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## CHAPTER 47

# Intracranial Embolectomy and Bypass Procedures for Occlusive Disease: Current Indications

Stephen L. Skirboll, David W. Newell, and M. Sean Grady

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Surgical intervention for cerebral ischemia due to intracranial occlusive vascular disease has been the subject of great controversy over the past decade. C. Miller Fisher, MD, predicted that "anastomosis of the external carotid artery or one of its branches with the internal carotid artery above the area of narrowing should be feasible" and that "someday vascular surgery will find a way to bypass the portion of the artery during the period of ominous fleeting symptoms" (1). With the development of microsurgical techniques in the 1960s, the first superficial temporal artery-middle cerebral artery (STA-MCA) bypass procedure was performed for cerebrovascular disease by Yasargil and associates (2). In subsequent years, this procedure gained increasing acceptance among neurosurgeons for a variety of indications associated with brain ischemia. In 1985, the EC/IC Bypass Study Group reported a 5-year prospective study of 1377 patients randomly assigned to surgical or medical management for symptoms of transient ischemic attack (TIA) or stroke (3). This study concluded that extracranial-to-intracranial (EC-IC) bypass was no more effective than aspirin therapy in reducing stroke or stroke-related deaths in all categories examined. Subsequently, controversy has surrounded the indications and efficacy of intracranial bypass procedures.

In this chapter, we will review the assessment of brain ischemia, indications for bypass procedures or embolectomy, and surgical techniques for bypass in the anterior or posterior circulations. We will conclude with an overview of the future of these procedures.

### RATIONALE FOR SURGICAL BYPASS PROCEDURES

Brain ischemia occurs when cerebral blood flow (CBF) is not sufficient to meet metabolic demands. Reduction of CBF may be caused by narrowing or occlusion of arteries leading to or situated within the intracranial cavity or by a reduction of cardiac output. Occlusion or narrowing of these major arteries may result from cardiac or artery-to-artery embolism, arterial dissection, atherosclerosis, or arterial thrombosis. This chapter will concentrate on the correction of flow-limiting lesions of arteries within the intracranial compartment caused by embolus or atherosclerotic narrowing.

The EC-IC bypass study has been criticized by a number of investigators and clinicians in the field of cerebrovascular disease. (4-6). However, it is clear that indiscriminate use of bypass procedures for brain ischemia is not justified. Over the past 5 years, a more comprehensive understanding of the pathophysiology of cerebral ischemia has been acquired, in no small part due to the EC-IC bypass study and the multicenter clinical trials comparing medical to surgical treatment of carotid artery disease. As a result, the selection of patients who may benefit from bypass procedures has become better defined.

Thromboembolism, the major cause of stroke or TIA, typically does not occur in intracranial vessels of sufficient caliber to permit open surgical embolectomy. For the most part, embolic material originates from the carotid bifurcation in the neck or from the heart. In cases of progressive intracranial arterial narrowing or occlusion, the brain accommodates to the reduction in CBF by developing collateral arterial supply. Patients whose symptoms of cerebral ischemia result

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from flow-limiting lesions of intracranial arteries (hemodynamic—rather than thromboembolic-related stroke or TIA) make up a small but important subset. It is the identification of this subset that is the key to determining the population that may benefit from bypass procedures.

## PATIENT SELECTION

### Clinical Characteristics

Patients should have a history of recurrent symptoms of focal cerebral or retinal ischemia, manifested by TIAs, reversible ischemic neurologic deficits, or mild completed strokes with subsequent fluctuations in neurologic function. Bypass surgery is not indicated in patients with acute or chronic major neurologic deficits. Similarly, an intracranial bypass procedure is not indicated in the asymptomatic patient or in patients with relief of symptoms by anticoagulation or antiplatelet drugs (7). Evaluation should include history and physical examination, chest radiograph, electrocardiography (ECG), and laboratory studies. Identification of stroke risk factors such as cardiac and vascular disease should be fully assessed by the appropriate consultants and tests. Finally, patients must demonstrate evidence of a flow-restricting lesion with insufficient collateral supply and impaired cerebrovascular reserve capacity by a combination of diagnostic anatomic and physiologic imaging techniques as described below.

### Diagnostic Imaging

The pattern of damage detected by computed tomography (CT) and magnetic resonance imaging (MRI) can be correlated with the cause of a stroke. Hemodynamic cerebral ischemia or infarction may be distinguished from that caused by thromboembolism by a location limited to transition zones between the vascular territories of the anterior cerebral, middle cerebral, and posterior cerebral arteries. Tissue damage in these regions is often termed a "border-zone infarction." MRI provides greater sensitivity than CT in detecting previous ischemic parenchymal damage, although it may not offer any increase in clinical usefulness. Instead, the degree of parenchymal abnormalities on MRI may better represent an overall description of chronic cerebrovascular disease that may be related to the risk of subsequent stroke (8).

Complete four-vessel cerebral angiography, including images of the external carotid arteries, is mandatory to fully appreciate the pathologic vascular anatomy underlying symptoms of stroke or TIA. Improved catheter design and techniques have reduced the morbidity and mortality associated with angiography, currently at approximately less than 3% in institutions experienced in studying patients for cerebrovascular disease. Only angiography can clearly discern the degree and nature of occlusive lesions, possible embolic

sources, and patterns of cerebral collateral supply. MR angiography has recently become available as a noninvasive technique to visualize the cerebral circulation. At present, this modality may serve as a potentially effective technique for screening or follow-up but does not yet provide optimal dynamic flow information and spatial resolution from which to best determine specific therapeutic interventions.

Advances in physiologic imaging have made the most impact in identifying the patient population that may benefit from an intracranial bypass procedure. Angiography does not provide sufficient insight into the physiologic response of the cerebral vasculature to identify those patients at the greatest risk for hemodynamic ischemia. Dilation of cerebral vessels is an important compensatory mechanism when cerebral perfusion pressure is decreased as a consequence of progressive vascular occlusive disease with poorly developed collateral supply (9,10). In these cases, it is important to evaluate the patient's ability to increase CBF above a resting state by determining the intrinsic vascular capacity to respond to physiologic challenges such as CO<sub>2</sub> or acetazolamide (11–13). This capacity has been termed the cerebrovascular reserve capacity (CRC), or vasomotor reactivity (VMR). Several techniques have been reported, including xenon Xe 133 CT (14) and transcranial Doppler ultrasonography (TCD) with acetazolamide or CO<sub>2</sub> challenge (11,13,15).

Such techniques were not widely available at the time of the EC-IC bypass study. Preliminary reports at that time showed that these tests might be quite important for identifying patients being considered for EC-IC bypass. However, the bypass study was not limited to patients with proven hemodynamic insufficiency and included patients with ischemia associated with a wide range of pathophysiologic conditions. Using techniques to measure cerebrovascular reactivity, investigators have better described and defined hemodynamic ischemia, as well as demonstrating that patients with this type of compromise may be at an increased risk of stroke. A prospective trial of patients with internal carotid artery (ICA) occlusion using TCD with CO<sub>2</sub> challenge found that individuals with diminished or exhausted reactivity had a significantly greater risk (30% vs. 8%) of stroke over a 3-year period compared with individuals with normal reactivity (16). Similar results were established in patients with ICA stenosis or occlusion, using <sup>133</sup>Xe-CT with acetazolamide challenge; patients with impaired blood-flow reactivity showed a 36% incidence of stroke over 2 years vs. 4.4% in the normal vasoreactivity group (17).

Significant improvement in CRC was demonstrated following EC-IC arterial bypass surgery in 28 patients with internal carotid artery occlusion and symptomatic hemodynamic cerebral ischemia (18). Before surgery, all patients had recurring episodes of neurologic deficits with angiographically defined ICA occlusion correlating to the side of ischemia, brain CT scans that were either normal or showed border-zone infarction, and severely impaired CRC using <sup>133</sup>Xe single-photon emission CT (SPECT) and acetazol-

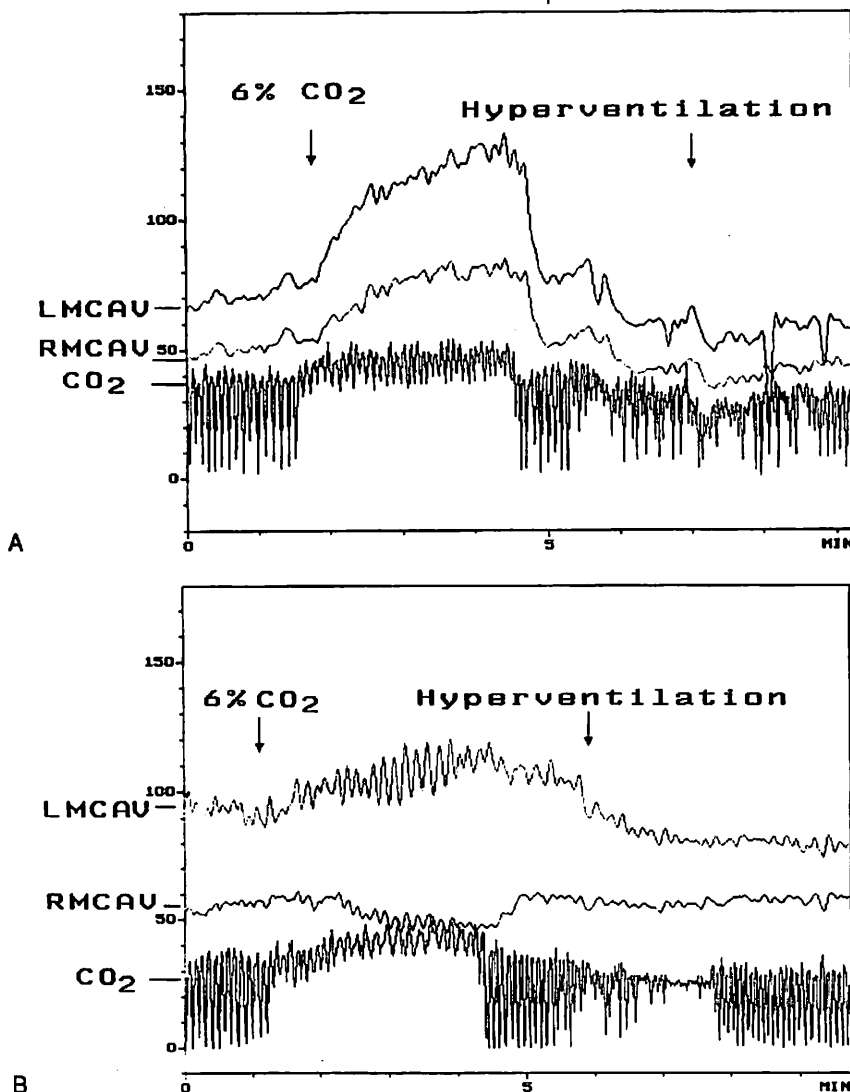
amide challenge. The immediate and long-lasting postoperative improvement in CRC was ascribed to the bypass-vessel contribution to CBF. In contrast, Piepgras et al (19) demonstrated no significant improvement after bypass in patients with cerebral occlusive disease and decreased CRC. Resting CBF was essentially unchanged following bypass surgery in this study, suggesting that appropriate candidates for bypass surgery may be those individuals with specific deficits due to ongoing hemodynamic cerebral ischemia and severely impaired CRC that affects the resting CBF.

### Cerebrovascular Reactivity Testing

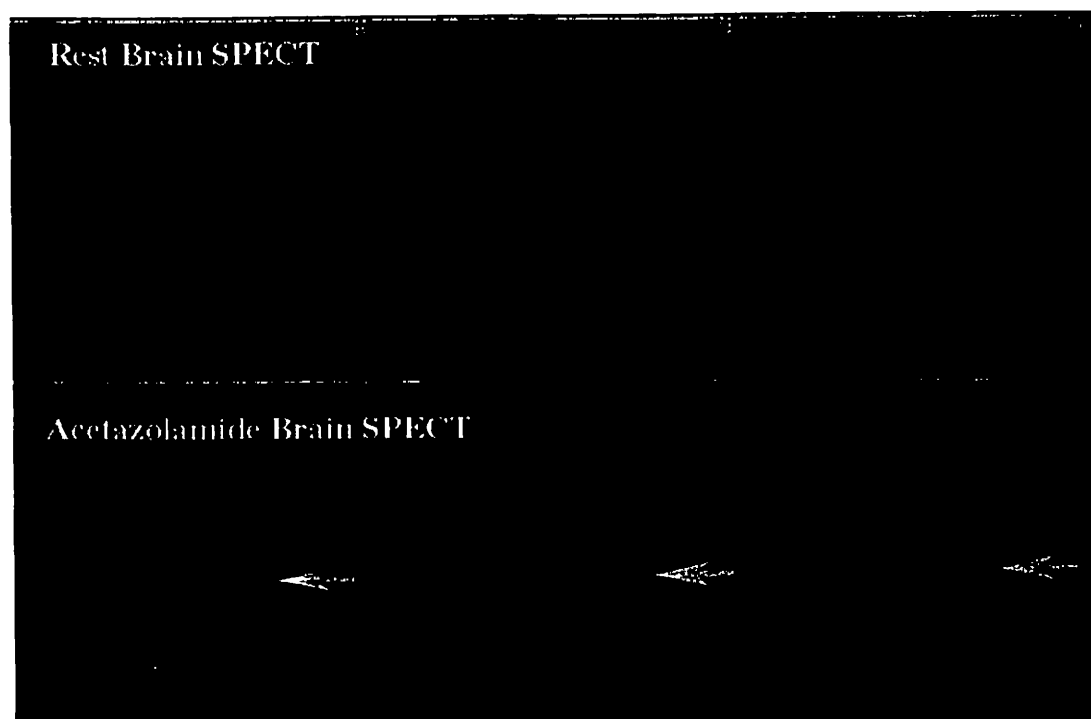
The stages of cerebrovascular compensation for reduced cerebral perfusion pressure caused by proximal arterial obstruction has been well defined using position emission tomography (PET) scan data (10). PET technology has not become widely available to clinicians with an interest in brain ischemia. Other technologies, however, have been able

to provide similar information, albeit not as extensive as PET scan data on reduced CRC in response to proximal arterial lesions. Direct studies of CBF can be performed using radioactive xenon with external detectors, as well as using cold xenon with CT-derived information on the regional distribution, the cold xenon being an index of CBF (17). SPECT can provide regional information on relative blood flow to various brain regions. TCD measurements can monitor the middle cerebral artery (MCA) velocity and thereby detect relative changes in blood flow in response to various stimuli.

To detect changes in cerebrovascular reactivity from proximal arterial obstruction, each of these techniques can be used before and after a vasodilatory or vasoconstrictive stimulus, and the vascular reactivity can be calculated. The commonly used vasodilatory stimuli include inhalation of  $\text{CO}_2$  and administration of acetazolamide. In general, as proximal arterial occlusions cause a progressive decrease in perfusion pressure, the first thing to be lost is the vasodilatory response to either acetazolamide or  $\text{CO}_2$ . Subsequently, the vasocon-



**FIG. 1.** Example of continuous TCD recordings of both middle cerebral arteries (MCAs) and end-tidal  $\text{CO}_2$  in response to changes in  $\text{CO}_2$ . **(A)** Normal response to inhalation of 6%  $\text{CO}_2$  and hyperventilation indicates an increase in blood flow velocity with  $\text{CO}_2$  inhalation and a decrease with hyperventilation. **(B)** Abnormal response to changes in  $\text{CO}_2$  concentration in the right MCA with more normal response in the left. With inhalation of 6%  $\text{CO}_2$ , there is an increase in the left MCA velocity and the subsequent paradoxical response in the right MCA velocity with a decrease in flow velocity. With hyperventilation, there is a decrease in flow velocity in the left MCA and no change in the right MCA velocity. This study indicates exhausted vasomotor reserve (VMR) on the right with a paradoxical or "steal" response (LMCAV, left MCA velocity; RMCAV, right MCA velocity; y axis, velocity in cm/sec; x axis, time in minutes).



**FIG. 2.** (A) Normal resting brain SPECT in a patient with limb-shaking TIAs that were brought on by decreases in blood pressure. (B) The same patient after administration of acetazolamide exhibits a decrease in perfusion in the MCA territory (arrows).

strictive response to hyperventilation is also affected, and then finally with a vasodilatory stimulus of either acetazolamide or  $\text{CO}_2$ , there is a paradoxical response of blood flow. In this situation, the blood flow decreases in the affected area with a vasodilatory stimulus. Various scales have been developed to grade responsiveness with each technique. It has now been demonstrated by both TCD with  $\text{CO}_2$  and VMR testing, as well as by cold xenon and acetazolamide testing that absent cerebrovascular reactivity in patients with carotid occlusion leads to an increased risk of stroke versus patients with carotid occlusion and intact or only moderately impaired VMR (16,17). Figure 1A shows a normal response in both middle cerebral vessels to hyperventilation and  $\text{CO}_2$  inhalation, and Fig. 1B shows an exhausted response with a slight "steal" phenomenon with inhalation of  $\text{CO}_2$  in a patient with an intracranial carotid occlusion. Figure 2 shows an example of SPECT imaging before and after acetazolamide administration, indicating a focal area of hypoperfusion during acetazolamide challenge.

Advantages of the TCD imaging include its low cost, ease of test performance, and no requirement of radioisotopes. Disadvantages include the fact that the regional resolution is poorer than tomographic techniques such as SPECT or cold-xenon CT. The direct measurement of autoregulation using TCD has also been performed in patients and is being investigated in those with occlusive disease. Preliminary results indicate that this technique correlates very well with  $\text{CO}_2$  reactivity (20). It appears that each of these techniques

is now becoming more readily available and can reliably identify patients with true hemodynamic insufficiency.

Table 1 lists the indications for bypass procedures.

## SPECTRUM OF PROCEDURES

### Anterior Circulation

Anterior circulation cerebral bypass surgery for occlusive disease is not restricted to a single standard procedure. De-

**TABLE 1.** Indications for bypass procedures in atherosclerotic occlusive disease

|                                                                                                                                                                                                                                                                                                                                            |
|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| TIAs, RINDs, or mild completed strokes with fluctuating neurologic symptoms                                                                                                                                                                                                                                                                |
| Cerebral ischemia or infarction in the border zone or terminal supply territories on imaging studies                                                                                                                                                                                                                                       |
| Angiographic demonstration of arterial stenosis or occlusion in region that correlates with neurologic symptoms                                                                                                                                                                                                                            |
| Failure of medical therapy including modification of risk factors (eg, hypotension) and use of antiplatelet and anticoagulant therapy                                                                                                                                                                                                      |
| Insufficient physiologic collateral blood supply (eg, inadequate ophthalmic, anterior or posterior circle of Willis collaterals, leptomeningeal, or transdural collaterals) associated with impaired cerebrovascular reserve capacity as determined by functional testing (eg, TCD with $\text{CO}_2$ /acetazolamide, SPECT-acetazolamide) |

**TABLE 2.** *Bypass procedures for anterior circulation ischemia*

| Proximal vessel                   | Interposition vessel | Recipient vessel                           |
|-----------------------------------|----------------------|--------------------------------------------|
| Superficial temporal artery (STA) |                      | Cortical middle cerebral artery (MCA)      |
| STA                               | Vein                 | M2/M3 of MCA                               |
| STA                               | Vein                 | M2/M3 of MCA                               |
| STA                               | Vein                 | Cortical MCA branch                        |
| Cervical carotid artery (CCA)     | Vein                 | Cortical MCA branch                        |
| CCA                               | Vein                 | Supraclinoid internal carotid artery (ICA) |
| CCA                               | Vein                 | Petrous ICA                                |
| Petrous ICA                       | Vein                 | Intradural ICA                             |
| External carotid artery (ECA)     | Vein                 | Cortical MCA branch                        |
| Subclavian artery                 | Vein                 | Cortical MCA branch                        |
| Occipital artery (OA)             |                      | Cortical MCA branch                        |
| OA                                | Vein                 | Cortical MCA branch                        |
| Middle meningeal artery           |                      | Cortical MCA branch                        |
| Retroauricular artery             |                      | Cortical MCA branch                        |
| STA placed directly on cortex     |                      |                                            |

pending on the clinical setting, the basic revascularization procedure varies with regard to location of donor vessel, need for an interposition graft, and location of recipient vessel. Donor vessels usually consist of the superficial temporal artery (STA) or its branches, the petrous ICA, or the internal or external cervical carotid artery (CCA). As a general rule, scalp vessels with angiographic lumen diameter <1 mm cannot be used. The patency rates are lower, technical difficulty with anastomosis is greater, and the potential blood-flow augmentation provided by such vessels is reduced. The larger of the two STA branches (anterior or posterior) is usually chosen, although the anterior branch is better situated to facilitate anastomosis to proximal MCA branches. Donor vessels may be directly anastomosed to recipient vessels, as in the STA-MCA bypass or in a petrous ICA-supraclinoid ICA bypass; or they may rely on an interposed vein graft, as in a CCA-petrous ICA or CCA-MCA bypass. Interposition bypass procedures offer the advantage of a larger blood flow channel and can be used when the distal STA is occluded or diseased. Table 2 lists various types of bypass procedures for the anterior circulation.

The advantages of the STA-MCA bypass include its shorter operative time, its less technically challenging nature, and its demonstrated high patency rate of 96% at 1 month after surgery (3). In addition, Schmiedek et al (18) demonstrated a substantial benefit of revascularization for hemodynamic ischemia using the STA-MCA bypass. The

advantages of a saphenous vein bypass graft procedure include a more proximal site of anastomosis, avoidance of abnormal "watershed" areas, and greater blood flow. It is particularly advantageous when suitable STA branches are lacking.

### Posterior Circulation

While vertebrobasilar insufficiency (VBI) from posterior circulation disease is not as common as anterior circulation ischemia, recurrent TIAs are associated with a similar 25–35% risk of subsequent infarction over the next 4–5 years (21,22). Moreover, posterior circulation infarcts may be associated with an increased morbidity and mortality (23,24). Caplan (25) and others have noted that lesions specific to the vertebrobasilar system compared with other areas are more prone to result in recurrent ischemic symptoms and infarction.

As with the anterior circulation, the most common pathologic process affecting the vertebrobasilar system is atherosclerosis, which can occur at any portion. Natural history studies of atherosclerotic disease in the posterior vs. anterior circulation suggest several possible distinctions (26,27). VBI develops more often secondary to intracranial as compared with extracranial disease (28,29). VBI symptoms usually result from arterial stenosis rather than emboli of the vertebrobasilar system, and ulceration of plaques in general is unusual in the posterior circulation (27–29). Thus, hemodynamic insufficiency, and not emboli, may be more common as a cause of posterior circulation stroke or TIA. In their review of cerebral revascularization, Onesti et al (30) noted that the vertebrobasilar system has fewer naturally occurring collaterals and bypass procedures in this region must perfuse less tissue compared with the anterior circulation. It is these differences that suggest that bypass procedures in the vertebrobasilar system may play a larger role than in the anterior circulation.

As with anterior circulation procedures, numerous surgical options are available for bypass of occlusive disease. Table 3 summarizes the majority of published surgical approaches.

### Patient Selection

According to Whisnant et al (31), VBI symptoms include two of the following: (a) motor and/or sensory symptoms occurring bilaterally or simultaneously, (b) dysmetria or gait ataxia, (c) dysarthria, (d) bilateral homonymous hemianopia, (e) diplopia. Other symptoms associated with VBI include tinnitus, vertigo, and multiple cranial nerve deficits, which usually are contralateral to the major sensory deficit. If other symptoms such as dizziness, syncope, drop attacks, or global amnesia occur singly, etiologies other than VBI should be investigated. The diagnosis of VBI should exclude cardiac pathology (dysrhythmias, cardiac insufficiency, emboli), blood disorders (thrombocytosis, sickle cell disease, hyper-

**TABLE 3.** *Revascularization procedures for the posterior circulation*

| Proximal vessel                   | Interposition vessel | Recipient vessel                            |
|-----------------------------------|----------------------|---------------------------------------------|
| Occipital artery (OA)             |                      | Posterior inferior cerebellar artery (PICA) |
| OA                                |                      | Anterior inferior cerebellar artery (AICA)  |
| Superficial temporal artery (STA) |                      | Superior cerebellar artery (SCA)            |
| STA                               |                      | Posterior cerebral artery (PCA)             |
| External carotid artery           | Saphenous vein       | PCA                                         |
| Cervical carotid artery           | Saphenous vein       | Vertebral artery                            |
| Vertebral artery                  | Radial artery        | PICA                                        |

coagulable states), basilar migraine headaches, demyelinating disease, intracranial neoplasms, and in some cases Meniere's disease. A detailed work-up should include complete differential blood count with smear, coagulation profile panel, metabolic screen, a 12-lead ECG, and Holter monitoring for 24–36 hours.

### **Diagnostic Imaging**

A CT scan is useful in delineating strokes and excluding the presence of nonvascular structural lesions. MRI allows a more sensitive description of ischemic areas in the posterior fossa and may afford the possibility of using MR spectroscopy to study these areas. Metabolic function can also be studied with PET, which may provide important physiologic background on the hemodynamic effects in patients with VBI. As with the anterior circulation, complete cerebral angiography remains the gold standard to delineate pathologic vascular disease and collateral supply. TCD has only more recently been used to evaluate vertebrobasilar disease, which is inherently more problematic because of vessel identification and the unusual hemodynamic characteristics of this vascular territory.

### **COMPLICATIONS OF BYPASS PROCEDURES**

Bypass procedures may result in the conversion of an intracranial stenosis to occlusion, leading to an acute neurologic deficit (32). The incidence of this can be reduced by treating with anticoagulants (eg, warfarin or heparin) for 3–6 months prior to surgical consideration (7). Many high-grade lesions will resolve during this treatment, obviating the need for surgical intervention. One theoretical advantage of a proximal placement of anastomosis is the improved perfusion to a greater number of vessels with a lessened chance

of stagnation in the vessel segment just distal to a stenotic site. If a deficit does occur as a result of a proximal intracranial occlusion with a patent graft, the patient can be managed by hydration and increases in the blood pressure. Symptoms persisting or beginning after several days may be treated with anticoagulants to reduce the risk of thrombosis.

Early postoperative deficits following EC-IC bypass have been well described (33). Most are transient and may be caused by cerebral edema, seizures, or dysautoregulation. These pathologic events are considered to be a direct consequence of the redistribution of flow surrounding the anastomotic site. Electroencephalography and close monitoring of anticonvulsant levels should be performed if seizures are suspected, and some groups maintain high steroid doses until the deficit has mostly resolved (34).

Late complications are usually due to ischemia. Delayed stroke may be caused by graft compromise or thrombosis, progression of atherosclerotic disease in the donor vessel, embolization from the graft, or the late conversion of an intracranial stenosis to an occlusion (32). Patients are maintained on daily aspirin to minimize some of these possibilities and control systemic risk factors (eg, hypertension, elevated cholesterol, diabetes, etc). Avoidance of smoking is mandatory. Anticoagulants may be necessary if embolization is suspected. TCD with emboli monitoring may help guide decisions regarding the administration of anticoagulants. If emboli observed by TCD are frequent and persistent, treatment with anticoagulants may be indicated. However, the value of emboli monitoring in this situation has not been established.

As with ischemia, bleeding complications may be seen in both the early and late postoperative periods. Early postoperative hemorrhage can be prevented with tight control of blood pressure (35). The incidence of hemorrhage may be further minimized by delaying the procedure after a recent stroke, if possible, and avoiding the placement of the anastomosis in an area of encephalomalacia. Bleeding from perfusion pressure breakthrough is a theoretical concern when higher flows are reestablished to an ischemic or poorly regulated vascular region. This phenomenon has been more frequently described with higher-flow vein grafts (36).

Early bleeding may occur at the anastomotic site despite strict blood pressure control and is obviously best controlled by preventative measures intraoperatively. Surgical evacuation should be performed when a hematoma causes a substantial degree of mass effect. Repeat bleeding or a new onset of bleeding may suggest the development of a pseudoaneurysm at the anastomotic site, which should be evaluated by angiography and treated with surgical revision.

Other potential complications include fluid collections in the subdural or subgaleal spaces in the early postoperative period; these usually resolve with time. In addition, scalp necrosis, wound infection, and meningitis are all potential issues complicating wound healing. Careful surgical exposure and technique help reduce the incidence of scalp necrosis and wound breakdown.



## INTRACRANIAL EMBOLLECTOMY

The lodging of an embolus in a major artery in the circle of Willis generally has catastrophic consequences, particularly if there is no well established collateral vascular supply distal to the embolus. Rather than the slow occlusion of focal atherosclerotic disease, which allows time for collaterals to develop, a large embolus causes an immediate reduction in cerebral blood flow. The subsequent stroke is usually large with devastating neurologic consequences. Surgical intervention is limited to arteries with a lumen diameter  $>1$  mm, typically the supraclinoid internal carotid artery (ICA) and middle cerebral artery (MCA). Several critical factors limit the usefulness of intracranial embolectomy, including length of time required to establish diagnosis and operative time required to expose the appropriate vessel and perform the embolectomy. Advances in endovascular neuroradiology and development of thrombolytic drugs such as tissue plasminogen activator (t-PA) and urokinase have relegated intracranial embolectomy to very rare occasions. Circumstances in which embolectomy may be reasonable are limited to identification of a thromboembolism in a major vessel during craniotomy. With the development of intraoperative TCD, major embolic events during carotid endarterectomy have been identified. While embolectomy may have been contemplated for such cases in the past, direct infusion of urokinase through a catheter placed directly into the carotid artery is much faster and consequently likely to be more efficacious (37).

Description of intracranial embolectomy includes case reports and very small series (38–40). All are in agreement that reperfusion must occur within 6–8 hours of onset of symptoms. Mortality and functional results vary considerably in these reports. Ipsilateral ICA occlusion is associated with a poor outcome, and collateral flow on angiography is the best predictor of outcome.

## CONCLUSIONS

Subsequent to the EC/IC Bypass Study (3), vascular bypass procedures have most frequently been employed to revascularize the brain when the parent artery must be sacrificed because of tumors or aneurysms. The development of imaging techniques to determine the physiologic response of the cerebral vasculature to stimuli such as  $\text{CO}_2$  challenge have enabled physicians interested in cerebrovascular disease to identify patients whose cerebrovascular reserve capacity (CRC) is exhausted. It is this population of patients with "isolated hemispheres," ie, a cerebral hemisphere with poor or absent collateral supply and clinical evidence of ischemia, that will benefit from intracranial bypass procedures. This group is a small subset of the population with cerebral ischemia; careful and thoughtful evaluation of a patient with symptoms of stroke or TIA must occur before the diagnosis of ischemia can be reached on a hemodynamic basis. How-

ever, when identified—and after failure of medical treatment—this group of patients can experience relief of their symptoms by intracranial bypass. Intracranial embolectomy, on the other hand, will likely be indicated only rarely but may be needed in certain situations in which acute embolism occurs and there is easy surgical access to the affected vessel.

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