

Endovascular Stenting of an Acutely Thrombosed Basilar Artery: Technical Case Report and Review of the Literature

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OBJECTIVE AND IMPORTANCE: The goal of this report was to describe the successful percutaneous endovascular use of a Gianturco-Roubin-2 coronary stent in the treatment of an acute atherothrombotic occlusion of the basilar artery. To our knowledge, the percutaneous endovascular deployment of an intra-arterial stent for the treatment of an acute atherothrombotic occlusion of the basilar artery and the percutaneous endovascular placement of a Gianturco-Roubin-2 stent in the basilar artery have not been previously reported.

CLINICAL PRESENTATION: An 83-year-old man presented with a recurrent, transient, locked-in syndrome resulting from a lower basilar artery occlusion caused by vertebrobasilar thrombosis superimposed on severe proximal basilar artery atheromatous stenosis.

INTERVENTION: After successful superselective intra-arterial thrombolysis of the vertebrobasilar clot, balloon angioplasty of the underlying basilar artery stenosis was performed, without significant angiographic improvement. Percutaneous endovascular deployment of a Gianturco-Roubin-2 coronary stent of 4-mm diameter was subsequently performed, with excellent angiographic results.

CONCLUSION: The patient made a very good neurological recovery but unfortunately died as a result of cardiogenic shock and sepsis. Detailed neuropathological follow-up results are presented; stent patency was revealed in the postmortem examination. The anatomic and pathophysiological considerations of basilar artery stent placement for the treatment of acute basilar artery occlusion related to atherosclerotic stenosis are discussed. (Neurosurgery 44:667-673, 1999)

Key words: Atherosclerosis, Balloon angioplasty, Thrombolysis, Vertebrobasilar insufficiency

The basilar artery is frequently a site for the development of atheromatous plaque (19). The clinical sequelae include ischemia, thromboembolisms,

dolichoectasia, aneurysm formation, and dissection. Patients presenting with symptoms of ischemia referable to atherosclerotic basilar artery stenosis are

usually initially treated medically using antiplatelet agents and/or anticoagulation therapy. For the subset of patients for whom medical management fails and for those who present with basilar artery occlusion as an emergency, the therapeutic options are limited. The results of surgical bypass grafting for basilar artery narrowing or occlusion are disappointing (25), especially if the grafting is performed during an episode of ischemia. Successful transluminal angioplasty of the basilar artery was first reported in 1980 by Sundt et al. (25); however, the body of data remains small. When angioplasty is unsuccessful in relieving stenosis, the treatment options are essentially exhausted. Endovascular stent placement has been successfully used in the treatment of atheromatous plaque in the coronary and peripheral arteries, and a recent report described the first successful percutaneous endovascular deployment of a stent within the basilar artery (15). We demonstrate the technical feasibility of endovascular stent deployment for the treatment of basilar artery atherosclerosis with acute thrombosis, and we discuss the relevant clinicopathological considerations.

CASE REPORT

An 83-year-old man with a prior history of hypertension, coronary artery disease, and hypercholesterolemia presented with a 6-day history of headaches and presyncopal episodes. A few days later he experienced two episodes, each lasting a few hours, of a fluctuating locked-in state associated with bilateral facial weakness, quadriparesis, and bilateral limb paresthesia. Consciousness was preserved throughout, suggesting preservation of midbrain perfusion. The patient spontaneously recovered from these episodes, with residual mild dysarthria and minimal right-sided hemiparesis. Magnetic resonance imaging scans demonstrated intraluminal thrombus within both distal vertebral arteries, extending into the proximal basilar artery. A small area of accompanying signal alteration, consistent with ischemia, was present in the left ventral

medulla. A cerebral angiogram confirmed bilateral distal vertebral artery occlusions above the level of the posterior inferior cerebellar arteries, with reconstitution of the rostral basilar artery via a right posterior inferior cerebellar artery to right anterior inferior cerebellar artery collateral anastomosis (Fig. 1). The vertebral arteries were codominant, of good caliber, and free of significant atherosclerotic disease in their extracranial portions. There was a

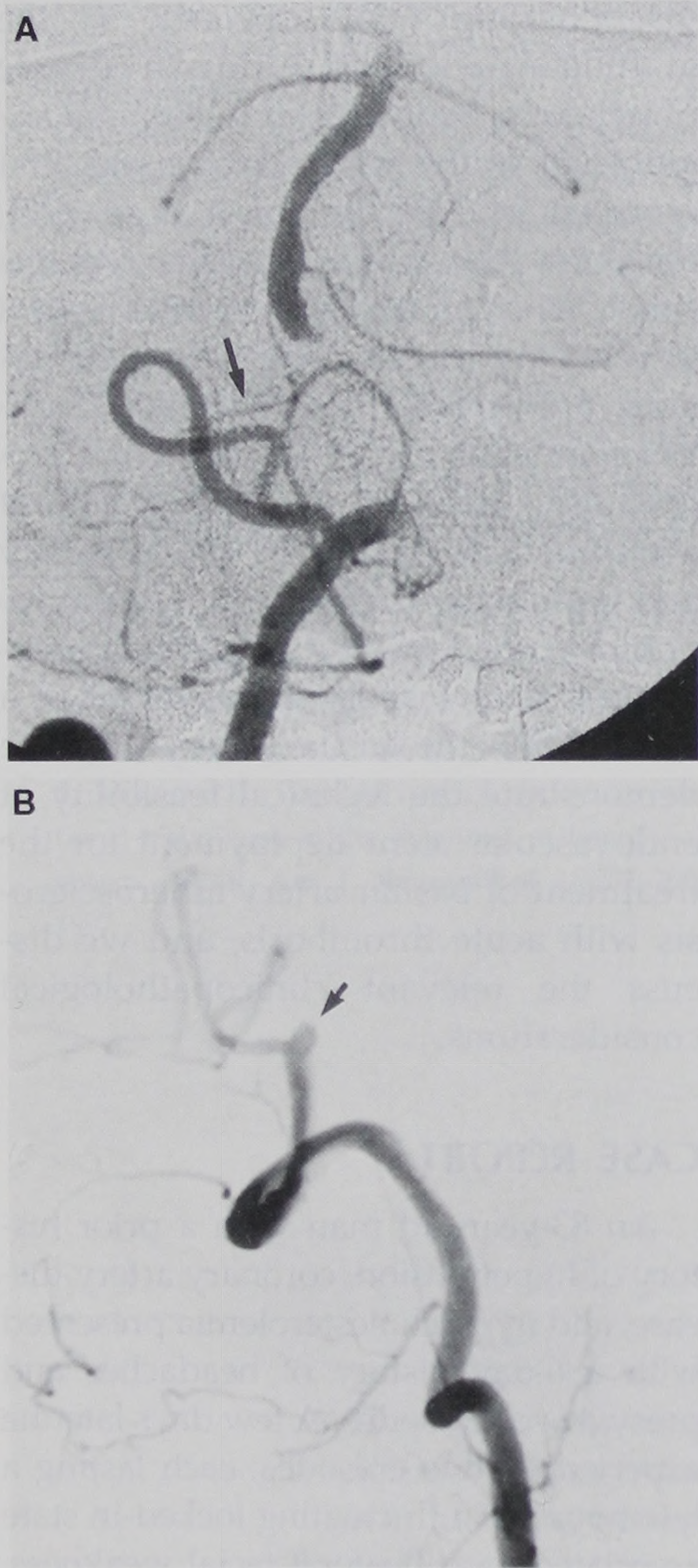


FIGURE 1. *A*, anteroposterior view of the posterior fossa during a right vertebral artery injection, showing reconstitution of the upper basilar artery via a right posterior inferior cerebellar artery-anterior inferior cerebellar artery anastomosis (arrow). *B*, lateral view of the posterior fossa during a left vertebral artery injection, showing complete occlusion of the left vertebral artery above the posterior inferior cerebellar artery origin (arrow), with no basilar artery reconstitution.

narrow left and an absent right posterior communicating artery.

At presentation, the patient was alert and orientated, with normal mental state examination findings. Cranial nerve examination results were unremarkable except for mild dysarthria; however, palatal movement was normal. Visual field testing yielded normal findings. There was very mild right hemiparesis (5-/5) with a right pronator drift. Plantar responses were downgoing. Sensory and coordination examination results were normal. The patient was transferred to our institution for emergency superselective intra-arterial thrombolysis.

TECHNICAL SUMMARY

Cerebral angiography performed immediately after arrival at our institution demonstrated findings similar to the previous angiogram (see above). Care was taken to ensure that the right femoral artery puncture was below the level of the inguinal ligament, because of the possibility of thrombolytic therapy.

General anesthesia was administered, and the patient underwent full heparinization (activated clotting time, >250 s), using an intravenously administered loading dose followed by a continuous intravenous heparin infusion. A 7-French guide catheter was selectively placed into the proximal left vertebral artery. A Rapid Transit microcatheter (Cordis Endovascular, Miami Lakes, FL) primed with an 0.014-inch steerable guidewire was advanced into the distal left vertebral artery, immediately proximal to the occlusive thrombus, and local thrombolytic therapy using urokinase (UK) was commenced. After infusion of 750,000 units of UK, there was substantial lysis of the proximal clot and reestablishment of antegrade basilar artery flow via the left vertebral artery, revealing severe underlying stenosis (14.5-mm length) of the basilar artery commencing at the vertebrobasilar junction and extending rostrally (Fig. 2A). The normal basilar artery diameter at its midpoint was approximately 3.6 mm. The microcatheter and guidewire were carefully advanced through this stenosis into the distal basilar artery clot, and an additional 100,000 units of UK were infused. The microcatheter was then retracted into the distal left vertebral artery, and an addi-

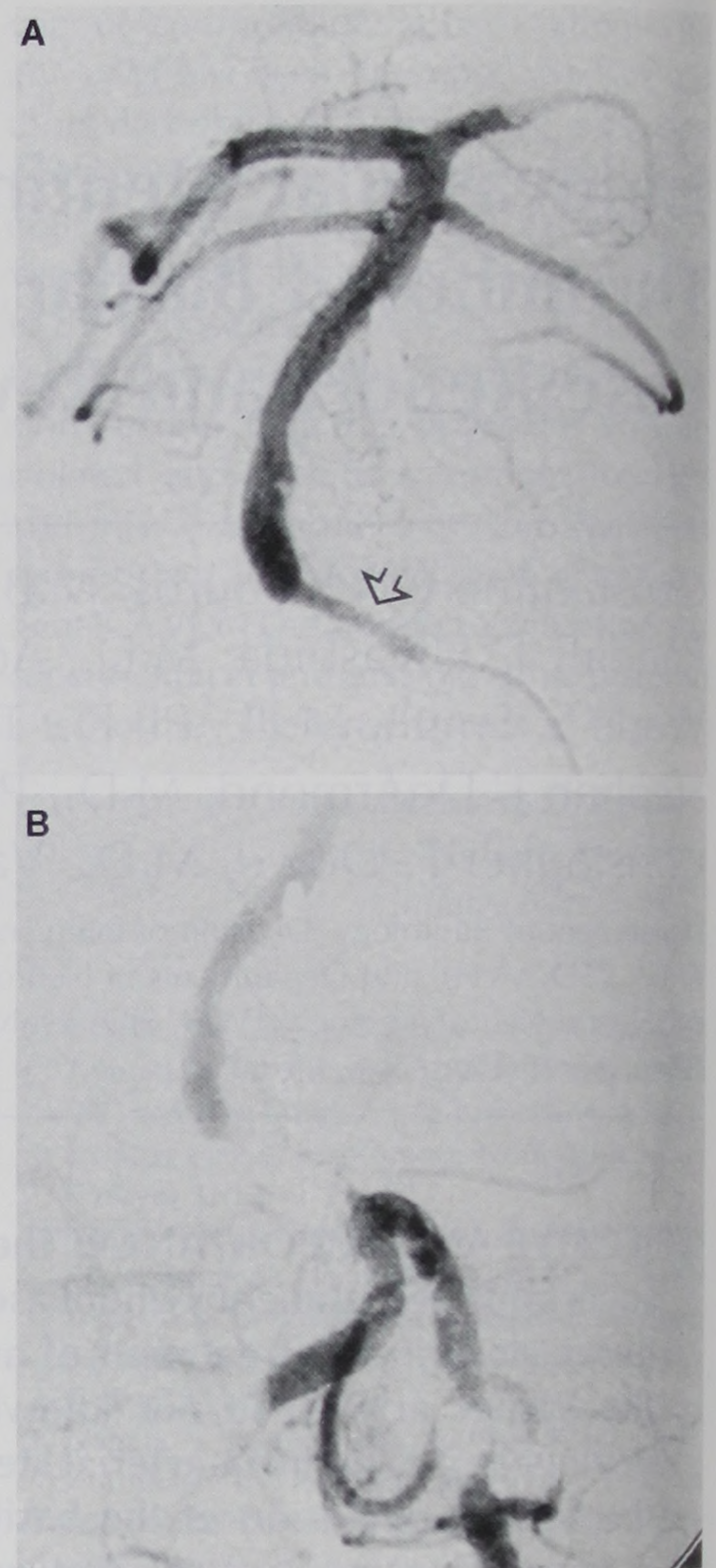


FIGURE 2. *A*, selective microcatheter injection showing antegrade flow within the basilar artery after local infusion of 750,000 units of UK and revealing underlying proximal basilar artery stenosis (open arrow). *B*, anteroposterior view of the posterior fossa after local infusion of 975,000 units of UK. There has been rethrombosis of the proximal basilar artery, with consequent weak filling distally.

tional 125,000 units were infused. After the administration of a total of 975,000 units of UK, there was only weak antegrade basilar artery flow via the left vertebral artery because rethrombosis at the site of basilar artery stenosis had occurred (Fig. 2B). It was also noted that a small hematoma was accumulating at the site of the previous right femoral artery puncture, necessitating the immediate application of a FemoStop compression device (Radi Medical Systems, Uppsala, Sweden), which remained in place for the remainder of the procedure.

With the tip of the microcatheter distal to the basilar artery stenosis, a 0.014-inch, 300-cm, exchange guidewire (Stabilizer; Cordis Endovascular) was placed in the distal left posterior cerebral artery. A coronary balloon angioplasty catheter (Valor; Cordis Endovascular) with a nominal balloon diameter of 3.5 mm and a length of 20 mm was then positioned across the basilar artery stenosis. Two brief balloon dilations were performed (with an intervening angiographic run), to pressures of 6 atmospheres (equivalent to a 4-mm diameter *in vitro*). *Figure 3* shows the final postangioplasty results, with severe residual basilar artery stenosis.

In view of the refractory nature of this plaque and the strong likelihood of rethrombosis if narrowing was not relieved, a balloon-mounted, Gianturco-Roubin-2 coronary stent (Cook Cardiology, Bloomington, IN), measuring 4 mm in diameter and 20 mm in length, was slowly and carefully advanced along the guidewire and positioned across the stenotic lesion. The balloon was inflated to a pressure of 6 atmospheres (recommended inflation pressure), resulting in satisfactory and uneventful stent deployment.

The balloon was carefully withdrawn into the distal left vertebral artery, taking care not to dislodge the stent, and

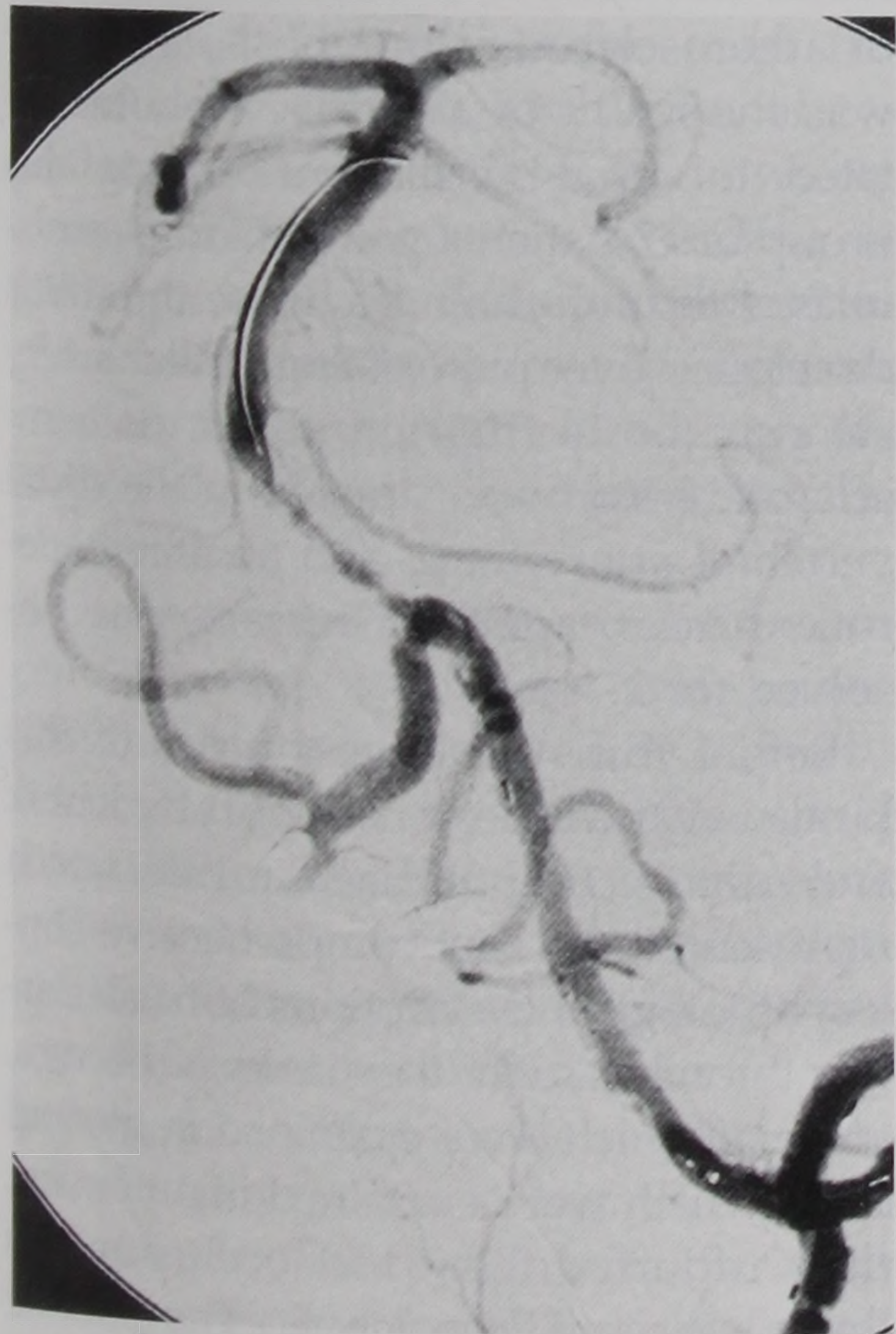


FIGURE 3. Anteroposterior view showing residual underlying severe proximal basilar artery stenosis after thrombolysis and angioplasty.

angioplasty of two moderately severe atherosclerotic lesions between the vertebrabasilar junction and the origin of the left posterior inferior cerebellar artery was undertaken, with satisfactory results. *Figure 4* shows the final angiographic result, with a basilar artery of normal caliber and patency of both vertebral arteries. There was evidence of small distal-branch occlusions within the right anterior inferior cerebellar artery and both posterior cerebral artery territories.

The right femoral sheath was left in place, as was the FemoStop device, maintaining constant pressure on the previous arterial puncture site. There was no evidence, however, of a significant groin hematoma, and hematocrit values remained stable for the next few days. Intravenous heparin treatment was continued, maintaining the activated partial thromboplastin time between 50 and 80 seconds. Treatment with aspirin (325 mg daily) and ticlopidine (250 mg twice each day) was commenced. The patient was transferred to the neurointensive care unit while intubated.

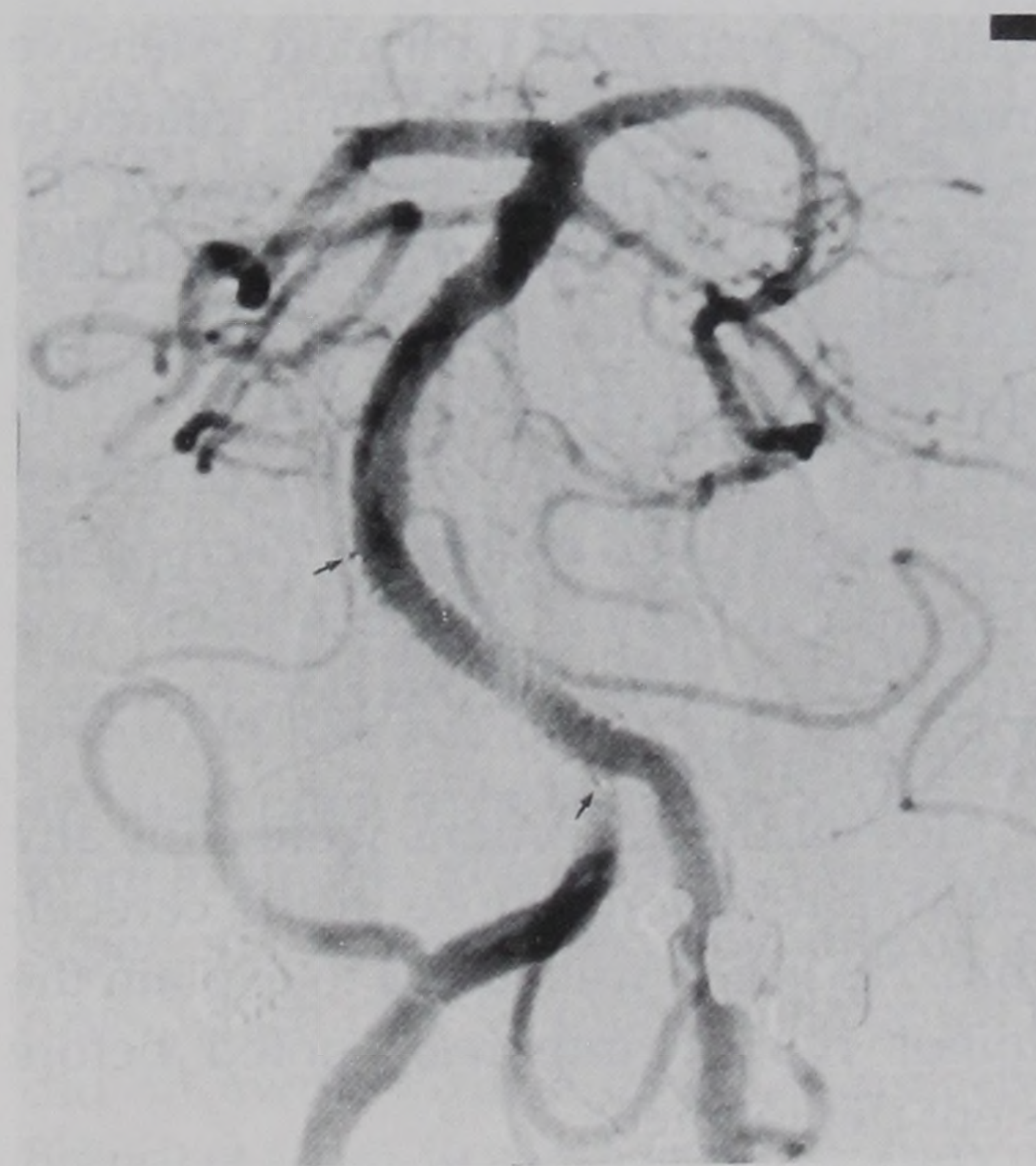


FIGURE 4. Anteroposterior view showing the final angiographic appearance after stent deployment within the proximal basilar artery and angioplasty of the distal left vertebral artery (small arrows, proximal and distal ends of the stent). The distal stent margin is just beneath the left anterior inferior cerebellar artery origin. The proximal stent margin extends a few millimeters into the left vertebral artery, partially covering the right vertebral artery outflow.

CLINICAL OUTCOME

The patient underwent extubation 24 hours later but required reintubation soon after because of pulmonary edema resulting from cardiac failure. Neurological examination results were normal except for minimal pre-existing right hemiparesis, which had not worsened. Eye movements, visual fields, and palate and tongue movements remained normal; however, the patient had acquired some pharyngeal dysmotility, which may have been the result of endotracheal intubation. Pharyngeal function significantly improved during the next few days, and the neurological status of the patient remained stable. His course in the intensive care unit was complicated by a periprocedural myocardial infarction (resulting in cardiac failure and pulmonary edema) and pneumococcal sepsis. The patient refused invasive cardiological therapy and, in view of ongoing coronary ischemia, tissue plasminogen activator was administered intravenously on the seventh postoperative day. This precipitated an acute decrease in the hematocrit value, as a result of a retroperitoneal hematoma necessitating blood transfusion. The patient died on the tenth postoperative day as a result of sepsis and intractable cardiac failure leading to cardiogenic shock.

PATHOLOGICAL RESULTS

Postmortem examination was performed approximately 15 hours after death, 10 days after the placement of the basilar artery stent. Gross examination of the basilar vessels revealed a metallic coil stent within the left vertebral and basilar arteries. The stent extended 3 mm into the distal end of the left vertebral artery and occupied two-thirds of the length of the basilar artery (*Fig. 5*). The distal vertebral arteries and basilar artery were patent. Atherosclerotic plaques measuring 0.5 and 1.0 cm in greatest dimension were noted rostral and caudal to the stent, respectively. These resulted in asymmetric circumferential luminal narrowing. Hemorrhage was noted within the wall of the basilar artery, over the rostral plaque. Microscopic examination of the vertebral and basilar arteries revealed severe atherosclerosis with recent hemorrhage between the atheromatous plaques and the



FIGURE 5. Photograph of the basilar artery in the postmortem examination. The stent can be clearly observed through the thin basilar artery wall (arrows). Note the relative wide spacing of the stent struts and extensive atherosclerotic disease. The distal stent margin is just beneath the left anterior inferior cerebellar artery origin, and this correlated with the junction of the superior two-thirds and inferior one-third of the pons in the gross specimen.

muscularis, consistent with mechanical disruption of the atherosclerotic plaque at the time of stent placement (Fig. 6).

Coronal sections of the brain revealed multiple areas of discoloration consistent with hemorrhagic infarctions. These areas were noted in the right parieto-occipital region and within the right occipital pole, in the distribution of the right posterior cerebral artery. The largest was a 1.5- × 1.0-cm hemorrhage that involved the inferior 20% of the visual radiations and adjacent occipital cortex. Bilateral superior cerebellar lesions that were approximately 1.0 cm in greatest dimension and consisted of multiple petechiae were present. In the pons, four petechiae of 0.2 to 0.3 cm in greatest dimension were lo-



FIGURE 6. Photomicrograph of the midbasilar artery, showing eccentric atherosclerotic plaque with hemorrhage (iatrogenic) between the plaque and the vessel wall (arrow).

cated in the right base. In the medulla, a 0.5-cm hemorrhagic lesion was located in the right dorsolateral tegmentum.

Microscopic examination of the brain, cerebellar, and pontine lesions demonstrated infarctions 1.5 to 2 weeks of age, corresponding to the time of the clinical locked-in episodes and stent placement. Microscopically, the hemorrhage in the right dorsolateral tegmentum of the medulla was associated with more extensive infarction of the medulla. This infarction extended from the posterior edge of the right pyramid to the hypoglossal nucleus and laterally to the right vestibular nuclei and descending spinal trigeminal tract. In addition to abundant vascular proliferation, there was an exuberant macrophage response, indicating that this lesion was up to 1 week older and therefore predated the intervention.

Most of the infarctions were clinically silent, particularly those in the cerebellum. No lesion was found to explain the minor right hemiparesis noted before stent deployment.

DISCUSSION

As early as 1934, Lhermitte and Trelles (17) noted that, of all of the cerebral vessels, the basilar artery was most commonly affected by atherosclerosis. In one of the largest series of its kind, Fisher et al. (12) in 1965, after examining the intracranial and extracranial cerebral vasculature in 178 routine autopsy cases, concluded that the basilar confluence was a site of predilection for ath-

erosclerosis. They observed that the lower part of the basilar artery, at the confluence of the vertebral arteries, was the primary site of affliction in the early stages of atheroma formation and that this pattern of distribution remained constant as the atherosclerotic disease progressed. In the proximal basilar artery, atherosclerosis was more severe on the side of the larger vertebral artery and involved the corresponding half of the perimeter of the basilar artery (the intracranial portion of the dominant vertebral artery was usually more severely affected). Those authors also noted a strong association between hypertension and the severity of atherosclerotic disease in the vertebrobasilar system. In their series, thrombotic occlusions of the larger intracranial arteries were limited to the vertebrobasilar system and were usually symptomatic, in contrast to extracranial vertebral artery occlusions, which were usually asymptomatic.

Eight years later, Castaigne et al. (7), noting the relative scarcity of postmortem data regarding the patterns of occlusions in the vertebrobasilar system, in an 8-year period collected 44 postmortem specimens demonstrating vertebrobasilar system occlusions. In their series there were 18 basilar artery occlusions, of which 17 (94%) were the result of atherosclerotic thrombosis; the other was the result of a cardiac embolus related to atrial fibrillation. Among this group of 17 patients with atherosclerosis-related thrombus, an occlusive thrombus developed over pre-existing basilar artery stenosis for 14 (78%), a primary atherosclerotic lesion was situated in the distal vertebral artery for 2, and an atherosclerotic basilar artery aneurysm was observed for 1.

Before this, the largest series of this kind was the milestone report by Kubik and Adams (16) published in 1946, which represented the first comprehensive clinicopathological descriptions of basilar artery thrombosis. For their series of 18 cases, some of which were examined in life and all of which were examined in autopsies, they reported 11 (61%) occlusions as thrombotic and 7 as embolic. The embolic source was cardiac in four cases (three cases of subacute bacterial endocarditis and one of atrial fibrillation), there was a single unusual case of a tumor embolus originat-

ing from invasion of the pulmonary vein by metastatic carcinoma, and the source was unknown in the remaining two cases. The discrepancy between the two sets of data with respect to embolic causes is partially explained by the increased incidence of subacute bacterial endocarditis in the older study. Kubik and Adams (16) also noted that in every case of thrombotic occlusion there was extensive accompanying basilar artery atherosclerosis and that atheroma-related occlusive thrombus was less frequent in the distal basilar artery, an observation in accordance with the data of Castaigne et al. (7).

According to the data cited above (7, 16) and other results (8), the chief cause of proximal basilar artery occlusion is *in situ* thrombosis. Indeed, these studies identified *in situ* thrombosis as the major cause of basilar artery occlusion; however, the true frequency of embolic occlusions may be underestimated because these are pathological data and some emboli may spontaneously lyse or fragment and pass distally. The true overall frequency of embolic occlusions is probably better assessed by angiographic studies; however, it is difficult to draw firm conclusions from the published data. This contrasts somewhat with occlusion of the major branches of the internal carotid artery, for which embolism is considered the most frequent cause (12, 18). Occlusion of the posterior cerebral arteries is, however, almost always embolic, according to Castaigne et al. (7), and occlusion of the distal basilar artery (above the superior cerebellar artery origin), producing the "top of the basilar" syndrome, is most often embolic (5).

Of interest in this regard are the recent growing awareness and understanding of the role played by hemodynamic factors in the pathogenesis of atherosclerosis. Although an extensive review is beyond the scope of this article, it is becoming increasingly well recognized that atherosclerosis has a propensity to develop in the region of the vessel wall exposed to low shear stress and complex flow patterns (6, 13). Much of this work has focused on the distribution of plaques at arterial bifurcations. Interestingly, the basilar artery is the only large artery in human subjects where two flows merge; in this particu-

lar anatomic arrangement, the low-wall shear theory predicts the development of plaque near the apex and at both lateral walls of the basilar confluence (22), a prediction strikingly in accordance with the aforementioned pathological data.

The diagnosis of basilar artery occlusion portends a grave prognosis. When this condition is untreated the mortality rate is greater than 70% (2, 3, 7, 8, 11, 16). Despite the increased time usually considered to be available between the onset of ictus and the initiation of thrombolysis (compared with anterior cerebral circulation stroke) and the advent of modern superselective thrombolytic techniques, death is still a probable outcome (4, 9, 14, 26). A recent retrospective study in which 20 patients with angiographic proof of basilar artery thrombosis were treated with local UK therapy reported a mortality rate of 65% at 3 months (8). Other reports have provided somewhat greater encouragement, generally correlating the presence of patent arteries after treatment with survival (4, 14, 26). In the previously cited study of Cross et al. (9), complete recanalization was achieved in one-half of the cases and 60% of these patients survived, although only one-third achieved a "good" neurological outcome (modified Rankin score of 0-2 and Barthel Index score of 95-100). In that study the best predictors of survival were distal clot location and complete recanalization. The survival rate for the group with proximal or midbasilar clots was only 15%. A possible explanation for these findings, as offered by the authors, is that distal clots are more often embolic and hence may be more amenable to lysis than is thrombus superimposed on pre-existing atherosclerosis, which may be prone to recur (as it did in two of their cases). Similarly, Brandt et al. (4), in a retrospective study of 51 patients with acute basilar artery occlusion treated with local thrombolytic therapy, reported a low recanalization frequency (19%) among those with atherothrombotic occlusive disease, noting that signs of subcortical arteriosclerotic arteriopathy in computed tomographic scans were correlated with nonrecanalization. The survival rate for patients with atherothrombotic disease was only 6% and that for those with caudal occlusion sites (inferior to the anterior inferior cerebellar

artery origin) was 13%. In that study, age was also identified as an independent covariable associated with atherothrombotic occlusion; the oldest surviving patient for whom recanalization was achieved was 63 years of age. Our patient was 83 years of age. Because most studies stress the importance of achieving recanalization, it seems likely that safe and effective methods of treating underlying atherosclerotic stenosis are desirable if patient outcomes are to be improved.

In our patient there was underlying stenosis of the proximal basilar artery, which caused rethrombosis after recanalization had been initially achieved with local thrombolysis. Balloon angioplasty was not successful (despite dilation to a diameter of 3.5 mm), and therefore stent placement was regarded as the only remaining therapeutic option. The data regarding the use of stents within the vertebral arteries for the treatment of atheromatous narrowing is sparse (10, 21, 24). Storey et al. (24) first reported the successful use of stents for the treatment of atherosclerotic narrowing of the vertebral artery origin in three patients. A report by Piotin et al. (21), in which five patients underwent endovascular placement of balloon-expandable coronary stents for symptomatic vertebral artery stenoses, described satisfactory angiographic results with resolution of symptoms for all patients at 14-month follow-up examinations. Higashida et al. (15) reported the first clinical use of a stent (Palmaz-Schatz; Johnson and Johnson Interventional Systems, Warren, NJ) in the basilar artery; however, this was for the treatment of a ruptured fusiform aneurysm, with the stent primarily acting as a scaffold to support endoaneurysmal, electrolytically detachable coils. Sekhon et al. (23) recently reported a similar procedure for the treatment of a wide-necked aneurysm arising from the intracranial vertebral artery.

We chose to use a coronary stent because high intrinsic stent flexibility is necessary for the safe and reliable navigation of the tortuosities of the vertebral artery. The Gianturco-Roubin-2 is a preloaded, balloon-expandable stent made of stainless steel. When deployed, the stent interstices are relatively wide-spaced (this can be clearly seen in the

postmortem specimen in Fig. 5). These relatively wide apertures may be disadvantageous if the stent is used as a scaffold to prevent herniation of electrolytically detachable coils into the parent arterial lumen in the treatment of fusiform aneurysms. In the basilar artery, however, wide apertures (and relatively thin struts) may be theoretically advantageous in preventing the covering and occlusion, by stent metal-work, of the ostia of small perforating vessels of the brain stem. In our case, pathological examination did not show evidence of brain stem infarction related to perforator vessel origin occlusion by the stent. The histological appearance of the right olivary lesion suggested that it predated the procedure, and the small petechial pontine hemorrhage sites were considered to be too focal to represent perforator origin occlusions by the stent. The wide stent apertures may have also helped to maintain the patency of the right vertebral artery, because the proximal end of the stent extended 3 mm into the left vertebral artery and therefore at least partially covered the distal right vertebral artery ostium. We chose to use a 4-mm-diameter stent in a basilar artery with a mid-diameter of 3.6 mm. Some authors previously advocated underdilating (no more than one-half of the normal mid-diameter) basilar artery atherosclerotic plaque when performing balloon angioplasty, because of the insubstantial muscularis and adventitial layers possessed by the basilar artery wall and the consequent elevated risk of perforation (1, 20). This may be of even greater relevance in the presence of eccentric plaque. Underdilation of plaque by simple balloon angioplasty may provide a sufficient increase in quantitative blood flow and may not result in a significant likelihood of restenosis or thrombosis (at least in the short term); however, preservation of stent patency at diameters of ≤ 2 mm seems less sure. Furthermore, the risk of vessel perforation may not be as great when a stent is used, compared with simple balloon angioplasty, with the stent possibly providing additional wall support.

Finally, the fact that, despite successful intervention and a good neurological outcome, the eventual death of the patient was at least partly caused by a

retroperitoneal hematoma, possibly related to a previous, relatively high inguinal puncture, should be considered a salutary warning. In any case of diagnostic angiography that may lead to subsequent thrombolytic therapy, particular care should be taken to ensure that the arterial puncture is below the inguinal ligament.

SUMMARY

The vertebrobasilar junction and proximal basilar artery are sites of predilection for atherosclerotic narrowing. In cases of basilar artery occlusion resulting from superimposed thrombus, attempted recanalization with thrombolytic therapy may be unsuccessful (resulting in a poor prognosis) unless primary treatment of the stricture is undertaken. If angioplasty fails, then stent placement remains one of the few remaining therapeutic options. This clinical scenario is most frequently encountered in elderly patients, for whom endovascular interventions are more likely to be hindered by vessel tortuosity. A flexible and conformable stent is desirable if successful endovascular delivery is to be routinely achieved. The authors report, for the first time, the feasibility of endovascular placement of a coronary Gianturco-Roubin-2 stent into the basilar artery for the treatment of an acute basilar thrombotic occlusion related to underlying atherosclerotic stenosis.

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COMMENTS

Phatouros et al. describe an 83-year-old male patient who presented with recurrent transient locked-in syndrome resulting from lower basilar artery occlusion. Despite attempts to reopen the vessel with 975,000 units of urokinase, there continued to be only very meager antegrade basilar artery flow, demonstrating significant stenosis as the cause of the occlusion. This case report also illustrates that in many instances angioplasty alone is ineffective in maintaining the patency of the vessel, because of recoil of the plaque. As we learn from our colleagues who perform cardiac and peripheral endovascular therapy, deployment of a stent after angioplasty is often essential to maintain the patency of the treated vessel. It is often difficult to determine whether a portion of the plaque is calcific, and certainly angioplasty is fraught with the potential for vessel rupture and immediate neurological demise. However, when weighed against the natural history of untreated basilar artery occlusion, this risk may be warranted. The authors also emphasize

that every detail is extremely important; in this case, something as basic as the site of the femoral puncture may have contributed to the hemorrhagic shock and ultimate cardiac demise. With continued development of flexible balloon and stent systems, reports such as this will become more common and will help to reduce the problems associated with intracranial occlusions and stenoses.

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This article describes a man with basilar artery thromboembolic occlusion. This disease may be very difficult to treat because of the atheromatous plaque that keeps the vessel stenosed even after the clot has undergone thrombolysis, leading to rethrombosis. Placement of the stent in this situation was probably the only way the artery could be kept open. This approach may become the standard method by which this disease is treated. Advances in stent technology allow this approach using smaller and more flexible devices. The authors also provided the postmortem examination results; without that additional information, the patency of the stent would always be questionable.

Stanley L. Barnwell
Portland, Oregon

The authors describe the percutaneous endovascular placement of a Gianturco-Roubin-2 coronary stent for treatment of an acute atherothrombotic occlusion of the basilar artery resulting from high-grade stenosis, which was identified after intra-arterial thrombolysis and which recurred immediately after percutaneous transluminal angioplasty. The patient, unfortunately, suffered a fatal myocardial infarction in the postprocedural period;

the authors obtained pathological and postmortem examination results. The authors also present a detailed review of the literature regarding vertebrobasilar atherosclerotic occlusive disease, as well as a review of methods of revascularization.

I am currently aware of only one other published report of such stent placement, by the same authors (1). Certainly, for the patient described here, the indications for stenting this vessel were appropriate. The authors chose the Gianturco-Roubin-2 stent because of the distance between stent struts, which theoretically would prevent occlusion of basilar and vertebral perforating vessels. The authors describe the procedure in exquisite detail, including a salutary warning regarding high inguinal punctures associated with retroperitoneal hematomas.

It should also be noted that the coronary AVE stent (Arterial Vascular Engineering, Santa Rosa, CA) has exceptional tracking ability around tight curves, which may be encountered in the intracranial vasculature in elderly atherosclerotic patients. Another great advantage of the AVE stent is its increased stability on the delivery balloon, which may be advantageous during treatment of these particular lesions. This excellent case report is a useful addition to our current body of knowledge and will be appreciated by many neurointerventionalists.

Donald W. Larsen
Interventional Neuroradiologist
Los Angeles, California

1. Higashida RT, Smith W, Gress D, Urwin R, Dowd CF, Balousek PA, Halbach VV: Intravascular stent and endovascular coil placement for a ruptured fusiform aneurysm of the basilar artery. *J Neurosurg* 87:944-949, 1997.

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