

Tandem Intracranial Stent Deployment for Treatment of an Iatrogenic, Flow-limiting, Basilar Artery Dissection: Technical Case Report

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OBJECTIVE AND IMPORTANCE: Intimal dissection constitutes one of the complications associated with angioplasty of intracranial vessels. We present a case of iatrogenic dissection of the entire basilar artery, which was induced by angioplasty and stenting of symptomatic, focal, intracranial vertebral artery stenosis, and its successful treatment with tandem deployment of a downstream stent.

CLINICAL PRESENTATION: A 61-year-old, hypertensive, renal transplant recipient presented with orthostatic vertebrobasilar insufficiency that was refractory to medical management, including anticoagulation therapy. Angiography revealed an occluded right vertebral artery and focal, high-grade, left intracranial vertebral artery stenosis. Magnetic resonance imaging showed multiple posterior fossa infarctions. The left intracranial vertebral artery stenosis was successfully treated with primary stent deployment and balloon angioplasty, with symptom resolution. On post-procedure Day 2, the patient noted worsening right hemiparesis.

INTERVENTION: Subsequent angiography revealed a flow-limiting, windsock-type, basilar artery dissection beginning at the distal end of the left vertebral artery stent and extending to the origin of the left posterior cerebral artery. A tandem stent was navigated intracranially and deployed past the first one, successfully sealing the dissection inflow zone and reconstituting normal flow to the top of the basilar artery. A clinical follow-up examination at 3 months revealed no further orthostatic symptoms and only mild residual right-sided weakness.

CONCLUSION: This is the first description of iatrogenic stent-induced dissection of the entire basilar artery that was successfully treated by inflow zone control via tandem intracranial stent deployment.

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Key words: Balloon angioplasty, Endovascular surgery, Intracranial atherosclerosis, Vertebral stenosis

Transluminal vertebral and basilar angioplasty has been shown to benefit select patients with posterior circulation ischemia who have experienced failure of maximal medical management (1, 7, 14). Recent advances in stent technology have produced more compact and flexible designs that enable intracranial navigation (10) and offer the ability to provide a scaffold to hinder restenosis and vessel recoil after angioplasty (11). Despite its potential benefits in select cases, intracranial angioplasty with stent placement remains a high-risk procedure, which can be complicated by thrombus formation and vessel dissection (7). We describe a case of extensive basilar artery dissection, originating proximal to the vertebrobasilar junction and extending distally to the origin of the left posterior cerebral artery. Dissection was induced by balloon angioplasty and stent placement for treatment of intracranial vertebral atherosclerotic stenosis. We report our clinical and endovascular management.

CASE REPORT

Clinical presentation

A 61-year-old man with a history of longstanding systemic hypertension and hypertensive nephrosclerosis, who had received a cadaveric kidney transplant, presented with a 1-week complaint of progressive loss of balance and ataxia. These symptoms were initially associated with generalized headache and malaise and eventually led to nausea and vomiting. Symptoms were clearly exacerbated by sitting or standing, forcing the patient to remain in bed, despite systemic intravenous anticoagulation therapy using heparin. A cardiac ultrasonographic examination revealed left ventricular hypertrophy and an ejection fraction of 70% but no evidence of mural or valvular thrombus. Of particular interest, the patient had undergone construction of a brachial Gore-Tex hemodialysis arteriovenous fistula (W.L. Gore & Associates, Inc., Flagstaff, AZ) (ipsilateral to the stenosed vertebral artery), which was still patent. During the neurological exami-

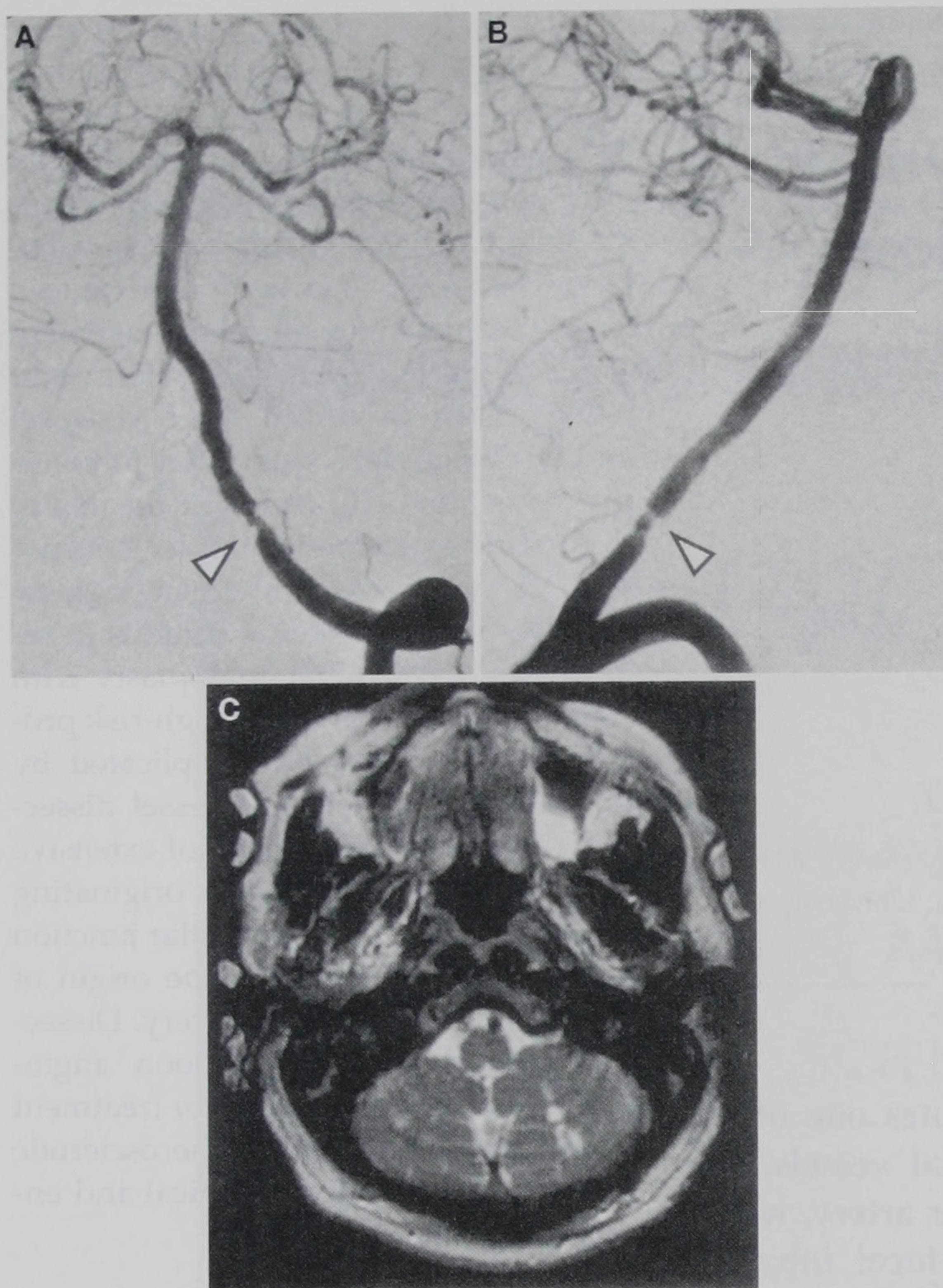


FIGURE 1. A and B, digital subtracted angiograms of the left vertebral artery (A, anteroposterior projection; B, lateral projection), revealing hemodynamically significant (70%) stenosis (arrowheads) in the intracranial portion. C, axial magnetic resonance imaging study, showing regions of strong signal in the left cerebellum consistent with infarction.

nation, which was performed with the patient supine, the patient was awake, alert, and oriented, with normal mental status, normal cranial nerve function, normal extraocular movement, and no evidence of nystagmus. Motor and sensory examination results were normal for all extremities. There was no dysmetria or incoordination in the extremities. When sitting, the patient became ataxic and nauseated and had a tendency to fall backward in his bed. Gait could not be tested. Magnetic resonance imaging scans of the brain showed multiple posterior fossa infarctions in the left cerebellum (Fig. 1). An angiogram obtained at the referring institution showed occlusion of the right vertebral artery in the intracranial segment, with flow stasis, and 70% stenosis of the intracranial left vertebral artery.

Intervention

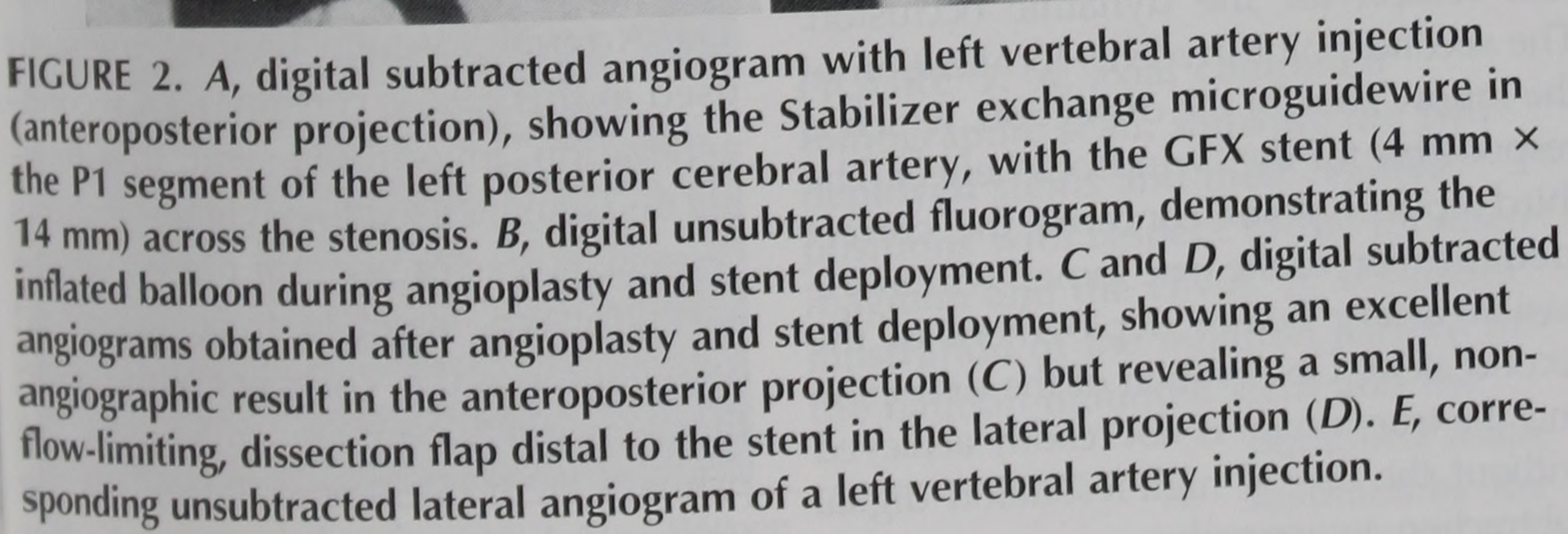
The patient showed no improvement in his orthostatic vertebrobasilar insufficiency with systemic anticoagulation therapy.

Therefore, the decision was made to proceed with an endovascular revascularization procedure. The patient was placed under endotracheally administered general anesthesia. Systemic heparin administration was increased to yield an activated clotting time of more than 250 seconds. Diagnostic angiography confirmed right vertebral artery occlusion and 70% stenosis of the intracranial segment of the left vertebral artery (Fig. 1), proximal to the origin of the posterior inferior cerebellar artery. A 7-French guide catheter (BriteTip; Cordis Endovascular, Miami Lakes, FL) was selectively placed in the proximal to mid-cervical portion of the left vertebral artery, with care being taken to avoid flow stasis. A RapidTransit microcatheter (Cordis Endovascular) was inserted coaxially with a 0.014-inch microguidewire (Transend 14; Scimed Life Sciences, Maple Grove, MN), to cross the lesion, and was placed in the midbasilar section. The microguidewire was then replaced by a 0.014-inch-diameter,

300-cm, exchange microguidewire (Stabilizer; Cordis Endovascular), the tip of which was selectively navigated into the left P1 segment of the posterior cerebral artery; the RapidTransit microcatheter was then withdrawn. A balloon-mounted, low-profile, coronary stent measuring 4.0 mm in diameter and 14 mm in length (GFX; Arterial Vascular Engineering, Santa Rosa, CA) was carefully navigated across the lesion. The stent position was confirmed by digital subtraction angiography before balloon inflation to 12 atmospheres and primary stent placement (Fig. 2). After stent placement, the balloon was deflated and carefully withdrawn. Post-deployment digital subtraction angiography showed excellent resolution of the stenosis, with the presence of a small, non-hemodynamically significant, dissection flap.

The patient was aroused from anesthesia and transferred to the intensive care unit according to the routine procedure. He continued to receive heparin therapy, as well as daily doses of clopidogrel (Plavix; Sanofi Pharmaceuticals, New York, NY) (75 mg) and aspirin (325 mg). Immediately postoperatively, the patient was noted to have transient right arm weakness (3–4 on a scale of 0–5), which was completely resolved by 6 hours after the procedure. On postprocedure Day 1, he was able to sit in a chair without any complaints of ataxia, nausea, or vomiting. However, in the evening of postprocedure Day 2, he was noted to have worsening right arm weakness, despite exhibiting a therapeutic partial thromboplastin time. The hemiparesis progressed to complete hemiplegia in the subsequent 12-hour period. The patient underwent computed tomographic scanning, which revealed a region of low attenuation in the left pons that was consistent with ischemia or infarction (Fig. 3).

The patient was taken to the angiography suite, where left vertebral artery injection showed intermittent occlusion of the left posterior cerebral artery and an extensive basilar artery dissection flap, originating distal to the stent and extending to the origin of the left posterior cerebral artery. The possibility of thrombus at the basilar apex was considered; therefore, a 6-French guide catheter (Envoy; Cordis Endovascular)



cerebral artery. A decision was then made to seal the inflow zone of the dissection. A 300-mm, 0.014-inch, exchange microguidewire (Stabilizer; Cordis Endovascular) was navigated into the true lumen of the basilar artery and into the origin of the left posterior cerebral artery. A second flexible stent (3.5 mm in diameter, 14 mm in length, GFX; Arterial Vascular Engineering) was then carefully navigated to traverse the first

The right hemiplegia of the patient was not immediately resolved after the second procedure. The patient continued to receive heparin, as well as Plavix and aspirin, and experienced a slow recovery, showing mild improvement in right arm (2–3 on a scale of 0–5) and leg (1–2 on a scale of 0–5) motor function before discharge to a rehabilitation facility. Significant recovery occurred after discharge from the hospital to the acute rehabilitation facility.

The patient returned 3 months after discharge from the hospital; he had been receiving Coumadin (DuPont Pharmaceuticals, Wilmington, DE) (international normalized ratio, 3), Plavix, and aspirin. His right-sided hemiparesis had nearly fully resolved, enabling him to walk independently and perform all activities of daily living without assistance. He denied headache, dizziness, numbness, and difficulty with swallowing or speech. His mental status was normal, with excellent cognition. He had intact sensation but mild residual left facial weakness and right hemiparesis (4 on a scale of 0–5 in the upper extremities and 4+ on a scale of 0–5 in the lower extremities), with resultant minimal circumduction on the right and decreased arm swing.

Percutaneous transluminal angioplasty for treatment of intracranial atherosclerosis has been shown to be beneficial in selected cases of symptomatic intracranial stenosis that is refractory to

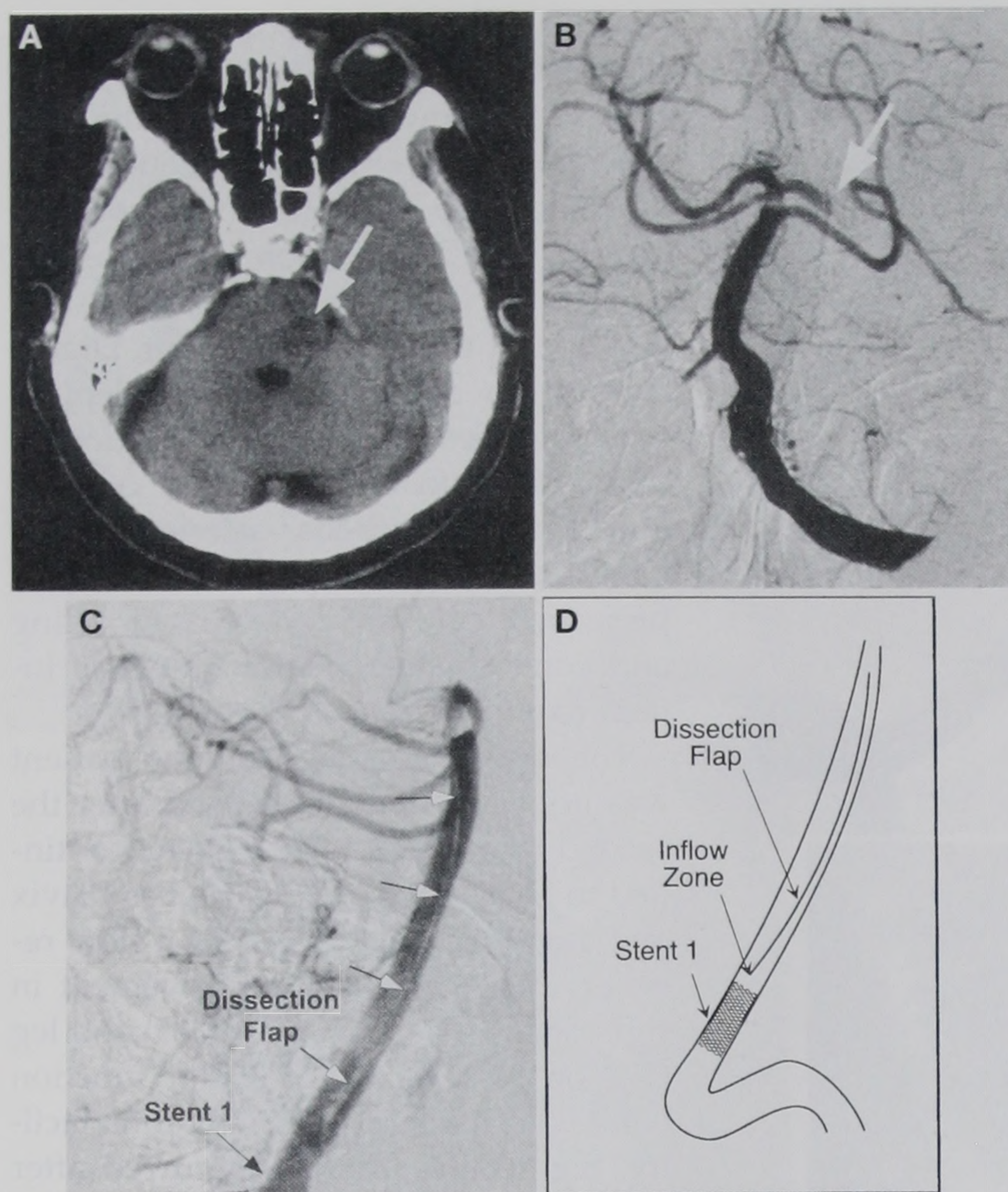


FIGURE 3. A, axial computed tomographic scan of the brain obtained on postprocedure Day 2, revealing the new finding of a left pontine focus of ischemia or infarction (arrow). B, subsequent digital subtracted angiogram with left vertebral artery injection (anteroposterior projection), showing intermittently poor filling of the left posterior cerebral artery (arrow), with no significant improvement after selective infusion of 400,000 units of

urokinase into the basilar apex. C, digital subtracted angiogram (lateral projection), revealing a sharply delineated (arrows) intimal dissection starting in the left vertebral artery, beyond the previously deployed stent. The dissection involves the entire basilar artery and ends at the origin of the left P1 segment. D, schematic illustration of the relative inflow zone of the dissection, with respect to the deployed stent.

maximal medical management (3), despite a 30-day morbidity rate of 11.7%. For a series of 12 patients who underwent balloon angioplasty for treatment of arteriosclerotic disease of the distal vertebral and basilar arteries at our center, Terada et al. (14) reported successful outcomes for 8 patients and a technical complication rate of 30%, including two dissections and two thromboembolic events. This case report illustrates a number of unusual and important features of vessel dissection in the vertebrobasilar system. The first point concerns the extent of propagation of the dissection to the apex of the basilar artery and its impingement on the left P1 origin. Although angiography performed after the neurological worsening on postprocedure Day 2 suggested thrombotic occlusion, the dynamic and intermittent filling of the left P1 segment during contrast injection, the lack of improvement in response to thrombolysis, and the angiographic dem-

onstration of the large extent of the dissection flap enabled recognition of the actual reason for the dynamic occlusion. The second point involves localization of the inflow zone of the dissection, which is responsible for the "windsock" effect. The third critical point concerns the correct identification of the true lumen within the basilar artery before deployment of the second stent. Observation of the dissection and the true lumen is not readily achieved without the use of high-resolution digital subtraction angiography.

Spontaneous dissections of the extracranial portion of the vertebral artery are associated with a higher risk of ischemic complications, particularly involving the brainstem (4). Intimal dissection is a well-recognized complication of percutaneous balloon angioplasty for treatment of atherosclerosis and is usually an acceptable consequence if it is not flow-limiting. Although the current case involves atherosclerotic stenosis, there have

been a number of recent reports of stents deployed in the intracranial vertebral artery as a scaffolding for treatment of sidewall pseudoaneurysms (9, 13) or fusiform aneurysms (6). Although there are significant differences in the physical properties and architecture of the coronary and cerebral vasculatures, some operators argue that the lack of dissection after percutaneous transluminal angioplasty in the coronary circulation indicates a greater propensity for restenosis (12).

Oversizing of the first stent used to treat the stenosis may have contributed to the initial dissection. Another possibility may have been a rapid rate of inflation. Blankenship et al. (2) reported an inverse relationship between the rate of inflation and the risk of dissection. Such a relationship seems counterintuitive, however. The Arterial Vascular Engineering stent is premounted on its delivery balloon catheter. During inflation, the balloon is designed to expand initially at the distal and proximal ends (resembling a dumbbell) and then in the center section containing the stent. This sequential expansion is intended to decrease the risk of stent migration or slippage across the area of stenosis during deployment. We hypothesize that a rapid rate of inflation might transiently overexpand the proximal and distal ends of the delivery balloon before stent expansion, as illustrated in Figure 5B. The Arterial Vascular Engineering stent used in this case is considered a second-generation, low-profile stent; it is flexible and can be used to navigate tortuous vessels. It was previously placed within a Wiktor-type stent to treat a residual dissection in the coronary circulation that was not adequately covered by the primary stent (5). It is doubtful that the uneventful passage of the second stent within the first would have been possible with an earlier-generation device, in the vertebrobasilar system.

It is theoretically possible that the presence of a brachial hemodialysis arteriovenous fistula, with consequently low downstream subclavian resistance, might have contributed to the symptoms exhibited by the patient. Kotval et al. (8) reported a case of subclavian steal induced by such an ipsilateral fistula.

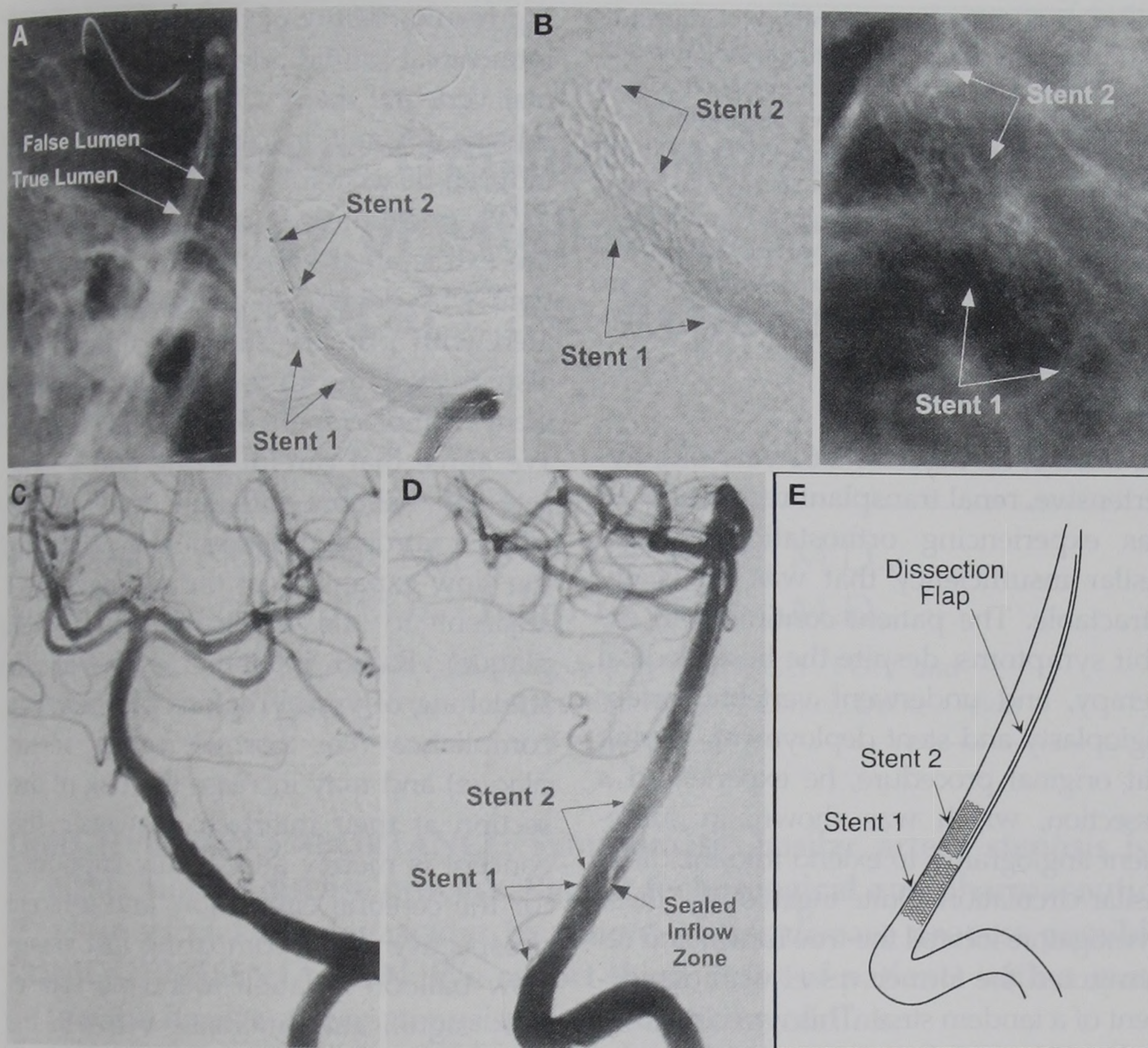


FIGURE 4. *A, left*, digital unsubtracted angiogram (lateral projection), showing the Stabilizer exchange microguidewire within the true (posterior) lumen, ending in the left P1 segment, along with the outline of the anterior false lumen; *right*, digital subtracted angiogram (anteroposterior projection), revealing the undeployed second GFX stent (3.5 mm × 14 mm) (Stent 2) across the inflow zone of the dissection, downstream from the previously placed proximal stent (Stent 1). *B, digital subtracted (left) and unsubtracted (right) angiograms (anteroposterior projection), showing the stent after deployment. C and D, digital subtracted angiograms (C, anteroposterior projection; D, lateral projection), showing resolution of the dynamic occlusion of the left P1 segment and excellent filling of both posterior cerebral and superior cerebellar arteries, as well as the tandem distal stent deployed at the site of the inflow zone of the dissection, with a significant decrease in the size of the anterior compartment, or false lumen, of the basilar artery dissection (D; compare*

with Fig. 3C). *E, schematic illustration of the relative positions of the two stents with respect to each other and to the inflow zone of the dissection, which has been sealed by the deployment of Stent 2.*

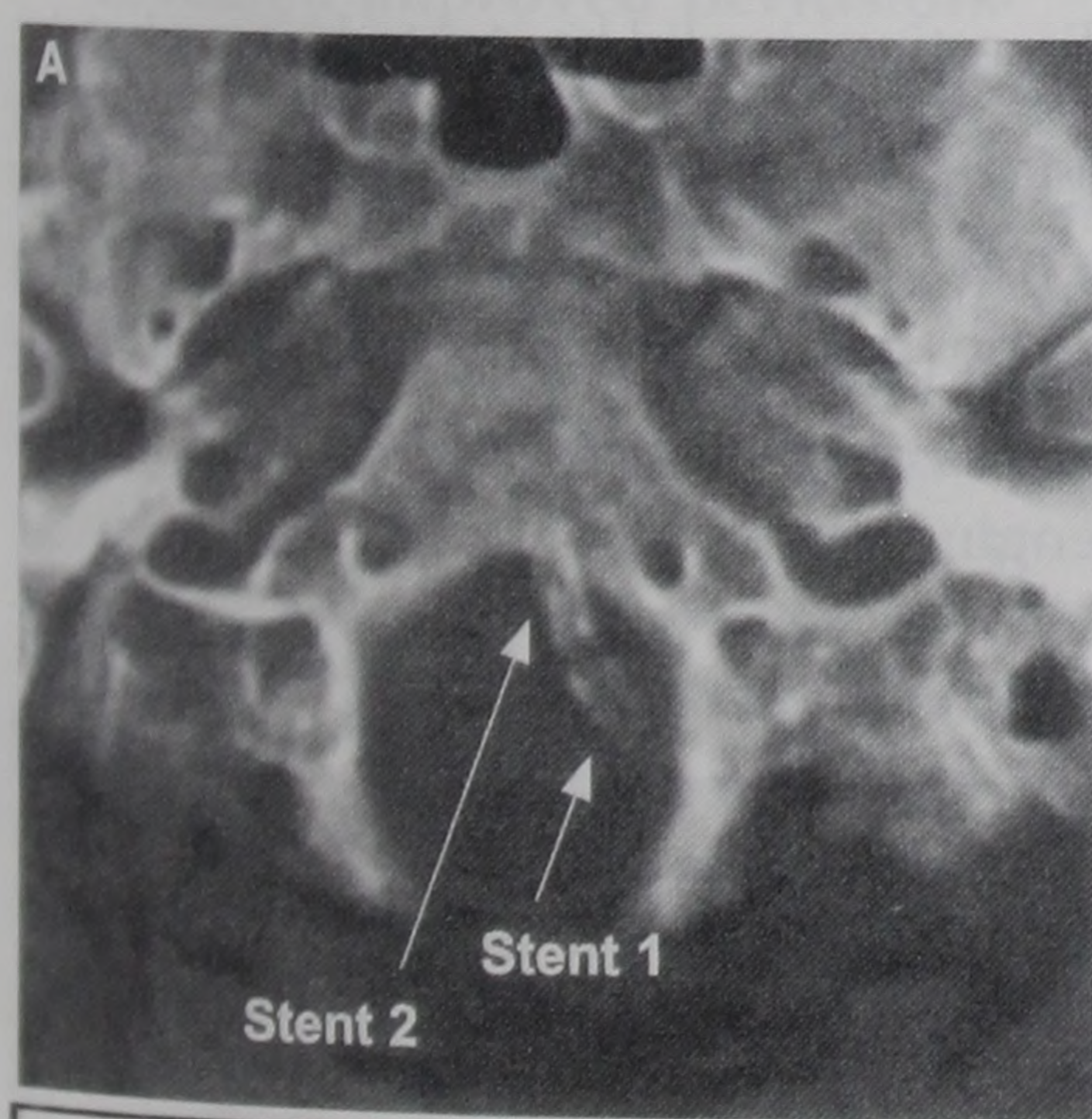
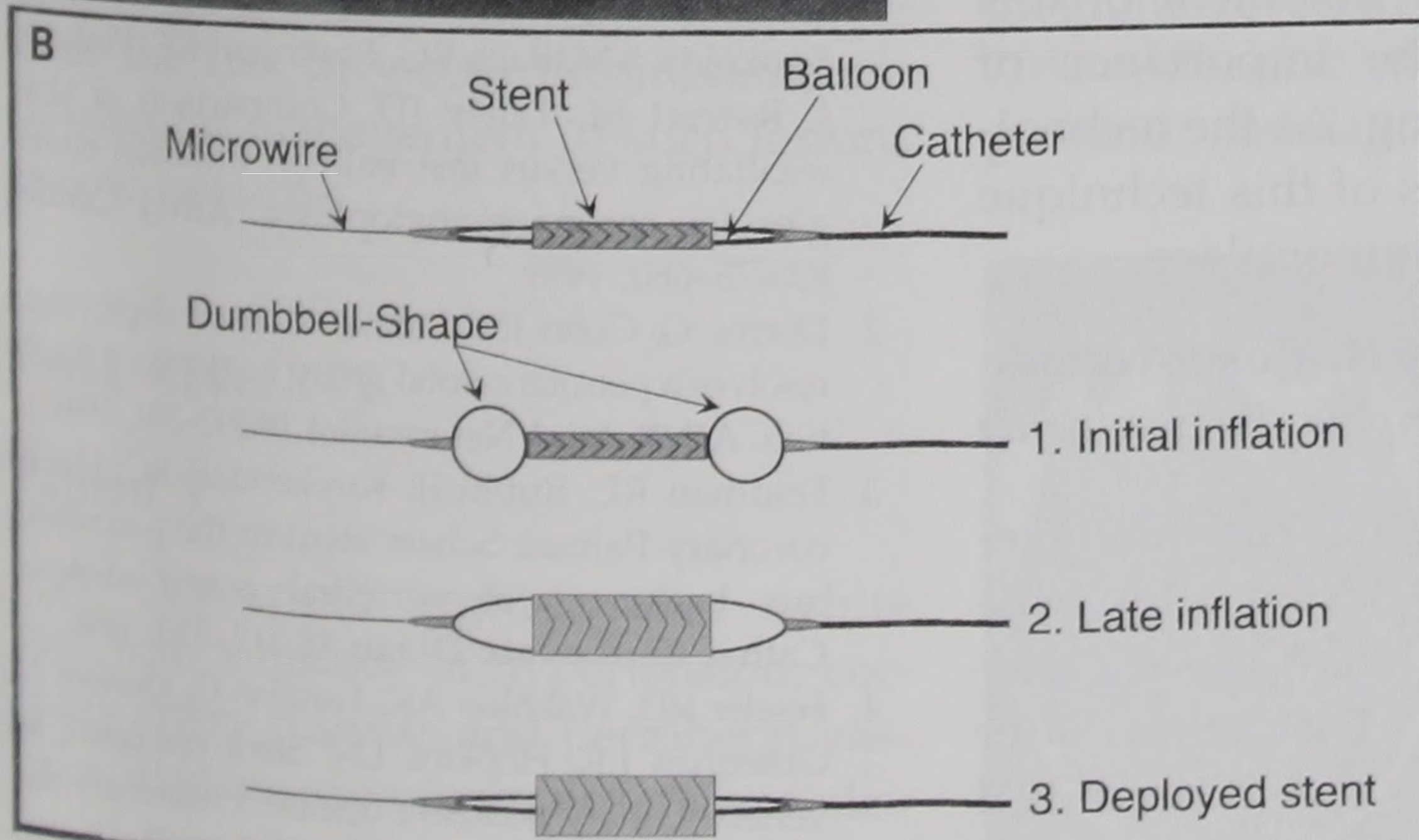


FIGURE 5. *A, axial computed tomographic scan, showing the two deployed stents and their relative positions with respect to the foramen magnum and the clivus. B, schematic illustration of the inflation pattern of the balloon-mounted stent and the*



dumbbell shape assumed by the distal and proximal portions of the balloon during initial inflation.

However, Doppler insonation of the left vertebral artery after deployment of the first stent showed no effect of pressure-cuff occlusion proximal to the fistula on systolic or diastolic velocities, arguing against such a phenomenon in this patient.

CONCLUSION

In summary, intracranial vertebral artery balloon angioplasty and stenting is a high-risk procedure reserved for patients who have experienced failure of maximal medical therapy. The complication and management presented in this report highlight the importance of careful selection and sizing of balloon and stent catheters and meticulous angiographic analysis. The need for proper preoperative analysis is made more pressing by the availability of a new generation of flexible stents that enable endovascular surgeons to readily navigate areas of the cerebral circulation that were previously considered unreachable.

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COMMENTS

Malek et al. describe a 61-year-old, hypertensive, renal transplant recipient who was experiencing orthostatic vertebrobasilar insufficiency that was medically intractable. The patient continued to exhibit symptoms, despite the best medical therapy, and underwent vertebral artery angioplasty and stent deployment. During that original procedure, he experienced a dissection, which was shown, in subsequent angiograms, to extend into the distal basilar circulation. Quite ingeniously, these investigators located the true lumen and reconstituted the former vessel with deployment of a tandem stent. This was obviously performed as a salvage procedure, and the patient did exhibit residual neurological deficits; however, it is likely that the outcome would have been worse if this procedure had not been performed.

Other important points can be obtained from this report, most importantly that overextension of a stent can lead to dissection, which is their hypothesis. I think that individuals who perform these procedures should pay great attention to this finding, which is supported by the interventional cardiac literature.

Oversizing of the first stent, as well as the weight of inflation of the balloon delivery device, might have contributed to the initial dissection, as the authors pointed out. However, as they also note, the complication and treatment of this patient emphasize the importance of stent and balloon sizing. As the technology improves, reports of this technique will become more commonplace.

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The authors describe the management of an iatrogenic basilar artery dissection after primary stenting of vertebral artery stenosis. Dissection after balloon angioplasty is a well-described complication, with potentially devastat-

ing results. Many of the early reports of intracranial carotid artery (2) and extracranial vertebral artery (3) stenting were for salvage therapy in cases of angioplasty-induced dissection.

We encountered similar problems in the extracranial vertebral artery of a patient who developed a dissection associated with primary stent placement. We also treated the dissection distal to the stent with placement of a second stent (4). We agree that careful sizing and a slow rate of balloon inflation may guard against iatrogenic dissection by allowing the slow expansion of the normal vessel adjacent to the diseased vessel (with plaque). Rapid inflation results in the stretching of vessel regions that differ in compliance (i.e., normal vessel versus plaque) and may increase the risk of dissection at their interface. However, this concept is merely anecdotally supported for the cerebral circulation, and a recent prospective study comparing fast versus slow balloon inflation found no statistically significant difference between the two techniques with respect to dissection in the coronary vasculature (1). It is important to carefully examine poststent angiograms, to rule out dissection, before the guidewire is removed. Any significant dissection should be stented immediately, if possible. The authors are fortunate that they were able to locate the true lumen of the dissected vessel 1 day after the initial procedure. The authors report excellent technical and clinical results for a challenging case.

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