Angiographic Changes after Pial Synangiosis in Childhood Moyamoya Disease

Richard L. Robertson, Patricia E. Burrows, Patrick D. Barnes, Caroline D. Robson, Tina Y. Poussaint, and R. Michael Scott

PURPOSE: To describe the angiographic changes accompanying the surgical treatment of moyamoya disease by pial synangiosis and to compare these changes with patient outcome. METHODS: The preoperative and postoperative cerebral angiograms, MR images, and clinical records of 13 children treated with pial synangiosis for moyamoya disease were reviewed. RESULTS: After synangiosis, 10 patients had significant neurologic improvement and three had minimal or no improvement. Postoperative MR images showed no new infarctions. Well-developed (grade A or B) transpial or transdural collaterals to the brain were present at the site of synangiosis in 84% of the surgically treated hemispheres. Cerebrovascular occlusive changes increased postoperatively in 76% of hemispheres. After synangiosis, moyamoya collaterals were increased in 48%, unchanged in 16%, and decreased in 36% of surgically treated hemispheres. All 10 patients with grade A or B collaterals bilaterally after synangiosis were asymptomatic or improved on follow-up. CONCLUSION: Pial synangiosis typically results in an increase in collaterals from the superficial temporal artery or middle meningeal artery to the brain. Synangiosis appears to result in stabilization or improvement in neurologic symptoms but does not prevent the angiographic progression of disease or the development of moyamoya collaterals. The angiographic demonstration of well-formed collaterals after synangiosis is associated with a favorable clinical outcome.

Index terms: Moyamoya disease; Children, diseases; Brain, surgery


Childhood moyamoya disease is a progressive cerebrovascular occlusive disorder that presents with recurrent transient ischemic attacks, strokes, headaches, and seizures. The angiographic features that define the disorder are progressive stenosis or occlusion of the internal carotid artery (ICA) bifurcation accompanied by parenchymal (moyamoya), leptomeningeal, and transdural collaterals (1–3). The posterior cerebral arteries may also be involved.

The angiographic appearance of the extensive parenchymal collaterals is responsible for the Japanese description of moyamoya, which translated means “something hazy, like a puff of smoke, hanging in the air.”

The goal of surgical intervention in moyamoya disease is to improve blood flow to hypoperfused cerebrovascular territories. At our institution, pial synangiosis is the surgical procedure of choice. In pial synangiosis, the superficial temporal artery (STA) is skeletonized and transposed to the pia through a craniotomy and openings in the dura and arachnoid. The adventitia of the STA is then sutured to the pia (4). Transpial and transdural collaterals subsequently develop from the STA and middle meningeal artery (MMA), respectively, to branches of the middle cerebral artery (MCA). A burr hole is also made over the frontal convexity in some patients to induce transdural collateral formation from the MMA to the anterior cerebral artery (ACA) territory.
The extent of collateralization to the brain is assessed by angiography typically at 1 year after synangiosis. The preoperative angiographic findings of moyamoya are well documented (1, 2). We reviewed the preoperative and postoperative angiograms of patients with moyamoya disease undergoing pial synangiosis in order to determine the nature of postoperative collateral flow, the relationship between the quality of collateral flow and the radiologic and clinical progression of disease, and factors related to poor postoperative collateral development.

Materials and Methods

The clinical records, magnetic resonance (MR) brain images, and preoperative and postoperative cerebral angiograms of 13 patients with moyamoya disease treated by pial synangiosis were reviewed (Table 1). The study population consisted of six boys and seven girls. At the time of surgery, the patients ranged in age from 2 to 16 years (average age, 7 years).

The disease was idiopathic in four patients, accompanied by Down syndrome in three patients, and associated with whole-brain irradiation and intrathecal chemotherapy for acute lymphocytic leukemia in one patient. One of the patients with Down syndrome also had been treated for acute lymphocytic leukemia. In five other patients, respectively, moyamoya was associated with localized radiotherapy for craniopharyngioma, neurofibromatosis 1, cloacal anomaly, Reye syndrome, and an unclassified generalized cerebrovascular dysplasia.

Bilateral pial synangiosis was performed in 12 patients and unilateral synangiosis in one. Of those patients undergoing bilateral synangiosis, surgery was performed in the second hemisphere during the same operation or within 7 days of the initial synangiosis in 11 patients. In two patients (cases 9 and 13), surgery on the second hemisphere was performed 30 months and 18 months, respectively, after unilateral synangiosis. Bilateral frontal burr holes were made in 10 patients and a unilateral burr hole was made in one patient.
Clinical follow-up data, preoperative and postoperative MR studies, and preoperative and postoperative cerebral angiographic studies were available in all patients. Four preoperative angiographic studies were performed at referring centers and nine at our institution. All postoperative angiography was performed at our institution; these studies were done with a digital subtraction technique and either a Toshiba RXO-250 single-plane or a General Electric Advantx DLX biplane angiographic unit. Informed consent was obtained for all angiographic examinations performed at our institution. Patients under 10 years of age had general anesthesia. Older patients were studied under deep sedation consisting of intravenous midazolam, fentanyl, and nembutal. Patients were hydrated intravenously during and after the procedure. Hyperventilation was avoided to minimize the risk of intracranial vasoconstriction. Infants weighing less than 16 kg routinely received a systemic anticoagulant (100 U/kg heparin sulfate intravenously) after the arterial puncture. Larger patients received an anticoagulant at the discretion of the radiologist. Preoperative cerebral angiography consisted of selective bilateral ICA and external carotid artery (ECA) injections with left or right vertebral artery injections in 11 patients. In one patient, only bilateral ICA injections were performed. In one patient, the left carotid circulation was evaluated before and after surgery with a common carotid artery injection because of the presence of an ICA occlusion. Postoperative angiography consisted of selective ICA and ECA injections with left or right vertebral artery injections in 12 patients.

The angiograms were evaluated by two independent observers and the results determined consensually. The severity or stage of disease was graded before and after surgery for each hemisphere using the Suzuki classification (2) (Table 2). In addition to the presence of parenchymal moyamoya collaterals and ICA stenosis or occlusion, Suzuki stages 3 through 6 are characterized by the progressive development of extracranial to intracranial collaterals. Since the synangiosis would be expected to alter the course of development of these collaterals, the staging criterion of ECA collateralization was not considered in assigning the Suzuki stage on the postoperative angiograms. The postoperative angiograms were evaluated for the extent and distribution of transpial and transdural collateral formation at the synangiosis (craniotomy) and burr hole sites. The transpial and transdural collaterals at the synangiosis site were graded according to the criteria proposed by Matsushima et al (5) for the evaluation of transdural collaterals after encephaloduroarteriosynangiosis (EDAS) (Table 3). Collaterals at the sites of the burr holes were scored in a similar fashion and were considered grade A if they supplied greater than two thirds of the ACA territory, grade B if they supplied between one third and two thirds of the ACA territory, and grade C if they supplied less than one third of the ACA territory. The caliber of the STA and MMA was measured before and after surgery. Postoperative angiograms were compared with the preoperative studies for the development of leptomeningeal collaterals from the MCA to the ACA and for changes in parenchymal (moyamoya) collateral extent. Postoperative angiograms were also assessed for regional differences in the time to the capillary phase of the injection.

Results

The criteria for surgical intervention were the presence of neurologic symptoms or signs likely to be related to cerebral ischemia and angiographic documentation of moyamoya disease. Seven patients had a completed stroke and two had a transient ischemic attack (Table 4). One patient had stroke and recurrent headaches. Two patients had seizures, and one patient had headache and vomiting without neurologic deficits.

The period of clinical follow-up after surgery ranged from 8 to 64 months (mean, 18 months). At follow-up, five patients had no residual neurologic deficits or seizures, four patients had improvement in their existing deficit and no further symptoms, and one patient had a marked reduction in the frequency of transient ischemic attacks. In the remaining three patients, one had persistent, unchanged hemiparesis; one had a slight reduction in the frequency of transient ischemic attacks; and one

### TABLE 2: Angiographic staging of moyamoya disease*

<table>
<thead>
<tr>
<th>Stage</th>
<th>Angiographic Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Narrowing of the internal carotid artery bifurcation without collaterals</td>
</tr>
<tr>
<td>2</td>
<td>Initiation of moyamoya collaterals</td>
</tr>
<tr>
<td>3</td>
<td>Intensification of moyamoya collaterals</td>
</tr>
<tr>
<td>4</td>
<td>Minimization of moyamoya collaterals</td>
</tr>
<tr>
<td>5</td>
<td>Reduction of moyamoya collaterals</td>
</tr>
<tr>
<td>6</td>
<td>Disappearance of moyamoya collaterals</td>
</tr>
</tbody>
</table>

* Adapted from Suzuki et al (2).

### TABLE 3: Postoperative collateral grading on external carotid angiography*

<table>
<thead>
<tr>
<th>Grade</th>
<th>Angiographic Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Area perfused by the synangiosis is greater than ⅔ of the MCA territory</td>
</tr>
<tr>
<td>B</td>
<td>Area perfused by the synangiosis is between ⅓ and ⅔ of the MCA territory</td>
</tr>
<tr>
<td>C</td>
<td>Area perfused by the synangiosis is less than ⅓ of the MCA territory</td>
</tr>
</tbody>
</table>

Note.—MCA indicates middle cerebral artery.

* Adapted from Matsushima et al (13).
had a residual but decreased hemiparesis and persistent headaches.

Preoperative cranial MR imaging showed infarctions in 10 patients (Table 1), bilaterally in five. MR studies performed 1 year after pial synangiosis showed no new infarctions. No patients had evidence of intracranial hemorrhage on either the preoperative or postoperative MR studies.

Preoperative angiography was performed to confirm the diagnosis of moyamoya disease, to document the severity of the disease, and to determine the presence of existing transdural collaterals to be preserved during surgery. Preoperative angiography was performed less than 60 days (range, 1 to 60 days; mean, 27 days) before synangiosis in all hemispheres.

A single postoperative angiographic study was obtained in 23 hemispheres between 7 and 13 months (mean, 12 months) after surgery. In two hemispheres (cases 9 and 13), a second postoperative angiogram was obtained at 42 and 30 months, respectively.

Preoperative angiography showed narrowing or occlusion of the ICA bifurcation bilaterally in 12 patients and unilaterally in one patient (Fig 1A). Stenoocclusive disease of the posterior cerebral artery was present in three patients (bilateral in two, unilateral in one). Native transdural collaterals were present from the MMA in six patients (nine hemispheres) on the preoperative examination. No native collaterals from the STA were evident before synangiosis (Fig 1B). No aneurysms were present before or after surgery.

The Suzuki stage ranged from 1 to 6 (mean, 2.92) before surgery and from 1 to 6 (mean, 3.68) after surgery. The two observers agreed on the angiographic stage of disease in 49 (88%) of 56 of hemispheres studied (two patients had two postoperative angiograms). The calculated $\kappa$ value for interobserver agreement on angiographic staging was .833. In no instance did the angiographic category differ by more than one stage between the two observers. An increase in Suzuki stage was documented in 11 patients (16 hemispheres) on follow-up angiography (Table 1). In three hemispheres, an increase in cerebrovascular stenoocclusive findings or moyamoya collaterals was evident at follow-up, although the angiographic changes did not meet the criteria for the next Suzuki stage (Fig 1C). In seven hemispheres, the moyamoya disease was angiographically stable after surgery. In the two hemispheres in which two postoperative angiograms were obtained, no changes were present from the first to the second examination.

In 25 surgically treated hemispheres (Table 1), transpial and transdural collaterals at the synangiosis site were grade A in 14 hemispheres (Fig 1D and E), grade B in seven hemispheres, and grade C in four hemispheres. The collaterals at the synangiosis site arose from the STA alone in seven hemispheres and from both the STA and MMA in 18 hemispheres. Transdural collaterals were present at the burr hole in 14 of 21 sites. Burr hole site collaterals were exclusively derived from the MMA and were minor (grade C) in extent in 12 hemispheres and moderate (grade B) in two hemispheres. These collaterals primarily supplied frontal branches of the ACA. The postoperative angiograms showed an increase in diameter and tortuosity of the STA in 21 hemispheres and of the MMA in 17 hemispheres (Fig 1D).

Leptomeningeal collaterals from the MCA to ACA territory developed in 11 hemispheres postoperatively and were accompanied by a preexisting or new A1 ACA segment occlusion in nine hemispheres. In one patient (two hemispheres), leptomeningeal MCA-to-ACA collaterals were present after surgery in the absence of A1 ACA occlusion. A reduction in PCA-to-MCA leptomeningeal collaterals was evident in one surgically treated hemisphere. After surgery, parenchymal collaterals increased in ex-

### Table 4: Clinical presentation and outcome

<table>
<thead>
<tr>
<th>Case</th>
<th>Presenting Sign or Symptom</th>
<th>Outcome after Pial Synangiosis</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>Stroke</td>
<td>Improved hemiparesis</td>
</tr>
<tr>
<td>2</td>
<td>Stroke</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>3</td>
<td>TIAs</td>
<td>Slight decrease in TIA frequency</td>
</tr>
<tr>
<td>4</td>
<td>Seizures</td>
<td>No seizures</td>
</tr>
<tr>
<td>5</td>
<td>Stroke</td>
<td>Hemiparesis unchanged</td>
</tr>
<tr>
<td>6</td>
<td>Seizures</td>
<td>No seizures</td>
</tr>
<tr>
<td>7</td>
<td>Stroke</td>
<td>Improved hemiparesis</td>
</tr>
<tr>
<td>8</td>
<td>Stroke</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>9</td>
<td>Stroke and headaches</td>
<td>Improved hemiparesis/persistent headaches</td>
</tr>
<tr>
<td>10</td>
<td>Stroke</td>
<td>Improved hemiparesis</td>
</tr>
<tr>
<td>11</td>
<td>TIAs</td>
<td>Marked reduction in TIA frequency</td>
</tr>
<tr>
<td>12</td>
<td>Headache and vomiting</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>13</td>
<td>Stroke</td>
<td>Improved hemiparesis</td>
</tr>
</tbody>
</table>

Note.—TIA indicates transient ischemic attack.
tent in 12 surgically treated hemispheres (48%) (Fig 1A and C), decreased in nine hemispheres (36%), and were unchanged in four hemispheres (16%) (Table 1).

Angiographically, a typical pattern of cortical branch filling was evident after pial synangiosis (Fig 1D and E). The ECA injection showed early filling of MCA branches in the sylvian or rolandic region via transpial or transdural collaterals from the STA or MMA. Antegrade filling of rolandic branches was evident in all patients in whom grade A or B collateralization was present. Contrast material was subsequently seen to reach other portions of the MCA and/or ACA territory. These regions displayed a delayed capillary phase (6 to 8 seconds) as compared with territories directly supplied by the synangiosis collaterals (4 to 5 seconds). Retrograde filling of M2 MCA branches to the M1 MCA segment bifurcation (Fig 1F) occurred in

Fig 1. Right ICA and ECA angiograms before (A, B) and 12 months after (C–F) pial synangiosis (patient 7).

A, Lateral projection of the preoperative ICA angiogram shows severe narrowing of the supraclinoid ICA (short arrow) with mild moyamoya collaterals (long arrow). Suzuki stage 3.

B, Lateral projection of the ECA angiogram during the same study as A shows no preexisting transdural collaterals. Note the size of the STA (long arrow) and the MMA (short arrow) before synangiosis.

C, Lateral projection of the ICA angiogram 1 year after synangiosis shows occlusion of the supraclinoid ICA (short arrow) and no antegrade filling of the MCA. New antegrade filling of the ACA is present owing to an increase in moyamoya collaterals (long arrow) and the development of contralateral ICA occlusion, resulting in loss of filling of the right ACA through the anterior communicating artery (see Fig 2). Suzuki stage 3.

D, Lateral projection of the postoperative ECA angiogram (early artery phase) shows increased caliber and tortuosity of the STA (long arrow) and MMA (short arrow) as compared with the preoperative examination. Transdural and transpial collaterals (arrowheads) from the STA and MMA to the sylvian branches of the MCA are present at the craniotomy site.

E, Lateral projection of the postoperative ECA angiogram (late arterial phase) shows slow antegrade filling of frontal, parietal, and temporal MCA branches.

F, Frontal projection of the postoperative ECA angiogram shows retrograde filling of the MCA to the M1 bifurcation (arrow).
14 hemispheres during the arterial phase of the ECA injection and was associated with the presence of well-formed (grade A or B) synangiosis collaterals in 13 hemispheres.

All 10 patients with grade A or B collaterals bilaterally were asymptomatic or had improvement in their symptoms (Tables 1 and 4). Of the three patients with grade C collaterals at the site of synangiosis on postoperative angiography, one patient (case 3), with grade C collaterals bilaterally, had a modest reduction in frequency of transient ischemic attacks (from approximately two per month to one per month); one patient (case 5), with grade C collaterals at a unilateral synangiosis site, had a persistent, unchanged hemiparesis; and one patient (case 9), with grade C collaterals to the right hemisphere and grade B collaterals to the left hemisphere, had continued headaches but improvement in the right hemiparesis.

Discussion

Moyamoya disease, or syndrome, is a progressive cerebrovascular disorder that causes neurologic deficits and intellectual deterioration. The disorder was described by Takeuchi and Shimizu in 1957 (6) and is characterized by severe stenosis or occlusion of the ICA bifurcation with the development of stereotypical parenchymal collaterals.

In Asians, moyamoya disease is most often idiopathic. Although in Western populations an underlying cause may not be identified, frequently the moyamoya angiographic pattern is associated with other disorders, such as neurofibromatosis 1, Down syndrome, and cranial irradiation for the treatment of leukemia or suprasellar tumors.

Childhood moyamoya disease differs from that in adults in that children have a higher prevalence of cerebral ischemia and a lower risk of aneurysms and hemorrhage (1, 7).

Both medical and surgical treatments for moyamoya disease have been attempted. The administration of corticosteroids, low molecular weight dextran, and antiplatelet agents has failed to alter the clinical course or angiographic progression of the disease, although reports have suggested that calcium channel blockers may provide symptomatic relief in some patients (8, 9). Surgical treatments for moyamoya disease are aimed at the revascularization of ischemic brain parenchyma by establishing collaterals from ECA branches to the ICA territories. Surgical treatment may be divided into direct and indirect revascularization procedures. The STA to MCA bypass is a direct procedure in which single or multiple anastomoses are created. The major disadvantage of the direct method is the necessity for temporary interruption of MCA branch blood supply, which is poorly tolerated by children with moyamoya. Additionally, the small caliber of the STA and the recipient MCA branch often makes these procedures technically difficult to perform (8).

An anastomotic procedure may be performed in isolation or in combination with one of the indirect revascularization procedures (10).

Indirect revascularization procedures rely on
the observed tendency of ischemic brain to recruit collaterals from any available source (11). A variety of indirect revascularization procedures have been devised. Encephalomyosynangiosis is the placement of the temporalis muscle with its vascular pedicle onto the cerebral cortex (12). EDAS is a procedure in which the intact STA and a strip of attached galea are sutured into a narrow dural opening beneath a linear craniotomy (13). Pial synangiosis is a technique in which the dura and the arachnoid are opened and the STA adventitia is sutured directly to the pia (4). Each of these procedures results in the development of transdural or transepial collaterals from the STA to the MCA branches in the perisylvian and rolandic regions.

In our series, synangiosis generally resulted in well-developed collateralization of the brain. In three patients (cases 3, 5, and 9) (four hemispheres); however, only minor collateralization (grade C) occurred at the synangiosis site. In one of these patients (case 3), the STA was of extremely small caliber on the preoperative angiogram. Other factors may also have affected the development of transpial collaterals in this patient, as another patient (case 1) also had small STAs at preoperative angiography but had grade A collaterals postoperatively. Patient 3 had received whole-brain radiation therapy and intrathecal methotrexate for the treatment of acute lymphocytic leukemia. Intracranial microangiopathy is reported to be associated with both these treatment methods (14). However, patient 1 was treated with whole-brain radiotherapy and intrathecal cytarabine (which is not known to be associated with small-vessel disease) and had a good angiographic result after bilateral synangiosis.

Poorly developed collaterals were present at the synangiosis site in patient 5. This patient had a generalized intracranial vascular dysplasia of unknown origin. angiographically, the dysplasia was characterized by extreme tortuosity of the intracranial vasculature. There were, however, no clinical stigmata of Menke kinky hair disease. Findings at aortography and renal angiography were normal. Follow-up angiography in this patient was performed only 7 months after surgery. The short follow-up interval may in part be responsible for the relatively poor angiographic demonstration of collaterals; Matsushima et al (13) documented progressive collateral development for at least 6 months after EDAS. However, even though follow-up angiography was performed in this patient at 4 months rather than at 1 year after surgery, the short interval may not be sufficient to completely explain the relative lack of collateral formation. Karasawa et al (10) noted the presence of transdural collaterals in as little as 1 week after encephalomyosynangiosis. The poor collateralization demonstrated by angiography in patient 5 may therefore have been due to both the short follow-up interval and the presence of underlying vascular dysplasia.

In patient 9, there was poor collateral development after right hemispheric synangiosis. Preoperative angiography in this patient showed no stenosis of the right ICA or MCA and only a moderate stenosis of the A1 ACA segment. This was the only hemisphere undergoing synangiosis in which no ICA or MCA stenosis was evident preoperatively. The lack of demand for increased blood flow in the MCA territory may have contributed to the poor surgical result.

The increase in cerebrovascular stenotic or occlusive changes commonly observed on postoperative angiography in our series may have been due to either mechanical alterations in flow related to the synangiosis or to progression of the disease itself. Conversion of a high-grade stenosis to a complete occlusion has been reported after successful STA-MCA bypass for atherosclerosis (15). Occlusion of the proximal MCA after the bypass procedure is presumably due to the reduction in flow across the stenosis accompanying the distal revascularization. It is possible in moyamoya disease that the development of extensive synangiosis collaterals decreases the flow within the proximal MCA, thereby predisposing this portion of the vessel to occlusion. In support of a flow-related explanation for the proximal occlusive changes after surgery is the absence of angiographic progression in three of the four hemispheres in which only minor collateralization was present at the site of synangiosis. However, proximal ACA occlusions also occurred in our patients during the follow-up period, even when only minimal collateralization of the distal ACA territory was present. Additionally, moyamoya disease whether treated or untreated is progressive. The rate of angiographic progression of untreated childhood moyamoya disease is poorly documented in the literature but, in general, the disease worsens rapidly in early child-
hood and stabilizes during adolescence (16, 17). In their series of six patients with moyamoya and angiographic follow-up ranging from 1 to 12 years, Ezura et al (17) found progression in all six patients prior to adolescence. As all but one of our patients underwent synangiosis before puberty, the observed worsening of stenoocclusive changes may simply reflect the natural tendency for the disease to progress. If collateral development is modulated by the presence of cerebral ischemia, then the development of extensive synangiosis collaterals would be expected to occur when the disease progresses.

Extensive basal ganglia collaterals have been implicated as a risk factor for intracranial hemorrhage in adults with moyamoya disease (2). Although hemorrhage is uncommon in children with moyamoya, a potential goal of surgical intervention is to stabilize or decrease the number of these parenchymal collaterals. Matsushima et al (13) reported that the formation of collaterals from EDAS results in a diminution in size of moyamoya collaterals at the base of the brain; however, no mention was made of the Suzuki stage preoperatively or postoperatively. In our series, 92% of the hemispheres in which the collaterals remained stable or decreased postoperatively had advanced disease (Suzuki stage 3 or greater) on preoperative angiography. Of the hemispheres in which the parenchymal collaterals were increased on postoperative angiography, 75% had early disease (Suzuki stage 1 or 2) on preoperative angiography that progressed to stage 3 on postoperative ICA angiography. These findings suggest that the observed changes in parenchymal collateral extent are a function of a natural progression of the disease rather than a result of the synangiosis, since parenchymal collaterals are expected to increase during the early stages of the disease and to decrease in its advanced stages.

None of the patients in this series had evidence of new infarctions on follow-up MR studies. The lack of new MR findings in those patients with well-formed collaterals may be due to improved or stabilized blood flow to the brain after synangiosis. In the three patients in whom the synangiosis collaterals were only minor, the severity of disease increased in only one hemisphere during the follow-up period. The relative lack of disease progression in these patients may be responsible for the absence of new post-operative MR findings.

The clinical utility of the collateral grading system was not assessed in this study and the collateral grade was not used to determine patient treatment. However, the grading system was used as an indication of the technical success of the procedure, and the angiographic demonstration of well-formed synangiosis collaterals did correlate with a favorable clinical outcome. In spite of angiographic evidence of increased stenoocclusive changes, all 10 patients with grade A or B synangiosis collaterals were clinically stable or improved at follow-up, suggesting that the synangiosis helped to stabilize the cerebral hemodynamics.

In conclusion, a characteristic angiographic pattern is seen after pial synangiosis. Preoperative angiographic features that may predict suboptimal collateral development are the absence of ICA and MCA stenosis or the presence of a diffuse cerebrovascular dysplasia. Synangiosis appears to result in neurologic stabilization or improvement. Synangiosis does not prevent and may even increase the likelihood of progressive occlusive changes. Synangiosis also does not appear to influence the development or evolution of parenchymal collaterals. The angiographic demonstration of well-formed synangiosis collaterals is associated with improvement in, or resolution of, neurologic symptoms and signs.

References


