Complications of intracranial bypass for vertebrobasilar insufficiency

LEO N. HOPKINS, M.D., AND JAMES L. BUDNY, M.D.

Department of Neurosurgery, State University of New York at Buffalo, and Dent Neurologic Institute, Millard Fillmore Hospital, Buffalo, New York

The authors' initial experience with intracranial revascularization of vertebrobasilar insufficiency, reported previously, fortunately yielded reasonably good results with high patency rates but in some cases there were significant, albeit temporary, complications. Since that time, major brain-stem strokes have occurred in two patients following superficial temporal to superior cerebellar artery bypass procedures. This occurrence has caused the authors to reassess their experience with this procedure and review the published literature with regard to complications. This review and the results of the international bypass study on anterior circulation ischemia suggest that a very cautious and conservative approach should be taken prior to considering intracranial bypass to the superior cerebellar or posterior cerebral artery.

KEY WORDS • vertebrobasilar insufficiency • posterior fossa bypass • extracranial-intracranial bypass • revascularization • anticoagulation

RECENTLY, we reported a combined series of extracranial to intracranial (EC-IC) bypass procedures for vertebrobasilar insufficiency (VBI). Those patients were operated on consecutively at the State University of New York at Buffalo between 1977 and 1984. Although there were some significant complications and one delayed death from septicemia, there were no brain-stem strokes or operative deaths.

Since completion of that series, major brain-stem strokes have occurred in two patients following superficial temporal artery (STA) to superior cerebellar artery (SCA) bypass procedures. Both patients eventually died. Our experience and the results of the international bypass study for anterior circulation ischemia prompted us to reexamine the role of intracranial posterior fossa bypass procedures in the treatment of VBI. A review of the natural history of this disease and the complications of these operative procedures helps to better define indications for intracranial bypass surgery. We first review the complications in our own cases.

Case Reports

Case 1

This 61-year-old hypertensive diabetic white man with a history of smoking 1½ packs of cigarettes for over 30 years underwent a femoral-popliteal bypass procedure 16 years prior to admission and a coronary artery bypass graft 12 years prior to admission. He suffered from chronic angina. He presented with bilateral visual field abnormalities and described diffuse loss of vision in both visual fields on several occasions. He also complained of episodic horizontal diplopia which was relieved by lying down. He was initially treated with aspirin. He presented again 8 months later with progressive unsteadiness of gait and speech difficulty described as word hesitation and slurring of speech. His gait difficulty was progressive to the point where he was unable to walk without assistance. He also complained of constant generalized and occipital headaches.

Examination. The patient had a left lower quadrantanopsia, ataxic gait, and horizontal nystagmus on left lateral gaze. Angiography revealed moderate stenosis of the left internal carotid artery at its origin in the neck and in the carotid siphon intracranially. There was a tight stenosis of the right vertebral artery just proximal to the origin of the posterior inferior cerebellar artery (PICA) (Fig. 1). An arch aortogram showed the left vertebral artery to be absent. Carotid artery studies showed no significant collateral blood supply to the posterior circulation.

Operation. The patient underwent an uneventful right-sided STA-SCA bypass on August 28, 1985. Initially, he did well except for a mild left-sided upper-
extremity weakness. Hydration was maintained with Ringer's lactate and Plasmanate (a plasma derivative). Nine hours postoperatively, he developed the rapid onset of shortness of breath and dyspnea. Tracheal suction yielded pink frothy material, and a clinical diagnosis of pulmonary edema was established. Arterial blood gas analysis revealed a pO₂ of 34 mm Hg. Shortly thereafter the patient was noted to have a marked left hemiparesis which rapidly progressed to hemiplegia in spite of aggressive diuretic and respiratory treatment. Over the next 24 hours his neurological status continued to deteriorate to a state of coma vigil. A computerized tomography scan showed occipital, cerebellar, and brain-stem infarcts. The patient developed pneumonia from which he eventually died. A postmortem examination was not carried out.

Case 2

This 71-year-old hypertensive black man suffered a left hemispheric partial nonprogressive stroke in 1979 which left him with a mild right-sided upper-extremity paresis and minimal mixed dysphasia. Two weeks prior to admission he experienced the sudden onset of a staggering and reeling gait which lasted about 5 minutes. Several days later he had a second similar episode from which he again recovered. One day prior to admission, his left upper extremity and shortly thereafter his right upper extremity became numb and weak. He noted loss of vision in the lower visual field bilaterally, and his speech became markedly slurred. This episode lasted about 15 minutes and then cleared completely. On the following day he had a second similar episode of bilateral upper-extremity weakness but this time associated with left lower-extremity weakness and perioral numbness, all of which resolved after about 30 minutes.

Examination. Examination showed a patient with mild dysphasia and minimal right upper-extremity weakness that was a residual effect from his previous stroke. He was otherwise neurologically normal. Cerebral angiography revealed that the left vertebral artery ended in the PICA. The right vertebral artery was severely stenosed just distal to the origin of the PICA, with almost no distal, vertebral, or basilar filling (Fig. 2). Both carotid arteries showed some filling of the upper basilar region through the posterior communicating arteries.

Operation. On May 28, 1985, the patient was taken to surgery where a right-sided STA-SCA anastomosis was carried out uneventfully. After surgery, the patient remained unchanged neurologically. On the 2nd postoperative day his speech was noted to be slightly slurred. Two days later a progressive left hemiparesis was ob-
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served which did not respond to volume expansion. Selective right external carotid angiography on Day 5 showed no filling of the bypass graft. Internal carotid injections demonstrated disappearance of the previously demonstrated upper brain-stem collateral supply. At this point, the patient was barely arousable, and despite continued volume expansion his condition deteriorated to a state of coma vigil. He progressed to complete brain-stem failure and died.

Discussion

The surgical treatment of VBI has recently been reviewed. The relative ease and safety of extracranial vertebral artery reconstruction makes extracranial revascularization preferable when possible. Symptomatic intracranial stenosis presents a more difficult problem. Our initial good results with revascularization of the upper brain stem via EC-IC bypass to the SCA and posterior cerebral artery (PCA) engendered in us a false sense of confidence. The known risks and technical difficulties with bypasses to the proximal SCA or PCA should have made us more circumspect in our indications for surgery and choice of operation. Our experience with these two cases prompted us to look more critically at the natural history and pathophysiology of VBI, to review previously reported complications of intracranial revascularization, and to reassess our approach to such patients.

There is little doubt that clinically significant intracranial vertebrobasilar disease, when left untreated, harbors a poor prognosis both from a neurological and from a cardiac viewpoint. Intracranial stenosis which involves the basilar artery or both vertebral arteries is associated with a particularly poor prognosis. This is at least in part due to the fact that the intracranial collateral circulation is usually poorly developed. As opposed to anterior circulation ischemia, which is more commonly embolic, posterior circulation ischemia from intracranial stenosis is usually due to hypoperfusion.

For this reason, postural changes are far more likely to cause symptoms in patients with VBI, as occurred in our series. Naritomi, et al., suggested that VBI may result in dysautoregulation which potentiates the effects of posture.

Compared to patients with anterior circulation ischemia, those with VBI tend to develop symptoms about one decade later and have a higher incidence of associated hypertension and diabetes. Both disorders are associated with a high incidence of coronary disease, resulting in a significant risk of congestive heart failure and myocardial infarction. The EC/IC Bypass Study Group has defined the natural history of anterior circulation ischemia treated with antiplatelet agents. The natural history of posterior circulation ischemia is not known, but there is a suggestion that antiplatelet and especially anticoagulant therapy are of help. The elegant chronological pathological studies of Castaigne, et al., demonstrated that most strokes in the posterior circulation result from thrombosis of a preexisting atherosclerotic lesion. This suggests that, barring contraindications, patients with VBI should be given a trial of anticoagulant drugs.

Angiographic assessment of our patients suggests that the majority of intracranial lesions (55%) occur in the proximal vertebral artery up to and including the origin of the PICA; distal vertebral and vertebrobasilar junction lesions account for 20%, and mid-basilar artery lesion for 9%. Therefore, if surgery is required, a proximal anastomosis to the PICA should suffice in the majority of patients.

The EC/IC Bypass Study Group has clearly shown that STA to middle cerebral artery (MCA) anastomoses were not effective in the population of patients entered in that study. We can be comforted by the fact that the STA-MCA bypass is a low-risk operation and relatively little harm was caused with respect to surgical morbidity and mortality. It is certainly possible that intracranial vertebrobasilar disease is so different from anterior circulation disease as to warrant more aggressive surgical therapy. On the other hand, the risk associated with a bypass of the SCA or PCA mandates that we approach vertebrobasilar disease with a new level of caution and conservatism.

We reviewed most of the major reported series more carefully to evaluate the risks associated with these procedures. Table 1 shows the mortality rates, patency rates, and complications associated with occipital artery to PICA bypass; Table 2 summarizes data for bypasses to the SCA and PCA. There are some ambiguities in these series so in a few instances there may be slight inaccuracies, but by and large the results are clear. A total of 90 cases of occipital artery to PICA bypass were reviewed: the overall patency rate was 91%, the mortality rate was 3%, and the total complication rate was 22%. Although many of these complications were transient, about 10% appeared to be serious, in the form of severe respiratory or cardiac problems or increased neurological deficit. Many of the respiratory complications may have been related to preexisting pareses of the ninth and 10th cranial nerves. There were three cases of significant aseptic meningitis and one of bacterial meningitis, in addition to the expected rare postoperative complications of hydrocephalus and epidural or subdural hematoma. Although the patency rates are quite good, it is our impression from the literature and from personal communications that a significant number of the postoperative angiograms showed less than luxuriant filling of the posterior circulation through the PICA.

Among the 86 bypasses to the SCA and PCA that were reviewed, there was a 12% mortality rate, which might be falsely low considering one group of STA-SCA bypasses in which the incidence of deaths was not discussed. The two recent cases we describe here would increase this fatality rate to 14%. Overall, there was a 79% patency rate. The total complications amounted to 55%. The majority of these were transient, but
TABLE 1
Summary of complications following occipital artery to posterior inferior cerebellar artery bypass

<table>
<thead>
<tr>
<th>Author &amp; Yr</th>
<th>No. of Cases</th>
<th>Mortality Rate</th>
<th>Patency Rate</th>
<th>Complications*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sundt &amp; Piepgras, 1978 &amp; 1985</td>
<td>39</td>
<td>5%</td>
<td>88%</td>
<td>minor increased neurological deficit (2); respiratory complications; often related to 10th cranial nerve palsy; epidural hematoma (1); subdural hematoma (1); aseptic meningitis (1); wound infection (1)</td>
</tr>
<tr>
<td>Ausman, et al., 1984</td>
<td>8</td>
<td>13%</td>
<td>100%</td>
<td>pulmonary edema (1 death)</td>
</tr>
<tr>
<td>Hopkins, et al., 1987</td>
<td>25</td>
<td>0%</td>
<td>100%</td>
<td>none permanent; hydrocephalus (2); respiratory complications (3); meningitis: bacterial (1), aseptic (1)</td>
</tr>
<tr>
<td>El-Fiki, et al., 1985</td>
<td>4</td>
<td>0%</td>
<td>100%</td>
<td>increased hemiparesis and pneumonia; gastrointestinal hemorrhage (1); increased dysarthria and ataxia (1)</td>
</tr>
<tr>
<td>total</td>
<td>76</td>
<td>4%</td>
<td>+91%</td>
<td></td>
</tr>
</tbody>
</table>

* Numbers in parentheses indicate the number of complications. The total complication rate was 22%.

TABLE 2
Summary of complications following superficial temporal artery or vein to SCA-PCA bypass

<table>
<thead>
<tr>
<th>Author &amp; Yr</th>
<th>No. of Cases</th>
<th>Mortality Rate</th>
<th>Patency Rate</th>
<th>Complications†</th>
</tr>
</thead>
<tbody>
<tr>
<td>El-Fiki, et al., 1985</td>
<td>14</td>
<td>21%</td>
<td>78%</td>
<td>temporal lobe retraction hematoma (2 deaths); myocardial infarction (1 death); wound infection (1); epidural hygroma (1); pneumonia (1); aseptic meningitis (1); hepatic infarct with (1); retraction hematoma (4)</td>
</tr>
<tr>
<td>Sundt &amp; Piepgras, 1985 (vein bypass)</td>
<td>33</td>
<td>15%</td>
<td>82%</td>
<td>21% (7 of 33) worse or dead; progressing strokes (4); graft failure (3); 50% with subdural hygroma with 2 graft failures; homonymous hemianopsia (1); venous infarct with hematoma (1); retraction hematoma (4)</td>
</tr>
<tr>
<td>Ausman, et al., 1984</td>
<td>14</td>
<td>7%</td>
<td>85%</td>
<td>meningitis (2); temporal lobe edema (5); subdural hematoma (1); delayed cerebrovascular accident (1)</td>
</tr>
<tr>
<td>Hopkins, et al., 1987</td>
<td>10</td>
<td>10%</td>
<td>90%</td>
<td>pneumonia/tracheostomy (1); midbrain contusion (1); transient coma (1); temporal retraction hematoma (2); delayed sepsis causing death (1)</td>
</tr>
<tr>
<td>Sundt &amp; Piepgras, 1985 (STA-SCA)</td>
<td>8</td>
<td>0%</td>
<td>88%</td>
<td>seizures (2); cardiac dysrhythmia (1); aseptic meningitis (2); hydrocephalus (1)</td>
</tr>
<tr>
<td>total</td>
<td>86</td>
<td>+12%</td>
<td>79%</td>
<td></td>
</tr>
</tbody>
</table>

* STA = superficial temporary artery; SCA = superior cerebellar artery; PCA = posterior cerebral artery.
† Numbers in parentheses indicate the number of complications. The total complication rate was 55%.

approximately 20% of the complications were serious. Many of the severe complications related to temporal lobe retraction, hematoma or edema, or cardiac and respiratory problems. Most of the subdural hygromas occurred in patients undergoing vein bypass, which may be a peculiarity of that procedure. Interestingly, only a small number of patients (including those with PCA bypasses) were reported to have visual field defects. Results of the entire series certainly point to a significant surgical risk, particularly with these more rostral revascularization procedures. A breakdown of our recently reported cases from the State University of New York at Buffalo and the Barrow Neurological Institute is seen in Table 2.

Several important points emerge. As has been previously noted, the medical and neurological condition of these patients is generally poor, and extreme care must be taken to minimize cardiorespiratory complications. The surgery is technically difficult, particularly bypass to the SCA and PCA, and the risk of temporal lobe retraction damage and other surgical complications is substantial. A significant number of surgical disasters appear to have occurred in patients with progressing stroke or stroke in evolution. It may be unwise to consider surgery in that group of patients. The group of patients who seemed to enjoy the best results with this type of surgery are those with clear-cut postural symptoms. Our review suggests that the occipital artery to PICA bypass has lower risk and somewhat better overall results, in spite of the fact that our own initial experience seemed to favor the more rostral bypass procedures, especially in terms of angiographic perfusion through the SCA and PCA.

In retrospect, a reassessment of the complications in the two cases reported here revealed several oversights. Neither patient was given a trial of anticoagulant medication. There is no proof in the literature that anticoagulation significantly reduces morbidity in cases of cerebrovascular disease, largely because of the complications inherent with anticoagulant therapy. On the other hand, review of the pathophysiology of intracranial vertebrobasilar disease suggests that the majority of strokes are caused by thrombosis in a preexisting atherosclerotic lesion. Anticoagulant therapy may, therefore, provide a margin of safety in these patients and should be tried unless contraindicated.
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Our initial success with PCA and SCA bypasses made us lean toward this procedure. Compared to our experience with occipital artery to PICA bypass, our rostral procedures showed more dramatic clinical and angiographic results with initially low morbidity. The patient in Case 1 could have been treated with an occipital artery to PICA bypass, or perhaps intracranial vertebral endarterectomy, rather than an STA-ACA bypass since the vertebral artery lesion was clearly proximal to the PCA.

The high incidence of cardiac disease in association with VBI is well known. The death of the patient in Case 1 was precipitated by an episode of severe congestive heart failure and acute pulmonary edema which potentially could have been avoided with more careful cardiac monitoring. A Swan-Ganz catheter might have prevented this complication. Case 2 involved a more elderly individual who may have benefited from anticoagulant therapy. An earlier postoperative angiogram might have prompted us to consider a second bypass operation, although the technical challenges associated with a second procedure in the face of a swollen temporal lobe are considerable. These operations were performed after we had accumulated a reasonable surgical experience and both went smoothly from a technical point of view. One patient (Case 1) actually died from cardiac complications, and the death of the other (Case 2) was likely due to a failure of patency in a critically compromized vertebrobasilar circulation causing postoperative brain-stem infarction.

When added to our previous series the complications in these two cases indicate a significant increase in the risk of bypass to the SCA and PCA, at least in our hands. This review has been helpful to us but still leaves a number of unanswered questions: 1) Do repetitive vertebrobasilar transient ischemic attacks imply an increased risk for stroke, or are they similar to transient ischemic attacks in the anterior circulation, which are extremely variable and of limited significance? 2) What constitutes a true failure of medical therapy? 3) What is the role of anticoagulation versus antiplatelet agents in VBI? 4) Are there any absolute indications for posterior circulation revascularization? 5) Is the risk of creating a bypass of the SCA or PCA worth taking?

The rarity of this disease probably precluded most surgeons from gaining a large experience and certainly makes a cooperative study very unlikely. We urge all surgeons involved in the care of these patients to maintain a conservative approach and exhaust all medical forms of therapy before opting for intracranial bypass surgery.

References
