The risk of fatal or disabling stroke in symptomatic intracranial athero-occlusive disease involving the vertebrobasilar circulation is high. In patients with symptomatic intracranial atherosclerosis, single-agent antiplatelet therapy with aspirin or anticoagulation with warfarin is associated with a 2-year ischemic stroke rate of 17%–20%. Although surgical bypass is a well-known treatment option for intracranial vertebrobasilar atherosclerosis, reported morbidity and mortality rates are high. Several authors have demonstrated the feasibility of basilar artery stent revascularization using current microcatheter and stent technology. Clinical outcomes and complication rates remain poorly understood because of limited collective experience. Recent studies suggest that the risk of procedure-related stroke is significant. Although short-term clinical outcomes after basilar artery stent placement have been reported previously, midterm clinical outcome data are lacking. We retrospectively reviewed our experience with patients who underwent basilar artery stent angioplasty to determine the frequency of ischemic stroke complications, the frequency and nature of nonischemic complications, and clinical outcomes. We also analyzed dependent variables for correlation with these end points including patient-related variables, technique-related variables, perioperative antithrombotic therapy, and complications. Operator experience was considered in the analysis.

Materials and Methods
This retrospective study was performed under a protocol approved by the local Institutional Review Board (IRB) in accordance with National Institutes of Health guidelines (IRB ID 606-2003). We reviewed all consecutive cases of basilar artery stent angioplasty performed for athero-occlusive disease at our Atlanta institution from March 1999 through March 2003. All procedures were performed on a Philips Integris biplane neuroangiography unit (Philips Medical Systems, Andover, Mass) by fellowship-trained interventional neuroradiologists (2) or neurosurgeons (1).

Clinical Data
Clinical data were obtained by retrospective chart review, including patient- and technique-related variables, perioperative antithrombotic therapy, and complications. Operator experience was consid-
ered a technique-related variable because we differentiated cases performed early (March 1999–July 2001) from those performed later (August 2001–March 2003). Technical success was defined as successful delivery of a stent to the target lesion with less than 50% residual stenosis. The timetable for administration of antithrombotic drugs in relation to stent placement was assessed in each patient. When available, intraoperative activated clotting times (ACTs) were evaluated.

Ischemic complications were assessed for temporal relationship to stent placement, background antithrombotic therapy, presentation, affected anatomy on brain imaging, acute therapy, maintenance therapy, and findings of diagnostic conventional angiography. When available, catheter angiograms were reviewed.

**Angiographic Data**

Anatomic characteristics, lesion characteristics, residual stenosis after stent placement, major branch artery jailing, and causes of ischemic stroke complications were evaluated by analysis of digital subtraction angiograms (DSA). Consensus agreement was reached by 2 fellowship-trained interventional neuroradiologists (T.A.A. and F.C.T.); neither radiologist was a primary operator for any of the stent placement procedures.

Pretreatment stenosis, pretreatment lesion lumen, residual stenosis, and lesion length were calculated electronically using EZ Vision software on an EZ Vision workstation (Philips Medical Systems). Stenoses were based on the normal basilar artery lumen (consensus judgment by T.A.A. and F.C.T.) as the denominator. Lumen and length of the lesion were measured using the distance between proximal and distal stent markers as an internal calibration standard. When measurements were made on pretreatment DSA, a retroanalysis was performed using a shared marker vessel as a calibration standard to bridge pretreatment and posttreatment DSA. Pretreatment stenosis, pretreatment lesion lumen, and residual stenosis were determined in the projection that demonstrated the most severe pretreatment luminal narrowing. Lesion ulceration was considered present if luminal contours were markedly irregular with overhanging margins.

Lesion angle was determined by tracing the central linear axes of the basilar artery on the proximal and distal sides of each lesion; the angle between the 2 axes was measured with a protractor in the projection that demonstrated the most severe angulation. Lesion length was determined in the projection that maximally elongated the lesion.

Lesion location was designated as distal if the cranio-caudal extent of the lesion was at or distal to the cranio-caudal median of the basilar artery or proximal if the lesion did not meet these criteria. For the evaluation of vertebral artery and basilar branch artery pathology, stenosis was considered severe if ≥50%, moderate if 21%–49%, and mild if ≤20%.

**Outcome Data**

Outcome data, including modified Rankin scores (mRS), were obtained by telephone interview of each patient. Interval events/changes occurred after stent placement and discharge from the hospital. Any event requiring hospitalization or a change in antithrombotic medication was considered clinically significant; details of clinically significant interval events were obtained by chart review. Clinical outcomes were defined as excellent (mRS 0–2), good (mRS 3), or poor (mRS 4–6).

**Statistical Analysis**

The Pearson correlation test identified statistically significant associations of clinical and angiographic variables (Table 1) with mRS, clinical outcome (excellent, good, or poor), and complications (ischemic and nonischemic). Analysis of variance determined statistically significant associations of mRS and clinical outcome with procedure-related complications (ischemic stroke, intracranial hemorrhage, access site complications, symptomatic restenosis) and non–procedure-related complications (cardiac events, pulmonary events, infectious complications, hematologic complications).

**Results**

**Patient-Related Variables**

Between March 1999 and March 2003, 2 women and 8 men ranging in age from 50 to 83 years underwent basilar artery stent placement for athero-occlusive disease at our institution (Table 2). Hypertension was the most common comorbidity, affecting 90% of patients. The indications for stent placement were refractory symptoms of vertebrobasilar ischemia despite medical therapy for the presenting index event (patients 1–7) or clinical factors (eg, chronic gait instability) that made patients 8–10 poor candidates for long-term anticoagulation. The onset of index symptoms was abrupt in 6 patients and...
progressive in patients 2, 3, 6, and 8. Medical therapies administered to treat the presenting index symptoms are listed in Table 2. All 10 cases were performed with the intention to stent. There were no “bail out” procedures to salvage failed balloon angioplasties. Only patient 6 did not undergo a prestent brain MR imaging; this patient had no clinical evidence of stroke before and during the first 3 days after stent placement. Three patients had MR imaging-proved acute strokes at the time they presented for stent placement.

Two patients underwent balloon angioplasty of the basilar artery 3–5 months before stent placement (Table 2). Patient 7 underwent balloon angioplasty for a Mori type C lesion (90% stenosis) 5 months before stent placement. This resulted in 50% residual stenosis that occluded 1 month after angioplasty, subsequently recanalized, and became symptomatic while the patient was taking aspirin. Patient 6 underwent balloon angioplasty for a Mori type B lesion (70% stenosis) 3 months before stent placement that resulted in 25% residual stenosis, which subsequently became symptomatic while the patient was taking clopidogrel (Plavix) and aspirin.

### Table 2: Patient demographics and clinical presentation

<table>
<thead>
<tr>
<th>No.</th>
<th>Age/Sex</th>
<th>Medical Comorbidities</th>
<th>Presenting Symptoms</th>
<th>Background Treatment at Presentation</th>
<th>Initial Treatment for Presenting Index Event</th>
<th>Acute Infarcts on Prestent Brain MRI</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>68/F</td>
<td>Hypertension, hypothyroidism</td>
<td>Hemi-numbness, hemiparesis, dysarthria</td>
<td>None</td>
<td>Heparin</td>
<td>Bilateral cerebellar, left parieto-occipital lobe</td>
</tr>
<tr>
<td>2</td>
<td>80/M</td>
<td>Hypertension, NIDDM, paroxysmal atrial fibrillation, ischemic coronary artery disease</td>
<td>Vertigo, ataxia</td>
<td>Aspirin 81 mg/day, warfarin (INR 2.17 seconds)</td>
<td>Heparin</td>
<td>None</td>
</tr>
<tr>
<td>3</td>
<td>76/M</td>
<td>Hypertension, tobacco abuse, dyslipidemia</td>
<td>Vertigo, drop attacks/syncope, ataxia, dysarthria, blurred vision</td>
<td>Clopidogrel</td>
<td>Warfarin</td>
<td>None</td>
</tr>
<tr>
<td>4</td>
<td>57/M</td>
<td>Hypertension, ischemic coronary artery disease</td>
<td>Hemiparesis, hemi-numbness</td>
<td>Clopidogrel, aspirin 325 mg/day</td>
<td>tPA, heparin, abciximab</td>
<td>None</td>
</tr>
<tr>
<td>5</td>
<td>83/F</td>
<td>Hypertension, ischemic coronary artery disease, congestive heart failure, hypothyroidism</td>
<td>Dysarthria, hemiparesis</td>
<td>Warfarin (INR unknown)</td>
<td>Heparin, aspirin 325 mg/day</td>
<td>None</td>
</tr>
<tr>
<td>6</td>
<td>50/M</td>
<td>NIDDM, dyslipidemia, tobacco and alcohol abuse</td>
<td>Hemiparesis, vertigo, dysarthria, ataxia</td>
<td>Clopidogrel, aspirin 325 mg/day × 3 months after balloon angioplasty of Mori B lesion (70% stenosis with 25% residual stenosis)</td>
<td>Heparin</td>
<td>No prestent brain MRI</td>
</tr>
<tr>
<td>7</td>
<td>61/M</td>
<td>Hypertension, NIDDM, alcohol abuse</td>
<td>Homonymous hemianopsia, drop attacks/syncope</td>
<td>Aspirin 325 mg/day × 4 months after acute thrombotic occlusion of basilar artery, 1 month after balloon angioplasty of Mori C lesion (90% stenosis with 50% residual stenosis)</td>
<td>Heparin</td>
<td>None</td>
</tr>
<tr>
<td>8</td>
<td>72/M</td>
<td>Hypertension, IDDM, dyslipidemia, paroxysmal atrial fibrillation, COPD, hypothyroidism, alcohol abuse, ischemic coronary artery disease</td>
<td>Vertigo, dysarthria, ataxia</td>
<td>Clopidogrel, aspirin 325 mg/day</td>
<td>Heparin</td>
<td>Left cerebellar</td>
</tr>
<tr>
<td>9</td>
<td>69/M</td>
<td>Ischemic coronary artery disease, hypertension</td>
<td>Dysarthria, hemiplegia</td>
<td>None</td>
<td>Clopidogrel, aspirin 325 mg/day</td>
<td>Left hemi-pontine</td>
</tr>
<tr>
<td>10</td>
<td>67/M</td>
<td>Hypertension</td>
<td>Hemi-numbness, hemiparesis</td>
<td>Aspirin (dose unknown)</td>
<td>Warfarin, aspirin 325 mg/day</td>
<td>None</td>
</tr>
</tbody>
</table>

Note: MRI indicates MR imaging; NIDDM, non–insulin-dependent diabetes mellitus; IDDM, insulin-dependent diabetes mellitus; COPD, chronic obstructive pulmonary disease; INR, international normalized ratio; sec, seconds; tPA, tissue plasminogen activator.

### Technique-Related Variables

Patients 1–5 and 7 underwent treatment between March 1999 and July 2001 and patients 6 and 8–10 underwent treatment...
between August 2001 and March 2003. Patients 1 and 3 were treated under conscious sedation, and the remaining received general endotracheal anesthesia.

Except for patient 6, all patients underwent anticoagulation with heparin intraoperatively. Intraoperative ACTs were above 250 seconds in patients 3, 7, 9, and 10 and were not recorded in patients 1, 4, and 8. During the procedure, patient 10 did not receive IIB/IIIa inhibitors, patient 7 received tirofiban (Aggrastat), and the remaining patients received abciximab (ReoPro).

Technical success was achieved in all 10 patients. Arterial access was transfemoral in 9 patients and transbrachial in 1 patient. The basilar artery lesion was predilated with an over-the-wire angioplasty balloon before stent placement in all patients except 9 and 10. Nine procedures were performed using only balloon-mounted coronary stents that included the PRIMO (Boston Scientific, Natick, Mass), Tristar (Guidant, Indianapolis, Ind), GFXAVE, S670AVE, S7AVE (Advanced Vascular Engineering; Medtronic, Santa Clara, Calif), and Sonic Hepacoat (Cordis, Miami Lakes, Fla). Patient 6 received a self-expanding Magic Wallstent (Boston Scientific) in addition to balloon-mounted coronary stents. Treatment included 1 stent in 7 patients (patients 1, 3–5, 8–10), 2 stents in patient 7, 3 stents in patient 2, and 4 stents in patient 6. The anterior inferior cerebellar artery (AICA) was jailed bilaterally in all patients except 4, 9, and 10; a posterior inferior cerebellar artery was jailed in patient 2.

Perioperative Antithrombotic Therapy

Preoperative antiplatelet therapy included aspirin and clopidogrel in 4 patients (patients 6, 8–10), clopidogrel only in patients 1 and 3, aspirin only in patients 5 and 7, and clopidogrel and abciximab in patient 4. Patient 2 was not given antiplatelet agents before treatment. Preoperative anticoagulation with heparin was administered to 6 patients (patients 1–3, 5–7). Patient 4 was given intravenous tissue plasminogen activator for acute stroke preoperatively.

Postoperative antiplatelet therapy included clopidogrel and aspirin in 6 patients (patients 1, 2, 5, 6, 8, 9), clopidogrel only in patient 3, and dipyridamole/aspirin (Aggrenox) only in patient 7. Patients 4 and 10 did not receive postoperative antiplatelet therapy. Antiaggregative agents were administered postoperatively for 24 hours in patients 2 and 4, for 48 hours in patients 1 and 7, and for 6 days in patient 8. All patients, except patients 1 and 7, who received postoperative anticoagulation therapy, were continued on warfarin after heparin was discontinued.

Anatomic Characteristics

Basilar artery stenosis was proximal in 7 (patients 1–5, 8, 9) and distal in patients 6, 7, and 10. Patients 1, 8, and 10 did not have athero-occlusive disease of the vertebral arteries. Intracranial vertebral artery stenosis was severe and bilateral in patient 2; severe and unilateral in patients 3, 5 and 9; and mild and unilateral in patients 7 and 4. In patients 5 and 6, one vertebral artery was occluded. In patient 5, the left vertebral artery was occluded, and the right vertebral artery was affected by tandem stenoses involving its origin and intradural segment.

No basilar branch artery pathologic lesions were demonstrated angiographically in patients 4, 5, 8, and 10. Moderate to severe stenosis of the AICA was bilateral in patients 7 and 9 and unilateral in patients 1–3. Patient 6 had occlusion of the left AICA and severe stenosis of the right AICA. Moderate stenosis was found in a posterior cerebral artery in patients 2, 3, and 7 and in a superior cerebellar artery in patient 2. Five (patients 3–5, 7, 10) had no demonstrable posterior communicating artery (PcomA), 3 (patients 1, 2, 9) had bilateral PcomAs, and 2 (patients 6 and 8) had unilateral PcomAs.

Lesion Characteristics

Mori classification of the basilar artery lesion was A in 2 patients, B in 6 patients, and C in 2 patients (Table 3). Mean measurements included pretreatment stenosis of 80.7%, prestent lesion lumen of 0.5 mm, lesion length of 11.6 mm, and lesion angle of 34.5°.

Ischemic and Nonischemic Complications

Within the first 30 days after basilar artery stent placement, 6 ischemic stroke complications affected patients 5–8 (Table 4). The postoperative course was complicated by multiple discrete strokes in patient 6 and a single stroke in 3 patients, including one that was fatal (patient 5).

Six patients (patients 2, 3, 5, 8–10) suffered nonischemic complications. Acute myocardial infarction occurred in patient 5 and supraventricular tachycardias occurred in patients 2, 5 and 8. Pulmonary complications included atelectasis (patient 5), pneumonia (patient 10), aspiration pneumonitis (patient 8), and pulmonary embolism (patient 9). The postoperative course was complicated by urosepsis in patients 5 and 8.
and catheter sepsis in patients 8 and 10. Patient 10 suffered from symptomatic anemia requiring blood transfusion. In patient 10, the basilar artery ruptured during stent deployment when the carrier balloon was inflated to nominal pressure (10 atmospheres). The resulting hemorrhage was controlled by tamponade with a HyperForm balloon occlusion catheter (ev3, Irvine, Calif) and reversal of anticoagulation with protamine. Postoperative hydrocephalus was managed by external ventricular drainage, and refractory intracranial hypertension was treated with osmolar therapy (hypertonic saline and mannitol), hypothermia, and pentobarbital coma. With no MR imaging evidence of brain ischemia, the patient was ultimately discharged to an inpatient rehabilitation facility, where he recovered and achieved an excellent outcome.

In patient 3, an attempted closure of a brachial arteriotomy with a Perclose device (Abbott Vascular Devices, Redwood City, Calif) was unsuccessful, resulting in a pseudoaneurysm that required immediate postoperative surgical repair. This patient subsequently developed chronic disabling median neuropathy.

Clinical Outcome after Stent Revascularization of the Basilar Artery

Late mid-term follow-up (12–46 months) in a cohort of patients with symptomatic basilar artery stenosis treated by stent placement is presented here for the first time. In our experience, most patients had good to excellent clinical outcomes and were free of vertebrobasilar ischemia (as defined by progressive or fluctuating symptoms) at 2–3 years after stent placement despite a significant incidence of ischemic complications.

### Statistical Analysis

Statistical analyses were performed using the chi-square test for categorical variables and the Student t test for continuous variables. Comparisons between groups were made using the Fisher exact test and the Mann-Whitney U test, respectively. The Fisher exact test was used for all comparisons involving fewer than 5 events. The significance level was set at P < .05.

### Discussion

Clinical Outcomes

One procedure-related death (patient 3) occurred 2 days after the procedure because of a large pontocerebellar infarction (Table 5). There were no poor clinical outcomes in surviving patients. Three patients had clinically significant interval events, including patient 3, who developed symptomatic restenosis. Patients 3 and 6 quit smoking at the time of their stent placement procedure.

Of 9 surviving patients (mean follow-up time, 31 months), 3 patients (patients 1, 2, 9) reported no symptoms at last follow-up and 5 (patients 4–8, 10) reported persistent fixed neurologic deficits that were stable or improving, consistent with the sequelae of completed ischemic strokes. Patient 3 complained of chronic fluctuating gait instability and positional vertigo.

### Table 4: Ischemic complications of basilar artery stenting

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Time of Onset Relative to Stenting</th>
<th>Anti-Thrombotic Medications at Time of Ictus</th>
<th>Clinical Presentation</th>
<th>Acute Infarcts (Imaging Modality)</th>
<th>Catheter Angiography</th>
<th>Acute Treatment</th>
<th>Maintenance Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>Immediate</td>
<td>Abciximab</td>
<td>Deep coma; after withdrawal of support, patient died on poststent day 10</td>
<td>Right cerebellum and anterior pons (CT)</td>
<td>Not performed</td>
<td>Not applicable</td>
<td>Not applicable</td>
</tr>
<tr>
<td>6</td>
<td>Day 4</td>
<td>Clopidogrel, aspirin*</td>
<td>Vertigo, ataxia</td>
<td>Left cerebellum (MRI)</td>
<td>No acute abnormality</td>
<td>Heparin, clopidogrel, aspirin</td>
<td>Clopidogrel, aspirin</td>
</tr>
<tr>
<td>7</td>
<td>Day 2</td>
<td>Ticagrel</td>
<td>Dysarthria, hemiplegia</td>
<td>Left pons (MRI)</td>
<td>Not performed</td>
<td>Heparin, tirofiban</td>
<td>Heparin, tirofiban</td>
</tr>
<tr>
<td>8</td>
<td>Day 1</td>
<td>Abciximab</td>
<td>Hemiplegia, respiratory failure</td>
<td>Right pons (MRI)</td>
<td>Stent thrombosis</td>
<td>Endovascular mechanical thrombectomy and tirofiban</td>
<td>Warfarin, ticagrel</td>
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Note: INR indicates international normalized ratio; MRI, MR imaging; *, when aspirin is indicated, 325 mg/day.

In patient 3, an attempted closure of a brachial arteriotomy with a Perclose device (Abbott Vascular Devices, Redwood City, Calif) was unsuccessful, resulting in a pseudoaneurysm that required immediate postoperative surgical repair. This patient subsequently developed chronic disabling median neuropathy.

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<td>Not applicable</td>
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<tr>
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<td>Day 4</td>
<td>Clopidogrel, aspirin*</td>
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<td>Left cerebellum (MRI)</td>
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<td>Heparin, clopidogrel, aspirin</td>
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<td>Ticagrel</td>
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</table>

Note: INR indicates international normalized ratio; MRI, MR imaging; *, when aspirin is indicated, 325 mg/day.
Predictors of Procedural Risk

Although the Mori classification did not correlate with stroke complications in our series, 2 components of the Mori scale did: lesion angle >45° was significant, and lesion length >10 mm approached significance. These findings emphasize that technical difficulty relates to the risk of ischemic complications. Mori et al have shown that technical difficulty in intracranial stent placement depends on a number of angiographic features incorporated into the Mori scale.8,13 On the basis of the Mori scale, most cases in our series were of intermediate difficulty (6 Mori B, 2 Mori A, and 2 Mori C).

We found that prestent lesion lumen ≤0.5 mm was associated with ischemic complications. This association may reflect the larger burden of embolic debris released when stents are advanced through a small-caliber lumen or redistribution of a larger plaque mass over the surface area incorporating perforator ostia. Other mechanisms may have been operant because most stroke complications in our patients presented in a delayed manner. Redistributed plaque may not immediately occlude perforator ostia but may encroach sufficiently to disturb local hemodynamics, thus enhancing platelet aggregation and thrombus formation. As subsequent downgrading of the antithrombotic drug regimen may push the rate of platelet aggregation toward the threshold for occlusion of perforator ostia, there may be a rationale to extend the period of anticoagulation in patients with critical luminal narrowing.

Although a smaller posttreatment lumen caliber may translate into less complete stent expansion and crowding of struts over perforator ostia, residual stenosis did not correlate with stroke risk in our series. In theory, a larger posttreatment lumen caliber should also decrease the need for anticoagulation and improve long-term patency rates. Marks et al, who advocated balloon angioplasty without stent placement for the treatment of intracranial atherosclerosis, emphasized that only a small change in lumen caliber is necessary to affect a large increase in flow.14 Theoretic advantages of primary stent placement over balloon angioplasty alone are both a reduction of complications resulting from acute vessel dissection and a reduction in the rate of restenosis.7 Proponents of balloon angioplasty alone have reported long-term results with a 5-year stroke-free survival of 85% when procedural complications are excluded14; they also suggest that procedural risks are lower than for primary stent placement. A recent series has

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Follow-Up (months)</th>
<th>mRS</th>
<th>Persistent Symptoms</th>
<th>Clinically Significant Interval Events</th>
<th>Maintenance Medical Therapy at Last Follow-Up</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>44</td>
<td>0</td>
<td></td>
<td>4 hospitalizations for gastrointestinal hemorrhage secondary to excessive anticoagulation</td>
<td>Clopidogrel, aspirin, atorvastatin</td>
</tr>
<tr>
<td>2</td>
<td>46</td>
<td>0</td>
<td></td>
<td></td>
<td>Warfarin, aspirin</td>
</tr>
<tr>
<td>3</td>
<td>45</td>
<td>3</td>
<td>Chronic imbalance, positional dizzy spells, dominant hand disability related to access site complication</td>
<td>MRI 44 months poststent performed to assess recurrent vertigo, diplopia, and gait ataxia while on clopidogrel showed no new infarcts. Angiogram showed 50% concentric stenosis just proximal to stent. Symptoms stabilized on heparin and remained stable on warfarin.</td>
<td>Warfarin, clopidogrel, simvastatin</td>
</tr>
<tr>
<td>4</td>
<td>39</td>
<td>2</td>
<td>Fixed difficulties with fine motor control</td>
<td>NA</td>
<td>Warfarin, aspirin, simvastatin</td>
</tr>
<tr>
<td>5</td>
<td>10 days</td>
<td>6</td>
<td>NA</td>
<td>NA</td>
<td>Warfarin, aspirin, simvastatin</td>
</tr>
<tr>
<td>6</td>
<td>29</td>
<td>3</td>
<td>Fixed gait ataxia, vertigo, dysarthria, dysphagia</td>
<td>Warfarin</td>
<td>Warfarin, aspirin, simvastatin</td>
</tr>
<tr>
<td>7</td>
<td>38</td>
<td>3</td>
<td>Fixed left hemiparesis, diplopia</td>
<td>Major myocardial infarction 30 days after discharge from stent hospitalization. Treated by coronary artery bypass grafting surgery and automated implantable cardiac defibrillator</td>
<td>Warfarin, clopidogrel, simvastatin</td>
</tr>
<tr>
<td>8</td>
<td>18</td>
<td>3</td>
<td>Fixed left hemiparesis, dysphagia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>12</td>
<td>0</td>
<td>Fixed gait ataxia, dysarthria, dysphagia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>8</td>
<td>2</td>
<td>Fixed gait ataxia, dysarthria, dysphagia</td>
<td></td>
<td>Warfarin, clopidogrel, simvastatin</td>
</tr>
</tbody>
</table>

Note: — mRS indicates modified Rankin score; MRI, magnetic resonance imaging; NA, not applicable.
reported the peri-procedural risk of stroke or death after intracranial vertebrobasilar angioplasty of less than 10%.14; however, others have found the risk to be as high as 28%.15

Ischemic complications did not significantly correlate with perioperative antithrombotic therapy; however, all but one stroke occurred on antiplatelet drugs alone (no anticoagulation). The one stroke that occurred in the setting of anticoagulation (patient 6) presented on day 17 (late delayed stroke)—a time when the effects of intimal hyperplasia could be operant. Because most stroke complications were early delayed, there may be a rationale to extend postoperative anticoagulation to 2 weeks, at which time patients would be managed with antiplatelet agents only.

Although ischemic complications did not correlate with clopidogrel preloading or postoperative dual antiplatelet therapy in our series, our small number of cases may have precluded detection of such an association. We could not assess the adequacy of perioperative platelet inhibition in the current series because platelet function assays were not obtained. In clinical practice and future studies, routine implementation of platelet function assays may help to prevent thromboembolic strokes that are the consequence of inadequate platelet inhibition secondary to insufficient dosing or patient resistance.16

**Lack of Correlation between Technical Success and Procedural Complications**

Our experience with basilar artery stent placement reveals a significant risk of peri procedural ischemic stroke that is consistent with other recently reported experiences.12 Although the technical success rate was 100% in our series, 4 patients suffered stroke complications. Our findings are similar to a series of 11 cases reported by Levy et al in which 4 patients died, 2 from basilar or vertebral artery rupture, and 2 from pontine stroke.12 In that series, patients with poor outcomes had Mori B and C lesions. In the SSYLVIA trial, 2 of 17 (12%) patients who underwent basilar artery stent placement suffered stroke within 30 days16; this relatively low stroke rate may have been related to the lesion characteristics of the patients selected for treatment in whom mean pretreatment stenosis was only 69.9%. Mori classifications were not reported.

**Timing of Ischemic Complications and Theoretic Mechanisms**

Acute intraoperative strokes that manifest immediately after stent placement may be the result of a “snow plowing” effect, thromboembolism, acute occlusion of perforator ostia by stent struts, or in situ thrombus.17 One such stroke was encountered in our series. Early delayed strokes that develop within the first few days after stent placement may be related to in-stent thrombus, occlusion of perforator ostia, or thromboembolism.

Early delayed strokes represented most stroke complications in our series (4 of 6). Patient 7 demonstrated stent thrombosis and the other 3 (patients 5, 6, and 8) with early delayed strokes had no associated angiographic evidence of in-stent/intraarterial thrombus, flow-limiting dissection, or branch artery occlusion. In each case, the stented basilar artery was widely patent. The pons was affected in 4 events, a middle cerebellar peduncle was affected in 1 event, and a cerebellar hemisphere was affected in 2 events. The results suggest that most early delayed strokes are related to small thromboemboli and perforator occlusions.

Late delayed strokes (≥2 weeks after stent placement) may be related to all of the above in addition to another potential mechanism caused by intimal hyperplasia within and around perforator ostia. In nonhuman primate models of postangioplasty restenosis, marked intimal thickening occurs between 14 and 28 days later.18 Side branch occlusion resulting from in-stent restenosis has been reported in the coronary circulation.19 In our series, only one late delayed stroke occurred, suggesting that if this phenomenon occurs after basilar artery stent placement, it does not account for most procedure-related strokes.

**Conclusions**

Despite technically satisfactory results, ischemic and nonischemic complications are common in patients who undergo basilar artery stent angioplasty. Lesion characteristics that predict technical difficulty, including present lumen diameter and lesion angle, also predict ischemic complications. Longer and more aggressive anticoagulation regimens may be beneficial because most ischemic complications have an early delayed presentation and affect patients unprotected by anticoagulation. Clinical outcomes correlate with ischemic complications and vertebrobasilar anatomy. Despite a significant complication rate, most of our patients experienced good to excellent clinical outcomes and were free of vertebrobasilar ischemia at late midterm follow-up. In consideration of these findings and the malignant natural history of symptomatic intracranial vertebrobasilar athero-occlusive disease, stent angioplasty may be a reasonably good treatment option for patients with technically favorable lesions, especially in those with medically refractory symptoms. Although we do not believe that our data should influence a change in the indications for basilar artery stent placement, our experience provides a basis on which patients and referring physicians can be informed about procedure-related risks. Whether the long-term risk-to-benefit ratio of stent placement will compare favorably to best medical therapy in all or select patients with symptomatic basilar artery atherosclerosis will need to be assessed in randomized clinical trials.

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**References**


