Staged stent-assisted angioplasty for symptomatic intracranial vertebrobasilar artery stenosis


Department of Neurosurgery and Toshiba Stroke Research Center, School of Medicine and Biomedical Sciences, University at Buffalo, State University of New York, Buffalo, New York

Object. Medically refractory symptomatic vertebrobasilar atherosclerotic disease has a poor prognosis. Studies have shown that longer (>10 mm), eccentric, high-grade (>70%) stenoses portend increased procedure-related morbidity. The authors reviewed their experience to determine whether a staged procedure consisting of angioplasty followed by delayed (≥1 month later) repeated angioplasty and stent placement reduces the morbidity associated with endovascular treatment of symptomatic basilar and/or intracranial vertebral artery (VA) stenoses.

Methods. The authors retrospectively reviewed the medical records in a consecutive series of eight patients who underwent planned stent-assisted angioplasty for medically refractory, symptomatic atherosclerotic disease of the intracranial posterior circulation between February 1999 and January 2002. Staged stent-assisted angioplasty was planned for these patients because the extent and degree of stenosis of the VA and/or basilar artery (BA) lesion portended an excessive procedure-related risk. The degree of stenosis, recent onset of symptoms (unstable plaque), vessel tortuosity, and lesion length and morphological features were contributing factors in determining procedure-related risk. Patient records were analyzed for location and degree of stenosis, preprocedural regimen of antiplatelet and/or anticoagulation agents, devices used, procedure-related complications, and clinical and radiographic outcomes.

Among the patients in whom staged stent-assisted angioplasty was planned, vessel dissection, which necessitated immediate stent placement, occurred during passage of the balloon in one of them. In a second patient, the stent could not be maneuvered through the tortuous VA. In a third patient, the VA and BAs remained widely patent after angioplasty alone, and therefore stent placement was not required. Significant complications among the eight patients included transient aphasia and hemiparesis in one and a groin hematoma that necessitated surgical intervention in another; there was no permanent neurological morbidity. The mean stenosis before treatment was 78%, which fell to 54% after angioplasty, and the mean residual stenosis after stent placement was 30%. At the last follow-up examination, none of the treated patients had further symptoms attributable to the treated stenosis.

Conclusions. The novel combination of initial angioplasty followed by delayed endoluminal stent placement may reduce the neurological morbidity associated with endovascular treatment of long, high-grade stenotic lesions. Attempting to cross high-grade stenoses with higher-profile devices such as stents may result in an embolic shower. Furthermore, neo-intimal proliferation and scar formation after angioplasty result in a thickened fibrous layer, which may be protective during delayed stent deployment. Larger-scale studies involving multiple centers are needed to elucidate further the lesion morphological characteristics and patient population most likely to benefit from staged procedures.

Key Words • angioplasty • endovascular therapy • arterial stenosis • intracranial stent
Staged stent-assisted angioplasty

suggested that the morbidity and mortality rates after VBA stent placement are as high as 30%, whereas in other series significantly lower rates of permanent neurological morbidity have been reported.13,14,15 Typically, long, eccentric lesions with severe luminal narrowing (that is, Mori26 Type C lesions) have the highest rate of complications, with procedural failure rates of 70%. Additionally, angioplasty alone has been associated with recurrence rates of stenosis ranging from 9 to 25%.9,45 These shortcomings led us to investigate the effectiveness of stent placement procedures. As our experience with intracranial stent placement developed, in conjunction with data in the coronary literature36 on disruption of plaque by angioplasty, we believed that we could minimize morbidity by reducing the percentage of stenosis in high-grade lesions, allowing time for the vessel to heal from the angioplasty, and then proceeding with stent placement if necessary.

To elucidate the risks associated with stent-assisted angioplasty of the posterior intracranial cerebrovascular system and to determine whether staging of this procedure (angioplasty followed by repeated angioplasty, with stent placement delayed by at least 1 month) reduces procedure-related morbidity while increasing rates of procedural success, we retrospectively reviewed our experience in a series of patients with severe, medically refractory symptomatic VBA stenosis.

Clinical Material and Methods

During a 35-month study period between February 1999 and January 2002, eight consecutive patients underwent planned stent-assisted angioplasty of the intracranial posterior circulation. Staged stent-assisted angioplasty was planned for these patients because the extent and degree of stenosis in their symptomatic, atherosclerotic lesions portended an excessive procedure-related risk. The degree of stenosis, recent onset of symptoms (unstable plaque), vessel tortuosity, and lesion length and morphological characteristics were contributing factors in determining the procedure-related risk. We reviewed these patients’ medical records for the following parameters: age, sex, location of symptomatic lesion, degree of stenosis before and after angioplasty and stent placement, clinical presentation, comorbidity conditions, antiplatelet and/or anticoagulation regimen, procedure-related complications, clinical and radiological follow up, and use of glycoprotein IIb/IIIa agents (abciximab or eptifibatide). Procedural success was defined as more than 50% endoluminal revascularization of the stenotic lesion (compared with the parent vessel lumen) after stent deployment, with resolution of symptoms at the time of clinical or angiographic follow up.

Measurements of lesion length, width, and percentage of stenosis (determined by dividing the width of the lesion by the width of the normal proximal parent vessel and multiplying by 100) were calculated using commercially available software (Image-Pro Plus version 3.0; Media Cybernetics, Inc., Silver Spring, MD). Angiograms were scanned using the same software package, and lumen diameters were calibrated based on the measurements of the parent vessel diameter that had been recorded in the angiography suite during the procedure.

Operative Technique

At our institute, endoluminal revascularization of the intracranial posterior circulation is typically performed in an awake patient with the aid of midazolam and/or fentanyl as sedatives. The procedure is begun by placing a diagnostic catheter in the VA, ipsilateral to the lesion. This catheter is advanced distally over a hydrophilic 0.035-in wire into the VA origin. Before the catheter is advanced into the VA origin, a single-dose bolus of heparin (70 μg/kg) is administered to achieve a target activated coagulation time within the range of 250 to 300 seconds. Roadmapping techniques are used to guide the wire to the C1–2 vertebral level. The wire is left in place, and the diagnostic catheter is exchanged for a guide catheter. In the cases presented here, glycoprotein IIb/IIIa inhibitors were administered during the procedure and continued for the next 12 hours, and heparin was given as a bolus dose of approximately 50 μg/kg to achieve a target activated coagulation time of 225 to 250 seconds.21,33 After the proper activated coagulation time has been achieved, high-magnification roadmapping techniques are used to advance first a 0.014-in navigational microwire, and subsequently a microcatheter, across the lesion. The microcatheter is seated in a normal distal branch of the stenotic vessel and the wire is exchanged for a stiffer, 300-cm coronary wire. The microcatheter is then removed.

In the cases presented here, the stenotic lesions were too severe and/or were believed to be too tortuous to cross safely with a stent without first performing angioplasty to increase the cross-sectional diameter of the arterial lumen. We chose the shortest angioplasty balloon that would fully cover the lesion and inflated this balloon to approximately 50 to 75% of the parent vessel lumen. A slow inflation was used, as has been described by others.9 The balloon was removed, and an angiographic study was performed. Stents were selected according to the following factors: length of the lesion, vessel diameter, and proximal vessel tortuosity. Although numerous types of stents are currently available, the choices were fewer at the time some of these patients were treated.

For adjunctive stent placement, the device is advanced toward the lesion, such that the tip of the guide catheter can be seen in the fluoroscopic field at all times during the procedure. The appropriate balloon-mounted stent is advanced over the wire across the stenotic area. The stent is deployed by toggling the insufflator at the appropriate nominal pressure, after which the stent delivery system is removed. Selective angiography through the guide catheter is then performed to visualize successful stent placement, degree of residual stenosis, and presence of dissection or thrombus.

Four days before angioplasty and stent placement, a regimen of aspirin (325 mg/day) and clopidogrel (75 mg/day) is prescribed for the patients. Also, 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors (statins) are included in the patient’s daily medical regimen. Findings on serial angiographic studies indicate an improvement in intracranial stenosis after daily administration of these inhibitors, which may augment the intrinsic lytic system.7

Results

The ages of the eight men in this study ranged from 52 to
90 years (mean 68 years, Table 1). These patients presented with symptoms of VBA insufficiency (five patients), stroke (two patients), or both (one patient). Symptoms of VBA insufficiency included a constellation of ailments such as dysarthria, vertigo, diplopia, motor and sensory dysfunction, syncope, and gait instability. In all patients treatment with antiplatelet and/or anticoagulant regimens had failed.

Lesion characteristics are presented in Table 2. All lesions involved the intracranial posterior circulation. Four lesions involved the midportion of the BA, three others were at the VB junction, and one was at the proximal intracranial segment of the VA. The mean diameter of the distal parent vessel lumen was 2.85 mm, compared with the mean lesion width of 0.63 mm. The mean preangioplasty stenosis was 77% (range 65–90%); the mean postangioplasty stenosis was 54% (range 32–69%); and the mean stenosis immediately after stent placement was 30% (range 20–45%).

Endovascular angioplasty balloons and stents used and complications encountered are presented in Table 3. One patient (Case 1) suffered a vessel dissection during balloon access, necessitating immediate stent placement. In a second patient (Case 7) tortuous vessel anatomy precluded the successful delivery of a stent. A third patient (Case 5) had excellent resolution of the stenosis and optimal vessel patency after angioplasty alone, as documented by postprocedural angiography, and did not require stent placement (Table 3). No permanent neurological complications occurred; however, in one patient (Case 1) transient hemiparesis and aphasia developed due to a pontine infarction following a BA dissection that occurred when we passed the angioplasty balloon. This patient improved to his baseline neurological status over the next few months. In one patient (Case 8) experienced some gait imbalance residually after stent placement, and he remains asymptomatic as of the last follow-up examination.

Discussion

Over the past 2 years, there has been marked advancement in stent technology. Clinicians are now able to deliver more pliable stents to the distal intracranial VB vasculature. There are cases in which treatment options are limited by excessive proximal tortuosity of or poor access to the lesion; however, with combined surgical procedures that expose the VA in the Cl−2 region or the foramen magnum, most atherosclerotic lesions of the intracranial VA or BA can now be effectively treated with endoluminal revascularization techniques.41

Limitations of Intracranial Angioplasty

Stent placement for medically refractory VBA stenosis...
Staged stent-assisted angioplasty

TABLE 3
Endovascular angioplasty balloons, stents, and associated complications*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Balloon (mm)</th>
<th>Stent (mm)</th>
<th>Complication</th>
<th>Complication Management</th>
<th>Follow Up (mos)</th>
<th>Clinical Angio</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Gemini, 2.5 × 10†</td>
<td>S670, 4 × 12‡</td>
<td>BA dissection when passing balloon; resulted in transient hemiparesis &amp; aphasia</td>
<td>stent placed for dissection</td>
<td>24</td>
<td>none</td>
</tr>
<tr>
<td>2</td>
<td>Predator, 2 × 20§</td>
<td>none</td>
<td>lesion too long for stent</td>
<td>repeated angio</td>
<td>33</td>
<td>32</td>
</tr>
<tr>
<td>3</td>
<td>Ninja, 2.5 × 20§</td>
<td>GFX, 4 × 16</td>
<td>none</td>
<td>NA</td>
<td>36</td>
<td>35</td>
</tr>
<tr>
<td>4</td>
<td>Ninja, 1.5 × 20§</td>
<td>S670, 3 × 9½ (predilated w/ a Ninja 2 × 20§)</td>
<td>none</td>
<td>NA</td>
<td>25</td>
<td>7</td>
</tr>
<tr>
<td>5</td>
<td>Ninja, 2 × 20§</td>
<td>none (no sig post-angiostenosis)</td>
<td>none</td>
<td>NA</td>
<td>37</td>
<td>32</td>
</tr>
<tr>
<td>6</td>
<td>Ninja, 2.25 × 10§</td>
<td>Bx Velocity, 2.75 × 18 (photon balloon predilated 1.5 × 20)§</td>
<td>retroperitoneal hematoma</td>
<td>surgical repair</td>
<td>23</td>
<td>none</td>
</tr>
<tr>
<td>7</td>
<td>Ninja, 2.25 × 20§</td>
<td>AVE INR, 3 × 12½</td>
<td>unable to pass stent: small VA dissection</td>
<td>procedure aborted</td>
<td>died</td>
<td>none</td>
</tr>
<tr>
<td>8</td>
<td>Cross-Sail, 2 × 10†</td>
<td>Bx Velocity, 3 × 8§</td>
<td>none</td>
<td>NA</td>
<td>6</td>
<td>none</td>
</tr>
</tbody>
</table>

* Angiog = angiography; sig = significant.
† Guidant, Santa Clara, CA.
‡ Medtronic, Minneapolis, MN.
§ Cordis Neurovascular, Miami Lakes, FL.
∥ Medtronic AVE, Santa Rosa, CA.

has been developed because of suboptimal long-term results after balloon angioplasty alone. Among the earliest reported cases of intracranial posterior circulation angioplasty are those reported by Sundt, et al.,37 who in 1980 performed angioplasty in two patients with severe BA stenosis. Since that time, many case reports and retrospective series1,6,9,10,19,25,26,29,31,38,40 have demonstrated the utility of this intervention in the short term. In a cohort of 50 patients with intracranial stenosis treated with angioplasty and reported on by Terada, et al.,40 the mean rate of posttreatment stenosis was 44%, with complications occurring in four patients. Moreover, restenosis of 50% or more occurred in 16%. In a review of 12 patients reported on by Terada, et al.,40 the mean rate of posttreatment stenosis was 44%, with complications occurring in four patients.

An important finding by Mori, et al.,26,29 is that lesion morphological features are predictive of restenosis and complication rates. Stenotic lesions that were long, eccentric, and ectatic were associated with greater rates of restenosis and lower rates of procedural success than those that were short, focal, and concentric. Although no evidence exists to demonstrate the superiority of stent placement over angioplasty alone in the intracranial circulation, available data indicate that it is superior for coronary vessels of similar caliber to the VA and BA. In one study of 43 lesions in coronary vessels less than 2.5 mm in caliber, 76% of patients remained symptom free or had patent target sites after coronary artery stent placement.20 Similar results were demonstrated by Morice, et al.,30 with 25% of patients requiring repeated intervention after stent placement in small coronary vessels. Perhaps the best evidence of the superiority of stent placement over angioplasty in small vessels (<3 mm) is gleaned from the results of the Stent Restenosis Study.31 Stent-assisted revascularization of coronary vessels resulted in a statistically significantly larger postprocedural lumen diameter in the periprocedural period and at the 6-month follow-up review, compared with angioplasty alone. Furthermore, restenosis of 50% or more occurred in 55% of the angioplasty group and in only 34% of the group receiving stents. The 1-year event-free survival rate was 78% in the stent-treated group and 67% in the group that underwent angioplasty. Although the coronary vasculature is histologically different from the intracranial vasculature, we cannot ignore these findings and we believe that stent treatment is likely to be superior to angioplasty alone.

Plaque Subtype Worsened by Aggressive Angioplasty

In a report about the histological subtyping of coronary atherosclerotic lesions, Stary, et al.,36 described the histological features of unstable plaques (on a I–VI grading scale). In a Type IV lesion, dense accumulation of extracellular lipid occupies a region of the intima, with no thrombosis or defects of the lesion surface. These are quite dangerous because they may easily progress to Type VI lesions (discussed later) and result in symptomatic occlusive disease. The region between the lipid core and the lesion surface contains proteoglycans, macrophage foam cells, and smooth-muscle cells, which can easily be exposed to blood from fissures in the intimal surface brought on by aggressive angioplasty or immediate stent placement. Submaximal angioplasty has a lower likelihood of creating breaks in the intimal surface, thereby preventing exposure of the lipid core and smooth-muscle cells to the bloodstream and coagulation cascade. Type V lesions, or fibroatheromas, have prominent new fibrous connective tissue with multiple layers of smooth-muscle cell proliferation overlying regions of the lipid core. These lesions tend to be thicker, with the fibrocellular response intermingled with calcium and lipid content. Type VI lesions, or complicated lesions, occur when Type IV or V lesions develop breaks in the integrity of the intimal surface or when they develop hemorrhage and/or thrombosis. The majority of symptomatic lesions occur following the progression to complicated (Type VI) plaques.

If we assume that similar plaque organization occurs in the intracranial circulation, aggressive angioplasty is likely to lead to disruption of the fragile plaque surface and increase the risk of progression to Type VI lesions. In turn, such progression may result in an embolic shower from the
fragmented lipid core, thrombosis, or dissection. With submaximal angioplasty, flow can be restored to relieve the patient’s symptoms without creating the same severity of intimal disruption (and plaque destabilization) as seen with more aggressive angioplasty. Furthermore, balloon-induced fibrocellular proliferation may result in a thickened neointima, which may be protective during stent placement in the ensuing weeks.34

Abundant histochemical evidence obtained in animal studies indicates that intimal healing and smooth muscle proliferation are active for 14 to 30 days after traumatic balloon angioplasty.39 Intravascular remodeling occurs in three phases: 1) an acute initiating phase in which platelets, thrombin, and leukocytes release biologically active mediators; 2) an intermediate phase in which activated medial smooth-muscle cells proliferate and migrate to the subintima; and 3) a chronic phase in which extracellular matrix production leads to remodeling.44 We recommend waiting a minimum of 1 month between angioplasty and staged stent placement to allow for intimal healing. Attempts to navigate devices across unhealed lesions may result in release of embolic debris or iatrogenic dissection. As demonstrated by the patient in Case 5 in our series, careful angiographic evaluation of a lesion previously treated with angioplasty is essential. Occasionally, modest inflation with the angioplasty balloon alone is sufficient to provide durable endoluminal recanalization, thereby obviating the need for stent placement.

Modest Endoluminal Revascularization Improves Blood Flow

As objective methods for the measurement of CBF become increasingly available, it is now possible to measure the presence of misery perfusion (insufficient flow to meet the metabolic demands of the ischemic territory) and flow augmentation after treatment of the stenotic lesion. In a report by Derdeyn, et al.,10 the achievement of a residual stenosis rate of 40% after balloon angioplasty was sufficient to improve hypoperfusion and normalize the oxygen extraction ratio. Thus, perhaps patients with high-grade or unstable (symptomatic) lesions are best treated with balloon angioplasty for partial revascularization. These pa-
patients would be readmitted for stent placement (pending an-
giographic findings) at a later date, when the lesion has
healed from the angioplasty.

Because flow is proportional to the radius of the lumen to
the fourth power (Poiseuille’s equation), a small augmenta-
tion in lumen diameter may be sufficient, as demonstrat-
ed in reports by Derdeyn, et al.,10 and Uchiyama, et al.,43
to provide transient relief of ischemic symptoms. Other ben-
efits of revascularization include reduced stroke risk as a
result of normalization of oxygen extraction fraction and in-
creased CBF.14 In a study conducted by Uchiyama, et al.,43
positron emission tomography was used to demonstrate
significant improvements in CBF and oxygen extraction ra-
tio after stent-assisted angioplasty of a severe BA stenosis.

Other modalities of blood flow assessment, such as single-
photon emission computerized tomography, have been used
to demonstrate successful flow augmentation after stent-as-
sisted angioplasty for ischemia resulting from posterior cir-
culation lesions.16,23

Risks Related to Intracranial Stent Placement

To date, various experiences with stent-assisted angi-
oplasy of the VB circulation have been documented in sev-
eral case reports and small retrospective series.2,13,18,22,24,27,28,32
In a recent review by Rasmussen, et al.,32 atherosclerotic le-
isons of the intracranial posterior circulation in eight pa-
tients were treated with endoluminal stent placement. These
authors reported technical success, with only 7 to 28% re-
sidual stenosis. In one patient with a 99% 14-mm-long ste-
nosis, the stent would not cross the lesion. This lesion was
dilated with two angioplasty balloons, after which a stent
was placed, resulting in a dissection and the patient’s even-
tual death due to a subarachnoid hemorrhage. These authors
concluded that the subarachnoid hemorrhage most likely re-
sulted from rupture of the VA after the dissection.

Symptomatic, high-grade, long stenoses are the types of
lesions that may be best suited for staged stent-assisted an-
gioplasty (Fig. 1). After multiple angioplasty treatments of the
lesion, the VA in the case reported by Rasmussen, et al.,
likely had small dissections not readily appreciable on an-
giography. Allowing time for a normal fibrotic reaction to
occur, during which the vessel can heal and “scar,” might
reduce the chance of vessel rupture or clinically relevant
dissection resulting from stent placement immediately after
angioplasty of such a tight lesion. In the aforementioned
large cohort series, Connors and Wojak4 stated that an
angioplasty site should not be recrossed immediately after
dilation because this maneuver has resulted in abrupt ves-
sel occlusion. As demonstrated by Rasmussen, et al.,32 Go-
mez and colleagues,12,13 and others,24 successful stent-as-
sisted angioplasty may be feasible in most lesions without
planned staging. For symptoms attributed to high-grade
stenotic lesions, however, partial endoluminal revascular-
ization with submaximal angioplasty alone followed by
stent placement (if needed) at a later date may help reduce
procedure-related morbidity.

In another series of VB A stent placement, two deaths
occurred perioperatively in patients with long, high-grade
stenoses.32 A third patient eventually died of a massive pon-
tine infarct thought to be related to an embolic shower that
occurred after stent deployment. In all but one patient with
MorI Type A lesions (< 5 mm, concentric stenosis), no
neurological procedural complications were noted. These
data further corroborate that stent-assisted angioplasty of
shorter lesions has a lower procedural risk and indicate that
longer, high-grade stenoses may have fewer neurological
complications after staged angioplasty and stent placement.
A caveat about angioplasty of long, high-grade lesions is
underscored by the results of Mori, et al.,26 in which a sig-
nificant increase in procedure-related morbidity was ob-
served after angioplasty of lesions they classified as Type C,
or those that were longer than 10 mm, ectatic or tortuous,
and/or occluded. For patients with Type C lesions, the pro-
cedure success rate was 33%, with one patient experiencing
a procedure-related stroke and six others requiring bypass
surgery. All patients with Type C lesions either suffered is-
silateral stroke or needed bypass surgery or repeated an-
gioplasty.

Conclusions

On the basis of previous studies, increased morbidity and
low procedure success rates may be associated with angi-
oplasy (when it is the sole method of endoluminal revascu-
larization) of long, tortuous, high-grade stenotic lesions of
the intracranial circulation.26,29 In light of these findings, in
conjunction with data that are available regarding lesion
morphological characteristics in the coronary circulation,
and normalization of CBF and metabolism after angi-
ographically suboptimal angioplasty (residual stenosis of ≥
50%), perhaps the goal of angioplasty of long stenotic le-
isons (Mori Grade C lesions) should be to achieve mod-
est luminal improvements (estimated residual stenosis of
50%) by use of undersized balloons and slow inflation tech-
niques.5 Restenosis rates are statistically significantly high-
er for Type C than for Type A lesions (< 5 mm, concentric).
Perhaps the higher morbidity and restenosis rates seen in
Type C lesions result from the fact that these are Type IV
and V lesions,30 which then progress rapidly to Type VI le-
isons due to balloon-induced surface disruptions. Therefore,
partial angioplasty should not be the definitive treatment,
but rather the initial stage for stent-assisted angioplasty.
Clearly, there are insufficient data regarding restenosis rates
for intracranial stent placement. With the development of
newer coated cardiac stents, restenosis rates with the stent
in place have declined from approximately 30% to 0% in
some early coronary artery stent trials.35

The data we have presented and the finding of no per-
manent neurological morbidity indicate a role for staged
angioplasty and stent placement. It is important to note that
the medical management and decision-making schema for
these eight patients were based on the complex morpholog-
ical features of their lesions. Only five patients, however,
were treated in a staged fashion; the other three were not
because of either vessel dissection, resolution of stenosis at
the time of angiographic follow up, or our inability to nav-
igate a stent successfully across vessel tortuositites. The fact
that only five patients were treated should not detract from
the fact that all eight patients were believed to be in a cate-
gory in which staged stent placement could lower the risk of
treating such complex lesions. Larger-scale studies in-
volving multiple centers are needed to elucidate the mor-
phological features of the lesions and the patient population most likely to benefit from staged treatment.

Acknowledgments

We thank Paul H. Dressel for preparation of the illustrations and the staff at Kaleida Gates Hospital Library for assistance in obtaining the reference articles.

Disclosure

Doctor Guterman is a consultant for Guidant Corporation, and Dr. Hopkins receives research support from and is a consultant for Boston Scientific; Cordis, Guidant, and Medtronic; in addition, he has a financial interest in Boston Scientific.

References


E. I. Levy, et al.
Staged stent-assisted angioplasty


Manuscript received March 19, 2002. Accepted in final form August 9, 2002. Doctors Bendok, Boulou, and Levy received funding from the American Association of Neurological Surgeons/Congress of Neurological Surgeons Joint Section on Cerebrovascular Surgery Mullan Neuroendovascular Surgery Award.

Address reprint requests to: L. Nelson Hopkins, M.D., Neurosurgery, University at Buffalo, 3 Gates Circle, Buffalo, New York 14209-1194. email: dzbuffs@aol.com.