</td>
</tr>
<tr>
<td>23</td>
<td>61/F</td>
<td>Incidental</td>
<td>M1/yes</td>
<td>100% None</td>
<td>11/No recanalization</td>
<td>11 months stroke improving</td>
</tr>
<tr>
<td>24</td>
<td>49/F</td>
<td>Headaches (history of trauma and coma)</td>
<td>P1/yes</td>
<td>100% None</td>
<td>7/No recanalization</td>
<td>7 months, no headaches</td>
</tr>
<tr>
<td>25</td>
<td>35/M</td>
<td>SAH</td>
<td>Paraceloid ICA/yes</td>
<td>&gt;90% Dome</td>
<td>3/Pseudoaneurysm formation at posterior aspect of aneurysm</td>
<td>15 months asymptomatic</td>
</tr>
<tr>
<td>26</td>
<td>71/F</td>
<td>Headaches, third nerve palsy</td>
<td>Cavernous ICA/yes</td>
<td>&gt;90% Base</td>
<td>6/No recanalization ≤10% ISS</td>
<td>6 months asymptomatic</td>
</tr>
<tr>
<td>27</td>
<td>58/M</td>
<td>Asymptomatic, angiography for ADPKD</td>
<td>Petrous ICA/yes</td>
<td>0% Dome</td>
<td>18/No recanalization</td>
<td>18 months asymptomatic</td>
</tr>
<tr>
<td>28</td>
<td>39/F</td>
<td>Headaches</td>
<td>Paraceloid ICA/yes</td>
<td>100% None</td>
<td>2/No recanalization</td>
<td>3 months asymptomatic</td>
</tr>
</tbody>
</table>

F indicates female; M, male; ADPKD, autosomal dominant polycystic kidney disease; ICA, internal carotid artery; VA, vertebral artery; PICA, posterior inferior cerebellar artery; MCA, middle cerebral artery; Pt, platinum; ISS, in-stent stenosis.
dizziness or gait disturbance. Eleven aneurysms (37%) were in the posterior circulation. More than two thirds (21 [70%]) of the aneurysms originated from arterial dissection.

**Aneurysm Treatment**

Patients were treated under general anesthesia using endovascular stenting or stent-assisted coiling techniques as previously described. In all cases, a stent was positioned into the lumen of the parent vessel either bridging the aneurysm or multiple stents were placed in a telescopic or Y configuration with reconstruction of the vessel comparable to an “endovascular bypass.” The stents available included coronary balloon-expandable stents and, more recently, the self-expanding neurovascular stents. Subsequently, the aneurysm was coiled as described previously with platinum coils (19 of 24), polymer-coated coils (4 of 24), or a combination of bare platinum and coated coils (one of 24). Immediate postprocedure angiograms were obtained and reviewed in multiple views, including standard lateral and anteroposterior projections.

Four patients (Cases 14, 19, 22, and 24) were treated in 2 stages. The first procedure included stenting followed by coil embolization within a few weeks. The remaining patients were treated in a single session.

The procedure was performed under therapeutic heparinization with activated clotting time of approximately 300 seconds. The patients were kept on heparin for at least 24 hours. In all procedures performed after 1999, patients were preloaded with 325 mg aspirin daily for 3 days before treatment and after 2001 were preloaded with the dual antiplatelet agents, 75 mg clopidogrel daily and 325 mg aspirin daily. If acute stenting was indicated, antiplatelet agents were given through the nasogastric tube. Postprocedure, the patients were kept on a dual antiplatelet regimen between 3 and 12 months.

**Clinical and Angiographic Outcome Measures**

Clinical data were obtained from examinations conducted by stroke neurologists or neurosurgeons before and after the procedure. Patients were re-evaluated generally within 30 days after the intervention. Long-term clinical evaluations were acquired in conjunction with the follow-up angiography. Technical success was defined by correct placement of the stent and successful positioning of the coils into the body of the aneurysm without compromising the parent vessel. Immediate postprocedure angiography measured aneurysm occlusion using a modification of the Raymond classification scale, which primarily was developed for berry-shaped aneurysms. In-stent stenosis, recanalization, need for retreatment, and patency of failed perforator arteries were evaluated on follow-up angiogram.

**Results**

**Postprocedural Angiographic and Clinical Outcome**

On postprocedural angiogram, 12 of 24 (50%) aneurysms treated with stent-assisted coiling were occluded without remnant. Eleven aneurysms showed subtotal occlusion. In one case, proper stent placement failed (Case 18). Of these 11 aneurysms with subtotal occlusion after treatment, 5 had residual dome filling (Raymond Class 3), whereas the other 6 had filling of a residual base (Raymond Class 2). In the 6 patients treated by stenting alone, contrast residual time within the aneurysm was increased moderately after the stent placement.

Excellent clinical and technical results were achieved in 23 of 28 patients (82%) without any periprocedural complications. Clinical adverse events occurred in 5 patients leading to 2 complications without any adverse outcome. There was one case of minor permanent morbidity (one of 28 [4%]), one case of major permanent morbidity (one of 28 [4%]), and one mortality (one of 28 [4%]).

In total, 25 patients (89%) retained no permanent deficits and returned to their baseline neurological examination. The 2 cases of resolved complications included one patient with a transient mild sixth cranial nerve palsy (Case 13), which resolved to baseline at follow-up visits. A wire perforation in another patient was observed during stenting that led to a SAH, which was controlled and did not have any clinical sequelae (Case 22). There were 4 patients (14%) who initially presented with SAH, none of whom experienced any periprocedural complications.

The 2 cases of permanent morbidity (one minor and one major) both involved infarctions. After placement of 2 overlapping stents into the basilar trunk, one patient (Case 10) had a minor stroke as documented by diffusion-weighted MRI associated with a small pontine perforator occlusion and retained permanent left fourth finger hypesthesia. An additional patient (Case 23) sustained a middle cerebral artery stroke 1 week after the procedure due to in-stent thrombosis despite dual antiplatelet therapy. Although the thrombosis was treated with tissue plasminogen activator, the patient retained some improving neurological deficit, including right leg weakness and partial expressive aphasia.

One patient (Case 18) died 2 weeks after discharge. The initial attempted endovascular treatment failed with secondary collapse of multiple stents (Neuroform I) placed in a telescopic fashion for the 30-mm giant and fusiform symptomatic vertebrobasilar junction aneurysm. Although an autopsy was not performed, it was assumed that the death was a result of the attempted treatment.

**Long-Term Angiographic and Clinical Outcome**

Of 27 patients who survived the intervention (96%), a follow-up angiogram was available in 23 patients (85%) at an average of 16.2 months (range, 1 to 108 months). These 23 patients had a total of 25 aneurysms. A total of 19 patients (83%), including Cases 20 and 24, each with 2 treated aneurysms, remained stable during the entire observation period (Table). None of the patients presenting initially with SAH experienced aneurysm rupture during the observation period. Four aneurysms in 4 patients (Cases 5, 10, 13, and 25) showed recanalization, all of which were treated initially with stent-assisted coiling. An additional patient received follow-up MR angiographic imaging at 11 months (Case 23), which showed no evidence of recanalization. Of the 6 aneurysms treated with stenting alone, follow-up angiography was obtained in 4 cases, which all demonstrated complete aneurysm occlusion without recanalization during the observation period. These included 3 petrous internal carotid artery dissecting aneurysms and one intradural vertebral artery dissecting aneurysm.

**Retreatment**

Of all 24 patients with follow-up imaging studies, 3 patients (13%) required retreatment. A patient with a dissecting basilar trunk aneurysm (Case 10) required recoiling and placement of a second stent 2 years after the initial procedure due to delayed growth of a small pseudoaneurysm proximal to the previously treated area (Figure 2). Another patient with a large atherosclerotic vertebrobasilar fusiform aneurysm
(Case #13; Figure 3) showed recanalization on 36-month follow-up angiogram. Successful recoiling was performed at 40 months through the original stent mesh; further follow-up at 20 months after the recoiling (60 months after the initial intervention) showed no further recanalization. In the third retreated patient, a complex dissecting paraophthalmic/paraclinoid internal carotid artery aneurysm (Case 25) involving the anterior choroidal artery that was initially treated by stent and coils showed 3-mm filling of a previously clotted pseudoaneurysm at the posterior–medial and superior aspect of the original dissection, which reached the terminal internal carotid artery on the 3-month follow-up angiogram. The origin of the anterior choroidal artery could be preserved during the recoiling with complete obliteration of the pseudoaneurysm at the posterior–medial and superior aspect of the fusiform dissection patent; no permanent neurological deficits were encountered. Of note, all patients requiring retreatment were treated initially with stent-assisted coiling and had angiographic evidence of aneurysm remnants immediately after the initial treatment. In an additional patient with an initial incompletely occluded M2/M3 middle cerebral artery dissecting aneurysm (Case 21), a reattempt at 6.5 months to obliterate the aneurysm through the existing stent struts failed. The follow-up angiography showed a crescent-shaped 1.5×2-mm calcified remnant associated with some coil rearrangement.

**In-Stent Stenosis**

Three patients with angiographic follow-up (3 of 23 [13%]) had angiographic evidence of in-stent stenosis at long-term follow-up; Case 4 had ≤50% stenosis at 5 months, Case 26 had <10% stenosis at 6 months, and Case 12 had ≥50% stenosis at 14 months. Case 12 was treated with stenting alone, whereas Cases 4 and 26 were treated with stent-assisted coiling. None of these patients were symptomatic and did not require retreatment for the stenosis. In both cases with ≥50% stenosis, the stent used was a coronary balloon-expandable stent, whereas a self-expanding stent was used in Case 26 with <10% stenosis.

**Jailed Perforators**

Of the 23 cases with angiographic follow-up, 22 (96%) did not have any symptoms from jailed perforator arteries. One patient (Case 10) had left fourth finger hypesthesia due to a small brainstem perforator occlusion confirmed by MRI diffusion-weighted imaging as previously discussed. Two patients had angiographic evidence of occlusions of named jailed arteries but remained asymptomatic. Although patent after stent-assisted coiling of a dissecting basilar trunk aneurysm, an occlusion of the anterior inferior cerebellar artery (AICA) was observed after placement of a second overlapping stent (Case 19). This patient did not present with any neurological sequelae and the AICA was patent on a 6-month follow-up angiogram. In a second patient (Case 13), the jailed right hypoplastic vertebral artery, patent at 1, 8, 16, and 36 months, was obliterated at the vertebrobasilar junction on the 40-month follow-up angiogram. The blood flow to the posterior inferior cerebellar artery and the anterior spinal artery was preserved. The patient currently remains asymptomatic at the time of this report, now with more than 5.5 years of clinical follow-up.

Figure 2. Case 10: A 49-year-old woman presented with severe headaches and neck pain. A, Right vertebral angiogram in a frontal view shows a large aneurysm of the basilar trunk with the area of dissection (arrow); note the AICA originating within the involved segment (open arrow). The aneurysm is stented (4×18-mm GFX coronary stent) and coiled with a small remnant (not shown). B, At 24-month follow-up, growth of a pseudoaneurysm proximal to the treated area. C–D, A second coronary stent (S7) is deployed in a telescopic fashion covering the previous stent. Coiling of the pseudoaneurysm is successfully accomplished with preservation of the AICA. A small area of the aneurysm still filled (arrow). Follow-up angiography at 108 months (not shown) and (E–F) at 108 months shows no coil compaction or aneurysm regrowth. Note the patency of basilar artery and right jailed anterior inferior cerebellar involved in the dissection.
Long-Term Clinical Outcome
A follow-up neurological examination was obtained in all 27 surviving patients with a range of 2 to 66 months (average, 15.4 months). Twenty-four of the 27 surviving patients (89%) had no new postprocedural deficit and showed significant improvement in their presenting symptoms. The patient with a delayed in-stent thrombosis 1 week after treatment (Case 23) and the patient with a minor brainstem perforator infarction (Case 10) were previously discussed. A third patient (Case 20) reported mild headaches that were not present as an initial symptom. Of note, mild headaches were present in 4 patients (15%) at clinical follow-up. Three of these 4 patients had chronic headaches as their presenting symptom.

Aneurysm Location
Of the 15 aneurysms in the internal carotid artery, follow-up angiography was available in 13 cases. One paraclinoid aneurysm showed recanalization (Case 25; one of 13 [8%]) and was retreated. All other aneurysms remained stable during the observation period. There were 4 aneurysms in the middle cerebral artery territory with long-term angiographic follow-up available in 3; none of those showed recanalization. The total recanalization rate in the anterior circulation was one in 16 (6%). Eleven aneurysms treated were in the posterior circulation. There was one death 2 weeks postprocedure (Case 18). Of the surviving patients, follow-up was available in 9 of 10 cases. Three of these aneurysms recanalized (6 of 9 [33%]) requiring retreatment in 2 cases.

Discussion
In our series of complex and surgically challenging fusiform and dissecting cranial aneurysms, stenting and stent-assisted coiling were feasible with an acceptably low periprocedural major morbidity (4%) and mortality (4%). Previously published series in which fusiform aneurysms of the vertebrobasilar system were repaired by endovascular techniques, including parent vessel sacrifice, have reported mortality between 0% and 20%.6,14,15 These data compare favorably to the natural history of the disease, which, in one study of 159
fusiform aneurysms with 719 patient-years of follow-up showed a mortality rate of 40%.19 Four patients (14%) presented with SAH, which is consistent with a previous report of a 17% incidence of SAH presentation in 120 fusiform aneurysms.20 Three of these 4 patients were completely asymptomatic after treatment on long-term follow-up, whereas the fourth patient reports mild headaches (this patient initially reported headaches before the SAH).

Stent-assisted coil embolization integrates 3 principles: (1) “endovascular bypass” with anatomic reconstruction of the diseased parent vessel; (2) mechanical support for coil embolization; and (3) flow diversion. In pseudoaneurysms, proper stent placement may help to reattach the dissected arterial tissue flap, thus closing off the false lumen. This may explain why all 4 of the dissection aneurysms (3 petrous carotid aneurysms and one intradural vertebral artery aneurysm) in which angiographic follow-up was available healed after stenting alone.

Currently available intracranial stents serve primarily as a mechanical buttress for coils and in wide neck and fusiform aneurysms help to define and remodel a new vessel wall. Because of their high porosity of approximately 80% to 85% free metal to metal ratio, these intracranial stents are not specifically designed to alter intra-aneurysmal hemodynamics.9,16,21 However, like in some of the patients treated in our series (Figure 1, Case 17; Figure 3, Case 13), the presence of the stent across the aneurysm may have modified the geometry of the parent artery/aneurysm complex and/or the blood flow into the aneurysm favorably enough to induce thrombus formation. Newer stent-like devices are being developed for intracranial aneurysms to serve as flow diverters within the parent artery.7–9,16,21–23

Recanalization
Among other current problems with intracranial stents such as stent movement, thromboembolic phenomena, dual antiplatelet treatment in ruptured aneurysms, and in-stent stenosis, long-term durability of the stented and coiled aneurysm remains a challenge and has not been studied.22,24–28 Aneurysms larger than 10 mm have reportedly the highest incidence of recanalization,29 most likely due to the neck size, low coil packing density,18,30 larger intra-aneurysmal clot burden, and probably the underlying biology of these aneurysms. Approximately 8% to 80% of coiled aneurysms show recanalization at angiographic follow-up, depending primarily on the size and location of the aneurysm.5,24,31–33 In our case series of fusiform and dissecting aneurysm treated with stent or stent-assisted coiling, recanalization was observed in 4 patients and successful retreatment was accomplished in 4 cases. We found a higher recanalization rate with posterior circulation aneurysms (33% versus 6%). This may be related to the size of the aneurysm and clot burden along the inner wall of the aneurysm. Interestingly, a large posterior circulation dissecting aneurysm (Figure 3, Case 13), which after initial occlusion remained obliterated over several years, showed delayed recanalization. This may suggest that these aneurysms may be biologically active with delayed neovascularization. It also emphasizes the need for long-term angiographic studies and challenges the initial perception that stents, or stent-like devices which may serve as flow diverters, may create stable aneurysm obliteration in larger aneurysms by thrombus formation alone.9,23

Histopathological studies have provided insight into the mechanism of recanalization.34–37 Neovascularization of the thrombus in giant aneurysms is considered to contribute to recanalization.38 Because the progression and organization of fresh clot into granulation tissue is necessary for long-term aneurysm stability when treated with coils,34 endovascular therapies are being developed to target both the reduction of neovascularization and the promotion of clot organization. Albeit the dissecting paracloidal internal carotid artery aneurysm in one patient (Case 25) was treated with Cerecyte “bioactive” coils and a stent, a recanalization was observed on the 3-month angiogram. A pseudoaneurysm had formed, which was occluded most likely by a thrombus on the initial angiogram.

Due to prevailing rupture risk, especially in dissecting aneurysms, retreatment may be required in case recanalization or regrowth of the pseudoaneurysm should occur. Our cases of dissecting and atherosclerotic fusiform aneurysms illustrate the need for both early (earlier than 3 months) and late (as late as 3 years or greater) angiographic follow-ups to rule out potential growth or regrowth of the (pseudo)aneurysm within the diseased arterial segment. In addition to development of “impervious endovascular bypass grafts,” the transformation of a clot within the aneurysm to a biologically silent scar tissue is a compelling concept but remains a challenge.

In-Stent Stenosis and Perforator Patency
The incidence of in-stent stenosis is likely related to neointimal growth as has been previously suggested,39 which can be secondary to endothelial injury. Endothelial cells play an integral role in the regulation of smooth muscle growth, and when that regulation is lost, neointimal proliferation can be seen that leads to stenosis. Unlike coronary balloon-mounted stents, self-expanding intracranial stents such as the Enterprise (Cordis Neurovascular J&J, Miami Lakes, Fla) or the Neuroform (BSC, Natick, Mass) have low radial force and are less traumatic. In our series, 3 patients (13%) with angiographic follow-up presented with an asymptomatic in-stent stenosis. Two of these cases, both with angiographic follow-up presented with an asymptomatic in-stent stenosis. Two of these cases, both with asymptomatic stenosis before availability of self-expanding stents. There was one case of in-stent stenosis of <10% stenosis with the newer self-expanding stent designed for the neurovascular realm. Moderate to severe in-stent stenosis with the Neuroform self-expanding intracranial stent has been reported to occur in 5.8% of patients treated.40

Although in vivo research data with high-porosity stents do not show occlusion of jailed smaller vessels,7,23,41 the potential risk of a stroke associated with perforating arteries being covered by stent struts is a consideration.42 In 11 patients, we implanted stents in the perforator-rich vertebrobasilar system and observed only one case of symptomatic perforator occlusion leading to a small pontine stroke. We also observed an angiographically visible asymptomatic occlusion of a jailed small AICA. It may be hypothesized that if the pressure
gradient across the perforators is maintained and the perforator is covered less than 50% by stent struts, the arteries will remain patent and remodel around struts or coils thereby preserving flow.\(^9,42\) However, the mechanism of delayed occlusion remains unclear. Great care is necessary to preserve perforators that exit from the stented arterial segment during aneurysm embolization by segmented coiling, the use of nondetachable balloons, or more recently steerable microcatheters. As discussed in the coronary literature,\(^43\) future development of absorbable stents should be considered to prevent permanent jailing of important perforators. Experience in cardiology teaches that it may be important to place patients on dual antiplatelet therapy to prevent thrombotic events. If detected during or early after the procedure, acute thromboembolic vessel occlusion may respond well to additional antiplatelet agents such as abciximab like in one of our cases (Case 25) with a failed anterior choroidal artery.

In conclusion, reconstructive endovascular repair using stents or stents and coils is an attractive therapeutic option for complex fusiform dissecting and atherosclerotic aneurysms in which a parent artery occlusion is not feasible. An excellent long-term outcome with low periprocedural morbidity and mortality can be achieved. However, due to both early and late recanalization, long-term angiographic studies may be required to assess the need for retreatment. In addition to introduction of stent-like flow diverters for parent arteries, development of treatment mechanisms to prevent clot revascularization remains a vital goal.

Acknowledgments
We thank Drs L. Nick Hopkins, Lee R. Guterman, and Giuseppe Lanzino who conducted or were involved in the treatment of the first few patients included in this series and published previously.\(^13\)

Disclosures
A.K.W. and M.J.G. infrequently work as consultants for Cordis Neurovascular J&K and have received in the past research grants from OmniSonic Medical Technologies, Boston Scientific Neurovascular, Micrus Endovascular, Microvention/Terumo, EV3 Neurovascular, Philips Medical Systems, Siemens Medical, and Aynlam Pharmaceuticals. A.K.W. has also served as a consultant for EV3 Neurovascular.

References


Stent-Assisted Reconstructive Endovascular Repair of Cranial Fusiform Atherosclerotic and Dissecting Aneurysms: Long-Term Clinical and Angiographic Follow-Up
Ajay K. Wakhloo, Jake Mandell, Matthew J. Gounis, Christopher Brooks, Italo Linfante, Jesse Winer and John P. Weaver

Stroke. 2008;39:3288-3296; originally published online September 4, 2008; doi: 10.1161/STROKEAHA.107.512996

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/39/12/3288

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org/subscriptions/