Elective Stenting of Symptomatic Basilar Artery Stenosis

Camilo R. Gomez, MD; Vijay K. Misra, MD; Ming W. Liu, MD; Van R. Wadlington, MD; John B. Terry, MD; Roekchai Tulyapronchote, MD; Morgan S. Campbell, MD

Background and Purpose—Percutaneous angioplasty of the intracranial arteries still carries the risk of dissection, with acute closure and embolization. Stenting has been shown to improve the safety and durability of angioplasty in every circulatory bed in which it has been applied. However, stenting of the intracranial arteries has been limited by the availability of stents that can be reliably deployed intracranially.

Methods—Twelve patients underwent elective stenting of the basilar artery after episodes of vertebrobasilar ischemia. In all patients, either medical therapy had failed or the patient had a contraindication for long-term anticoagulation. Information from independent neurological examinations, quantitative angiography, and clinical follow-up was collected. Differences between pretreatment and posttreatment degree of stenoses were subjected to 1-way ANOVA for repeated measures.

Results—There were 10 men and 2 women, all white, aged 40 to 82 years (mean age, 62.6 years). Stent placement was successful in all patients, leading to statistically significant changes in the degree of stenosis, from 71.4% (range, 53% to 90%) to 10.3% (range, 0% to 36%) (P<0.0001). There were no deaths, stent thromboses, perforations, ruptures, or myocardial infarctions. Clinical follow-up was available for 0.5 to 16 months (mean, 5.9 months). One patient had nonspecific symptoms, and another had a transient ischemic attack. All other patients remained asymptomatic.

Conclusions—Elective stenting of the basilar artery is feasible, with minimal risk to the patient. Its impact on long-term stroke prevention and its durability are unknown and will require further study. (Stroke. 2000;31:95-99.)

Key Words: angioplasty ■ basilar artery ■ stenosis ■ stents

The application of percutaneous balloon angioplasty to the treatment of intracranial atherosclerotic arterial stenoses has been tempered by the increased risk of stroke resulting from distal embolization, vessel dissection, or arterial rupture.1-4 In particular, concerns about balloon angioplasty of the basilar and middle cerebral arteries have included the risk of occluding the ostia of the penetrating small arterial branches that originate from these vessels, namely, the pontine perforators and the lenticulostriate branches, respectively, by compression of the plaque with the angioplasty balloon. Stenting has been shown to increase the safety and efficacy of balloon angioplasty in the treatment of extracranial carotid artery atherosclerosis.5-8 More recently, elective stenting of atherosclerotic lesions of the intracranial arteries has been possible with newer, more flexible coronary stents.9-13 Still, stenting of the cerebral arteries in the subarachnoid space has been limited by the lack of stents suitable for this purpose. We report our experience with elective stenting of symptomatic basilar artery atherosclerotic lesions refractory to medical therapy.

Subjects and Methods

Between March 1998 and August 1999, 12 patients underwent elective stenting of the basilar artery in our institution, as part of a feasibility protocol approved by the Institutional Review Board. The study population is composed of individuals who were found to have basilar artery stenosis in the course of an evaluation for ischemic events in the vertebrobasilar system. In general, the procedure was offered to patients who met the following criteria: (1) previous symptoms (ie, transient ischemic attack or nondisabling ischemic stroke in the vertebrobasilar system); (2) angiographically proven focal significant basilar artery stenosis (ie, causing >50% diameter reduction); and (3) either recurrent symptoms while being treated with optimal doses of heparin or warfarin or a contraindication for long-term treatment with warfarin. This report does not include any patients treated as part of emergency protocols or while having active neurological symptoms referable to basilar stenosis or occlusion.

A complete neurological history was taken, and an examination was performed on all patients by an independent neurologist not involved in the interventional procedure. Such a neurological evaluation was performed before the procedure and again at 24-hour, 4-week, and 6-month follow-ups. Preprocedural measurement of stenoses was performed either by hand with calipers on radiographic films or with the use of online quantitative angiography (Integris, Phillips Medical Systems). The quantitative angiography measurements were also performed on all vessels before and after stenting, as well as at follow-up angiography when this was carried out. The degree of stenosis was calculated in relation to the adjacent distal normal vessel diameter (analogous to the method of the North American Symptomatic Carotid Endarterectomy Trial for grading carotid stenosis), and calibration of the measurements was performed with the contrast-filled guiding catheter used as the reference.

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All clinical, angiographic, and stenting data were recorded on case report forms by a physician. The clinical end points were as follows: (1) any stroke (disabling or not), myocardial infarction, or death within the first 30 days of the procedure; (2) repeated intervention by angioplasty or surgery within the 6 months that followed the procedure; and (3) clinical status at the 6-month follow-up visit. Angiographic end points were as follows: (1) minimum lumen diameter and percent stenosis after stenting and (2) minimum lumen diameter and percent stenosis on any follow-up angiogram performed during follow-up. Restenosis was defined as a stenosis >50%.

The preprocedure and postprocedure protocols were analogous to those previously described. All patients were premedicated with aspirin and either ticlopidine (Ticlid, Roche, Inc) or clopidogrel (Plavix, Sanofi, Inc). Percutaneous access was obtained via 1 of the femoral arteries, where a 6F or 7F catheter sheath was introduced. The subclavian artery from which the larger vertebral artery originated was engaged with a 5F Newton 5 catheter (HN5), which was directed into the vertebral artery over a 0.038-inch hydrophilic-coated wire (Glidewire, Meditech, Inc). Heparin, at a dose of 100 U/kg, was administered through the diagnostic catheter. This was then exchanged by a 6F or 7F multipurpose or straight guiding catheter, whose tip was advanced up to the level of the second cervical vertebra. Preprocedural angiographic images were then obtained in orthogonal planes (Figure). The lesions were crossed with a hydrophilic-tipped 0.014-inch wire (Trascend, Meditech, Inc) or Choice PT, SciMed, Inc, and this was advanced within a flexible microcatheter (ie, Turbo Tracker, Target Therapeutics, Inc or Transit, Cordis, Inc) or directly within the predilation balloon. The tip of the wire was positioned in the second portion of 1 of the posterior cerebral arteries to ensure maximum support. The lesions were then predilated with a 2.0- to 3.0-mm×10- to 20-mm coronary balloon (ie, Predator XL or Ninja, Cordis, Inc) at 4 to 6 atm for 30 to 45 seconds with the use of a standard insufflator. The maximal pressure
of inflation was achieved by very slowly and gently inflating the balloon until no waist was visible. The original wire was then exchanged for a 260-cm, 0.014-inch floppy-tipped exchange support wire (ie, Luge Sci Med, Inc). A 3.0- to 3.5-mm×8- to 9-mm flexible coronary stent (ie, Microstent II or GFX, Advanced Vascular Engineering, Inc or Multilink Duet, Guidant, Inc) was then deployed across the point of stenosis (Figure ). Deployment of the stents was performed by inflating the balloon to 9 to 10 atm. Both antiplatelet agents were continued for 4 weeks, after which the patients were only treated with aspirin 325 mg/d.

The results of all measurements were entered into a computerized database and subjected to statistical analysis with the use of dedicated software (GB-Stat, Dynamic Microsystems, Inc). Demographic variables were analyzed with descriptive statistics. The differences between preprocedural and postprocedural degree of stenosis were compared by 1-way ANOVA for repeated measures.

### Results

The clinical characteristics of the patients are summarized in Table 1. There were 10 men and 2 women, all white, aged 40 to 82 years (mean age, 62.6 years). Balloon angioplasty and stent placement were successful in all patients. The distal arterial reference size varied from 2.70 to 4.28 mm (mean, 3.44 mm). The mean stenosis before the procedure varied from 53% to 90% (mean, 71.4%), while the residual stenoses after the procedure varied from 0% to 36% (mean, 10.3%). The preprocedural minimum lumen diameters measured 0.32 to 1.81 mm (mean, 0.97 mm). Typical technical and angiographic results are shown in the Figure. All patients had 1 stent deployed, except 2 patients who had 2 stents. One of these patients had a plaque that extended into the terminal portion of the dominant vertebral artery. The other had a second lesion in the distal vertebral artery, which required treatment for the basilar stenosis. There was a statistically significant difference between the percentages of stenoses before and after stenting.

<table>
<thead>
<tr>
<th>No.</th>
<th>Age, y</th>
<th>Sex</th>
<th>Race</th>
<th>RD, mm</th>
<th>SMLD, mm</th>
<th>Stenosis Before Procedure, %</th>
<th>Stenosis After Procedure, %</th>
<th>Stent No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>60</td>
<td>M</td>
<td>W</td>
<td>4.28</td>
<td>0.96</td>
<td>79</td>
<td>16</td>
<td>Microstent II</td>
</tr>
<tr>
<td>2</td>
<td>72</td>
<td>M</td>
<td>W</td>
<td>3.38</td>
<td>0.32</td>
<td>90</td>
<td>8</td>
<td>Duet</td>
</tr>
<tr>
<td>3</td>
<td>51</td>
<td>M</td>
<td>W</td>
<td>3.40</td>
<td>0.88</td>
<td>74</td>
<td>36</td>
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<td>M</td>
<td>W</td>
<td>2.80</td>
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<td>74</td>
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<td>GFX</td>
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<tr>
<td>5</td>
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<td>M</td>
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<td>0.96</td>
<td>77</td>
<td>13</td>
<td>Duet</td>
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<tr>
<td>6</td>
<td>40</td>
<td>F</td>
<td>W</td>
<td>2.99</td>
<td>1.00</td>
<td>62</td>
<td>3</td>
<td>Duet</td>
</tr>
<tr>
<td>7</td>
<td>67</td>
<td>M</td>
<td>W</td>
<td>3.60</td>
<td>1.34</td>
<td>62</td>
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<td>Duet</td>
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<td>66</td>
<td>M</td>
<td>W</td>
<td>3.40</td>
<td>0.72</td>
<td>80</td>
<td>0</td>
<td>Duet</td>
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<tr>
<td>9</td>
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<td>F</td>
<td>W</td>
<td>3.00</td>
<td>1.00</td>
<td>64</td>
<td>6</td>
<td>Duet</td>
</tr>
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<td>10</td>
<td>75</td>
<td>M</td>
<td>W</td>
<td>3.73</td>
<td>1.04</td>
<td>75</td>
<td>7</td>
<td>Duet (2)</td>
</tr>
<tr>
<td>11</td>
<td>59</td>
<td>M</td>
<td>W</td>
<td>3.85</td>
<td>1.81</td>
<td>53</td>
<td>7</td>
<td>Duet (2)</td>
</tr>
<tr>
<td>12</td>
<td>70</td>
<td>M</td>
<td>W</td>
<td>2.70</td>
<td>0.97</td>
<td>67</td>
<td>6</td>
<td>Duet</td>
</tr>
</tbody>
</table>

RD indicates reference diameter; SMLD, stenosis minimal lumen diameter; and W, white.

He had a normal neurological examination, and his double vision disappeared within a few days. Another patient complained of blurred vision after the procedure and was found to have sixth and seventh nerve pareses; these completely resolved within 8 weeks. None of the patients experienced wound infections, bleeding requiring transfusions, or significant bradycardia, hypotension, or loss of consciousness with balloon inflations. There were no stent thromboses, wire perforations, or arterial ruptures.

Clinical follow-up was available in all patients, with the follow-up period varying from 0.5 to 16 months (mean, 5.9 months) (Table 2). Angiographic follow-up was available in 2 patients. In 1 asymptomatic patient, angiography failed to show any evidence of restenosis. The second patient (patient 6) suffered an episode of seeing “flashing lights” 4 months after basilar artery stenting. This prompted a repeated angiogram, which disclosed occlusion of the basilar artery proximal to the stent. The vessel was successfully recanalized by balloon angioplasty; at present the patient is being treated with warfarin, and a follow-up angiogram is planned in 6 to 8 weeks. The only other patient with symptoms during the follow-up period complained of nonspecific dizziness and lightheadedness. He suffers from a severe cardiomyopathy, and his current complaints are different than those that prompted basilar stenting. Immediately after stenting, all patients were treated only with antiplatelet therapy, even those who had required warfarin for symptom control before the procedure. The 1 exception is patient 1, who also suffers from atrial fibrillation and requires long-term anticoagulation.

### Discussion

Percutaneous transluminal angioplasty is an established technique for the treatment of atherosclerotic lesions of the coronary, iliac, and renal arteries. Its use in the cerebral vessels, however, has traditionally been tempered by concerns of causing distal embolization of plaque material, dissection, or vessel rupture.1–4 More recently, balloon angioplasty has been demonstrated to be safer than previously suspected, although the risk of dissection continues to be an
issue. Theoretically, stenting improves acute and long-term patency and minimizes the risk of acute closure from dissection by trapping plaque material between the stent and the vessel wall. Several reports have described the safety and efficacy of extracranial carotid stenting with low mortality, low stroke rates, and very favorable long-term patency. Despite these advances, only recently has it been possible to use stents to support angioplasty of the intracranial arteries. The delay has been largely due to the inability of the previously available stents to track well into the cranial portion of the vasculature. However, the recent explosion in stent technology has resulted in more flexible stents that may be tracked more easily and are potentially suited for intracranial deployment.

In comparison with other vascular beds, balloon angioplasty of the cerebral arteries within the subarachnoid space represents an interventional challenge. First, these are more delicate, thin-walled vessels, with greater risk for rupture with significant morbidity and mortality. Second, the more delicate, thin-walled vessels, with greater risk for atherosclerotic lesions. We attribute the success of the procedures performed in our patients. It is possible to argue that the 2 patients with visual complaints had small brain stem infarctions. However, no evidence of such was found by imaging. An alternative explanation is that, as the deployed stent expands the wall of the basilar artery, the adventitia irritates the adjacent cranial nerves, producing temporary symptoms.

Although balloon angioplasty of the basilar artery has been reported, only a few cases of basilar stenting can be found in the existing literature. Higashida et al described a patient with a dissecting traumatic aneurysm of the basilar artery, which required stenting of the parent vessel to allow its coiling. In another institution, a patient with acute stroke caused by occlusion of the basilar artery was treated by intra-arterial thrombolysis, but recurrent occlusion of the vessel despite balloon angioplasty prompted the authors to place a second-generation flexible coronary stent (GRII, Cook Cardiology, Inc) to maintain vessel patency.

To our knowledge, ours is the only small series of successful elective stenting of the basilar artery for symptomatic atherosclerotic lesions. We attribute the success of the procedures to the considerable advances made recently in terms of interventional equipment. The guidewires used have hydrophilic-coated soft tips, allowing their placement in the distal intracranial vasculature while providing adequate support for stent deployment. In our experience, it seems essential that the wire tip be anchored in the posterior cerebral artery to facilitate tracking of the balloons and the stents. Although this can be accomplished by having the microwire and the predilation balloon advance as a unit, it is sometimes necessary to advance the wire with a microcatheter and then switch the former for an exchange length 0.014-inch wire. The balloons used have very low profiles and are easily tracked, making them capable of reaching even the cortical branches of the middle cerebral and posterior cerebral arteries. The stents used, all of which were of the newer generation branches of the middle cerebral and posterior cerebral arteries, have excellent longitudinal flexibility, provide excellent coverage of the vessel surface, and have minimal recoil. We consider the significant difference in stenosis reduction of major clinical importance, since previous reports of balloon angioplasty of intracranial lesions have shown only modest overall improvement of lumen diameter.

### TABLE 2. Available Follow-Up Data and Medications Before and After Procedure

<table>
<thead>
<tr>
<th>No.</th>
<th>Med Before Procedure</th>
<th>Med After Procedure</th>
<th>Follow-Up Period, mo</th>
<th>Follow-up Angiography</th>
<th>Clinical Status at Follow-Up</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>W,† A</td>
<td>W,† A</td>
<td>16</td>
<td>No</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>2</td>
<td>W</td>
<td>A</td>
<td>12</td>
<td>No</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>3</td>
<td>W, A, C</td>
<td>A, C</td>
<td>12</td>
<td>No</td>
<td>Dizziness, cardiomyopathy</td>
</tr>
<tr>
<td>4</td>
<td>T</td>
<td>A</td>
<td>11</td>
<td>Yes</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>5</td>
<td>W</td>
<td>A</td>
<td>6</td>
<td>No</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>6</td>
<td>W</td>
<td>A</td>
<td>5</td>
<td>Yes</td>
<td>Transient ischemic attack</td>
</tr>
<tr>
<td>7</td>
<td>W, A, C</td>
<td>4</td>
<td>No</td>
<td>Asymptomatic</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>W, A, C</td>
<td>2</td>
<td>No</td>
<td>Asymptomatic</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>W, A, C</td>
<td>1</td>
<td>No</td>
<td>Asymptomatic</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>H, A, C</td>
<td>1</td>
<td>No</td>
<td>Asymptomatic</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>W, A, C</td>
<td>1</td>
<td>No</td>
<td>Asymptomatic</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>W, A, C</td>
<td>0.5</td>
<td>No</td>
<td>Asymptomatic</td>
<td></td>
</tr>
</tbody>
</table>

Med indicates medication; W, warfarin; A, aspirin; C, clopidogrel; T, ticlopidine; and H, heparin.

*After the first 4 weeks.
†Warfarin continued for atrial fibrillation.
ently improved angiographic outcome may affect the ability to
discontinue long-term anticoagulation, which is also a
shortcoming seen in at least some angioplasty series.21
Clearly, our angiographic follow-up is not optimal since only a
minority of patients returned for angiography. Future studies
will have to take this into account, since it has been
our experience that noninvasive methods are inadequate to
assess patency of the stent.

The demonstration of carotid endarterectomy as an effec-
tive method for reducing the risk of stroke in patients with
significant extracranial atherosclerotic carotid artery lesions
has made carotid revascularization the standard of care for
these patients.23 However, for patients whose lesions are not
readily accessible to surgery, medical therapy has been the
only therapeutic option. For patients with intracranial stenotic
lesions, this often implies long-term anticoagulation with
warfarin.24 It seems intuitive that balloon angioplasty repres-
sents a potential solution for effective reduction of stroke risk
in these patients, yet the inherent risk of vessel dissection,
distal embolization, or rupture has limited its widespread
use.1–4 Although recent experience suggests that there have
been significant improvements in technique and materi-
als,16,20–22 the impact of balloon angioplasty on the degree of
stenosis is minimal, and most patients continue to be treated
with warfarin after the procedure. On the other hand, our
patients had significant reduction of the stenosis by stenting
and did not require any further long-term anticoagulation.

Our cases demonstrate that angioplasty and stenting of the
basilar artery are technically feasible and that they may be
performed without causing significant neurological injury.
Still, it is impossible to know whether the results we achieved in
the treatment of our patients are exceptional or if they can
be reproduced consistently in this subset of patients at risk for
stroke. Furthermore, it is impossible to know whether stent-
ing will lead to better outcomes and reduced stroke risk or
how it will compare with medical treatment in the future.
Greater experience with the technique described should allow
a better definition of the role of stenting in the treatment of
atherosclerotic lesions of the basilar arteries as well as other
arteries in the subarachnoid space.

References
Safety and efficacy of percutaneous transluminal angioplasty for intra-
2. Takis C, Kwan ES, Pessin MS, Jacobs DH, Caplan LR. Intracranial
3. Purdy PD, Devous MD Sr, Unwin DH, Giller CA, Batjer HH. Angio-
plasty of an atherosclerotic middle cerebral artery associated with
improvement in regional cerebral blood flow. AJNR Am J Neuro-
4. Volk EE, Prayson RA, Perl J. Autopsy findings of fatal complication
of posterior cerebral circulatory angioplasty. Arch Pathol Lab Med.
5. Theron J. Protected carotid angioplasty and carotid stents [in French]. J
6. Roubin GS, Yadav S, Iyer SS, Vitek J. Carotid stent-supported angi-
oplasty: a neurovascular intervention to prevent stroke. Am J Cardiol.
1996;78(3A):8–12.
for restenosis after carotid endarterectomy: initial experience. Stroke.
Elective stenting of the extracranial carotid arteries. Circulation.
1997;95:376–381.
intracranial internal carotid artery stenosis: a case report. AJNR Am J
10. Dorros G, Cohn JM, Palmer LE. Stent deployment resolves a petrous
stent in the percutaneous treatment of an intracranial carotid artery ste-
12. Emery DJ, Ferguson RDG, Williams JS. Pulsatile tinnitus cured by
angioplasty and stenting of petrous carotid artery stenosis. Arch Oto-
13. Mori T, Kazita K, Mori K. Cerebral angioplasty and stenting for intra-
14. Houdart E. Angioplasty of three intracranial vertebro-basilar athero-
comment 339–340.
1996;60:377–381.
16. McKenzie JD, Wallace RC, Dean BL, Fлом RA, Khayata MH. Pre-
liminary results of intracranial angioplasty for vascular stenosis caused by
atherosclerosis and vasculitis. AJNR Am J Neuroradiol. 1996;17:
263–268.
transluminal angioplasty of stenotic basilar artery [in French]. Neu-
18. Houdart E. Angioplasty of three intracranial vertebro-basilar arterio-
comment 339–340.
19. Higashida RT, Smith W, Gress D, Urwin R, Dowd CF, Balouose PA,
Halbach VV. Intravascular stent and endovascular coil placement for a
ruptured fusiform aneurysm of the basilar artery: case report and review of
MC. Transluminal angioplasty for middle cerebral artery stenosis in
patients with acute ischemic stroke. AJNR Am J Neuroradiol. 1999;20:
553–558.
21. Marks MP, Marcellus M, Norbash AM, Steinberg GK, Tong D, Albers
GW. Outcome of angioplasty for atherosclerotic intracranial stenosis.
22. Mori T, Fukuoka M, Kazita K, Mori K. Follow-up after intracranial
percutaneous transluminal cerebral balloon angioplasty. AJNR Am J Neu-
23. Biller J, Feinberg WM, Castaldo JE, Whittenmore AD, Harbaugh RE,
Dempsey RJ, Caplan LR, Kressowik TF, Matchar DB, Toohe JF, Easton
JD, Adams HP Jr, Brass LM, Hobson RWN, Brott TG, Stenauer L.
Guidelines for carotid endarterectomy: a statement for healthcare profes-
sionals from a Special Writing Group of the Stroke Council, American
Pessin MS, Weichell E, Sila CA, Furlan AJ. The Warfarin-Aspirin Symp-
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