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. Procedural 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.
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
One patient complained of double vision after the procedure. The headache improved significantly after balloon deflation and abated over a period of a few hours in 2 patients and within a few days in the third. 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

<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>
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<tbody>
<tr>
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RD indicates reference diameter; SMLD, stenosis minimal lumen diameter; and W, white.
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 of coronary stents, are provided sheathless and premounted on a balloon with a nominal pressure of 9 to 10 atm. These stents 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. This appar-

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**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>
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<td>W,† A</td>
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<td>A</td>
<td>12</td>
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<td>3</td>
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<td>A, C</td>
<td>12</td>
<td>No</td>
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<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>
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<td>W</td>
<td>A, C</td>
<td>4</td>
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<td>Asymptomatic</td>
</tr>
<tr>
<td>8</td>
<td>W</td>
<td>A, C</td>
<td>2</td>
<td>No</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>9</td>
<td>W</td>
<td>A, C</td>
<td>1</td>
<td>No</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>10</td>
<td>H</td>
<td>A, C</td>
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<td>W</td>
<td>A, C</td>
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<td>No</td>
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<td>12</td>
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<td>A, C</td>
<td>0.5</td>
<td>No</td>
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</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 effective 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 represents 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 materials,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 stenting 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

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