

Posterior circulation revascularization

Superficial temporal artery to superior cerebellar artery anastomosis

JAMES I. AUSMAN, M.D., PH.D., FERNANDO G. DIAZ, M.D., PH.D.,
R. A. DE LOS REYES, M.D., HOOSHANG PAK, M.D., SURESH PATEL, M.D.,
BHARAT MEHTA, M.D., AND ROUSHDY BOULOS, M.D.

Departments of Neurological Surgery and Neuroradiology, Henry Ford Hospital, Detroit, Michigan

✓ Eight patients were evaluated for severe vertebrobasilar insufficiency (VBI). There were five males and three females, with an average age of 60.2 years (range 42 to 67 years). Three were diabetic and five hypertensive, including two patients who had both diseases. Seven of the eight had ongoing episodes of VBI refractory to anticoagulant and/or antiplatelet agents. Symptoms included two or more of the following in all patients: dizziness, diplopia, hemiparesis, hemihyesthesia, perioral numbness, bilateral visual blurring, dysarthria, and ataxia. Angiography revealed severe atherosclerotic stenosis of the proximal or midsection of the basilar artery in all patients.

A 10-cm segment of the anterior or posterior division of the superficial temporal artery (STA) was anastomosed to a proximal segment of the superior cerebellar artery (SCA) through a right subtemporal approach. Seven of eight (87%) postoperative angiograms demonstrated patency as evidenced by filling of the SCA and, in most cases, of the basilar artery. Six of the eight patients were improved or asymptomatic after the operation, one was unchanged, and one died. The average follow-up period was 14 months, with a range of 4 to 23 months. Transient morbidity included temporal lobe swelling in four patients and a subdural hematoma in one. Anastomosis of the STA to the SCA is a feasible therapeutic option in the patient with VBI secondary to stenosis of the proximal or midsection of the basilar artery.

KEY WORDS • vertebral artery • basilar artery • anastomosis • microsurgery • cerebral angiography • superior cerebellar artery • stenosis

THE syndrome of vertebrobasilar insufficiency (VBI) may be caused by a variety of disorders, both structural and nonstructural. The most common structural lesions are atherosclerotic plaques, which may occur anywhere from the proximal subclavian artery to the basilar bifurcation.⁹

While stenotic lesions in the extracranial vertebral system have been approached surgically for years, those involving the intracranial vertebral and basilar arteries awaited further refinements in microsurgical techniques. These techniques, pioneered by Yasargil, *et al.*,¹⁸ in the anterior circulation, were first applied to the posterior circulation by us⁴ in the form of an occipital artery to posterior inferior cerebellar artery (PICA) anastomosis. Sundt and Piepgras¹⁶ and Khodadad, *et al.*,¹³ reported their further experiences with

this operation. A recent report by Allen, *et al.*,¹ describes microsurgical endarterectomy of the intracranial vertebral artery for stenosis proximal to the origin of the PICA. Thus, stenosis of the intracranial vertebral artery proximal to the origin of the PICA became surgically approachable.

There remained, however, the problem of stenosis distal to the PICA. For vertebrobasilar junction stenosis distal to the PICA but proximal to the anterior inferior cerebellar artery (AICA), we have reported the technique of occipital artery to AICA anastomosis.² Still to be resolved was the problem of midbasilar stenosis. That portion of the basilar artery between the AICA's and the superior cerebellar arteries (SCA's) represents a very frequent site of atherosclerotic disease in the intracranial vertebrobasilar

Anastomotic revascularization of posterior circulation

TABLE 1
Summary of course in eight patients with anastomosis*

Case No.	Age (yrs), Sex	Diabetic	Hypertensive	Lesion in BA	Graft Patency	Operative Complications
1	61, M	yes	no	occlusion	yes	TLS
2	63, M	no	yes	occlusion	yes	TLS, SDH
3	64, M	no	yes	stenosis	yes	TLS
4	67, F	no	yes	stenosis	yes	TLS
5	60, M	yes	yes	stenosis	yes	none
6	42, F	no	no	stenosis	yes	none
7	58, M	no	no	stenosis	yes	none
8	67, F	yes	yes	stenosis	no	BA thrombosis

* BA = basilar artery; TLS = temporal lobe swelling; and SDH = subdural hematoma.

system.¹⁰ We have attacked stenosis at this level by performing a superficial temporal artery (STA) to proximal SCA bypass.³ In this present paper, we report our experience with eight such STA-SCA anastomoses and review the indications, operative technique, complications, and results of this operation.

Summary of Cases

Superficial temporal artery to SCA anastomosis was performed on eight patients between November, 1979, and December, 1980 (Table 1). There were five males and three females with an age range of 42 to 67 years. All except one (Case 7) were experiencing ongoing episodes of VBI, despite treatment with anticoagulant and/or antiplatelet agents. The patient in Case 7 had suffered a completed brain-stem infarct several months previously, and despite lack of persistent symptoms, underwent a bypass procedure because of the precariousness of his posterior circulation. There were three diabetic patients and five who were hypertensive, including two patients who had both diseases. Dizziness, although common, was not considered diagnostic of VBI unless associated with cranial nerve, sensory, cerebellar, and/or motor symptoms. Table 2 summarizes the symptoms encountered in our series of patients. Findings on neurological examination on admission to the hospital were minimal in this series (Table 3); however, two patients (Cases 2 and 8) deteriorated during the course of their diagnostic work-up. Both patients had extremely tight basilar stenoses which proceeded to occlusion (one with fatal results) during hospitalization. These cases will be considered in more detail below.

Case Reports

Case 1

This case has been reviewed in detail in a previous paper.³ Briefly, this 61-year-old man presented with episodes of light-headedness, near syncope without loss of consciousness, and headache associated with nausea; he also had episodes of dysarthria and circum-oral paresthesias. Cerebral angiography performed elsewhere revealed mid-basilar artery stenosis just distal to the origins of the AICA's (Fig. 1A). The

TABLE 2
Occurrence of symptoms

Symptoms	No. of Cases
dizziness	7
diplopia	6
hemihyesthesia	6
dysarthria	5
hemiparesis	5
ataxia	3
perioral numbness	2
tinnitus	2

TABLE 3
Findings on physical examination

Findings	No. of Cases
none	3
Babinski sign	2
hemiparesis (mild)	2
ataxia	1
nystagmus	1

patient was started on intravenous heparin therapy and referred to Henry Ford Hospital. Despite the heparin, the patient continued to have episodes of circumoral numbness, transient right lower extremity monoparesis, and alternating numbness of the right or left side of the body. A repeat cerebral angiogram revealed complete occlusion of the previously stenotic basilar segment (Figs. 1B-D and 4, Case 1). Due to the persistence of his symptoms, the patient underwent a right STA-SCA anastomosis on November 26, 1979. The use of the posterior cerebral artery as the recipient vessel was rejected because it was the only visible source of supply to the basilar artery. Its temporary interruption during surgery was believed therefore to be contraindicated. The patient's postoperative course was unremarkable, and he remains asymptomatic and off anticoagulant therapy 2 years following the procedure. Postoperative angiograms demonstrate patency, with enlargement of the anastomosis between

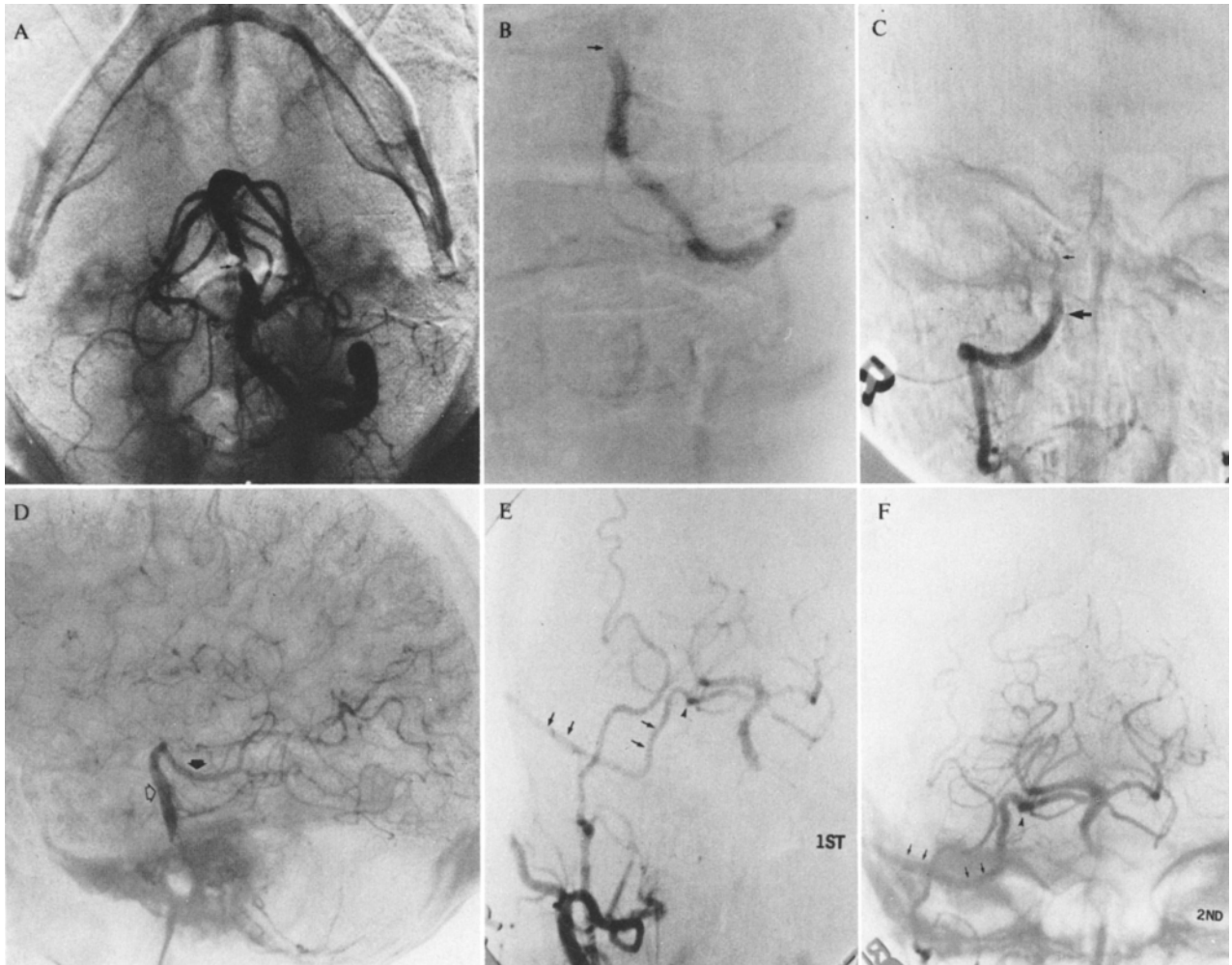


FIG. 1. Case 1. A: Left vertebral angiogram, base projection, showing a high-grade stenosis of the midportion of the basilar artery (*arrow*). B: Left vertebral angiogram, frontal projection, revealing complete occlusion of the basilar artery (*arrow*) where previously there was a high-grade stenosis. C: Right vertebral angiogram showing the right vertebral artery (*large arrow*), which opacifies a small right posterior inferior cerebellar artery (*small arrow*). D: Left internal carotid artery, lateral projection. The left posterior cerebral artery is opacified retrogradely from pial collateral anastomoses from the left middle cerebral artery (*dark arrow*). The basilar artery is opacified retrogradely from the left posterior cerebral artery (*open arrow*). E: Right external carotid angiogram 10 days postoperatively showing a patent anastomosis (*arrowhead*) between the superficial temporal artery (STA, *arrows*) and the right superior cerebellar artery (SCA). There is filling of the distal basilar and both superior cerebellar and posterior cerebral arteries. F: Right external carotid angiogram 5 months postoperatively. The size and caliber of the right STA has increased (*arrows*). The *arrowhead* shows the site of STA anastomosis to the right SCA. The distal basilar and posterior cerebral and superior cerebellar arteries are visualized.

the first and second postoperative angiograms (Fig. 1E and F).

Case 2

This 63-year-old man experienced several episodes of transient dizziness in November, 1979. His local physician found him to be hypertensive and started him on antihypertensive drugs in December, 1979. The patient then developed transient left amaurosis,

followed by a prolonged left hemihyesthesia and mild left hemiparesis. Cerebral angiography revealed a high degree of basilar artery stenosis. The patient was anticoagulated with warfarin and referred to Henry Ford Hospital. In spite of anticoagulation therapy, during the 10 days before admission the patient continued to experience increasingly frequent episodes of dizziness, disorientation in space, loss of balance, nausea, vomiting, headaches, and left hemi-

Anastomotic revascularization of posterior circulation



FIG. 2. Case 2. *Upper:* Frontal (*left*) and lateral (*right*) projections of the right vertebral (*large arrowhead*) angiogram revealing complete occlusion (*small arrowhead*) of the basilar artery. There is reflux of contrast material into the opposite left vertebral artery (*small arrows*), with opacification of the left posterior inferior cerebellar artery (*large arrows*). *Lower:* Right external carotid angiogram, frontal (*left*) and lateral (*right*) projections, revealing an enlarged-caliber right superficial temporal artery (*small arrows*) anastomosed to the right superior cerebellar artery (*large arrowheads*). The *small arrowhead* points to the site of anastomosis. Opacification of the basilar artery (*large arrow*) is seen, with visualization of both superior cerebellar arteries. Note other areas of narrowing of intracranial vessels.

paresis. Neurological examination on admission on March 2, 1980, revealed an alert, oriented man with slightly decreased recent memory, a mild left hemiparesis, left hemihypesthesia, left hyperreflexia, and poor tandem gait.

Because the original angiogram did not show the origin of the great vessels or his STA, it was decided to repeat the study. The patient was taken off heparin and underwent repeat angiography on March 6. This

study revealed occlusion of the previously stenotic basilar segment (Fig. 2 *upper*) and other intracranial stenoses (Fig. 4, Case 2). After the angiogram, the patient experienced increased somnolence and some progression of his left hemiparesis. An arterial line and Swan-Ganz catheter were inserted, and arterial blood pressure and pulmonary wedge pressures were elevated by volume expansion, with improvement in sensorium and motor strength on the left. Heparin

anticoagulation was reinstated. During the next several days, the patient's somnolence and left hemiparesis improved but continued to fluctuate, and it was decided to proceed with the bypass because of his unstable clinical state. On March 12, the patient underwent right STA-SCA anastomosis.

The postoperative course was stormy. Although initially unchanged from his preoperative status, the patient's level of consciousness and hemiparesis deteriorated on the 2nd day after surgery. A computerized tomography (CT) scan revealed right temporal lobe swelling, and osmotic diuretic therapy was instituted. After this swelling had partially subsided, a repeat CT scan showed a right temporoparietal subdural hematoma, which was evacuated on March 27. Over the next several weeks the patient showed slow but steady improvement. Repeat angiography on April 4 revealed a patent anastomosis (Fig. 2 lower). The patient was discharged to a rehabilitation center on April 29, 1980, where he was given anticoagulant therapy prophylactically. A follow-up visit in March, 1981, showed the patient to have a moderate left hemiparesis and difficulty with recent memory. The rest of the neurological examination was unremarkable, and the patient was free of transient ischemic attacks (TIA's) but still on anticoagulant drugs.

Comment

This case serves to illustrate two important points. The first concerns the role of blood pressure in the etiology and treatment of VBI. This patient's initial symptoms (as well as those of Case 8) were exacerbated by the initiation or augmentation of antihypertensive therapy. Similarly, both patients required the actual induction of hypertension to ameliorate their ischemic symptoms during the course of their treatment. This pressure-dependence of the posterior circulation has been alluded to by others.⁷ It is not our intention to condemn antihypertensive therapy. We would, however, like to point out that in the patient with VBI, exacerbation of symptoms by lowering the blood pressure with medication or position of the patient is highly suggestive of high-grade stenosis in the vertebral or basilar artery, with marginal perfusion pressure across the stenosis, and requires prompt angiographic investigation.

The second point is in regard to angiography. All of our patients tolerated their initial angiograms well. In this patient, however, the basilar artery became occluded prior to the second angiogram, resulting in clinical deterioration. Whether this was caused by the discontinuation of anticoagulant therapy in preparation for the angiogram, by technical factors during angiography, or a combination of these, repeat angiography in patients with high-grade basilar stenosis should be undertaken with extreme caution, and other measures should be initiated to prevent coagulation, such as partial heparinization, if angiography is essential.

Case 3

This 64-year-old man was admitted on August 26, 1979, with a 4-week history of transient episodes of dizziness, left hemihypesthesia, and diplopia, increasing in frequency to one per day on the several days prior to admission. These spells had been refractory to aspirin therapy.

Neurological examination on admission was negative. The patient underwent angiography which revealed approximately 90% stenosis in the midportion of the basilar artery just distal to the AICA's, and other cerebral vascular lesions (Fig. 4, Case 3). He was placed on warfarin anticoagulation therapy and sent home. However, he continued to have vertigo and diplopia increasing to approximately one to two times a week associated with nausea and vomiting.

On March 2, 1980, repeat angiography revealed no change in the lesions. The patient underwent a right STA-SCA anastomosis on March 17. The postoperative course was complicated by the development of a right sixth nerve palsy and lethargy secondary to right temporal lobe edema. These signs gradually resolved. A postoperative angiogram on April 1 revealed a patent anastomosis. The patient continued to improve, complaining only of slight difficulty with memory and a few episodes of diplopia. These symptoms cleared prior to his discharge. On his last clinic visit on March 26, 1981, 1 year after surgery, the patient was neurologically intact and had no further episodes of VBI.

Case 4

This 67-year-old hypertensive woman presented with a 3-month history of intermittent dizziness and a 2-week history of intermittent right-sided facial and hand numbness, dysarthria, and ataxia, with a tendency to veer to the right. Neurological examination on admission was normal. Angiography revealed mid-basilar stenosis and other cerebral vascular lesions (Fig. 4, Case 4).

She was placed on a course of oral anticoagulant drugs and was discharged; however, her symptoms increased in frequency and she was hospitalized again 2 months later. On April 14, 1980, the patient underwent a right STA-SCA anastomosis. The initial postoperative angiogram revealed only marginal filling of the anastomosis, but a subsequent angiogram on November 14, 1980, revealed better filling. At her last clinic visit on January 13, 1981, the patient was asymptomatic except for occasional right-sided tinnitus, which was thought by the ear, nose, and throat consultant to be due to high-frequency hearing loss.

Case 5

This 60-year-old man experienced three episodes of transient dizziness, diplopia, dysarthria, and left hemiparesis and hemihypesthesia in the 2 months prior to admission. The episodes lasted approximately 20 min-

Anastomotic revascularization of posterior circulation

utes and then completely cleared. Cerebral angiography performed elsewhere revealed a high-grade stenosis of the basilar artery between the AICA and SCA, and other cerebral vascular lesions (Fig. 4, Case 5). Warfarin was prescribed, but this was subsequently discontinued due to gastrointestinal bleeding. The patient experienced another similar TIA on discontinuation of anticoagulation therapy, and was referred to Henry Ford Hospital for further evaluation and treatment.

The neurological examination on admission was negative. The patient underwent a right STA-SCA anastomosis on July 21, 1980. The postoperative course was uneventful. Angiography performed 10 days after surgery revealed a patent anastomosis with filling of the upper basilar system. The patient complained of slight difficulty with recent memory postoperatively, but was otherwise asymptomatic. He remained free of symptoms at his last clinic visit in February, 1981.

Case 6

This 42-year-old diabetic hypertensive woman presented with a 1-year history of increasingly frequent episodes of dizziness, tinnitus, diplopia, and left hemiparesis. For other medical reasons she could not be given oral anticoagulant therapy. The episodes were unaffected by therapy with aspirin and dipyridamole. Neurological examination revealed slight left-sided hyperreflexia and bilateral extensor plantar responses, but was otherwise unremarkable. The patient underwent angiography, which revealed proximal basilar stenosis and other cerebral vascular lesions (Fig. 4, Case 5). She underwent a right STA-SCA anastomosis on August 20, 1980. The postoperative course was unremarkable. Postoperative angiography revealed a patent anastomosis. On her last clinic visit on March 26, 1981, the patient stated that she had experienced two further episodes, of a lesser magnitude, of dizziness and left-sided face and hand numbness since the operation. She was otherwise neurologically intact and asymptomatic. She was admitted to the hospital and underwent angiography the following day. This revealed enlargement of the STA and increased filling of the basilar artery and its branches.

Comment

This patient's episodes of VBI were not entirely halted by the surgery. They have, however, decreased in both magnitude and frequency, while at the same time the angiographic appearance of the bypass graft has enlarged. Further follow-up review is necessary to see whether her episodes will cease altogether.

Case 7

This 58-year-old man experienced three episodes of dizziness, diplopia, dysarthria, and right hemihypesthesia in November, 1979. The first two episodes were

transient, and the last resulted in a prolonged deficit. At the time of his last episode he underwent cerebral angiography, which revealed a right vertebral artery occlusion, a left vertebral artery which ended in the PICA, and evidence of mid-basilar stenosis (Fig. 4, Case 7). The patient was placed on aspirin and dipyridamole therapy and referred to Henry Ford Hospital.

It was uncertain whether he should be followed medically or undergo a surgical bypass procedure. It was elected to follow him clinically, and a repeat angiogram was planned to evaluate the status of his vertebrobasilar circulation and its collaterals. Angiography was repeated on November 5, 1980, revealing no change in the angiographic lesions. Because it was thought that the patient was at risk for a major brainstem infarct due to his severely compromised circulation, and because of the patient's concern about the possibility of a subsequent infarction, he was admitted for a basilar artery revascularization. The neurological examination on admission was negative except for bilateral extensor plantar responses. The patient underwent a right STA-SCA anastomosis on November 11, 1980. Postoperative angiography on March 2, 1981, revealed a patent anastomosis but poor filling of the posterior fossa vasculature through the anastomosis. At his last clinic visit on March 6, 1981, the patient remained asymptomatic (as he had been preoperatively) and neurologically intact.

Comment

This patient underwent an essentially "prophylactic" bypass. In spite of anatomically significant lesions, the lack of demand for extra circulation (as evidenced by his lack of ongoing symptoms which implied adequate circulation) may explain the poor filling through the anastomosis.

Case 8

This 67-year-old diabetic hypertensive woman presented to the emergency room on October 16, 1980, with a 1-day history of transient nausea, vomiting, bifrontal headache, left upper extremity paresis, and dysarthria. She had experienced a transient episode of bilateral visual blurring 1 week previously. In the emergency room, the patient was noted to have an elevated blood pressure (182/110 mm Hg). She was told to increase her antihypertensive medication. Later that day, she experienced another transient episode of all of the same symptoms plus bilateral blurred vision. She returned to the emergency room and was admitted to the medicine service.

Neurological examination on admission was normal. Shortly after admission the patient developed cardiac dysrhythmias which required that she be transferred to the coronary care unit for prolonged evaluation and treatment. After her cardiac status was stabilized, she continued to have episodes of VBI. On November 3, 1980, the patient underwent angi-

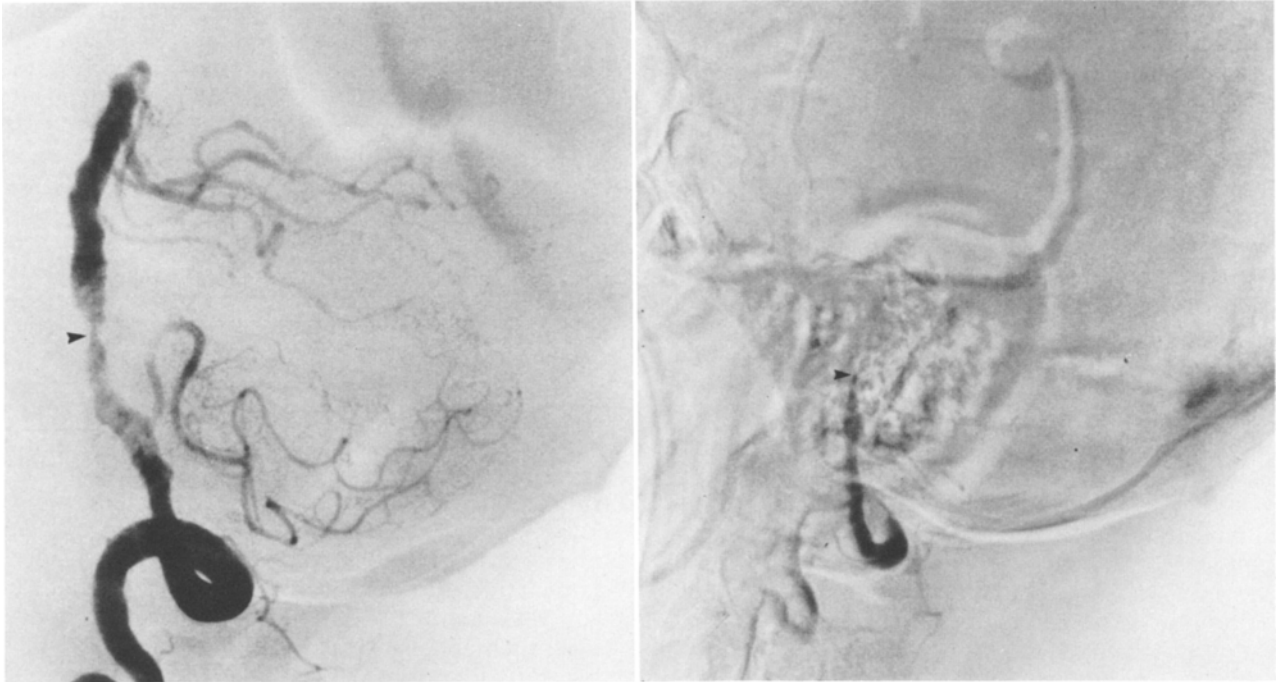


FIG. 3. Case 8. *Left:* Left vertebral angiogram, lateral projection, demonstrating high-grade stenosis of the basilar artery (*arrowhead*). The marked irregularity of the basilar artery indicates atherosclerotic changes. *Right:* Postoperative left vertebral angiogram showing complete occlusion of the basilar artery (*arrowhead*).

ography which demonstrated a high degree of basilar artery stenosis and other cerebral vascular lesions (Figs. 3 *left* and 4, Case 8). She was placed on full intravenous heparin anticoagulation therapy but, despite this, developed episodes of dizziness and visual blurring. On November 4, she experienced a transient (5-minute) episode of quadriparesis and unresponsiveness. She was transferred to the neurosurgical intensive care unit and underwent intravascular volume expansion in addition to anticoagulation, and was taken off her antihypertensive medications. During the following month, numerous attempts were made to decrease the patient's blood pressure, which was in the range of 180/110 to 200/130 mm Hg. However, each time this was done she became symptomatic, requiring re-elevation of her blood pressure.

Because of this refractoriness to medical therapy, basilar artery revascularization was thought to be the only alternative. On December 9, 1980, the patient underwent a right STA-SCA anastomosis. Postoperatively, she never regained consciousness. A repeat angiogram revealed occlusion of the entire basilar artery (Fig. 3 *right*). The patient had a progressive downhill course and died on January 4, 1981. An autopsy was refused by the family.

Comment

This case reinforces the point made in the comment on Case 2 concerning the role of hypertension in

posterior circulation insufficiency. This patient required not only full anticoagulation therapy but hypertension greater than 100 mm Hg diastolic (a potentially dangerous combination) in order to remain asymptomatic. It was believed that surgical treatment offered her the only chance to avoid a potentially catastrophic stroke. Nevertheless, her entire basilar artery thrombosed perioperatively. Whether this occlusion was related to the discontinuation of heparin anticoagulation in preparation for surgery or to the operation itself is speculative. However, the fact that the entire basilar artery as well as the bypass graft became thrombosed would tend to support the former conclusion. It is quite possible that in a patient with a critically tight stenosis such as this, thrombosis is the inevitable result. However, if faced with the same situation in the future we would consider tapering the patient's heparin while administering intravenous low-molecular-weight dextran and possibly a vasopressor, such as dopamine, before and during surgery. Only further experience with similar patients will tell us whether this is a reasonable approach.

Operative Technique

Prior to intubation, an arterial line is inserted to constantly monitor blood pressure. In neurologically unstable patients, a Swan-Ganz flow-directed balloon-tipped catheter is placed percutaneously to

Anastomotic revascularization of posterior circulation

closely monitor fluid balance and cardiovascular function during and after the operation. The catheter is usually placed just before the operation in those patients whose heparin anticoagulation has been discontinued several hours prior to surgery. A Foley catheter is inserted to monitor urinary output and to allow the use of osmotic diuretics during the surgery. A lumbar catheter is inserted for intraoperative cerebrospinal fluid (CSF) drainage. It is critical that the CSF drainage be functional because of the necessity for retraction and elevation of the temporal lobe in order to reach the tentorial incisura.

The patient is placed supine with the right shoulder and hip elevated and the head horizontal, hyperextended, and the left side down. The right side is always used to avoid retraction of the temporal lobe on the dominant side. Both anterior and posterior branches of the STA are traced on the scalp with the use of a directional flow Doppler probe. The anterior branch is preferred for use in the anastomosis. It is traced from the area anterior to the pinna to the most distal portion visualized on the angiogram, usually for 8 to 10 cm or more. The scalp is shaved and prepared with povidone-iodine solution in the usual manner. A direct incision is made over the STA, and approximately 10 cm of the vessel is dissected under the microscope, from its most distal to its most proximal location. All side branches are individually cauterized and transected. Anatomical continuity is maintained only at the proximal and distal ends of the vessel. The incision is then extended posteriorly from the pinna above the ear in a parieto-occipital direction so that the incision from anterior limb to posterior limb represents a **V**, with the apex of the **V** at the pinna. The flap is reflected and retracted superiorly. The temporalis muscle is incised in a cross-bow fashion and the flaps are retracted laterally. A free bone flap is raised by placing a single burr hole on the anterior inferior temporal region just above the ear at the point through which the STA will enter. With the electrical drill, a quadrangular free bone flap is elevated. The inferior portion of the flap is brought down to the base of the middle fossa by extending the craniectomy with a Leksell rongeur. After hemostasis is achieved, the dura is tacked to the bone to prevent intraoperative bleeding. The spinal drain is opened at this time, and the patient is given 40 mg of furosemide intravenously, followed 10 to 15 minutes later by a 50-gm bolus of intravenous mannitol.

A dural incision is made at the base of the craniotomy and the dura is reflected superiorly. The temporal lobe is elevated until the tentorial incisura is identified. Care is taken not to compromise the vein of Labbé or posterior temporal veins as they enter the sigmoid sinus. Self-retaining retractors are placed. The edge of the tentorium is elevated, cauterized, and transected to provide more exposure. The microscope is again utilized and the arachnoid over the brain stem is picked up and opened, revealing the SCA. The flap

of the tentorium is retracted and reflected, and is anchored to a more lateral aspect of the tentorium with a single suture. Care is taken not to damage the fourth nerve. The SCA is identified in its lateral circum-mesencephalic portion. The vessel generally has no perforating branches as it traverses from the lateral portion of the midbrain to the superior portion of the cerebellum. However, if it does have branches to the brain stem, a portion of the artery beyond the area with the brain-stem branches is selected for the anastomosis. This artery can be single but frequently divides into rostral and caudal branches.¹⁵ The larger of the two branches is identified, and all arachnoid adhesions are carefully dissected under the microscope. The vessel is generally fairly loose and can be gently elevated by placing a small cotton ball or a small piece of Gelfoam underneath the vessel. A rubber dam is placed on top of the Gelfoam and underneath the vessel to isolate the segment of the artery to be used. The self-retaining retractors are now temporarily removed as attention is again directed to the STA. The distal portion of the STA is transected and its proximal portion clipped with a broad Mayfield clamp, partially sprung to prevent STA endothelial damage. The vessel is irrigated thoroughly with heparinized saline. The STA is used in its entirety and only the most distal portion is carefully cleaned of all adventitial tissue. The vessel is brought down to the area of the SCA, and a fish-mouth stoma is made in the STA.

Now the patient is given 120 mg of intravenous thiopental for cerebral protection, and Kleinert-Kutz clamps are placed on the SCA. A longitudinal elliptical arteriotomy is made on the SCA to equal the length prepared on the STA. A Silastic stent is placed inside the SCA after the vessel is irrigated with heparinized saline. With the aid of the long bayonet microinstruments, including locking needle holders and fine-tipped forceps, an end-to-side anastomosis is completed with interrupted 10-0 nylon sutures. To facilitate suturing in this depth, half-circle needles are utilized. The stent is removed before completion of the sutures, and the area of the anastomosis is once again irrigated with heparinized saline. The temporary clips are removed by first taking off the distal SCA clip, then the proximal SCA clip and, last, the STA clip. A small amount of bleeding is generally observed and, if necessary, an additional suture is added to the area of the anastomosis. The operative field is thoroughly irrigated with normal saline, and the retracted temporal lobe is carefully inspected for bleeding points or contusions. Hemostasis in this area and on the proximal portion of the STA pedicle is achieved. The incision is closed in layers in a routine manner. Care is taken to allow sufficient room for the STA to enter the dura and bone undisturbed. A major portion of the dura is closed and the bone flap is replaced. The skin and muscle are closed in a routine manner.

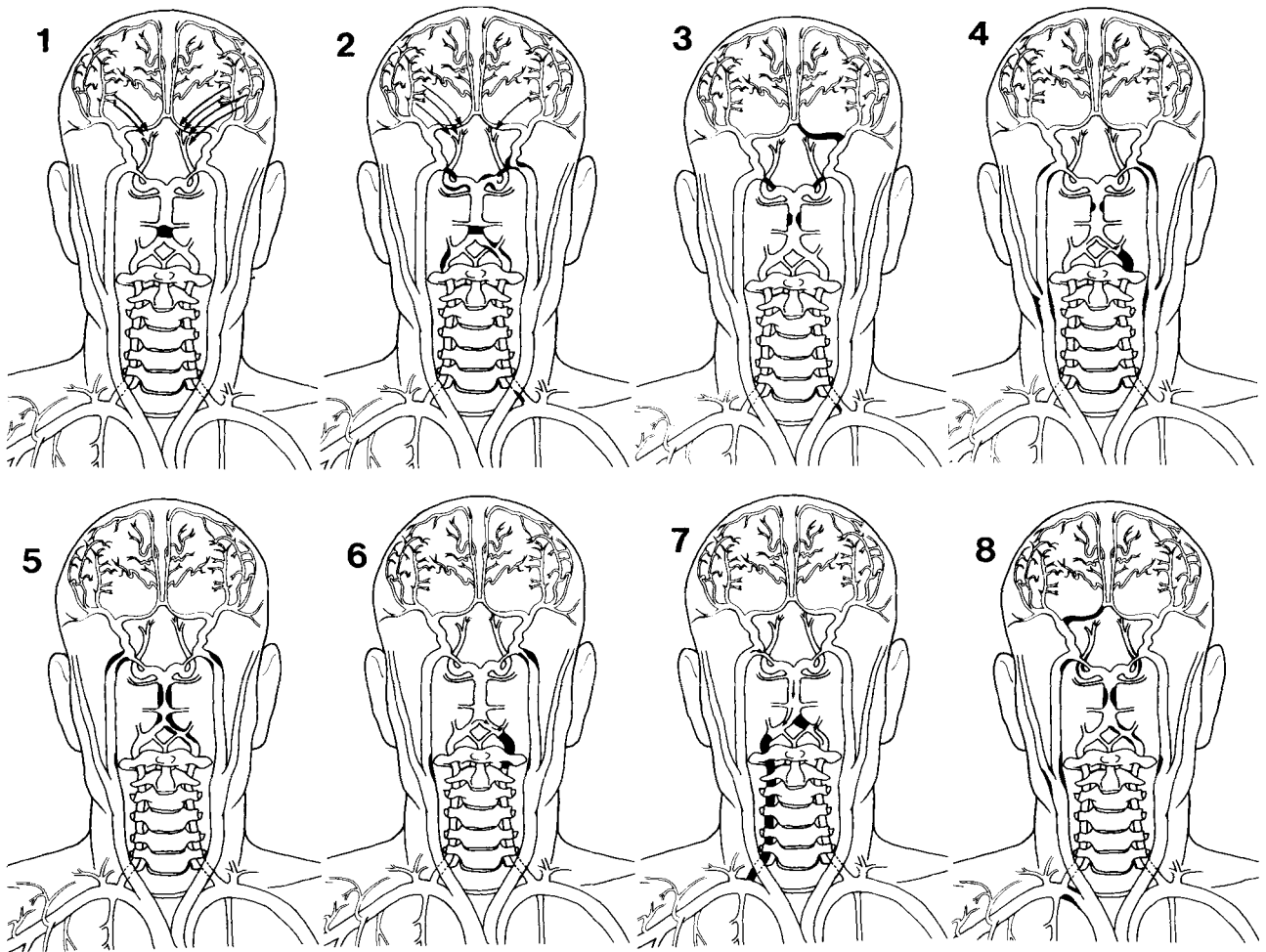


FIG. 4. Diagrams of the major extracranial and intracranial vessels, anteroposterior view, in all eight patients. *Dark areas* represent areas of occlusion or stenosis. *Arrows* represent collateral circulation.

Postoperative Management

Concern for temporal lobe swelling is paramount. A CT scan is usually performed within the first 6 hours after surgery to ensure that there is no temporal or subtemporal clot. None was found at this stage. Swelling of the temporal lobe was found in four of these eight patients within 3 days of surgery, and was treated with steroids and/or mannitol, if necessary, with complete resolution of the edema. Otherwise, routine postoperative management is given. The patients are not restarted on heparin therapy. All patients were started on aspirin postoperatively, 5 grains four times daily.

Follow-up angiograms were obtained in all patients. Six had angiograms within 2 weeks of surgery and the remainder within 5 weeks of the operation. Five patients had two postoperative angiograms and one patient three. Patency, defined as any intracranial filling through the anastomosis, was found in seven of the eight cases. Filling of a single SCA branch was seen in two cases, and filling of the basilar artery and

usually some or all of its branches to the site of the stenotic occlusion was seen in five of eight cases. In the six patients with two or more postoperative angiograms, increased filling of the recipient vessel tree was demonstrated in two cases. To properly visualize the STA and SCA, a selective external carotid injection was usually necessary because the internal carotid artery was usually patent in these patients. A Townes projection with this injection, directed as if looking at the vertebral basilar circulation, is necessary to visualize filling of the system through the STA-SCA anastomosis. Figure 4 summarizes the angiographic results.

Discussion

The syndrome of vertebrobasilar insufficiency (VBI) is predicated upon a history of transient episodes indicative of ischemia in the brain stem, cerebellum, or occipital cortex.⁵ This posterior circulation insufficiency may be characterized by a variety of symptoms, including cranial nerve dysfunction such

Anastomotic revascularization of posterior circulation

as diplopia or dysarthria, cerebellar findings such as ataxia or dysmetria, occipital cortex symptoms such as bilateral amaurosis or hemianopsia, and motor or sensory alterations involving any combination of the four extremities. Dizziness, although probably the most common single symptom seen in VBI, rarely occurs in isolation, and, when unaccompanied by other symptoms, is usually the result of labyrinthine disease and not vertebrobasilar ischemia.¹² Table 1 lists the symptoms, in order of frequency, observed in our series. This is generally representative of their incidence in other series in the literature.^{5,12,14}

The incidence of VBI appears to be approximately half that of carotid circulation TIA's.⁸ Although some studies have suggested a worse prognosis for carotid than vertebrobasilar TIA's,¹⁴ this appears to be a function of the rigidity of criteria for the diagnosis of VBI: if dizziness alone is considered indicative of VBI, the subsequent "stroke" rate will be lower than that for carotid TIA's. Carlidge, *et al.*,⁸ using strict criteria for the diagnosis of VBI, found no significant difference in the probability of the occurrence of stroke between patients (without angiography) who suffered TIA's related to the carotid or the vertebrobasilar circulation. Both were in excess of 35% over 5 years.

Atherosclerotic stenosis or occlusion in the posterior circulation is the most frequent cause of VBI.⁹ These atherosclerotic plaques may occur anywhere from the proximal subclavian artery to the distal basilar artery. While some present as distinct clinical syndromes (such as subclavian steal or "top of the basilar" syndrome⁶) which suggest the etiology, the vast majority require cerebral angiography for localization of the lesion.

Modern angiographic techniques allow accurate diagnosis with an acceptable risk in the patient with VBI. Of 88 consecutive vertebral angiograms performed for posterior circulation insufficiency syndromes at Henry Ford Hospital, five (5.6%) have been associated with worsening of the patient's neurological status, with three of these (3.4%) resulting in persistent minor deficits. There have been no deaths. This compares favorably with the complication rate in the literature, which has mostly been reported to be in the range of 3% to 9%.¹¹ Both surgical and angiographic risks appear to be increased in the patient with high-grade stenosis, but one would also assume that these are the patients who are at the greatest risk of infarction in the natural history of the disease.

Anticoagulation has been the treatment of choice for basilar artery stenosis in the past, but its long-term value has not been documented. There is, additionally, a high risk of hemorrhage associated with its use.¹⁷ Five of these patients were experiencing ongoing symptoms while on heparin or warfarin. For these patients, there was no medical alternative to the operation. For those patients with less severe symptoms, a choice could be made between medical and surgical

therapy. Whether these patients would have been better treated medically can be debated but, considering the severity of the vascular compromise, the mortality rate (13%, or one of eight cases) and the permanent morbidity rate in this series was quite limited.

While the angiographic and surgical morbidity in this series might, at first glance, seem excessive, it should be recalled that many of the patients were suffering from unstable VBI secondary to critically tight stenoses. Although there are no available data on the natural history of a similar (untreated) population, one would expect that the stroke rate in this group would be extremely high.

As more experience is gained in the surgical treatment of basilar artery stenosis, perhaps a clearer definition of the criteria for surgery will emerge. For the present time, this series stands as a point of reference with which to compare a similar group of patients on strictly medical therapy.

References

1. Allen GS, Cohen RJ, Preziosi TJ: Microsurgical endarterectomy of the intracranial vertebral artery for vertebrobasilar transient ischemic attacks. *Neurosurgery* 8:56-59, 1981
2. Ausman JI, Diaz FG, de los Reyes RA, et al: Anastomosis of occipital artery to anterior inferior cerebellar artery for vertebrobasilar junction stenosis. *Surg Neurol* 16:99-102, 1981
3. Ausman JE, Diaz FG, de los Reyes RA, et al: Superficial temporal to proximal superior cerebellar artery anastomosis for basilar artery stenosis. *Neurosurgery* 9:56-60, 1981
4. Ausman JI, Lee MC, Klassen AC, et al: Stroke: what's new? Cerebral revascularization. *Minn Med* 59:223-227, 1976
5. Bradshaw P, McQuaid P: The syndrome of vertebrobasilar insufficiency. *Q J Med* 32:279-296, 1963
6. Caplan LR: "Top of the basilar" syndrome. *Neurology* 30:72-79, 1980
7. Caplan LR, Sergay S: Positional cerebral ischaemia. *J Neurol Neurosurg Psychiatry* 39:385-391, 1976
8. Carlidge NEF, Whisnant JP, Elveback LR: Carotid and vertebral-basilar transient cerebral ischemic attacks. A community study, Rochester, Minnesota. *Mayo Clin Proc* 52:117-120, 1977
9. Castaigne P, Lhermitte F, Gautier JC, et al: Arterial occlusion in the vertebro-basilar system. A study of 44 patients with post-mortem data. *Brain* 96:133-154, 1973
10. Cornhill JF, Akins D, Hutson M, et al: Localization of atherosclerotic lesions in the human basilar artery. *Atherosclerosis* 35:77-86, 1980
11. Faught E, Trader SD, Hanna GR: Cerebral complications of angiography for transient ischemia and stroke: prediction of risk. *Neurology* 29:4-15, 1979
12. Fisher CM: Clinical syndromes in cerebral thrombosis, hypertensive hemorrhage, and ruptured saccular aneurysm. *Clin Neurosurg* 22:117-147, 1975
13. Khodadad G, Singh RS, Olinger CP: Possible prevention of brain stem stroke by microvascular anastomosis in the vertebrobasilar system. *Stroke* 8:316-321, 1977

14. Marshall J: The natural history of transient ischaemic cerebro-vascular attacks. *Q J Med* **33**:309-324, 1964
15. Saeki N, Rhoton AL Jr: Microsurgical anatomy of the upper basilar artery and the posterior circle of Willis. *J Neurosurg* **46**:563-578, 1977
16. Sundt TM Jr, Piepgras DG: Occipital to posterior inferior cerebellar artery bypass surgery. *J Neurosurg* **48**:916-928, 1978
17. Terént A, Andersson B: The outcome of patients with transient ischemic attacks and stroke treated with anti-coagulants. *Acta Med Scand* **208**:359-365, 1980
18. Yaşargil MG, Krayenbühl HA, Jacobson JH II: Microneurosurgical arterial reconstruction. *Surgery* **67**: 221-233, 1970

Manuscript received August 6, 1981.

Accepted in final form January 19, 1982.

Address reprint requests to: James I. Ausman, M.D., Ph.D., Department of Neurological Surgery, Henry Ford Hospital, 2799 West Grand Boulevard, Detroit, Michigan 48202.